

Emotion Expression in Post-MI/PCI patients – the Effects of Two Types of Social Sharing on Psychological and Physiological Outcomes

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Declaration

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Summary

Cardiovascular Disease (CVD) is currently the world's major cause of death, killing 17 million people a year (World Health Organisation; WHO, 2011). In Ireland 10,000 people die each year from CVD (33% of all deaths), half of whom die due to a heart attack (Irish Heart Foundation, 2012). Numerous demographic, clinical and behavioural characteristics have been associated with the higher risk of development of CVD and recovery from cardiac events, including psychological distress (e.g., depression, anxiety), negative emotions (e.g., anger) and emotion inhibition (e.g., Type D personality, anger suppression).

Brief interventions inducing the expression of emotions have been tested for over 25 years; some have proved effective (expressive writing paradigm), while the procedure of others (social sharing) is still being improved. In this study, social sharing of facts *and* emotions was compared to natural social sharing, without an added instruction for emotion expression. Outcomes examined are psychological distress and affect, as well as blood pressure and heart rate. Additionally, heart rate variability is recorded during sharing interviews. The study also investigates possible moderating effects of Type D personality and Alexithymia, and mediating effects of emotion regulation.

Chapter 1 introduces the concept of emotion inhibition and its deleterious effects on health and wellbeing, and outlines emotion regulations theories, highlighting emotion inhibition-related regulation strategies. The chapter concludes that whereas the act of emotion inhibition may initially serve adaptive goals, when it is applied continuously, as a trait or coping style, it leads to adverse outcomes on psychological and physical health.

Chapter 2 discusses emotion expression in general, as well as interventions designed to induce emotion expression, such as the writing task, and outlines their effects (positive and negative) on various outcomes. This chapter concludes that, in general, written expression is

a promising method of improving psychological and physical health both through catharsis and cognitive processing of a negative experience.

Chapter 3 introduces the concepts of social sharing and verbal emotion expression. First, the chapter defines social sharing, discusses initial retrospective/diary studies, as well as subsequent laboratory studies. Second, it introduces verbal emotion expression and its effects. The chapter concludes that although natural social sharing has no apparent benefits, a social sharing intervention with induced emotion expression has not been thoroughly studied.

Chapter 4 outlines Coronary Heart Disease, its aetiology, treatment and risk factors. In particular, psychological factors are discussed, as well as psychological treatments offered to cardiac patients.

Chapter 5 presents the study rationale and the main model, as well as the results of the feasibility study that was conducted to test the study procedure, before implementing it with a clinical sample. Results suggested the procedure was understandable for participants, easy to follow, and safe, as it did not generate distress.

Chapter 6 outlines the main study hypotheses, and main study procedure including results of reliability analysis for all the scales used.

Chapters 7 and 8 present the results. Although for general post-MI sample the intervention did not bring expected effects on distress, a positive impact on physiology was observed. Furthermore, the intervention had some potential benefits for Type D individuals (mediated by rumination change) and people with Alexithymia.

Chapter 9 presents qualitative analysis of participants' narratives.

Chapter 10 discusses the study findings and concludes with an outline of main implications for theory and research, such as the necessity of focusing social sharing interventions on Type D individuals, as well as post-MI care.

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Emotions and Emotion Inhibition

The present chapter describes what emotions are and how people respond to them – the concept of emotion regulation is introduced with a focus on the suppression strategy. Next, the chapter outlines the concept of emotion inhibition – its definition and history, the distinction between suppression and repression, and a description of several separate psychological constructs (coping strategies and personality traits), which have the inhibition of emotions at their core.

1.1. Emotions and Emotion Regulation

This section describes emotions and emotion regulation. It introduces the concept of suppression, which is further described in the following sections.

1.1.1. Emotions and emotion regulation.

Among a vast amount of emotion definitions, the one that is used in the present thesis describes emotions as whole-body phenomena affecting three areas of human experience: subjective feeling, behaviour, and physiology (Iris B Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). This means that when experiencing an emotion, a person is aware of the emotion they feel (subjectivity), he/she can associate it with the somatic changes (physiology), and he/she can feel like doing something with it (behavioural expression). In fact, however, some people are unable to recognise which emotion they are experiencing at a given time, having a condition categorised as Alexithymia (section 1.4.3.). Other people, although understanding their own emotions, may choose to suppress them (not to show any signs of those emotions) or to regulate them differently.

Emotions are complex phenomena and before they occur, as an effect of a emotion-generating situation, two processes usually occur: attention and appraisal. In other words, for an emotion to exist, one must first notice the situation and then evaluate it in terms of its potential to generate emotion (*Fig. 1.*)(the "modal model" of emotion; James J Gross & Thompson, 2007).

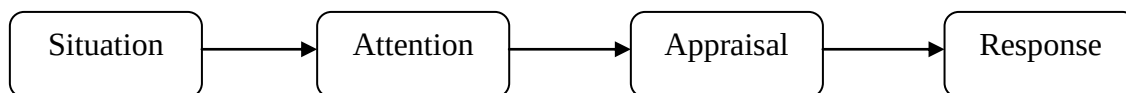


Fig. 1. The “Modal Model” of Emotion.

Emotion regulation is a term used to describe different techniques that people apply in order to avoid, block or decrease an unpleasant emotion, or thought or a behaviour deriving from that emotion. Emotion regulation theory has its roots in theories on coping with stress. For example, a distinction has been made between active coping (situation change), altering the meaning of the situation (reappraisal), and controlling the affective reaction generated by the situation (Morris & Reilly, 1987; Pearlin & Schooler, 1978). Gross (1998a) analysed the processes underlying diverse emotion regulatory acts, and distinguished five emotion regulation strategies (*Fig. 2*). Four of them are antecedent-focused regulation strategies, which occur before one responds to the situation. These are situation selection (e.g., trying to avoid a stressful situation), situation modification (influencing the situation in a way so it does not seem threatening anymore), attention deployment (i.e., directing attention elsewhere to distract oneself from the situation, or directing attention on thoughts and emotions - rumination, see below), and cognitive change (trying to see the situation in a different, non-threatening way, e.g., reappraisal, see below). The fifth strategy Gross describes is response

modulation, of which suppression is the most commonly researched form of such modulation. Other response-focused strategies may focus on intensifying affect, prolonging it, or inhibiting the physiological components of emotion (e.g., breathing exercises to decrease sympathetic response, or muscle relaxation). The latter, although may be understood as a type of emotion inhibition, does not relate to the psychological process of emotion inhibition as viewed in the present thesis. By targeting the physiological component of emotion, one switches off emotion, as affect does not exist without physiology (Keltner & Gross, 1999). In case of emotion inhibition, or suppression, the overt expression of emotion is inhibited, which not only does not lead to a decrease in physiological arousal, but increases it even further (see section 1.3.).

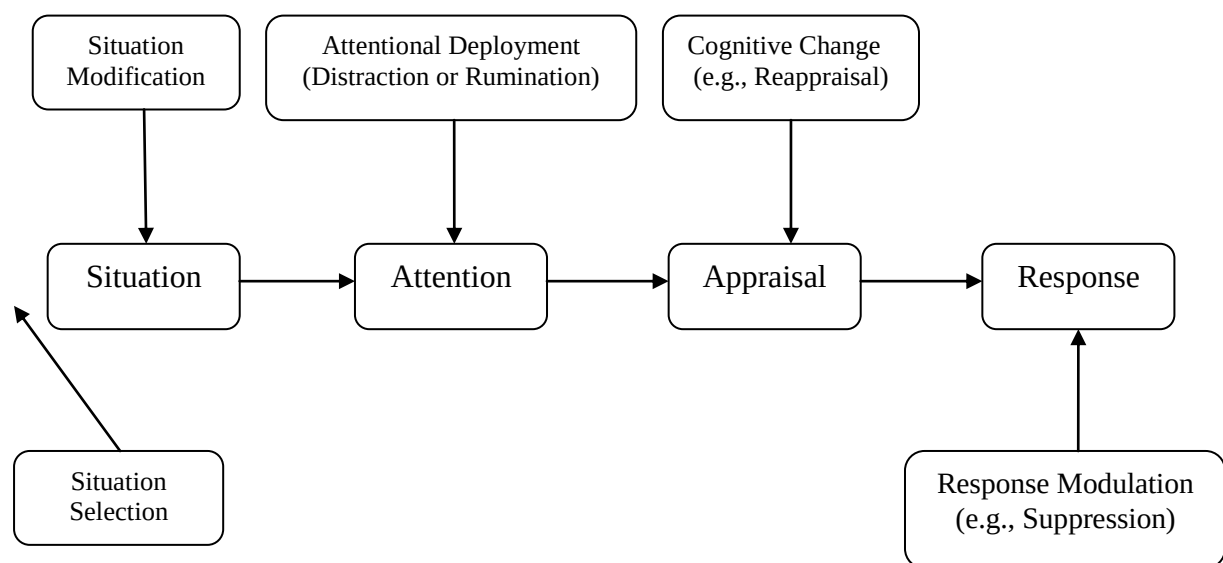


Fig.2. Emotion Regulation Strategies (James J Gross, 1998a)

Three strategies that are included in Gross' model and have been researched by psychologists, especially in relation to health, are rumination, suppression and reappraisal. The simplified emotion regulation model that is of relevance to this thesis is presented in *Fig. 3.*

Suppression and reappraisal have been studied and compared by Gross in his research. He described reappraisal (a type of cognitive change strategy) as the altering of the meaning of the situation, through which one can “switch off” the emotion before it fully appears. Suppression is a response-focused regulation strategy, which is a voluntary and conscious inhibition of emotions, or thoughts, or expressive behaviour related to these emotions, for example trying to change how one feels (experiential aspects; e.g., suppressing grief when planning a funeral) or how one expresses emotions (behavioural and verbal aspects; e.g., hiding one’s fear from others). Although Gross did not study rumination, he acknowledged it to be one of the types of attention deployment. Rumination is a spontaneous uncontrolled returning of negative thoughts and feelings to consciousness, and it is believed to be one of the negative effects of emotion inhibition (Pennebaker, Barger, & Tiebout, 1989). Rumination’s relation to physiology and health is further described in section 1.3.4.1. Furthermore, there is an association between suppression and rumination, in which suppression of negative emotions leads to rumination, which is further discussed in section 1.3.4.1.

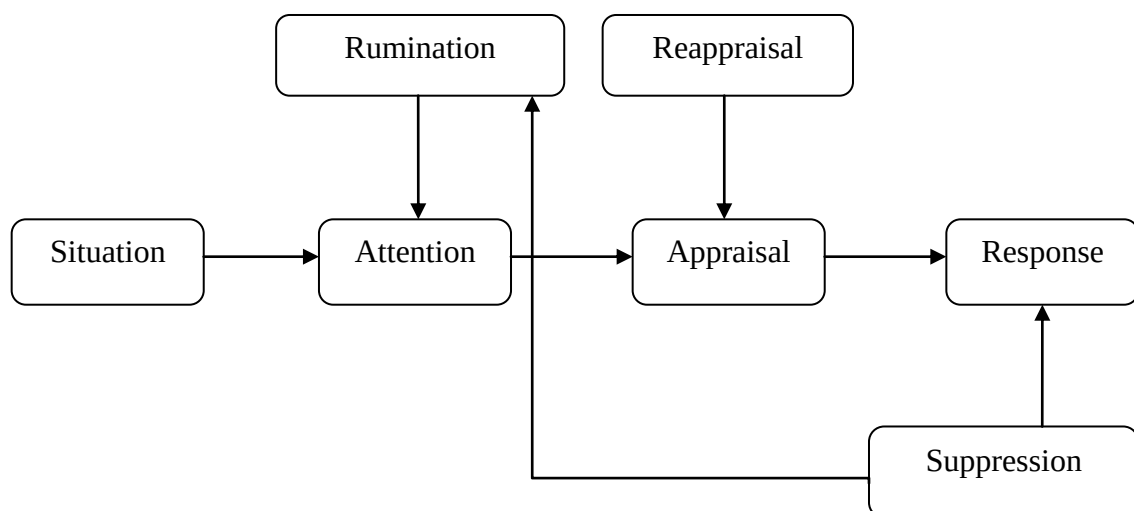


Fig.3. Emotion Regulation Strategies in the Focus of the Present Thesis

The strength of Gross' model of emotion regulation lies in its simplicity and its close relationship to emotion theory; however, it is not clear from his theory whether his approach to emotion regulation is focused on automatic (unconscious) and deliberate (conscious) techniques, or only on the latter. Although he made a distinction between emotion regulation and ego defences (James J Gross, 1998b), he also stated that emotion regulation strategies may be conscious and unconscious. It is unclear for example whether he would regard repression as attention deployment or a response-focused strategy.

Other regulation models (see below) add certain dimensions and may complement Gross' model; however, they do not explain at which point of emotion process the regulation strategy is applied, rather they explain emotion regulation out of the context of emotion construct.

For example, Parkinson and Totterdell's (1999) classification of emotion regulation strategies differentiated between cognitive and behavioural regulative strategies further divided to diversion strategies or engagement strategies. After adding an active vs. passive continuum to the theory they established the following diversion strategies: passive avoidance (not thinking about the problem or avoiding the situation) and active distraction (thinking about or doing something pleasant/relaxing or thinking about or doing something else). Emotion inhibition is such a diversion strategy and can be both passive (not thinking about a problem), or active (suppression by distraction), although it seems in Gross' theory this would be similar to attentional deployment.

Among the engagement strategies they initially distinguished passive engagement (i.e., acceptance) and active engagement (cognitive reappraisal, active problem solving, social support, and venting). Of note, Gross' strategy of suppression would here be classified as a diversion, whereas reappraisal is an engagement strategy.

Grouping the findings of reviews and research on emotion regulation, Koole (2009) distinguished between need-oriented, goal-oriented and person-oriented emotion regulation. Need oriented-emotion regulation (e.g., emotion inhibition) is a way of maximising short-term positive emotional state and can be disruptive if it is in conflict with long-term goals. Research on repressive coping (excluding distressing thoughts and emotions from one's conscious awareness; outlined in section 1.4.1.) supports the theory of need-oriented avoidance (in this case, automatic) of attention and knowledge (Derakshan, Eysenck, & Myers, 2007). Similarly, active suppression is a regulation technique serving a short-term need; however, it may be ineffective in the long-term (James J Gross, 1998b)(see section 1.2.).

Goal-oriented emotion regulation depends on existing goals or tasks, which either promote or inhibit certain affective states. Suppression is an example of this type of regulation, as is cognitive reappraisal. Person-oriented emotion regulation differs from the two previously described types of regulation in that instead of addressing a certain need or goal, it serves the purpose of sustaining the whole person's integrity. Of note, all of Gross' regulation strategies can be either goal-, need-, or person-oriented. Although Koole's classification adds an interesting dimension to emotion regulation theory, it does not explain how people regulate their emotions, whereas Gross' theory does.

1.1.2. Research into the effectiveness of emotion regulation strategies.

Most research confirms reappraisal to be the most effective regulation strategy in decreasing subjective experience of negative emotion, whereas suppression, although effective in decreasing the expression of emotion, to be one of the least beneficial strategies. For example, when looking at regulative strategies from the Parkinson and Totterdell's taxonomy, empirical studies suggest that engagement strategies (especially behavioural

diversion, such as exercise, and cognitive reappraisal) seem to be the most effective in regulating emotions, whereas venting and avoidance are the least effective (R. E. Thayer, Newman, & McClain, 1994; Totterdell & Parkinson, 1999). Of note, although the venting category includes verbal emotion expression, it is a broad category comprising of different types of “letting one’s feelings out” (e.g., crying, shouting, or hitting a pillow), and should not be mistaken for verbal emotion expression, which is a focus of the present study.

According to Gross, antecedent-focused strategies are more effective in minimising negative affect than response-focused ones. To test this, Gross compared the influence of reappraisal or suppression on subjective emotional experience and on the physiological response (James J Gross, 1998a) and observed that although emotion expression was successfully inhibited by both strategies, subjective negative emotion experience was only altered by reappraisal, whereas suppression led to an increase in physiological response. Contrary to the pattern hypothesised, reappraisal did not decrease the physiological response (in fact, it was similar to that of the control group), which Gross explained by the fact that the process of reappraisal may not necessarily affect physiology in the first place. Contrary to Gross’ view on the effectiveness of antecedent-focused strategies, rumination has been linked to increases in negative affect in depressed samples (Donaldson & Lam, 2004; Lyubomirsky, Tucker, Caldwell, & Berg, 1999; Watkins & Moulds, 2005).

Another study that tested the effectiveness of regulation techniques found that reappraisal, acceptance and problem solving were all associated with a decrease in psychopathological symptoms (Aldao, Nolen-Hoeksema, & Schweizer, 2010). The most recent meta-analysis testing the effectiveness of different strategies (Webb, Miles, & Sheeran, 2012) found that cognitive change (reappraisal) was effective when affect was measured using self-reports and behavioural measures, but not physiological measures. Response modulation (suppression) had an effect on behavioural measures, no effect on self-reported affect, and a small negative

effect on physiological measures, suggesting increases in arousal following the application of this strategy. Overall, across all outcomes, cognitive change was the most effective strategy of all and attentional deployment was least effective.

1.1.3. Summary.

The first part of this chapter introduced emotion regulation theory in relation to the “modal model of emotion” (Gross, 1998b), which is adapted in this thesis. In general, emotion regulation relates to the strategies that either prevent emotion from occurring, or change (suppress or intensify) the emotion response. Whereas some theories focus on the motives behind regulation, or the level of engagement in emotion experience, Gross’ model is simplistic, based on emotion theory, and explains when and how people regulate emotions. Out of different types of strategies Gross enlisted, two (reappraisal and suppression) were studied by him and compared, whereas one (rumination), although only mentioned by Gross, has an important place in psychology (and health psychology) research. The new emotion regulation model presented here illustrates the focus of this thesis. Of those three strategies, reappraisal has been found to be the most effective and beneficial in decreasing emotion, whereas suppression, although effective in behaviour control, led to increases in physiological arousal and had no effect on subjective emotion experience (Webb, et al., 2012). Rumination has been associated with increases in negative affect (e.g., Watkins & Moulds, 2005). Emotion regulation theory does not explain what happens when emotions are expressed, instead of regulated, and why some people express, while others inhibit them. The next section of this chapter deepens the understanding of emotion inhibition and its types, and presents different personality traits/types or coping styles that are closely related with inhibition.

1.2. Emotion Inhibition and Inhibitive Personality Types

This section outlines the construct of emotion inhibition and reintroduces suppression as defined in the present thesis, as well as repression. Personality traits and personality types, which underlining mechanism is emotion inhibition are also described here.

1.2.1. Emotion inhibition.

Inhibition (latin: *inhibere*; to “hold in”, to restrain; Oxford English Dictionary) was described in medicine in the context of neurophysiology in the middle of the nineteenth century (Aron, 2007; R. Smith, 1992). Inhibition is an important aspect of our life. It enables movement (selective inhibition of antagonist muscles), it regulates hormonal secretion, it is vital for nervous system functioning (e.g., in an initial stress response the parasympathetic nervous system is inhibited), but it also regulates our perception and cognition by blocking unimportant stimuli that we encounter, or excluding some information from processing. Believing that psychologists should distance themselves from physiological definitions of inhibition in favour of understanding the concept in purely psychological terms, Skaggs (Skaggs, 1929) proposed a dual nature of inhibition in psychology, as he distinguished between voluntary and involuntary inhibition. Indeed in psychology, inhibition can refer to the mechanism of repression (automatic inhibition of thoughts and/or emotions; also called *blocking*) or suppression (conscious inhibition of thoughts and/or emotions), or behavioural restraint (MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003). Behavioural and emotional inhibition is often construed as a sign of maturity and emotional stableness, as it is adaptive to suppress emotions we can not deal with (e.g., grief when planning a funeral), or to behave in a socially accepted way, ignoring internal needs and drives (e.g., remain calm when one wants to burst in tears). Inhibition of thoughts, on the other hand, helps us to concentrate on a task despite external thoughts. Although initially adaptive, inhibition can have detrimental

effect on psychological and physiological wellbeing, which will be further explained later in the chapter, after a short history of the construct is presented.

In psychoanalysis, Breuer and Freud worked together on a patient with hysteria (Freud & Breuer, 1896), which formed the basis for the later development of the theory of repression and conversion mechanisms (Freud, 1957). Freud believed that although an impulse (e.g., anxiety) cannot be destroyed, its expression can either be suppressed (actively inhibited) or it can be repressed without one's will being involved (e.g., in case of traumatic memories). Freud distinguished between two types of repression – primal repression and after-expulsion (repression proper). Primal repression meant blocking from consciousness a mental representation of an instinct, whereas repression proper was blocking all material (such as thoughts, emotions, or memories) associated with that instinct, regardless whether this material was, in itself, painful (Rosenstock, 1951). The primal target of Freudian repression was therefore a drive (or a “wish”) unaccepted by the Ego (Boag, 2006). To Freud, repression was an underlying factor of psychological disorders such as phobias and neuroses, and of somatic complaints (e.g., headaches being the result of conversion hysteria; Breuer & Freud, 1895/1955). Freud proposed that, in order to treat psychopathology, an uncovering of hidden impulses, thoughts and emotions had to occur. In psychoanalysis, this was achieved through methods such as dream analysis and free association.

Behaviourism described a different type of inhibition – inhibition of behaviour – which was believed to have an important role in learning: when new stimuli interfere with existing responses (external inhibition) or when conditioned responses interfere with unconditioned ones (internal inhibition; Pavlov, 1928, as cited in MacLeod, et al., 2003). Cognitive psychology, on the other hand, focused on inhibition of cognitive processes (e.g., thoughts). In the middle of the 20th century, cognitive psychologists defined inhibition as a slowed response to a stimulus compared to baseline. Most definitions of inhibition in cognitive

psychology were related to research on attention and memory – among these were attentional processes such as negative priming and inhibition of return, and directed forgetting and retrieval-induced forgetting, which describe inhibition in memory research (MacLeod, et al., 2003).

The present work is focused on a particular type of psychological inhibition, namely inhibition of emotions, recognised by Freud as one of the types of repression proper (when emotions associated with the repressed instinct are blocked from consciousness). Currently, emotion inhibition is a general term for an underlying process of some coping styles, regulatory and defence mechanisms or personality traits that have a common factor of non-expression of emotions, which, in most cases, are negative emotions (Garssen, 2007). The approach applied here is based on Skaggs' distinction between automatic (unconscious) and conscious inhibition. Emotion inhibition is therefore a process of removing or blocking emotional material (thoughts and/or emotions related to an emotional event) and/or expressive behaviour deriving from it, out of the present experience. The two main types of emotion inhibition are:

- 1) *Repression*: Emotional material is removed from consciousness completely without one being aware of the process. It can be inhibited before or after it reaches the conscious mind. Blocking occurs before thoughts and emotions reach attention. If, however, a person experiences these thoughts/emotions they can still be removed from the conscious mind together with the memory of them occurring. These processes describe the mechanism of repression.
- 2) *Suppression*: Emotional material is experienced, but a person actively chooses to remove it from attention (e.g., by using distraction). In this case a person can retrieve emotional material at will or given a cue (e.g., someone asking how they felt). This mechanism is called suppression. One may suppress emotions and thoughts that they

choose not to experience, but suppression is also used to describe inhibition of behaviour. The scope of the present study does not extend onto suppression of expressive behaviour, therefore (unless stated otherwise) here, the term suppression is applied strictly to emotion and/or thought inhibition.

Evidence for repression and suppression being separate and independent comes for example from a study by Tamagawa and colleagues (2013; outlined in section 1.3.4.), who found no significant correlation between repression (measured with the Weinberger Adjustment Inventory (Daniel A Weinberger, 1990) and suppression (The Courtauld Emotional Control Scale)(Maggie Watson & Greer, 1983). Two constructs linked to repression that have been widely studied are Type C personality and repressive coping style (see section 1.4.1.), whereas Type D personality seems to be based on the suppression construct (see section 1.4.2.).

1.3. Inhibition, Physiology and Disease (The Inhibition Theory)

Health problems are common for people who don't discuss their feelings (e.g. Pennebaker & O'Heeron, 1984) and repress traumatic memories (Kilpatrick, Resick, & Veronen, 1981). In a cohort study (B. P. Chapman, Fiscella, Kawachi, Duberstein, & Muennig, 2013) data from the 2008 General Social Survey–National Death Index were presented. This survey is conducted at yearly basis by the National Opinion Research Center. Chapman et al. presented data between 1996 and 2008, including data received from the Emotion Suppression Scale of the General Social Survey (Ross & Mirowsky, 2008), which was administered to 729 adults in 1996. Results showed that people high in suppression (scores within the 75th percentile) had higher all-cause mortality ($HR = 1.35$) than those low in suppression (scores within the 25th percentile). Similarly, high level of suppression was

associated with increased risk of cancer death ($HR = 1.70$). Different pathways linking emotion inhibition with physical or psychological disease, such as physiology, immune functioning, and psychological factors (negative emotions and mood) are discussed in the following sections.

1.3.1. Physiological effects of inhibition – the Inhibition Theory.

Emotion inhibition has been found to adversely affect health due to its impact on physiology (J W Pennebaker, 1997b) and Pennebaker's inhibition theory (*Fig. 4.*) was a foundation for the initial research on expressive writing (see Chapter 2).

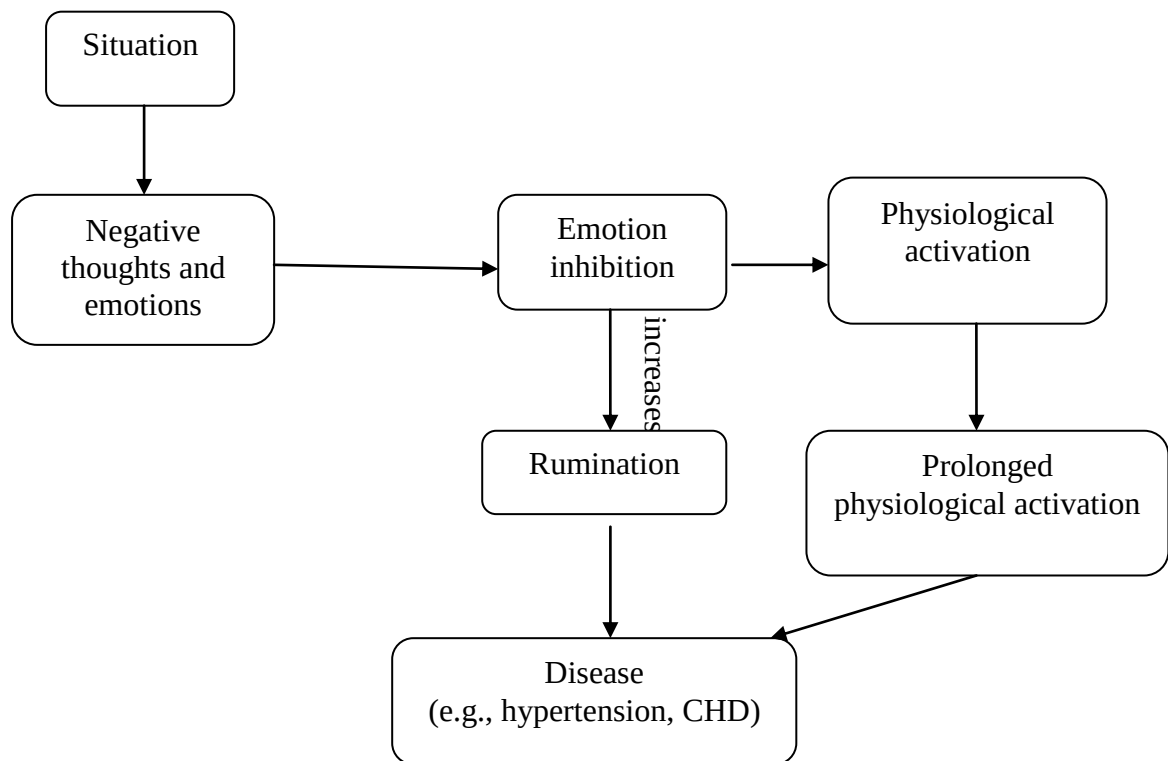


Fig.4. The Inhibition Theory

According to inhibition theory, if negative thoughts and emotions related to an emotional life event are inhibited (suppressed/repressed), the act of inhibition acts as a stressor and leads to a sympathetic activation (e.g., increase in blood pressure). If inhibition is applied frequently,

the physiological cost is over-activation of the sympathetic nervous system (SNS) and cardiovascular system. Inhibition also affects health indirectly, through increased rumination (see section 1.3.4.1.).

An example of research supporting the inhibition theory was carried out by Gross and Levenson (1993). In their study, participants watched a disgust-generating film, after which the suppression condition participants were asked to inhibit their reaction (emotional expression) to the film, whereas other participants were allowed to express their emotional response to the material. As a result, suppression participants showed greater sympathetic activity (an increase in skin conductance, blood pressure and respiration rate). In addition, disgust has been previously found to have a different physiological pattern than anxiety, anger or sadness, being associated with lower heart rate, whereas anger, fear and sadness were all linked to increases in heart rate (Levenson, 1992). In the study presented in section 1.1.2., Gross found that suppression led to an increase in physiological response, suggesting suppression is a stressor itself, however it did not lead to decreased heart rate activity. After detailed analysis, this was explained by a possibility of a floor effect (the control group's heart rate activity slightly decreased).

Similarly, Mauss and Gross (2004) suggested that inhibition that is maintained as a coping style leads to prolonged activation of the SNS and to an exaggerated sympathetic response of the cardiovascular system to a stressor *in future*. This strains the cardiovascular system causing changes to its structure (compromising its functioning through “wear and tear”) and inducing chronic release of glucose and lipids into the blood stream (I.B. Mauss & Gross, 2004). Sympathetic activation has also been associated with an increase in cortisol levels, a long-term immune system suppressor (Segerstrom & Miller, 2004), which is a proposed mechanism by which inhibition can adversely affect the immune system (e.g. B. Esterling, Antoni, Kumar, & Schneiderman, 1990). More recently, suppression has been linked to

increases in physiological measures of stress, such as muscle tension, cardiopulmonary symptoms, sympathetic arousal, neurological symptoms, upper respiratory symptoms (Tamagawa, et al., 2013).

1.3.2. Emotion inhibition and immune system.

There is some evidence for the adverse effects emotion inhibition on the immune system, coming from research on repressive coping and Type C coping (see section 1.3.1.), as well as from studies on suppression. An association between repressive coping and immune system was found in a study on outpatients having been treated for stress-related disorders in a behavioural medicine clinic (Jamner, Schwartz, & Leigh, 1988). Patients were assessed for repressive coping and had their blood drawn for analysis. Results showed immunosuppressive effects of inhibition in repressive copers.

Type C coping has also been found to adversely affect immune system activity: it predicted disease progression in patients with HIV, assessed by the CD4 cell count and clinical symptoms (Solano et al., 2002; Solomon, Kemeny, & Temoshok, 1991; Solomon & Temoshok, 1987).

Another type of emotion inhibition, (active) suppression, was a focus of Petrie et al.'s (1998) study on the inhibition – immune system link. Participants who had just completed an emotional vs. non-emotional writing task were asked to either suppress their thoughts about what they wrote about, or to think of what they wrote about for a few minutes. Consequently, two suppression groups (suppression of emotional material vs. suppression of non-emotional material) were compared with two non-suppression groups. Results showed an adverse effect of emotion inhibition on immune system, as suppression was associated with decreases in circulating T lymphocytes (CD3).

1.3.3. Inhibition and chronic illness.

Repressive coping has been linked to both cancer and hypertension. Mund and Mitte's (2012) meta-analysis of studies testing physiological associations between repressive coping and illness (cancer, cardiovascular disease, asthma, and diabetes) concluded that repressive copers have 31% more probability of suffering from somatic diseases and are at an 80% greater risk of hypertension; however, the latter result cannot be generalised due to a small sample and the significant heterogeneity of the studies. The effect size for the relationship between repressive coping and cardiovascular disease was non-significant ($OR = 1.23$). Some studies found adverse effects of repressive coping on cancer prognosis (e.g., Giese-Davis, Sephton, Abercrombie, Durán, & Spiegel, 2004; Jensen, 1987) and cancer mortality (e.g., Weihs, Enright, Simmens, & Reiss, 2000), but others suggest repressors (i.e., individuals with a repressive coping style) cope better with their illness (A. F. Pedersen & Zachariae, 2010; Ward, Leventhal, & Love, 1988). An association has been noted between emotion control and psychological adjustment to cancer; however, the direction of causality remains unclear (Classen, Koopman, Angell, & Spiegel, 1996; Maggie Watson et al., 1991).

Repressive copers were found to be at a 51% greater risk of cancer; however, based on two studies that measured repressive coping before cancer diagnosis, the authors put forward a possibility that this type of coping can be a result of the diagnosis of cancer, rather than a preceding factor. For example, a prospective cohort study conducted by White et al. (2007) measured anger control in healthy participants before they were followed-up 9 years later. After testing the relationship between anger control and onset of different types of cancer, the authors only observed a small significant effect for prostate cancer, but not for any other types of cancer.

1.3.4. Inhibition and psychological health.

Suppression has been associated with adverse psychological outcome. For example, it has been linked to increases in psychopathological symptoms such as anxiety, depression, eating, and substance-related disorders (Aldao, et al., 2010). Here, suppression was induced by asking participants to inhibit negative thoughts related to a negative emotion, rather than inhibiting emotional behaviour; therefore, a different aspect of a negative emotion was being suppressed compared to Gross' study described above (subjective experience vs. expressive behaviour). This study suggests that during an emotional episode, when one is confronted with negative thoughts, suppressing those thoughts can be counterproductive. Similarly a recent study (Tamagawa, et al., 2013) has linked suppression to increases in negative mood (e.g. anxiety, depression, fatigue and confusion) and in cognitive disorganisation, and decreases in activity, whereas repression was found to negatively correlate with anxiety, depression, anger, fatigue and confusion, which suggests possible different effects of each of the two types of emotion inhibition on psychological health.

The impact of inhibition on one's social life is also a factor of interest to the present thesis. People who often inhibit their thoughts and emotions are viewed as either good copers (who don't need support), or "closed" type of people, who don't want to talk about their problems (consequently support is not offered to them), and this lack of support may adversely affect their health (I.B. Mauss & Gross, 2004). Similarly, inhibition can impact physical disease prognosis indirectly through miscommunication with medical staff. For example, it has been suggested that hypertensive patients, who may be less likely to express their negative emotions than non-hypertensive patients, failed to communicate with medical staff on their emotional distress, which lead to this distress being unnoticed and unaddressed by clinicians (Roter & Ewart, 1992).

1.3.4.1. Rumination.

As was briefly mentioned in section 1.1.1, inhibition facilitates mental rumination (Pennebaker, et al., 1989). Active inhibition of thoughts around an emotional event may paradoxically lead to obsessive thinking about that event (Kelly & Kahn, 1994; Roemer & Borkovec, 1994; Wegner, 1989, 1997). This generates more inhibition, and therefore more physiological arousal. In a study mentioned above (Pennebaker & O'Heeron, 1984) the more participants *thought* about the spouse's death, the more health problems they experienced. Lepore et al. (1996) investigated the effects of rumination on distress in bereaved women who were either high or low in social constraints (similar to social inhibition). Results showed that in highly inhibited women high levels of rumination was related low level of social sharing and high levels of distress, whereas in uninhibited ones increased rumination was associated with increased sharing and decreased distress.

Rumination has been linked to increased risk of depression among healthy people (e.g., Just & Alloy, 1997), increased levels of depression among the bereaved (e.g., Nolen-Hoeksema & Davis, 1999) and increases in anxiety (e.g. Dickerson & Kemeny, 2004). Rumination has also been linked to increases in saliva cortisol levels, which in turn is an indicator of distress (e.g., Lovallo & Thomas, 2000). It has been suggested that prolonged rumination acts as a chronic stressor activating the sympathetic nervous system (Brosschot, Gerin, & Thayer, 2006), which is a similar mechanism as the one proposed for prolonged inhibition (Pennebaker, Hughes, & O'Heeron, 1987). Indeed, increased rumination may be a result of the increased use of suppression (Abramowitz, Tolin, & Street, 2001). Rumination has been linked to elevated blood pressure (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; L. M. Glynn, N. Christenfeld, & W. Gerin, 2002), CHD (Caprara, Barbaranelli, Colombo, Politi, & Valerio, 1995), adverse CHD prognosis (Fernandez et al., 2010), and cardiovascular health (L. Glynn, N. Christenfeld, & W. Gerin, 2002).

1.3.5. Positive aspects of emotion inhibition.

Emotion inhibition can be adaptive in some life situations. When facing thoughts and emotions that one can not deal with, inhibition can have positive effects on psychological health and coping with trauma. For example, Posttraumatic Stress (PTSD) patients who repressed the traumatic memories of childhood sexual abuse cope better than those who did not (Bonanno, Noll, Putnam, O'Neill, & Trickett, 2003). These findings are supported by Coifman et al.'s study on bereavement (Coifman, Bonanno, Ray, & Gross, 2007), where repressive coping was not only associated with decreases in distress (anxiety and depression), but also better health, and faster adaptation to a new situation. Similarly, in a study on a group of patients after an MI, repressive copers were found to show better long-term adaptation to life with CHD and less PTSD symptoms, compared to non-repressors (Ginzburg, Solomon, & Bleich, 2002).

There is much less evidence for positive effects of emotion suppression. One study found an association between suppression and an increase in positive emotions in students with borderline personality disorder (A. L. Chapman, Rosenthal, & Leung, 2009); however, the majority of research agrees that suppression, while effectively decreasing the expressive behaviour, has an adverse effect on physiology (Webb, et al., 2012). This may be due to the fact that while in repression the cognitive component of emotion disappears from consciousness, in suppression it does not and, moreover, may reappear with increased intensity (rumination, see section 1.3.4.1.).

1.3.6. Summary.

This section outlined the theory of emotion inhibition and illustrated how it (adversely) affects different psychological and biological outcomes (B. P. Chapman, et al., 2013).

Although an unconscious type of inhibition, repression, may have some positive effects on (Coifman, et al., 2007), research on (active) suppression suggests that it may adversely affect physiology (James J Gross & Levenson, 1997) and it leads to unwanted rumination (Wegner, 1997). This may be partly due to the fact that in suppression, the cognitive components of negative emotions are still accessible for people, whereas in repression they are unaware of experiencing those.

The illustration of the inhibition theory in this section of the chapter provides an understanding of the links between emotion inhibition and health through mediators such as physiological over-activation and increased rumination. In Chapter 2, the model is developed further, complemented with emotion expression and mediators of the expression effects on health (section 2.3).

1.4. Inhibitive Personality Traits and Coping Styles

This section outlines three dispositional constructs related to emotion inhibition. The first, Type C/repressive coping is related to repression, whereas the second, Type D personality – to suppression. The last construct described is Alexithymia, which is not emotion inhibition *per se*, but rather it reflects the inability to understand (and therefore express) one's own emotions. In case of Alexithymia, emotion inhibition is a result of this inability. Research has investigated these traits and their relationship to disease.

Understanding the character of each of them helps better understand the other constructs relating to emotional inhibition.

1.4.1. Type C coping and repressive coping.

Type C coping was first described in 1981 among cancer patients (L Temoshok & Heller, 1981) and it was later proposed that it was related to poor prognosis and adverse immune response (L. Temoshok, 1985). However, even before Temoshok started her research on psychosocial aspects of autoimmune diseases other researchers noted abnormal emotion expression in cancer patients (Greer & Morris, 1975). Greer and Morris' study compared a group of 69 patients with malignant breast tumours with a group of 91 patients with benign breast disease. Results showed a pattern of consistent inhibition of anger and other negative emotions in cancer patients as compared with controls. Later, Temoshok described Type C coping pattern among female patients with malignant melanoma (1987). Such patients were emotion repressors, tended to direct their anger inwards, were overly nice ("people pleasers"), and put others' needs before their own (L Temoshok, 2003). To measure Type C coping, Temoshok initially used Weinberger's technique for assessment of repressors (D.A. Weinberger, Schwartz, & Davidson, 1979) and combined two scales: Taylor's Manifest Anxiety Scale (MAS)(J. A. Taylor, 1953), and Marlowe-Crowne's (M-C) Social Desirability Scale (that measured defensiveness) (Crowne & Marlowe, 1960). As opposed to the other types distinguished by Weinberger - truly low-anxious, high-anxious, and defensive high anxious - repressors avoided confronting their negative emotions. Type C individuals were predicted to score high on the defensiveness scale, but low on the anxiety measure, due to the mechanism of repression. This way of measuring Type C coping was soon abandoned, as it was clear to Temoshok that cancer patients would often express high anxiety right after the diagnosis, which was when she assessed them. These high anxiety scores however, were a result of "getting the bad news", not an indication of the patients' usual tendency to cope (L Temoshok, 2000).

The next Type C assessment method involved testing the discrepancy between reported anxiety and physiological activation (skin conductance) (Kneier & Temoshok, 1984). Melanoma patients were compared with cardiovascular disease patients and a healthy sample. Cancer patients indeed showed heightened skin conductance levels during a stressful task (disturbing slides) despite reporting low anxiety. Of note, cardiovascular patients showed both high reported anxiety and high arousal levels, opposite to Type C coping style. Temoshok changed the way of assessing Type C pattern furthermore by using a measure of immune system activity. Cancer patients were interviewed, and levels of their expressiveness were assessed from the video tapes. Temoshok found that non-expression of negative emotions was directly related to adverse changes in immune system, therefore she concluded that it is emotion inhibition that is the pathogenic factor in Type C coping (L. Temoshok, 1985). It is not clear from the literature whether this emotion inhibition is an involuntary process (repression) or an active one. Temoshok uses both terms interchangeably. It seems though that the main negative emotion that is inhibited by Type C's is anger (L Temoshok, 2000). Of note, currently Temoshok uses a vignette method to assess Type C coping, asking patients to rate how similar their own reactions are to the individual on the vignette. Through this projective method she believes to be minimizing participants' defensiveness, as they do not discuss own maladaptive coping style directly (L Temoshok, 2000). Using this method suggests that the author herself believes that Type Cs repress, rather than suppress, their negative affect.

Repressive coping is believed to be a separate construct from Type C coping (e.g. Garssen, 2007). Repressive copers are unaware of the negative emotions they experience, therefore they often report low levels of stress and anxiety; however they score high on trait social desirability, measured by The Marlowe-Crowne Social Desirability Scale (the same technique Temoshok initially used in her research on Type C pattern). Contrary to self reports

of anxiety, physiological activity of those repressive coping individuals (such as heart rate, blood pressure, and electrodermal response) is heightened (D.A. Weinberger, et al., 1979). Of note, before Weinberger's method was constructed, repressors were assessed using the Repression-Sensitization (R-S) scale (Byrne, Barry, & Nelson, 1963). Repressors were on the other pole of a continuum from sensitizers, who were individuals who tend to express their emotions with exaggeration.

The reasons for the lack of consensus in the literature as to the differences between Type C and repressive coping may arise because repressive copers are believed to inhibit all types of the negative emotions (Garssen, 2007), whereas Type C individuals mainly inhibit anger (M. Watson, Pettingale, & Greer, 1984). Furthermore, anger expression style (inhibition vs. expression) has been found to be a separate form of emotion regulation from other emotions' regulation types (e.g. anxiety, sadness) (Burns, Quartana, & Bruehl, 2007). Temoshok suggested that discrepancies in cancer research as to the direction of the relationship between Type C coping and cancer outcome are a result of different time points during disease that these constructs are measured (L Temoshok, 1987). She admitted that early in her research on Type C coping, she made a mistake to assess anxiety levels in cancer patients immediately after the diagnosis, which is why some psychologists tend to put Type C personality together with anxious-defensive coping (Garssen, 2007). In fact, Type C coping and repressive coping have many similarities, and one might even assume that Type C coping is a specific focused aspect of repressive coping. Similarities are not only conceptual, but also extend to practical research, linking both constructs to autoimmune disorders. Finally, it is also possible that repression is a continuous construct and that Type C lies in a different point on the dimension, than repressive coping. Although it is not within the scope of this work to solve the issue of whether the constructs are in fact one single type of coping, the fact that they both rely on the mechanism of repression (repression of negative emotions versus repression

of anger) allows one to use the term repressive coping as a general term, remembering however about the discrepancy between both coping styles in the breadth of emotion repressed.

It has been suggested that repressive coping is characteristic of cancer patients, however it does not characterise cardiac patients (Kneier & Temoshok, 1984). The next section outlines another emotionally inhibitive personality type, high prevalence of which has been found in cardiac patients.

The main difference between Type C/repressive coping and Type D personality (see below) is that in repressive coping the mechanisms of inhibition are involuntary and occur most likely due to defensiveness, whereas in Type D emotion inhibition is conscious and deliberate (suppression) and it occurs in social situations. This is a very important distinction as it suggests that people with Type D personality are aware of their negative emotions, to which they are prone, however they do not inform others about them.

1.4.2. Type D personality.

Before Denollet developed the Type D personality construct, he noticed that cardiac patients are somewhat defensive, socially inhibited, and (as he then believed) unaware of their negative emotions. He initially studied negative affectivity (NA) in comparison with repressive coping, treating both these constructs as coping styles, which is now established NA is not. As Type D construct developed, a second trait, social inhibition, was added. Type D (“distressed”) personality is therefore composed of two unrelated (J Denollet & De Potter, 1992) traits: negative affectivity (NA; similar to neuroticism) and social inhibition (SI; inhibition of affect and behaviour in a social setting)(J. Denollet & D. Brutsaert, 1998), as measured by the DS-14 scale (Johan Denollet, 2005). NA is a predisposition to experiencing negative emotions (D. Watson & Clark, 1984), and SI is the tendency to suppress emotions

and behaviour in a social situation (Asendorpf, 1993). The fact that Type D personality requires both traits to be above average to high suggests that people with Type D personality must self-rate their negative affectivity as being high. If Type D was in any way related to repressive coping, self-ratings of NA would have to be low, as repressive copers are simply not aware of what negative emotions they had (after repression occurs those emotions are no longer present, although they may leave physiological traces responsible for disease). It is evident that Type D describes individuals who on the one hand report experiencing increased levels of negative emotions, however, on the other hand they report not being open about it to other people. Social inhibition, in Denollet's understanding, therefore seems to comprise a mix of introversion ("*I'm a closed kind of person*"; DS-14, see Chapter 5, section 5.3.2.2.) and behavioural and emotion suppression ("*I often feel inhibited in social interactions*"). Of note, the prevalence of Type D personality was found to range between 23% (women) to 27% (men) in Germany (Hausteiner et al., 2010) and even 38.5% in the general British population (L. Williams et al., 2008).

The argument of adverse health effects of emotion inhibition, as well as suppression specifically, was outlined in section 1.3.; however it is worth noting that within research on Type D numerous studies have linked this personality type to the progression of various cardiovascular disorders (e.g. J. Denollet & D. Brutsaert, 1998), response to conventional treatment (e.g. J Denollet et al., 2006; J. Denollet, J. Vaes, & D. Brutsaert, 2000), negative prognosis in CHD patients (Denollet & D. L. Brutsaert, 1998; Denollet, J. Vaes, & D. L. Brutsaert, 2000), cardiac mortality (e.g. Denollet, Sys, & Brutsaert, 1995; J. Denollet et al., 1996), or impaired health and depression in heart failure patients (A.A. Schiffer et al., 2005). More recent studies suggest an association between Type D and maladaptive illness perceptions (Williams, O'Connor, Grubb, & O'Carroll, 2011 a), unhealthy lifestyle (Gilmour

& Williams, 2012; Svansdottir, van den Broek, Karlsson, Gudnason, & Denollet, 2012), as well as poor adherence (Williams, O'Connor, Grubb, & O'Carroll, 2011 b), maladaptation to stress (Howard, Hughes, & James, 2011), and increased health care utilisation (Michal, Wiltink, Grande, Beutel, & Brähler, 2011).

Separately from SI, NA has been linked to angina pectoris and other CHD-related complaints, although it is unrelated to objective cardiac health indices (D Watson & Pennebaker, 1989). This implies that some people may complain about their health because of high NA even though their health is not worse than low-NA people. Another theory suggests that NA is a secondary trait that occurs in the presence of an illness, rather than NA causing the illness (D Watson & Pennebaker, 1989). However chronic NA makes people more prone to experience increased stress (e.g., H S Friedman, 2000; Suls, Green, & Hillis, 1998) and stress has been linked to the development and progression of CHD (e.g., Iso et al., 2002). The link between SI and CVD is similar to that of emotion inhibition and CHD, as outlined in the previous chapter. Additionally, socially inhibited people are often isolated and such isolation is associated with poor CHD prognosis (e.g., Carpeggiani et al., 2005). Decrease in social support has also been linked to cardiac mortality (Bucher, 1994; Frasure-Smith et al., 2000).

It has been suggested that diabetes patients with Type D personality were more depressed, lonely, anhedonic and anxious, and had less social support while having experienced more stressful life events than non Type D patients (Nefs, Pouwer, Pop, & Denollet, 2012). A more recent study found a relationship between Type D and emotionally triggered stress cardiomyopathy, suggesting a possible effect of Type D on bioreactivity to emotional stress (Compare et al., 2013).

Research has linked Type D personality to adverse emotional health (e.g., depressive symptoms, anger, decreased well-being, tension, low self esteem, low positive affect, to name

a few), poor perceived health, and lower quality of life (for a review see S. Pedersen & Denollet, 2003). Type D personality may also predispose individuals to cardiovascular health problems (S. Pedersen & Denollet, 2003). The next chapter develops this issue further.

According to some, the area of research on Type D personality, which focused mainly on effects of Type D on cardiovascular health, presents a problem of “same-team replication” (Ioannidis, 2012), as a majority of studies linking Type D with disease was conducted by Denollet and his colleagues (the “Tilburg group”). However, this group have also published studies that reported null findings (e.g. Pelle et al., 2010; Angélique A Schiffer, Pedersen, Widdershoven, & Denollet, 2008), whereas other researchers have reported adverse effects of Type D on physical health (Whitehead, Perkins-Porras, Strike, Magid, & Steptoe, 2007), increased reactivity to stress (Habra, Linden, Anderson, & Weinberg, 2003) and a decrease in health behaviours and social support (L. Williams, et al., 2008). The effects of Type D personality on various outcomes have been analysed in four meta-analyses (Grande, Romppel, & Barth, 2012; O'Dell, Masters, Spielmans, & Maisto, 2011; J. Reich & Schatzberg, 2010; Versteeg, Spek, Pedersen, & Denollet, 2012). As all meta-analyses reviewed only research on cardiovascular disease samples, they are described in detail in the next chapter. Furthermore, a number of independent studies on different clinical or healthy samples have been published, showing adverse effects of Type D on adherence (sleep apnea sufferers; Broström et al., 2007), health behaviours and social support (students; L. Williams, et al., 2008), perceived occupational stress, psychological health and somatic complaints (healthcare professionals; Ogińska-Bulik, 2006), and quality of life (Parkinson's disease patients; Dubayova et al., 2009).

1.4.3. Alexithymia.

Alexithymia is a construct introduced by Sifneos (1973), and is described as an *inability* to identify and describe emotions and it is viewed as an impairment, rather than an act of inhibition (such as repression or suppression). Alexithymia is typically measured by Toronto Alexithymia Scale (G. J. Taylor, R. M. Bagby, & J. Parker, 1992). The three subscales in the TAS-20 are: difficulty identifying feelings, difficulty describing feelings, and externally oriented thinking. For individuals with Alexithymia, the physiological aspects of emotion are still experienced, but they are not associated to an emotion, or even when they are, the type of an emotion is not recognised. This leads to emotion non-expression; however this is not due to social inhibition, suppression or repression, but rather, it is because individuals do not know what emotion to express (e.g. Newton & Contrada, 1994; G. J. Taylor, Bagby, & Parker, 1999).

Alexithymia is a separate construct both from Type D personality (Williams, Curren, & Bruce, 2011) and from Type C coping (L. R. Temoshok et al., 2008), in that it describes a cognitive deficit in recognising and processing emotions, rather than social inhibition or a coping mechanism, although all three constructs ultimately lead to the same end result: non-expression of emotions. The prevalence of Alexithymia ranges between 13% in Finland (Salminen, Saarijärvi, Äärelä, Toikka, & Kauhanen, 1999) and 18% in Canada (Parker, Taylor, & Bagby, 1989).

Alexithymia is believed to increase reactivity to stress (Martin & Pihl, 1985, 1986) and immunosuppression (Dewaraja et al., 1997; Guilbaud et al., 2009; Mandarelli et al., 2010; McIntosh et al., 2014; L. R. Temoshok, et al., 2008; Todarello et al., 1997; Todarello et al., 1994). People with Alexithymia are also prone to depression and anxiety (Bogdanova, Díaz-Santos, & Cronin-Golomb, 2010; Hendryx, Haviland, & Shaw, 1991; Lumley, Stettner, & Wehmer, 1996; Marchesi, Brusamonti, & Maggini, 2000), as well as psychological stress

(McIntosh, et al., 2014). Although some researchers found a physiological link between Alexithymia and cardiovascular disease (e.g., Grabe et al., 2010), others suggested the association, if exists, is psychosocial, not physiological (Peters & Lumley, 2007; Valkamo et al., 2001).

1.4.4. Summary.

In this section of the chapter three inhibitive constructs were outlined – Type C/repressive coping, Type D personality, and Alexithymia. The differences between them (especially between Type C and Type D coping) are not always clear as there is no consensus in the literature regarding Type C coping and whether emotions are repressed or suppressed (although the method used by the author in her research suggests repression). What has been established is that repressive copers repress, and are not aware of either their negative emotions, or the fact that they are repressing them; Type Ds are high in negative affect of which they are aware, however they do not discuss it with others due to a combination of introversion and social inhibition; people with Alexithymia are not aware of inhibiting their negative emotions as they are not able to recognise them.

Although all these constructs have clear differences between them, the fact that emotion inhibition is the underlying factor in all of them (whether repression, or suppression, or non-expression due to inability to recognise emotions) is most likely the reason why they all have been linked to somatic disease.

1.5. Chapter Summary

The chapter described emotions as subjective feelings combined with physiological reactions and expressive behaviour, and outlined how all these aspects can be regulated by emotion regulation techniques. Combining the findings of psychological research, three

regulation strategies stand out as having the most impact on psychological and physical health: reappraisal, suppression, and rumination. Reappraisal is the most effective regulation technique (Webb, et al., 2012), as it targets the subjective feeling of emotion. Suppression, although effective in regulating the visible aspects of emotion has negative impact on physiology (Webb, et al., 2012) – a finding in line with the inhibition theory. Rumination not only leads to adverse psychological (Dickerson & Kemeny, 2004) and physical outcomes (Caprara, et al., 1995). It also has been proposed to be closely related to emotion inhibition (Pennebaker, et al., 1987).

Inhibition of emotions is a general term for all processes that block some aspect of an emotional experience. Research focusing on automatic and passive inhibitive processes investigates constructs such as repression, repressive coping or Type C coping, whereas research focused on active suppression investigates Type D personality. Separate area of psychological research focuses on Alexithymia, which is neither conscious, nor automatic inhibition, but rather a cognitive impairment in identification and description of emotions. Continuous use of emotion inhibition has been linked to over-activation of the sympathetic nervous system (SNS) and cardiovascular system (I.B. Mauss & Gross, 2004), increased cortisol levels and suppressed immune system (Segerstrom & Miller, 2004), and therefore to physical disease (e.g., hypertension, CHD, cancer)(Mund & Mitte, 2012). Additionally, inhibition has been associated with adverse psychological outcome (Aldao, et al., 2010; Tamagawa, et al., 2013). The inhibition theory was at roots of the initial emotion expression interventions, further described in the next chapter.

Emotion Expression and Health

Emotion regulation theory and the inhibition theory both consistently treat emotion inhibition as a maladaptive strategy, although often capable of decreasing negative affect. Unlike emotion regulation theory, which proposes a better regulation strategy should be used instead of suppression, the inhibition theory proposes a productive response to emotion – emotion expression. Whereas regulation strategies are focused on different types of modulation of emotion response, there is considerable amount of research confirming the beneficial character of responding to emotion by discussing it. This chapter outlines the construct of emotion expression and presents a review of positive and negative consequences of emotion expression for psychological and physiological functioning, as well as general and cardiac health.

2.1. Definition

There has been a debate whether expression of emotions is an innate ability (Darwin, 1872, 1998) or an effect of upbringing within a certain culture (Klineberg, 1938; Landis, 1924). In his book “The Expression of The Emotions in Man and Animals” Darwin (1872/1998) suggested that expression of emotions was a result of an emotion experience and that it could be both adaptive and serving interpersonal communicative purposes. He proposed that some expressions are habitual and adaptive (e.g., raising one’s eyebrows when surprised), however some ceased to be adaptive in humans (e.g., baring one’s teeth during anger). Darwin also believed that some emotion expressions have a calming effect on the body, i.e., their role is to relieve excess arousal of the nervous system.

In psychology, emotion expression is defined as a behaviour that informs others, or self, of one's emotional state. It can be a direct verbal (e.g., "I feel sad"), indirect verbal (e.g., shouting) or non-verbal (e.g., facial expression or hitting something as an indication of anger) expression. Only the direct verbal type of emotion expression requires conscious knowledge of one's emotions, and an ability to describe them. However even then, it does not follow that these emotions will be verbally shared to other people.

In the present thesis emotion expression relates to an intentional act of disclosing one's emotional experience. This expression may be either written (expressive writing) or verbal (social sharing). Both types of emotion expression are reviewed in the following chapters.

2.2. Emotion Expression in the Therapeutic Context

Retrieval of repressed memories, thoughts and feelings was achieved in psychoanalysis through free associations and dream analysis (Freud, 1926/1953). Although psychoanalysts, analysed clients' memories and thoughts, as well as feelings, abreaction of negative affect related to these cognitions was a vital part of therapy, despite the fact that usually emotion expression seemed to work only short-term (W. Reich, 1949). It was proposed that during the analysis of the unconscious only a part of related affect is released, therefore it cannot lead to long term benefits (Nunberg, 1932, as cited in W. Reich, 1949). For Freud (1890, 1895, as cited in Sletvold, 2011) emotion expression was defined as changes in body movement, facial expression and tone of voice, following an onset of an emotion. Of note, he did not differentiate between emotion types, but instead used the term "anxiety" for negative affect in general. Freud's therapeutic technique through association was used to uncover repressed memories, thoughts, and emotions. This kind of emotion expression was later studied by Reich and incorporated in his "character analysis" theory as a therapeutic technique ("reading of emotion expression", W. Reich, 1949). Reich believed that

Freud's association techniques rely too much on language, whereas analysis of emotion expression allows one to "read the mind" (Higgins & Raphael, 1967/1972, as cited in Sletvold, 2011). Emotions, for both these theorists, were a result of excessive drive that needed to be discharged in order to attain wellbeing. Following Freud, many psychotherapists and counsellors noted the positive effects of retrieving and verbalising negative emotions and thoughts (e.g. Alexander & French, 1946; Brenner, 1982), wherein the focus was on emotions in the context of personal relationships and an interaction with a therapist (transference). In this view, emotional episodes were not separate individual experiences, but rather a result of interacting with others, within a society. Emotion expression was therefore a way of communication and satisfying need (e.g., Shaffer, 1983).

In the second half of the 20th century, cognitive psychologists proposed that those emotions that result from distorted thoughts and irrational beliefs can be altered by addressing those cognitions (Ellis, 1962). For the underlying negative thought to be analysed, an emotion first had to be realised and expressed.

Humanistic psychology viewed emotions as change-motivating factors, which had to be experienced in therapy as they had an adaptive character (Maslow, 1979). Similarly, inhibition of negative emotions, especially anger, was considered detrimental for psychological health by Gestalt therapists (Perls, Hefferline, & Goodman, 1951), and experiencing them consciously, as well as expressing them, was a focus of therapy.

The approaches to emotion expression outlined above valued the role of expression in psychological and somatic health, and different pathways, through which this effect was achieved, were proposed. First, there was catharsis, the release of negative energy (Freud & Bonaparte, 1954). Cognitive psychology found that changing negative thoughts and beliefs resulted in a positive shift in affect, as emotions do not exist without a preceding thought

(e.g., Ellis, 1962), and Gestalt psychologists believed in becoming aware of, and solving interpersonal problems, were a way to better functioning (Perls et al., 1951). Finally, humanists trusted that goal achievement and self-fulfilment lead to happiness and wellbeing (Maslow, 1979). It was not until the theory of stress emerged that a psychoneurobiological link between emotions and health was proposed (Selye, 1956). Although Selye viewed stress as a nonspecific reaction to environmental demands, emotions were among stressors that he took into account in his theory. He did not differentiate between negative and positive stressors, as he believed that any demand placed upon a person can lead to a physiological reaction resulting in activation of the hypothalamus-pituitary-adrenal axis (HPA; see section 4.1.5.1), leading to cortisol (and other glucocorticoids) secretion; this idea complemented earlier theories (Cannon, 1939; as cited in Goldstein & Kopin, 2007) that saw pathological potential in stress-induced activation of the sympatho-medullary system, which resulted in the release of lipids and glucose to the bloodstream. Of note, within the study of emotion expression some researchers limited their focus to one emotion (e.g. anger; Spielberger et al., 1985), which subsequently lead to a separate field of study testing the effects of different anger expression styles (overt vs. covert) on physical health (including cardiac health)(e.g., Brosschot & Thayer, 1998; Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003).

2.3. Expressive Writing Method

To counteract the adverse effects of emotion inhibition, which Pennebaker believed were physiological and similar to stress (e.g., 1987), in the 1980s, he designed an intervention based on an in-private writing task facilitating emotion expression. The theory that underlined his research (outlined in the previous chapter, section 1.3.) stated that the physiological over-activation in inhibition should be balanced by disinhibition (down-regulation of physiological arousal), and that putting emotions into words was sufficient to

achieve this effect (*Fig. 5*). In this model, disinhibition mediates the relationship between emotion expression and physical health, whereas decreased rumination mediates the effect of expression on both psychological and physical outcomes. Section 2.3.4 of this chapter develops this issue further.

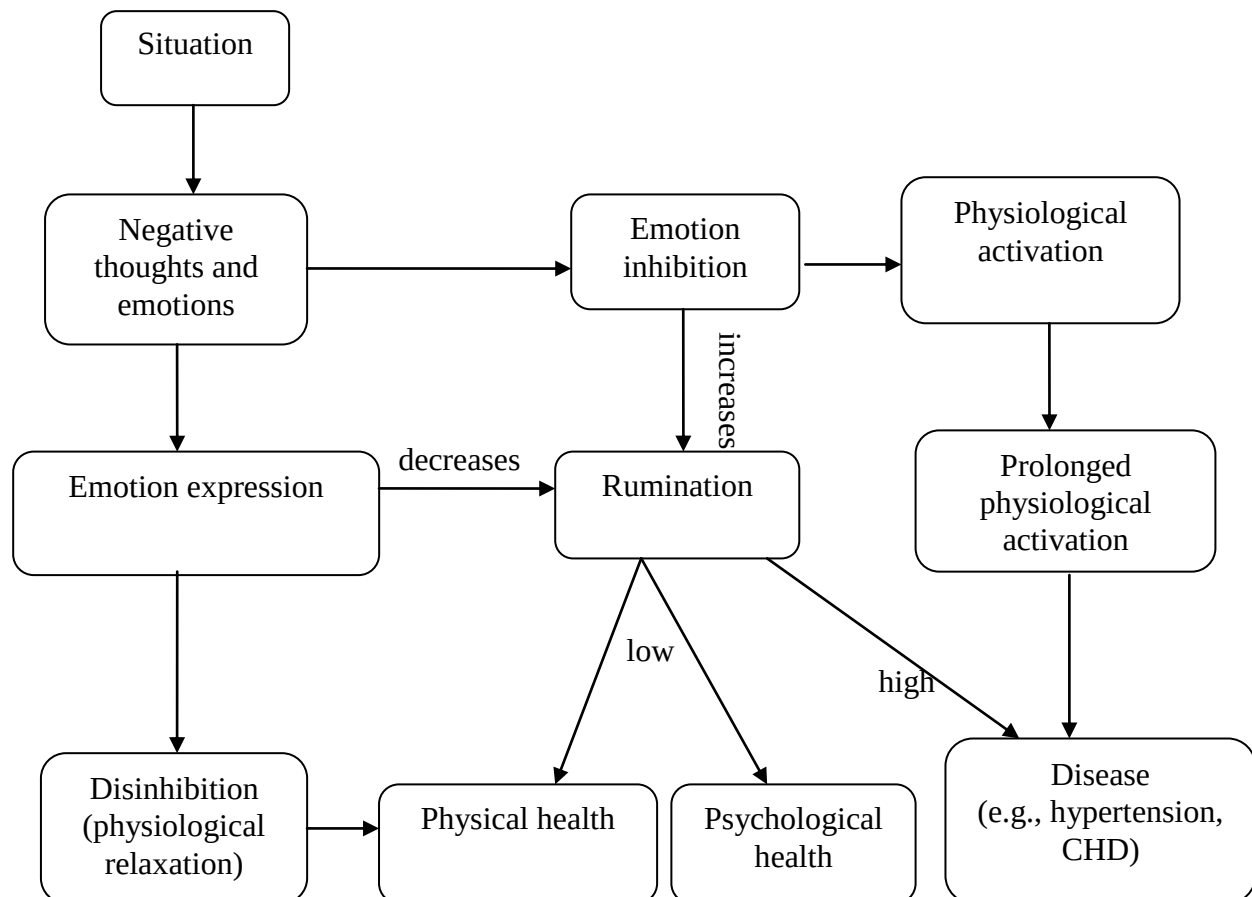


Fig.5. The Working Model of Emotion Expression

Pennebaker also stressed, and based his method on it, that expressing facts surrounding traumatic events did not bring the required effects and that the expression of emotions had to occur. In the early interventions (e.g. J.W. Pennebaker & S.K. Beall, 1986; J.W. Pennebaker, J.K. Kiecolt-Glaser, & R. Glaser, 1988) participants were asked to write about either the most

traumatic experience of their life and their deepest thoughts and feelings related to it, or about a stressful event that happened recently. Control groups wrote about trivial subjects such as plans for the day. Participants typically wrote for 15-20 minutes each day for several consecutive days. Early findings showed benefits of writing for physical health (J.W. Pennebaker & S.K. Beall, 1986) and immune system (J.W. Pennebaker, et al., 1988), despite negative mood effects immediately after disclosure (J.W. Pennebaker & S.K. Beall, 1986). This was followed by numerous other studies investigating into the effects of expressive writing on different outcomes, some of which studies are reviewed below. Reviewed studies test the effects of writing on one or more of the following outcomes: physiology, immune system functioning, general health, chronic illness, psychological health, or various outcomes for trauma patients and for people with Alexithymia.

2.3.1. Effects of expressive writing.

Expressive writing has been found to affect people in different ways – through physiology, immune functioning, or psychological functioning (emotions, cognitions and behaviour). These pathways are described below.

2.3.1.1. *Physiological outcomes.*

Several expressive writing interventions tested its impact on physiology, using measures such as skin conductance level, heart rate and blood pressure. For example in Pennebaker and Beall's study (1986) students' blood pressure and heart rate were measured before and after each writing session. Results showed that all participants' systolic blood pressure decreased from before to after each session; no significant effects were visible for diastolic blood pressure or heart rate. A subsequent study (J W Pennebaker, J K Kiecolt-Glaser, & R Glaser, 1988) confirmed this result.

In Petrie et al.'s study (1995) students from the experimental condition reported increased heart pounding after each session, indicating increased distress in those participants; however throughout the study a significant decrease in their skin conductance was recorded, relative to the control group where skin conductance increased toward the end of the study. These changes in skin conductance indicated that experimental participants' stress levels decreased as a result of this study, whereas control participants' stress levels increased. This was partly confirmed in another study, where experimental participants' emotional arousal (measured by saliva cortisol level) decreased from before to after a writing intervention, whereas the controls' arousal did not change during the course of the study (D. Sloan & B. Marx, 2004). In the same study, physiological arousal at the first writing intervention has been found to predict better psychological functioning 4 weeks after the intervention (depression and PTSD symptoms), which was related to the unpleasantness of the task itself. Experimental participants' baseline arousal was significantly higher than that of the control participants, as was the rating of unpleasantness of the task. None of these measures were significant at the second and third writing session. In Smyth's et al.'s study on PTSD sufferers (J.M. Smyth, Hockemeyer, & Tulloch, 2008) experimental participants' saliva cortisol levels decreased more than those of the control participants from before to after each session, indicating a decrease in emotional distress in this group.

In another study, expressive writing was associated with decreased sympathetic arousal (McGuire, Greenberg, & Gevirtz, 2005). Physiological measures such as blood pressure, skin conductance and heart rate variability (outlined in Chapter 4, section 4.1.5.) were recorded a week before the first writing session. After writing, physiological measurements were recorded at 1- and 4-month follow-ups. At both follow-ups emotion expression participants blood pressure decreased relative to baseline and their heart rate variability indicated less sympathetic arousal. This is consistent with current knowledge of a link between inhibition

and physiology (Brosschot & Thayer, 1998). Emotion inhibition, which acts as a stressor, if used continuously it has an adverse effect on the body's ability to cope with the sympathetic response (via vagal tone; see Chapter 4, section 4.1.5.). When the vagal tone is low, HRV is low, whereas BP and HR increase, as they cannot be balanced with the parasympathetic system. When aiming to answer the question about the effects of emotion expression on physiology, measuring just the BP and HR is insufficient and the measurement of HRV should be added as a measurement of vagal tone.

2.3.1.2. Expressive writing and immune function.

An effect of expressive writing on the immune system has been observed. For example in Pennebaker et al. (1988) 50 healthy students engaged in 20-minute writing sessions for 4 consecutive days, writing about either the most traumatic event in their life or about trivial topics (such as daily activities). Students' blood samples were taken a day before the intervention started, on the day of the last session and six weeks after. These were used to assess immunological parameters. Other outcomes measured were the amount of health centre visits, distress and physiological indices (this is further explained in following sections). Results indicated a significant higher immunological response throughout the writing sessions in the expressive writing participants than in the controls, compared to baseline (pre-intervention) levels.

Petrie et al. (K J Petrie, R J Booth, J W Pennebaker, K P Davison, & M G Thomas, 1995) confirmed these results in their study on medical students ($n = 40$) who tested negative on the hepatitis B (Hep B) antibody test. After four days of writing, a Hep B vaccination was administered to all participants. The writing method applied here and the types of outcomes measured resembled those of the study described above (Pennebaker, et al., 1988), however different immune indices were measured (Hep B antibody levels). As a result, expressive

writing group had significantly higher levels of Hep B antibody levels, an increased number of circulating CD4 cells (T helper cells) and total lymphocytes, which indicates a positive effect of disclosure on immune system. In a similar study, Petrie et al. (1998) asked participants to write about an emotional vs. non-emotional writing task. Results showed that expressive writing lead to increases in total lymphocyte count.

Immune function following emotional expression was also investigated by Esterling and colleagues (1990); a writing intervention was delivered to participants who were later tested for antibody levels to Epstein-Barr viral capsid antigen (EBV-VCA). Emotional expression was linked to lower levels of antibody to EBV-VCA, after controlling for all other confounding variables. This study however lacked a comparison to a control group, therefore conclusions should be drawn with caution. Research on HIV patients also supports the association between expression and immune system (e.g. O’Cleirigh, Ironson, Fletcher, & Schneiderman, 2008; K. Petrie, Fontanilla, Thomas, Booth, & Pennebaker, 2004). For example, in Petrie et al.’s (2004) study HIV patients were randomized to either a trauma writing group (writing about either their HIV experience or another traumatic event), or a control group (trivial topics). Their immune function was assessed 2 weeks, 3 months, and 6 months after the intervention was completed. There were positive effects of emotional writing on HIV viral load, whereas it increased in the control group. Additionally, the CD4 lymphocyte counts increased for the experimental group 6 months after the intervention indicating improvement in immune function.

2.3.1.3. Expressive writing and general health.

A number of studies investigated the impact writing had on general health, an outcome often operationalised as reported or objective number of health centre/GP visits (health care utilization) during months following the intervention (e.g., Pennebaker & Beall, 1986) or as a

health status assessed by medical staff (Cameron & Nicholls, 1998; e.g. M. Greenberg & Stone, 1992; M. Greenberg, C. Wortman, & A. Stone). In an early study by Pennebaker and Beall (1986) college students were randomized to either an emotion expression condition (n = 12), a factual writing condition (describing facts surrounding the traumas; n = 11), a combination condition (describing facts and feelings; n = 11) and a control condition (neutral writing, n = 12). Participants' health was assessed by recording their physical symptoms (e.g. headache, dizziness, heart pounding; assessed before and after writing sessions), a number of health centre visits (before the study and approximately 6 months after its completion) and a number of days they were restricted from daily activities due to illness (at a 4-month follow-up). Four months after the study the control group reported the greatest amount of days their activities were restricted due to illness, whereas the combination group had the fewest number of such days. At a 6-month follow up, there was a tendency towards an increase in the number of health centre visits for all groups except the combination group. This study suggests that expressive writing may have a positive long-term effect on health care utilization and general health, although some limitations may derive from small sample sizes in each cell. A subsequent study (Pennebaker et al., 1988) that compared disclosure of traumatic events and feelings with trivial writing among healthy students confirmed the finding regarding positive effects of disclosure on the number of health centre visits.

2.3.1.4. Chronic illness samples: cancer.

Disclosure has been found to improve physical symptoms in chronically ill patients. For example, in Rosenberg et al.'s (2002) study, 30 prostate cancer patients were randomized to either the experimental group, where they wrote for 20 minutes daily, for four consecutive days, about their deepest thoughts and feelings regarding their condition or another chosen traumatic experience (n = 16), or the no-intervention control group (n = 14). Experimental

patients reported lower pain severity than controls: these differences were significant at both the 3-months, and the 6-months follow-up. The writing intervention also resulted in a decline in health care utilization, such as doctor's appointments and medication use, although the trend was non-significant.

Sixty women with breast cancer were randomized to one of three conditions, emotion expression group (writing about the deepest thoughts and feelings, $n = 21$), benefit-finding condition (writing about positive thoughts and feelings, $n = 21$), and facts condition (writing about just facts, $n = 18$), in all of which they were asked to write about their illness (Stanton et al., 2002). They completed four 20-minute sessions over a period of three weeks. At both follow ups, 1 and 3 months after the intervention, emotion expression was associated with decreases in distress in those patients who scored low on cancer related avoidant coping, whereas the opposite was found for women with high avoidance. Emotion expression was also related to decreases in physical symptoms at the 3 months follow-up, compared to the facts group. Additionally, at the same follow-up, participants from the emotion expression and benefit-finding groups reported fewer cancer related doctor's appointments than the facts group. Of note, Morgan, Graves, Poggi, and Cheson (2008) reported a positive effect on leukemia and lymphoma patients' quality of life 3-weeks post emotional writing.

2.3.1.5. Chronic illness samples: cardiac patients.

There is limited research on expressive writing among cardiac patients. Recently, written emotion expression was tested among 153 patients after their first MI (Willmott, Harris, & Gellaitry, 2011). For 3 consecutive days, participants in this study wrote for 10-20 minutes about either their feelings about their recent MI (experimental group) or about daily activities (controls). They were assessed at three time points (1, 2 and 5 months after the intervention, in relation to quality of life (self-report), clinical health (assessed by a cardiac nurse), the

number of subsequent GP/hospital visits and amount of prescribed medications. Participants were also asked whether they returned to work, or why they did not do so and when they thought they would. Results showed that, at 5 months follow-up, experimental participants experienced less cardiac-related symptoms and they had made fewer GP/hospital visits than those in the control group. Additionally, a decrease was observed in medicines prescribed for the experimental participants, whereas control participants had more medicines prescribed over time. At the 5 months follow-up, expressive writing participants had lower diastolic blood pressure, less GP/hospital visits than controls, and the number of prescribed medications decreased in this group over the months (an opposite trend was found for the control group patients regarding the medicines). Of note, patients who took part in the expressive writing task attended more cardiac rehabilitation sessions than neutral writing participants, which might have been an important mediating factor in the relationship between expressive writing and outcome measures.

A more recently published study found positive effects of writing on health related quality of life (HRQoL) in cardiac patients (D. Hevey, Wilczkiewicz, & Horgan, 2012). Post-MI patients wrote for 20 minutes a day, for 3 consecutive days, either revealing their MI-related feelings (experimental condition, n = 43) or discussing trivial topics (controls, n = 46). Patients' HRQoL, measured before and after writing intervention, and at a 3 months follow-up, was significantly higher in the intervention group than the control group. HRQoL increased significantly for the expressive writing group compared with the control group. Furthermore, individuals with Type D personality benefited more from the intervention than non-Type Ds.

2.3.1.6. Other chronic disease samples.

In Smyth's et al.'s study asthma and rheumatoid arthritis patients were randomized to either an experimental group (39 asthma and 32 rheumatoid arthritis) or the control group (22 asthma, 19 rheumatoid arthritis)(J M Smyth, Stone, Hurewitz, & Kaell, 1999). Experimental participants wrote about the most traumatic experience in their life, whereas the controls wrote about trivial topics. Outcomes measured were spirometry test results for asthma patients and disease severity for RA patients as assessed by a rheumatologist. The experimental asthma patients' lung function improved 4 months after the intervention, compared to the control asthma patients, and experimental RA patients benefited in terms of disease symptoms compared to controls with RA. In a study with fibromyalgia patients (J. Broderick, D. Junghaenel, & J. Schwartz, 2005), participants were randomized to either the emotional writing condition, neutral writing condition, or the usual care (control) condition. Expressive writing was associated with increases in psychological wellbeing (quality of life, anxiety and depression levels), and with a reduction of pain and fatigue 4 months post-intervention, although these outcomes were not sustained at a 10 months follow up. Fibromyalgia patients were also randomised to either a writing (n = 38) or the control condition (n = 34)(Gillis, Lumley, Mosley-Williams, Leisen, & Roehrs, 2006). The experimental participants wrote about a stressful event that still bothered them, and its effects on their illness, whereas control participants wrote about neutral topics. At the 1-month follow-up disclosure participants reported better sleep quality than control participants, however control participants had less negative mood and more social support at the 1-month follow-up than did the experimental participants. On measures of disease impact, sleep quality and health care utilisation the writing group improved more at 3-months follow-up than the control group, which reported worse sleep quality and increased health care utilization.

2.3.1.7. Expressive writing and psychological wellbeing.

Findings from most studies described above give insight into the effects of expressive writing on psychological wellbeing. Research on healthy students supports the notion that written emotion expression reduces long-term distress despite inducing it immediately after the disclosure session (Pennebaker, et al., 1988; Petrie, et al., 1995). In Pennebaker and Beall (1986) immediate increases in negative mood (e.g. anxious, sad, guilty) were observed in all three trauma writing conditions, whereas in the control group an increase in positive mood was noted. Of note, experimental participants' negative mood increased throughout the three writing days. In Pennebaker et al., (1988) higher distress levels (physical symptoms and negative mood) were found in the expressive writing group compared to the control group, immediately after the study; however experimental group had better mood at the 3-month follow-up. Subsequent studies reported similar effects (James W Pennebaker, 1997; K.J. Petrie, R.J. Booth, J.W. Pennebaker, K.P. Davison, & M.G. Thomas, 1995).

In Murray et al. (E. J. Murray, Lamnin, & Carver, 1989) and Donnelly and Murray (D A Donnelly & E J Murray, 1991) investigated whether the writing task is comparable to psychotherapy, participants either wrote about, or attended psychotherapy sessions, and discussed traumatic or trivial topics. In in the second study (Murray et al., 1991), when both interventions were conducted over 4, rather than 2 days, the results showed positive changes in cognitive processing, self esteem and adaptive behaviour in both groups. Although the benefits were initially greater in the psychotherapy group, the difference became less obvious over the course of the intervention. Still, the writing induced immediate (pre- to post-session) increases in negative mood. This was investigated further (D.A. Donnelly & E.J. Murray, 1991) and again it was observed that although both groups reported high painfulness of the event they disclosed, worse mood disturbances were observed for the writing task

participants. After the intervention however, both groups reported perceived benefits of the interventions, relative to controls. The authors explained this in terms of differences between the mode of expression – writing vs. talking. Verbal emotion expression is further discussed in Chapter 3, section 3.2.

Participants with previous psychological trauma (e.g., sexual abuse, severe illness) benefit from engaging in expressive writing, showing positive changes in psychopathological indices, and a decrease in rumination and avoidance (Schoutrop, Lange, Hanewald, Duurland, & Bermond, 1997), and furthermore, better physiological (J.M. Smyth, et al., 2008) and physical functioning (M. Greenberg, et al.; King & Miner, 2000). In a study conducted on female students with PTSD symptoms (n = 49), outcomes such as depression, PTSD symptoms, physical symptoms and the amount of sick days were all at significantly lower levels in participants who engaged in expressive writing than in controls (D. Sloan & B. Marx, 2004).

Research also suggested positive effects of expressive writing for posttraumatic stress patients (J.M. Smyth, et al., 2008). Participants in this study (n = 24; all diagnosed with PTSD) rated their mood, PTSD symptoms, post-traumatic growth (the degree of positive changes reported after experiencing a traumatic event as measured by the Post-Traumatic Growth Inventory) and saliva cortisol level (the latter outcome was measured before and after each session). Although participants who wrote about their traumas experienced less positive emotions and their physiological arousal levels were higher by the end of the study they had greater decreases in anger and emotional tension. Moreover, the experimental group cortisol levels decreased more than those of the control participants from before to after each session. Finally, post-traumatic growth was greater in the experimental group relative to the control. In another study, experimental participants (suffering bereavement) were asked to write about the loss of their loved one, for 15 minutes daily for four days (n = 30) (Range, Kovac, &

Marion, 2000). The control group wrote about neutral topics (n = 34). Participants in the experimental condition have been grieving for significantly less period of time than the trivial writing participants.

2.3.1.8. Expressive writing and Alexithymia.

Expressive writing may have positive health effects for patients high in Alexithymia. In Solano et al.'s study (Solano, Donati, Pecci, Persichetti, & Colaci, 2003). Forty patients awaiting a bladder papilloma resection were assessed for Alexithymia, distress and postoperative course. Half of the patients engaged in 20-minute expressive writing sessions, for 3 consecutive days, starting 3-4 days before the surgery. The rest of the patients, who constituted a control group, did not take part in the writing task, but were assessed at the same time points as the experimental participants. As a result, patients who engaged in expressive writing had a shorter time of post-operative hospital stay and lower general score on the symptom check list. *Post hoc* tests showed that these differences between the experimental and control groups were only significant for patients high in Alexithymia, who benefited more from the intervention. This suggests that encouraging emotion expression in those people who don't naturally express their emotions might be of benefit. This has been confirmed in another study on students with Alexithymia (Paez, Velasco, & González, 1999) which also suggested that in the case of inhibited individuals brief expression interventions may have adverse effects as compared to in-depth disclosure.

2.3.1.9. Negative or null effects of expressive writing.

Immediate negative mood effects of expressive writing have been reported in some studies, however these effects usually disappeared a few weeks after the intervention. More profound adverse effects of writing were observed in studies with PTSD patients. For example, in

Gidron et al.'s study (Gidron, Peri, Connolly, & Shalev, 1996) participants were randomised to either the disclosure group (n = 8) or the neutral writing condition (n = 6). They wrote daily for three days, and were followed-up at 5 weeks post-intervention. The results of this study were that the disclosure patients had an increase in health centre visits at 5 months post-intervention, and an increase in avoidance symptoms. Written disclosure was also suggested to have a negative impact on psychological functioning in patients after limb amputation (Gallagher & MacLachlan, 2002). In Gallagher and MacLachlan's study forty eight participants were randomly assigned either to the emotional writing group or to the neutral writing group. Worse social adjustment was associated with being more affected by the essay topic. Moreover, participants writing about more severe topics reported restrictions in physical activity. Finally, participants who wrote about previously undisclosed topics reported more functional satisfaction with the prosthesis also admitted to – a finding that actually indicates a positive effect of writing in this sample showing that among lower limb amputees writing about previously withheld experiences may have a positive impact on some aspect of living with the prosthesis. This study was however limited by a high attrition rate. High-risk samples, such as PTSD patients, or limb-amputees should, in future, be approached with care when it comes to emotion expression interventions.

In addition some studies fail to find an effect of expressive writing, e.g., in suicidal college students (Kovac & Range, 2002), students with negative body image (Earnhardt, Martz, Ballard, & Curtin, 2002), bereaved persons (Stroebe, Stroebe, Schut, Zech, & van den Bout, 2002), patients with Rheumatoid Arthritis (J. Broderick, A. Stone, J. Smyth, & A. Kaell, 2004), or asthma patients (A. Harris, C. Thoresen, K. Humphreys, & J. Faul, 2005). However, null findings from some studies suggest that future research could focus on examining moderators of expressive writing's effects.

2.3.2. Effects of expressive writing: meta-analyses.

Table 1 presents the results of meta-analyses investigating the effects of expressive writing on different outcomes. Smyth's (1998) meta-analysis included 13 RCTs and reported the overall effects size of expressive writing to be $d = 0.47$ ($r = .23$) (and after excluding an outlier study: $d = 0.41$ ($r = .20$, $p < .0001$)). After comparing effect sizes in different samples the only significant difference emerged between students and non-students in psychological outcomes: students' benefited more ($d = 0.76$) than non-students ($d = 0.34$).

Smyth also compared studies with different number of writing sessions (range: 1 - 5), different length of each sessions (range: 15 – 30 minutes) and different duration of the entire intervention (range: 1 – 28 days). Studies that spaced out the writing sessions over a longer period produced a greater overall effect size, although these longer interventions had no significant impact on psychological or physiological outcomes' effect sizes. Greater effect sizes in psychological wellbeing were observed in experimental groups where participants wrote about current rather than past traumas. Additionally, greater effects sizes were found in physiological functioning in studies where participants wrote about either past or current traumas rather than only past traumas. There was an overall rise in distress in expressive writing groups from before to immediately after the intervention but this was unrelated to health outcomes.

A small meta-analysis was conducted by Frisina et al. (Frisina, Borod, & Lepore, 2004) in which 9 RCTs were analysed. The analysis only investigated the effects of expressive writing on health in clinical samples. A small, but significant mean effect size for health outcomes was found ($d = 0.19$). Furthermore, expressive writing seemed to have a positive effect on physical health ($d = 0.21$).

In Meads and Nouven's meta-analysis (2005) 61 RCTs were analysed. Separate analyses were conducted for the following samples: 1) physical conditions (e.g. cancer, reumatoid

arthritis, HIV, headaches; $n = 13$; e.g. J. E. Broderick, D. U. Junghaenel, & J. E. Schwartz, 2005; Kelley, Lumley, & Leisen, 1997), 2) psychosocial stressors (e.g. PTSD, child sexual abuse, frequent clinic visitors, exams, unemployment; $n = 18$; e.g. Batten, Follette, Rasmussen Hall, & Palm, 2003; M. A. Greenberg, C. B. Wortman, & A. A. Stone, 1996) and 3) healthy samples (including children; $n = 28$; e.g. Reynolds, Brewin, & Saxton, 2000; Sheffield, Duncan, Thomson, & Johal, 2002). They concluded that there is insufficient evidence for positive effects of expressive writing on psychological or physical health. Harris' meta-analysis (2006) analysed the effects of expressive writing on health care utilization among healthy participants (13 RCTs), participants with medical conditions (e.g. cancer, asthma; 6 RCTs) and participants with psychiatric conditions (e.g. depression, PTSD; 10 RCTs). This partition was a result of heterogeneity analysis, which indicated that studies with different samples should not be combined in one analysis. Harris' meta-analysis showed that expressive writing had a positive effect on health care utilization in healthy participants ($g = 0.16$; $CI_{95} = 0.02$ to 0.31), but it had no such effect on medical ($g = 0.21$; $CI_{95} = -0.03$ to 0.43) or psychiatric populations ($g = 0.06$; $CI_{95} = -0.12$ to 0.24); although in case of psychiatric populations the effect increased with more writing sessions. This meta-analysis concluded that expressive writing might have a different effect on different samples, and that although writing does not decrease health care utilization for some samples, it might still have an effect on other outcome measures in these samples (e.g., mood, or specific illness symptoms).

In a meta-analysis that analysed 30 RCTs on expressive writing, Mogk et al. (Mogk, Otte, Reinhold-Hurley, & Kröner-Herwig, 2006) concluded that there are no health or psychological benefits of expressive writing ($g = 0.04$, $CI_{95} = -0.08$ - 0.15), for psychological outcomes ($g = .01$, $CI_{95} = -0.17$ - 0.19), and for physical health outcomes ($g = 0.07$, $CI_{95} = -0.06$ - 0.19). Additionally, no effect was found for subjective health and objective health ($g =$

-.05, $CI_{95} = -.18 - .09$, and $g = .01$, $CI_{95} = -.27 - .29$, respectively). Of note, in Mogk's meta-analysis different health outcomes were analysed together (e.g. immune factors and breathing parameters), whereas these were separated in Frattaroli's one (2006).

Frattaroli (2006) analysed 146 studies, out of which 102 (70%) had a positive effect size.

Results of this analysis gave an overall (unweighted) effect size of .075, a highly significant result ($p < 0.001$), suggesting a positive overall effect of expressive writing. Positive effects of writing were observed on overall psychological health ($n = 112$ studies, $r = .056$, $p < .00014$), and on separate psychological variables such as anger ($r = .183$), distress ($r = .102$), anxiety ($r = .051$), depression ($r = .073$) and positive human functioning ($r = .045$).

Significant effects were also found on overall physiological functioning ($n = 30$ studies; $r = .059$, $p < .0075$), and separately on immune parameters ($n = 13$, $r = .099$). These results suggest that expressive writing has positive effects on both psychological and physical health.

Table 1
Results of Meta-Analyses Investigating the Effects of Expressive Writing

Meta - analysis	Number of RCTs	Type of analysis and Sample categories (number of RCTs)	Outcomes analysed (number of RCTs) and effect sizes:	Overall Effect Size	Limitations
Smyths, 1998	13	<u>Analysis of studies on the following samples:</u> <u>Healthy Samples:</u> Healthy college students (9) University employees (1) Adolescents (1) Prison inmates (1) Unemployed (1)	<u>Outcomes:</u> Reported health (9) $d = .42$ Psychological wellbeing (9) $d = .66$ Physiological functioning (4) $d = .68$ General functioning (5) $d = .33$ Health behaviours (6) $d = .03$	$d = .47$ $r = .23$	
Frisina et. el., 2004	9	<u>Physical illness samples:</u> Cancer patients (4) Asthma and rheumatoid arthritis (1) <u>Psychological samples:</u> PTSD patients (2) Depressed, suicidal patients (1) Psychiatric prison inmates (1)	<u>Outcomes:</u> Physical health (8) $d = .21$ Psychological health (8) $d = .07$	$d = .19$ $r = .10$	Limited number of studies Limited samples within some of the studies

Meads & Nouwen, 2005	61	<u>A combined analysis as well as separate analyses within the following sample categories:</u> <u>Healthy samples (28)</u> including one with children <u>Physical illness (14)</u> <u>Cancer (5)</u>, RA (2), college students with physical symptoms (2), asthma (1), HIV (1), headaches (1), fibromyalgia (1), orthopaedic patients (1) <u>Psychosocial stressors (16)</u> PTSD (2), trauma (3), child sexual abuse (1), having a baby in intensive care (1), bereavement (3), natural disaster sufferers (1), prisoners (1), unemployed (1), relationship breakup (1), undertaking exams (1), frequent clinic visitors (1)	<u>Outcomes:</u> <u>Health centre visits:</u> overall WMD = -.09 healthy samples WMD = -.11 <u>Subjective health measures:</u> overall WMD = -.95 <u>Psychological outcomes</u> Positive mood SMD = .56 Negative mood SMD = .37 Anxiety SMD = .16 Depression SMD = .22 Avoidance WMD = -.06 Intrusion WMD = .17 <u>Objective health measures:</u> no effect reported <u>Performance:</u> no effect reported	Not measured	6 studies measuring physiological and immunological outcomes were excluded from this analysis High heterogeneity of studies

Harris, 2006	30	<u>Separate analyses within each of the following sample categories:</u> <u>Healthy samples (14)</u> College students (13) Employed adults (1) <u>Physical illness samples (6)</u> Fibromyalgia (1) Asthma (1) Cancer (prostate, breast; 2) Irritable bowel syndrome (1) Cystic fibrosis (1) <u>Psychological samples (10)</u> Sexual abuse (1) Trauma (2) Bereavement (due to natural death or suicide; 3) Suicidal thoughts (1) High in somatic symptoms (1) Psychiatric prison inmates (1) Romantic breakup (1)	<u>Outcome: Health Care Utilisation</u> <u>Samples:</u> <u>Healthy samples:</u> $g = .16$, $CI_{95} = .02$ to $.31$ <u>Physical illness samples:</u> $g = .21$, $CI_{95} = -.03$ to $.43$ <u>Psychological samples:</u> $g = .06$, $CI_{95} = -.12$ to $.24$		High heterogeneity of studies Limited number of studies within analysed categories
Mogk et. el., 2006	30	<u>Healthy samples (17)</u> Students (16; including 2 studies on children) University employees (1) <u>High risk samples (7)</u> Trauma (sexual abuse, natural disaster), negative body image, bereaved, unemployed, parents of chronically ill children <u>Clinical samples (4)</u>	<u>Outcomes:</u> <u>Physical illness (24)</u> (Including physiological functioning and somatic symptoms): Overall $g = .07$, $CI_{95} = -.06$ to $.19$ Self reported health $g = -.05$, $CI_{95} = -.18$ to $.09$ Objective health $g = .01$, $CI_{95} = -.27$ to $.29$ <u>Psychological health (19)</u> (PTSD, depression, anxiety, negative affect): $g = .01$, $CI_{95} = -.17$ to $.19$	$g = .04$, $CI_{95} = -.08$ to $.15$	No heterogeneity test reported Division into sample categories not thoroughly explained, i.e. out of 30 studies, 16 were analysed within the “normal” (healthy) group, 7 within the “high risk” group, 4 within the “clinical” group and 8 within the “miscellaneous” group, meaning that groups overlap and some studies were included

		Cancer, PTSD, asthma, psychiatric prison inmates Other Frequent clinic visitors, students with hep. B inoculation	<u>Health behaviour variables (16)</u> (clinic visits, sick days, smoking, alcohol use, drug use, diet, exercise, sleep patterns) $g = .20$, $CI_{95} = .04$ to $.36$ <u>Samples:</u> Healthy student samples (16): $g = .07$, $CI_{95} = -.03$ to $.22$ High risk samples (7): $g = -.03$, $CI_{95} = -.06$ to $.20$ Posttraumatic samples (6): $g = -.10$, $CI_{95} = -.25$ to $.46$ Clinical samples (4): $g = .08$, $CI_{95} = -.15$ to $.31$ Miscellaneous (8) $g = 0$, $CI_{95} = -.19$ to $.26$		in more than one group.
Frattaroli, 2006	146		<u>Psychological health (112) $r = .034$ (weighted, random effects; $p < .0001$)</u> Significant effects: Depression $r = .07$ Positive functioning $r = .05$ Distress $r = .10$ Anger $r = .18$ Anxiety $r = .05$ <u>Physiological functioning (30) $r = .06$ (unweighted, $p = .008$, random effects)</u> Immune functioning $r = .10$ (random effects) <u>Reported health (95) $r = .07$ (unweighted, $p = .0001$,</u>	.063 (weighted) 70% of studies with a positive effect size ($p < .001$)	

			random effects) Disease outcomes $r = .13$ (RE) Illness behaviours $r = .07$ (RE) <u>Health behaviours (10) $r = .007$ (ns)</u> <u>General functioning (43) $r = .05$ ($p = .006$, random effects)</u> Social relationships $r = .06$ Cognitive functioning $r = .06$ <u>Subjective impact of the intervention (33) $r = .16$ ($p = .00004$, random effects)</u> Positive attitude towards intervention (26) $r = .27$ Attempts to process the event (21) $r = .13$		

Note. Types of effect sizes: d : Cohen's d ; r : coefficient r ; g : Hedges g ; WMD: Weighted mean difference (random effects), SMD: Standardised mean difference (random effects)

2.3.3. Moderators of the effects of expressive writing on different outcomes.

A number of different theories have been proposed to explain who can benefit from expressive writing interventions (see Pennebaker & Chung, 2007). The inhibition theory suggests that emotion expression is most needed where emotions are the most inhibited, which implies that personalities for which inhibition is an underlying factor should benefit from expression. Indeed, Type D personality was found to moderate the effect of writing on HRQoL (D. Hevey, et al., 2012) among cardiac patients in the way that post-MI patients with Type D personality benefited more from emotion expression than patients without it.

Alexithymia is another moderator of the effect, as studies have found expressive writing is more beneficial for people with Alexithymia, rather than those without this trait (O'Connor & Ashley, 2008; Páez, Velasco, & González, 1999; Solano, et al., 2003). The fact that people with either Type D personality or Alexithymia may benefit more from emotion expression suggests that certain emotion inhibition must pre-exist for the intervention to bring positive results. Repressive coping, on the other hand, has not been found to moderate the effects of expressive writing (Baikie, 2008; Lumley, Tojek, & Macklem, 2002) suggesting that brief expression interventions may not be suitable for individuals who have removed negative affect from consciousness.

Other moderators of the effect of emotional disclosure on outcomes have been suggested in the largest and most recent meta-analysis conducted by Frattaroli: sample type (effects greater in physical illness samples than in no illness samples), study setting (effects greater for private room setting than public room), psychological factors (effects greater for participants higher in stress, but also for participants high in optimism), follow-up length (effects greater when outcomes were measured less than 1 month after disclosure than more than 1 month), length and number of disclosure sessions (effects greater for longer sessions and for at least three sessions), months since the event (effects greater for more recent topics),

topic of disclosure/specific instructions (greater when participants disclosed previously unshared topics).

The fact that people with either Type D personality or Alexithymia may benefit more from emotion expression suggests that certain emotion inhibition must pre-exist for the intervention to bring positive results, therefore encouraging emotion expression in those people might be of benefit.

2.3.4. Mechanisms (mediators) underlying the health effects of written disclosure.

Sloan and Marx (2004b) reviewed three theoretical models that aimed to explain the mechanisms behind written disclosure. The first explanation of the effect was put forward by Pennebaker in his inhibition theory. While emotion inhibition was associated with “physiological work” and with adverse health effects, emotion expression (disinhibition) was proposed to reverse these effects. The pathway was physiological and similar to that of the stress-disease association. The second model is the cognitive adaptation model (J W Pennebaker, 1997a; J. Smyth, True, & Souto, 2001) which relates to the fact that a traumatic experience generates a cognitive dissonance, which needs to be resolved in order to recover from trauma (Horowitz, 1986). Cognitive processing theory emphasizes the need to make sense of, organise and integrate thoughts and feelings about an event to accrue benefit (Pennebaker, 1993). This can be achieved when negative experiences are expressed with the use of language, facilitating the organization of the knowledge surrounding the experience (Sloan & Marx, 2004a; Smyth et al., 2001) and reappraisal (Pennebaker & Beall, 1986). This cognitive processing leads to understanding, which decreases the need for rumination, and therefore stress, and may positively impact body and mind (Pennebaker, 1989; J W Pennebaker, 1997a). In order to test the cognitive adaptation model, written narratives have

been subjected to either content analysis (Creswell et al., 2007) or linguistic analysis with an emphasis on the use of cognitive words (e.g., insight or cause words; Shaw, Hawkins, McTavish, Pingree, & Gustafson, 2006). An increased use of cognitive words (e.g., “cause”, “know”) has been associated with increased benefits of expressive writing (Pennebaker, Mayne, & Francis, 1997). Linguistic analysis of a narrative is described in detail in section 2.4. It has been proposed that research on rumination might further support the cognitive adaptation model if expressive writing was found to decrease intrusive thoughts (Lepore, Greenberg, Bruno, & Smyth, 2002). Similarly, in a study on distressed students (Gortner, Rude, & Pennebaker, 2006), the benefits (decrease in depression symptoms) of expressive writing were mediated by rumination.

The emotional exposure-habituation model is theoretically linked to exposure-based treatments within behavioural therapy (Foa & Kozak, 1986; Meadows & Foa, 1999), where a classically conditioned link between an event and the individual’s reaction to it can be extinguished during repeated exposure. One of the features of expressive writing is reliving negative experiences and emotions from the past, exposing a person to stress (Lepore et al., 2002; Sloan & Marx, 2004b). By repeatedly exposing the individual to the aversive stimulus, expressive written disclosure may facilitate habituation to both stimulus and the negative affective response, consequently extinguishing negative emotional associations, which produces beneficial outcomes (Lepore, 1997). Such habituation of negative affect during the course of expressive writing sessions has been reported in the literature (Sloan, Marx, & Epstein, 2005) and decreases in negative emotion word use would reflect such a process. To test this model, some researchers focused on analysing changes in affect during a series of writing sessions, while others concentrated on the link between intrusive thoughts (rumination) and adverse physical and psychological outcomes (Sloan & Marx, 2004a).

Following the first approach, in support of the model, it has been found that within-session increases in negative affect became less profound over the course of several sessions and the opposite was true for positive affect (Lepore et al., 2002). Elsewhere however no association between changes in affect and outcome has been reported (Kloss & Lisman, 2002).

More support for the model comes from Lepore and Greenberg's study (2002) where expressive writing was reported to reduce the adverse effects of rumination, therefore decreasing negative symptoms that were associated with it.

Sloan and Marx (2004a) suggested that it is a mixture of processes, rather than one model, that is responsible for positive effects of expressive writing. Subsequent research distinguished between written expression of insights or written expression of negative emotions, and suggested that each is associated to a different explanatory model - cognitive adaptation or exposure-habituation, respectively (Shim, Cappella, & Han, 2011). It was observed that disclosure of insight words (e.g., "understand", "know") was associated with less illness-related concerns (in cancer patients) at 4-month follow-up, whereas emotion words (both positive, e.g., *happy*, *joy*, and negative e.g., *angry*, *frustrated*) were unrelated to this outcome. This supports the cognitive adaptation model. The exposure-habituation model was tested in the same study by analysing the use of emotion words. Whereas positive emotion words were unrelated to outcomes, negative emotion word use was associated with better functional well-being in cancer patients at follow-up.

2.3.5. Summary.

The inhibition theory views emotion expression as an antidote to the adverse effects of inhibition. Research on expressive writing indeed suggests that expression has a number of positive effects on psychological health (e.g., decreasing negative affect), physiological

functioning, and immune parameters (Frattaroli, 2006), although negative effects were found in PTSD samples (e.g., Gidron, et al., 1996).

An important moderator of the effects of writing is emotion inhibition – expression seems to be more beneficial for those, who are suppressors (e.g., Gortner, et al., 2006), which is consistent with the inhibition theory. In line with this, Alexithymia was found to moderate the effects of writing on outcomes (O'Connor & Ashley, 2008; Páez, et al., 1999; Solano, et al., 2003) whereas Type D was found to moderate the positive effects of expressive writing among cardiac patients (D. Hevey, et al., 2012). Beside the fact that expression is more effective for suppressed samples, it works possibly through three different channels (D. M. Sloan & B. P. Marx, 2004b): physiology (disinhibition), emotional habituation, and cognitive change (decrease in rumination and increase in reappraisal). Whereas physiological changes can be assessed by measuring indexes such as BP, HR, and HRV, the degree of emotional habituation can be investigated through linguistic analysis of a narrative (see below). Cognitive change can be measured by changes in reappraisal and rumination, as well as through analysis of language.

2.4. Linguistic Analysis of a Narrative

The mere emotional expression of a trauma is not sufficient (Krantz & Pennebaker, 2007); translating experiences into language appears to be essential for the writing to be effective, as it allows the person to assign meaning, coherence and structure to the event, which facilitates assimilation of the event: such assimilation may enhance coping responses, and subsequently result in better adjustment post-event (Pennebaker & Chung, 2011).

The idea that underlined the initial expressive writing studies (e.g., Pennebaker & Beall, 1986) was that writing allowed participants to express previously inhibited thoughts and emotions related to a traumatic/stressful experience. By allowing previously avoided material

to be processed and organized, such expression would have psychological and physical health benefits. As people's thought processes and emotional states are reflected in the words they use (Tausczik & Pennebaker, 2010), linguistic analysis of a narrative has a potential to test what mechanisms lie behind the benefits of emotion expression (Shim, et al., 2011). For example, greater use of causal (e.g., *because*, *effect*) and insight (e.g., *think*, *consider*) words facilitates the building a narrative framework over the course of writing, which may result in a reappraisal of the experience in a less threatening manner (Pennebaker & Chung, 2011) and, consequently, in a reduction of negative emotions, as indexed by less use of negative emotion words (e.g., *hurt*, *nasty*), arising when thinking and writing about the experience.

Research supports the argument about the importance in expressive writing of reappraisal, understanding and making sense of the negative event (Pennebaker, 1993); a mediating effect of cognitive processing has been reported from expressive writing studies showing that those people who used more cognitive processing words benefited most (Ulrich & Lutgendorf, 2002). Other studies have similarly reported that positive effects of expressive writing are strongest when an increased use of these cognitive processing words over time is observed (Pennebaker, 1993; Rivkin, Gustafson, Weingarten, & Chin, 2006), which indicates active reappraisal of the events (Tausczik & Pennebaker, 2010). With regards to emotion words, it has been proposed that the benefits of expressive writing were either associated with increases in negative words use and decreases in positive words use (Pennebaker, 1993), or to simultaneous increases in positive and negative words use (Pennebaker & Francis, 1996). In general, findings have been mixed; health improvements have been linked with greater use of positive emotion words in some studies (Danner, Snowdon, & Friesen, 2001), but negative emotion words in others (Pennebaker, 1993). After combining research data it was suggested that a typical process of healing from negative experience would involve a decrease in

negative emotion words use combined with increased use of cognitive words and positive emotion words (e.g., Pennebaker, Mehl, & Niederhoffer, 2003). Later, Pennebaker and Chung (2007) explained that using insight and causal words in conjunction with positive emotion words is a language pattern that reflects a positive reappraisal of the experience, which may facilitate cognitive broadening (Fredrickson, 2001) to produce expressive writing's benefits. Indeed, in a very recent study, among those MI patients in the expressive writing condition, greater improvement in HRQOL was associated with greater increases from the first to the third writing session in use of positive words and cognitive processing words (David Hevey & Wilczkiewicz, 2014).

Pennebaker, Francis, and Booth (2001) developed a computer programme called Linguistic Inquiry and Word Count (LIWC), which quantifies the language statistics (e.g., word count, sentence length) and linguistic dimensions (e.g., pronoun use) of written text. LIWC is described in detail in Chapter 5, section 5.9.3.

2.5. Chapter Summary

Emotion expression is a behaviour (verbal or non-verbal) that informs others, or self, of one's emotional state. It has been studied in relation to psychological wellbeing (Pennebaker, 1997), physiology (e.g., Petrie et al., 1995), and somatic disease (Willmott, Harris, Gellaitry, Cooper, & Horne, 2011) through pathways such as catharsis, reappraisal, awareness or neurophysiology. The most well studied technique of emotion expression is the expressive writing task (Pennebaker & Bell, 1986), based on writing either about deepest thoughts and feelings regarding the most traumatic experiences of one's life, or about trivial topics. Benefits of writing for physical and psychological health have been observed in numerous studies, although null and adverse effects have been also noted (Frattaroli, 2006). The results of the most recent meta-analysis, which combined the greatest amount of

expressive writing research, suggest that expressive writing has positive effects on psychological and physiological functioning, as well as on the immune system. The effects of expressive writing have been found to be moderated by emotion inhibition (Gortner et al., 2006), including Type D personality (e.g., Hevey et al., 2012) and Alexithymia (e.g., O'Connor & Ashley, 2008), in that the benefits of emotion expression were greater for those high in inhibition. Expressive writing was also more effective for people with physical illness or for people higher in stress (Frattaroli, 2006). As people with Type D or Alexithymia are at the same time higher in stress, and more prone to illness than those without those traits, emotion expression interventions in those groups may bring required effects and should be investigated even further.

Three mechanisms that were proposed to be underlying the health effects of written disclosure, disinhibition (decrease in physiological indexes), the cognitive adaptation model and the emotional exposure-habituation model (Sloan & Marx, 2004b). Disinhibition is typically tested by measuring participants' blood pressure, heart rate, and more recently, also heart rate variability, as an index of the vagal tone. Both other models can be tested with the use of linguistic analysis of a narrative. The cognitive model can additionally be tested by measuring changes in reappraisal and rumination. Findings of research on language use during disclosure suggest that greater use of cognitive words (or cognitive words in conjunction with positive emotion words) facilitates reappraisal of the experience and reduces negative emotions, whereas increased use of positive and moderate use of negative words is associated with mood benefits (Pennebaker et al., 1997).

Social sharing and Verbal Emotion Expression

Although social sharing has been defined as the sharing of thoughts and emotions related to an emotional life event, there may be a number of important differences between actual verbal emotion expression and social sharing. Both of these concepts are outlined below.

3.1. Social Sharing

After experiencing a negative life event people often share their thoughts and emotions, and they do so as they believe that discussing their experiences with other people helps them to cope (Pennebaker, Zech, & Rimé, 2001). Mixed definitions of social sharing have been used in research: some studies view social sharing as being with others during an emotional episode (e.g., Wagner et al., 2014), rather than disclosing one's thoughts and emotions to others. The next section of this chapter describes the social sharing definition used in the present study.

3.1.1. Definition of Social Sharing.

Social sharing of memories, thoughts and emotions is defined as "...a process that takes place in the minutes, hours, days, even weeks and months – and sometimes years, or even an entire life – following an emotional episode" (B. Rimé, 2009, p. 18). In this process, memories, thoughts and emotions associated with a life event are discussed with one or more people, or with an imaginary (e.g., a diary) listener (B. Rimé, 2009). Most people engage in social sharing (for a review, see Rime, 2009); for example, Rime et al. (B Rimé, Philippot, Boca, & Mesquita, 1992) found that up to 96% of people talk about negative experiences, thoughts, and feelings with others, and over 60% of stories are shared in the first 48 hours after the

event occurred. Similarly, a study testing the extent of sharing after 9/11 and Madrid 2004 attacks (Bernard Rimé, Páez, Basabe, & Martínez, 2010) found that over 80% of people engaged in spontaneous social sharing as a result of these events. Social sharing occurs for people of different ages, gender, education levels and culture. The extent of this process is unrelated to emotion type, one's age, culture and level of education (e.g. B. Rimé, Finkenauer, Luminet, Zech, & Philippot, 1998; Rime, Mesquita, Boca, & Philippot, 1991b).

3.1.2. When, with whom, and how much do people share?

People share emotions naturally and the extent of sharing is unrelated to the valence of emotion, with the exception of emotions of shame and guilt, which are associated with avoidance rather than seeking of support (B. Rimé, 2009). According to Rime, social sharing is secondary to one's need to talk about emotional events, whether it is with a group of people, one person, or even a symbolic recipient (e.g., a diary). Sharing is usually initiated on the day of the event and it lasts (with varying intensity) for weeks, months and sometimes even years. Most emotions are shared immediately after the event, and repeatedly, with the exception of shame and guilt. People tend to share life events if they are distressed by them, but inconsistencies in literature exist regarding the intensity of emotion required for sharing to occur; some believe a certain threshold must be reached and another not to be exceeded for people to disclose an event (e.g., Gilbert, 1976), while others maintain that emotion must be intensive (O Luminet, Zech, Rimé, & Wagner, 2000). Many people ruminate about past traumas even 20 years after they experienced a stressful event and some of them still wish they could discuss it with others (Tait & Silver, 1989). For sharing partners people usually choose their intimates (children choose their parents, adults choose spouses and friends) (B. Rimé, 2009). However people do not limit themselves to one sharing partner, but usually

discuss the same topic with different people, especially if the emotions generated by the event were intense.

3.1.3. Social sharing research: methodology and findings.

Early research on social sharing tested the extent to which people share emotions, how soon they do so, whether demographic variables (i.e., age, education, personality, and cultural differences) influence the frequency or intensity of social sharing, and whether either of these variables impact recovery from an emotional event (B. Rimé, et al., 1998). Those initial social sharing studies applied retrospective autobiographical procedures, i.e., participants were asked to think of an emotional event they had experienced and to state whether they talked about it to anyone, to whom, how soon after the experience, and how often they did so (B. Rimé, 2009). Rime's six studies into social sharing (Rime, Mesquita, Boca, & Philippot, 1991a) are an example; healthy volunteers were asked to recall an emotional life event from the past (that induced a particular emotion) and subsequently answered questions about their sharing of the event. Results showed that more than 88% discussed their experience with others and most of them did so the same day or soon after. These conversations were most often held with friends and loved ones, although men were more likely to discuss their experiences with their spouses than friends, whereas women did not have a preference. People were quicker in sharing events that generated anger or anxiety, than those that induced other emotions; shameful experiences were shared as often as other emotions, however not as soon after the event, and mostly with family, as opposed to with friends or strangers. The unreliableness of the retrospective method, related to the amount of time that passed between the negative life experience and recalling of that experience, encouraged researchers to use a daily diary method - a more reliable procedure, as the time between an event and retrospection was less than a day (B. Rimé, et al., 1998). Of note, disadvantages of diary

methods have been noted; for example, the need to ensure that participants understand the instructions and follow the protocol, and participants' commitment and compliance need to be considered (Iida, Shrout, Laurenceau, & Bolger, 2012). Furthermore, writing a diary can influence the individual's behaviour, or, as result of daily writing, people may learn more about the concept that is being studied, therefore their answers might change with time (Iida, et al., 2012).

In studies using daily dairy method, participants were followed up for weeks after the negative event (such as bereavement, exams, first blood donation, first childbirth, and performing a first dissection) answering questions about the amount they shared that experience (Rimé, et al., 1998). All the experiences were later shared by participants, not only in the first week after they occurred, but also during subsequent weeks.

As initial research on the effects of sharing was prone to selective and reconstructed memory biases, investigators also designed laboratory-based social sharing experiments (e.g., Luminet IV, Bouts, Delie, Manstead, & Rimé, 2000), where participants would be exposed to an emotional event, after which the extent of their social sharing to a friend would be recorded. In those studies, participants watched three-minute emotion-generating films (the groups differed in the intensity of emotion generated, but not in the emotion type), after which they waited outside the lab with a friend, who also took part in an experiment (Luminet IV, et al., 2000). Participants' conversations were later analysed and the amount of words related to a watched film was assessed. Results were in line with previous research on social sharing: participants who watched the most emotional film shared this experience with a friend the most (40% of words referred to the event), and participants from the other two conditions (no emotion and moderate emotion) barely mentioned the films (less than 5 %). This suggests that in order to be shared, an emotion must reach certain intensity, a finding also supported by other studies (e.g., studies on dream sharing; A. Curci, Rimé, Gisle, & Baruffol, 2007). This

study can be criticised for not investigating the participants' narratives further, in order to test the percentages of emotion words used (if any). If, for example, it was found that people do not typically express emotion during spontaneous sharing, it would help understand the differences in the effects of expressive writing and social sharing.

Addressing this, Zech and Rime (2005) designed two studies inducing social sharing, including specific instructions as to what was to be discussed. In study 1, fifty one students were instructed to recall the most distressing emotional life event from their past, and to either discuss the experience with the experimenter (emotion sharing vs. factual sharing conditions), or to discuss trivial topics. In the emotion sharing condition participants were asked to concentrate on their emotions related to the event, whereas the other sharing group was asked to only share the facts. Additionally, the factual sharing group was specifically asked not to express any emotions. The control group talked about their typical day at the university. The impact of the experience and recovery from it were investigated, as well as perceived benefits of part-taking in the sharing study. All participants rated their emotional recovery from the experience at the beginning of the study and at 7 days and 2 months follow-ups. Additionally, perceived benefits of sharing and beliefs about the effects of sharing the event during the study were measured. Results showed that although 86% of recalled experiences had been previously shared with others and that the extent of sharing was unrelated to current emotional impact of the event. Despite significant time effects on emotional recovery, no differences in recovery were observed between the conditions, suggesting that even the control group recovered from the event in a similar manner. There were however significant differences between conditions in perceived benefits of sharing: the emotion expression participants rated laboratory sharing as more beneficial than the participants from the just facts group, and the just facts condition participants rated sharing as more beneficial than the control participants.

In study 2, students interviewed their acquaintances using a slightly different procedure. Instead of a just facts condition, a second emotion expression condition was added where participants were asked to share a different emotional episode than the one they initially thought of, when they entered the study. A second control condition was also added where participants did not engage in any type of sharing, but were assessed in the same way as the other three groups. As expected, participants in study 2 also viewed the sharing intervention as beneficial regardless of whether they disclosed the same event as the recalled one, or a different emotional experience. Perceived benefits in both sharing groups were significantly greater than in the control groups; however, no group differences in recovery from the event were observed. The second study additionally found that emotional recovery was slower in people who naturally shared the experience outside of the laboratory setting. Furthermore, those who ruminated more about the event were more upset, which is in line with previous research (e.g., Just & Alloy, 1997). There was no follow-up assessment in this study, as the post-intervention assessment was conducted 3 days after the intervention.

There are several shortcomings of the procedure applied in those studies that may have had an impact on the findings. First, there was only one sharing session, whereas in expressive writing research the norm was 3-4 sessions over consecutive days or weeks; in fact, the more sessions, the better the outcomes were (Frattaroli, 2006). Second, participants in the emotion expression group were asked to share “various feelings and emotions they felt during this episode, to examine and express them, to explain why they felt them and describe the implications the episode had for them” (p. 273), which suggests that they were not describing the event in detail, but rather concentrating on their emotions. In the writing task instructions, the emotion expression group described the event in detail and discussed related deepest feelings. Third, the outcome measured here was self-rated recovery from the event, whereas in expressive writing research benefits were found for general psychological, physiological

and behavioural outcomes. Given these limitations, there is a need for a study that would apply the instructions from the writing task to *multiple* social sharing sessions, measuring outcomes similar to those measured in expressive writing research.

3.1.4. The effects of social sharing.

The main presumption that drove social sharing research was that socially sharing an emotion would contribute to emotional recovery. Emotional recovery was defined as a “significant alleviation of the impact the memory of the episode had for the person” (Rime et al., 1998, p. 5). Rime et al.’s review (1998) presented the findings of several studies monitoring the amount of sharing participants engaged in immediately after an emotional event, including the laboratory studies (Luminet et al., 1996; in Rime, et al., 1998), and concluded that there was no relationship between the extent of sharing and emotional recovery from the experience. This was later confirmed in the laboratory studies on induced sharing (Zech & Rime, 2005). In their review, Rime et al. (1998) note, that there is a possibility that social sharing has benefits for some people, but not others; therefore, they recommend that future studies take into consideration individual differences such as emotion inhibition. This is in line with the inhibition theory (Pennebaker, 1985), and research, which found that people who naturally inhibit their emotions have been found to benefit more from emotion expression (D. Hevey, et al., 2012; Páez, et al., 1999).

Rime et al. (1998) presented other potential benefits of social sharing, including how social sharing may help construct a social narrative and consolidate the memory of the event, improve interpersonal relationships and social integration, and enhance the social knowledge about emotions. In terms of investigating the benefits of social sharing, future research would benefit from studies applying similar instructions, and focusing on outcomes similar to those

studied in expressive writing research (e.g., psychological distress, physiology, general health).

3.1.5. Negative effects of sharing.

Curci and Rime (2012) investigated whether the extent of sharing has an effect on emotional recovery. Participants were grouped with regards to how much they socially shared a negative life event of their choosing. They were subsequently grouped with regards to how much they shared the experience. Results showed that self-reported recovery was poor in participants who constantly shared the negative life event (no extinction of sharing with time), as compared to other participants who either shared the event as it initially happened and not after that, or shared it again (with family) during the time of the study, but to a small extent. This study not only contradicted the notion that disclosure is beneficial for emotional recovery, but suggested that there may be negative results of over sharing.

Some noted that verbal disclosure is not as natural a component of emotion expression as are facial expressions or behavioural changes induced by an affective state; as such, verbal expression may be viewed as counterproductive (Wilson & Schooler, 1991). Additionally, disclosers are often faced with difficulties in the form of negative listener's responses (e.g., Nolen-Hoeksema & Davis, 1999), which is the main downside of sharing of negative emotions. These reactions vary from a mere non-understanding to behaviours such as rejection and non-acceptance (Silver, Wortman, & Crofton, 1990). It seems people do not feel comfortable with hearing about negative experiences of others and possibly also with witnessing those people reliving the distress during disclosure. They might therefore discourage the disclosers from sharing (Coyne, Wortman, & Lehman, 1988; Gottlieb & Wagner, 1991).

3.1.6. Other directions of research: Quality of social sharing and social sharing of positive experiences.

Although the present study was designed to investigate the effects of social sharing with induced emotion expression on psychological and physiological outcomes in a clinical sample, other directions of research are acknowledged.

For example, as research failed to show emotional recovery resulting from social sharing, investigators focused on the quality of sharing from sharer – listener interactive perspective (Panagopoulou, Maes, Rimé, & Montgomery, 2006). Consequently, Rime (2009) proposed that two types of sharing should be considered in order to discuss impact of sharing on recovery from an emotional event: socio-affective (or emotional) mode of sharing (support, empathy and consolation) and a cognitive mode (listener – induced reappraisal, challenging of schemas, motive and goal reorganisation)(B. Rimé, 2009). Rime's predictions with relation to the two sharing types' impact on recovery were that emotional sharing is important for relationship maintenance, reassurance and temporary emotional support, thus helping a person cope with emotions (anxiety, helplessness) and bringing a sense of relief all of which can be mistaken for emotional recovery. Cognitive sharing, on the other hand, has a potential for complete recovery from an event. Subsequent studies showed that whereas cognitive sharing proved the most beneficial for recovery from stress, socio-affective mode showed perceived benefits such as lower loneliness scores and social benefits (e.g., Zech & Rimé, 2005).

A different direction of research suggests that sharing of positive experiences is as common as sharing of negative ones (A. Curci, et al., 2007) and has a potential of prolonging one's happiness (Langston, 1994), as opposed to the need for catharsis induced by negative experiences and emotions (e.g., Reis et al., 2010). Indeed, retelling positive events and reliving emotions associated to them are linked to increases in positive affect (Langston,

1994). Research based on a diary method indicated that people were happier and more satisfied with life on the same days when they shared positive experiences with other people, compared to days when they did not (Gable, Reis, Impett, & Asher, 2004). It is plausible, however, that due to one's happiness an individual has more people around to discuss happy events with (Gabriel, Carvallo, Jaremka, & Tippin, 2008).

3.1.7. Summary.

Social sharing is a process, following a negative life event, of discussing memories, thoughts and emotions associated with a life event with a listener (B. Rimé, 2009). Between 80% (Bernard Rimé, et al., 2010) and 96% (Rime et al., 1992) of people engage in sharing, as they believe it is beneficial to them.

Initial social sharing research investigated retrospective autobiographical self reports of sharing or applied daily diary methods (Rime, 2009) and observed no benefits of social sharing in terms of emotional recovery from the event. As these methods were prone to selective and reconstructed memory biases, laboratory experiments were designed (e.g., O Luminet, Bouts, Delie, Manstead, & Rimé, 2000; Zech & Rimé, 2005); however, no effects of sharing on emotional recovery were observed here either. The criticisms of the laboratory studies include not analysing participants' narratives from the linguistic perspective (i.e., investigating the percentages of emotion words used) to determine whether they, in fact, engaged in emotion expression (O Luminet, Bouts, et al., 2000), or applying different instructions, one session intervention, and testing different outcomes, at different follow-up periods than was the case in the expressive writing studies (O Luminet, Bouts, et al., 2000; Zech & Rimé, 2005). Rime et al.'s review (1998) concluded that there was no relationship between the extent of sharing and emotional recovery from the experience, which was later

confirmed again, in the laboratory studies inducing social sharing (using special instructions; Zech & Rimé, 2005).

In terms of investigating the benefits of social sharing, Rime et al. (1998) recommended that future studies take into consideration individual differences, such as emotion inhibition. Research would also benefit from studies applying similar instructions, and focusing on outcomes similar to those studied in expressive writing research (e.g., psychological distress, physiology, general health), to better understand the gap between the effects of expressive writing and those of social sharing.

3.2. Verbal Emotion Expression

Verbal emotion expression is essentially expression through words, therefore in a sense, expressive writing is a type of verbal expression. On the other hand, vocal emotion expression is expression through voice, which does not imply the use of words. To avoid confusion, and for a lack of a better word, here the term “verbal” emotion expression will relate to verbal vocal expression (i.e., talking, rather than writing about own emotions). Verbal emotion expression reduces distress about distress, promotes insight, and influences interpersonal relationships (Kennedy-Moore & Watson, 2001). It has been found to have immunological benefits (B. A. Esterling, Antoni, Fletcher, Margulies, & Schneiderman, 1994) and perceived benefits (D.A. Donnelly & E.J. Murray, 1991; E. Murray & Segal, 1994), similarly to expressive writing.

Although verbal emotion expression have not been studied extensively, its importance for positive outcome has been acknowledged in psychotherapy (Bady, 1985; L. S. Greenberg & Webster, 1982; Rice & Wagstaff, 1967). Within the paradigm of clinical psychology, additional information about the effects of brief verbal expression interventions comes from studies on psychological debriefing (see below).

3.2.1. Evidence from psychological debriefing studies.

Psychological debriefing was developed as a brief crisis intervention, administered within days of a traumatic event (Raphael & Wilson, 2000) for trauma-exposed staff members (e.g., police, fire fighters), as a group intervention. Talking about feelings and reactions to the traumatic incident was proposed to decrease the duration, severity or impairment from traumatic stress (Everly & Mitchell, 1999) for secondary trauma victims.

Of note, CISD typically involves excessive factual sharing and sharing of thoughts and emotions related to a traumatic event. Studies that tested the efficacy of CISD on psychological wellbeing in secondary trauma victims found significant positive changes in anger, depression and stress levels among police officers (e.g., Bohl, 1991), decreases in depression, anxiety and PTSD symptoms among medical emergency staff (Jenkins, 1996), or decreased anxiety among fire fighters (e.g., Bohl, 1995). Later, psychological debriefing was applied to primary trauma victims and usually as an individual, rather than group intervention, both of which defied the original guidelines (e.g., Mitchell & Everly Jr, 1995). In subsequent research (e.g., Forneris et al., 2013; Gartlehner et al., 2013) psychological debriefing did not alleviate the symptoms of distress and even was detrimental for psychological wellbeing (Van Emmerik, Kamphuis, Hulsbosch, & Emmelkamp, 2002). All studies reviewed in Van Emmerik et al.'s meta-analysis (2002) comprised of one debriefing session, conducted in the first month following trauma. These once-off interventions typically had a duration between 30 minutes (in case of non-CISD debriefing) to 1-3 hours (in case of CISD). Outcomes measured were PTSD symptoms, depression, anxiety, and other psychological symptoms. The findings can be explained in terms of disruption of natural event processing or disallowing participants the time for habituation to trauma. Consistently, in earlier studies on bereaved people similar patterns were observed, namely that participation in mourning rituals did not have any positive effects on mood and grief, but was

detrimental for emotional health (Weiss & Richards, 1997). Furthermore, Rime et al. (2010) discovered that more intensive social sharing in the first days following the events in Madrid 2004 was associated with higher levels of rumination and emotional arousal at 3- and 8-week follow ups.

Psychological debriefing therefore is not an appropriate method for PTSD patients (being originally designed for secondary victims of trauma), which is consistent with findings of research on expressive writing in PTSD sufferers (outlined in Chapter 2, section 2.3.1.10.). This however does not imply that social sharing interventions, with induced emotion expression would have similar adverse effects, as the method applied in the proposed study involves three, rather than one, sharing sessions and it will be applied to a general cardiac sample, not to PTSD patients. Furthermore, different evidence comes from studies using the method of verbal expression to a symbolic or passive listener (see below).

3.2.2. Verbal expression without an active listener.

One of the types of verbal emotion expression that has been tested and compared to the writing task is in-private, out-loud verbal disclosure without a listener. Pennebaker and colleagues (Pennebaker, et al., 1987) investigated the impact such verbal expression had on physiological measures, conducting two studies. In the first study, they used a repeated-measures design with a sample of 24 students. While alone in a room, participants were asked to talk for 6 minutes about traumatic life events (facts only), and, after a 3-minute rest period, to talk about trivial topics for another 6 minutes. Their skin conductance, blood pressure and heart rate were recorded during the intervention. Additionally, the students were classified as low-disclosers or high-disclosers (using objective analysis of disclosure by independent judges, combined with participants' subjective views). It was observed that high-disclosers

had low levels of skin conductance during the trauma topic and higher levels during the trivial one, whereas the opposite was true for low-disclosers.

In the second study they compared two groups of students ($n = 24$ in each group). One group was asked to talk to a tape recorder, and the other to an undisclosed person hidden behind a curtain (the “confessor”). For nearly 4 minutes participants talked about a traumatic event they experienced, after which they were asked to think for 4 minutes about a traumatic event, then again talk about a trivial topic, and, finally, to think about a trivial topic (counter-balancing method was used when administering these instructions). Of note, the “confessor” did not talk to participants during the whole session, except for reading the instructions to them at the beginning of it. Students’ skin resistance and heart rate were recorded during. Their levels of disclosure were also rated, the same way as in the first study. Results showed decreases in skin conductance in high disclosers during focusing on traumatic events, but not during trivial events. This trend was reversed in low disclosers. This suggested that discussing a traumatic event was associated with lower sympathetic arousal.

In a different study, Murray and Segal (1994) compared the writing task with disclosure to a tape recorder. In both conditions, participants either discussed their thoughts and emotions related to an emotional event, or talked about trivial topics. Post-intervention benefits of emotion expression (i.e., decreased pain and upset of the event and positive changes in thinking about it) were similar for both types of expression. No effects were found on psychological and physical health at 3 months follow-up; however, this was most likely due to a floor effect.

Another study compared talking to a tape recorder with expressive writing (B. A. Esterling, et al., 1994). In both expression conditions participants disclosed stressful events and emotions, whereas control participants wrote or spoke about trivial topics. Both expression groups showed significant improvement in immune function over the four week period.

Interestingly, post-intervention immunological benefits in the verbal expression group were significantly greater than those in the written expression group (which were still greater than those in the control group).

Although lacking a visible listener, those studies suggest potential health benefits of verbal emotion expression.

3.2.3. Mediators of effects of verbal expression.

In their studies, Murray et al. (1989) and Donnelly and Murray (1991)(Chapter 2, section 2.3.1.7) concluded that greater benefits of psychotherapy, than expressive writing, may derive from an interpersonal interaction that is absent in studies using the writing task, as well as from the fact that emotions are expressed verbally. According to Kennedy-Moore and Watson (Kennedy-Moore & Watson, 2001), there are three ways in which verbal emotion expression (with a listener) can influence psychological and/or physical health and wellbeing. First of all, it reduces distress about distress; second, it promotes insight; and finally, it influences interpersonal relationships, although it may have a negative effect on mood immediately after disclosure (for a review, see Kennedy-Moore & Watson, 1999).

Although the first two pathways can also explain the effects of expressive writing, the social (interpersonal) component of emotion expression is distinctive for verbal emotion expression with a (not symbolic) listener. If negative emotions arise from problems within an interpersonal relationship, sharing these emotions with that person would be adaptive, promote mutual understanding and help solve problems (Kennedy-Moore & Watson, 2001).

3.2.4. Summary.

Verbal emotion expression has been found to have positive and negative effects on psychological or physical outcomes, depending on procedures applied by researchers.

Some found evidence that verbal expression reduces distress, facilitates social support and helps cognitively process the emotional event (Kennedy-Moore & Watson, 2001), whereas others found immunological benefits (B. A. Esterling, et al., 1994) and perceived benefits (D.A. Donnelly & E.J. Murray, 1991; E. Murray & Segal, 1994).

Verbal expression may have received negative press from multiple studies using a psychological debriefing method, initially designed for secondary trauma victims, on primary trauma victims. Although initial studies found an array of positive effects of this method among police officers and fire fighters (decreases in anger, depression and stress; e.g. Bohl, 1991, 1995), or medical emergency staff (decreases in depression, anxiety and PTSD symptoms; Jenkins, 1996), when debriefing was applied to primary trauma victims, null and adverse effects on psychological outcomes were observed (Van Emmerik, et al., 2002).

Other types of verbal expression were also tested, for which outcomes were much more positive. For example verbal emotion expression to either a tape recorder or an undisclosed listener had positive effects on physiology (decreasing arousal)(Pennebaker, et al., 1987), immune system (B. A. Esterling, et al., 1994), or on recovery from the event (decreased upset of the event and positive changes in thinking about it)(E. Murray & Segal, 1994).

Other authors suggest that the existence of a listener in verbal expression may add a component of social interaction/perceived support to methods such as expressive writing (e.g., Donnelly and Murray, 1991).

3.3. Chapter Summary

This chapter adds two new dimensions to research on expressive writing outlined in the previous chapter – a social interaction and (out-loud) verbalisation of experience and emotion (Donnelly and Murray, 1991).

Social sharing research highlights that people in general discuss their emotional experiences with other people and the more intensive the emotion they feel the more likely it will be shared (Rime, 2009). Numerous studies, applying different methods consistently failed to find evidence of positive benefits of social sharing on emotional recovery from the event. Even laboratory studies observing natural social sharing at the time it occurred (O Luminet, Bouts, et al., 2000), or inducing social sharing (Zech & Rime, 2005) only found evidence for perceived benefits of sharing.

Of note, whether people expressed emotions during sharing was either not investigated (O Luminet, Bouts, et al., 2000), or the instructions for participants did not match those from the expressive writing research (Zech & Rime, 2005). Furthermore, one session interventions were insufficient in expressive writing studies, and yet were widely applied in the social sharing literature, and a limited range of outcomes were measured in these studies.

Research on verbal emotion expression found both positive (B. A. Esterling, et al., 1994; Kennedy-Moore & Watson, 2001; E. Murray & Segal, 1994; Pennebaker, et al., 1987) and negative effects on outcomes (Van Emmerik, et al., 2002), depending on procedures applied by researchers, although psychological debriefing when applied in accordance with its intended use also proved to be beneficial (e.g., Bohl, 1991, 1995; Jenkins, 1996). Rime et al. (1998) recommended that future studies take into consideration individual differences, such as emotion inhibition. Furthermore, future studies should apply similar instructions, and focusing on similar outcomes, that were applied in expressive writing research, to make possible the comparison between the effects of expressive writing and social sharing. At

present, it is still unknown whether social sharing indeed induces emotion expression, and whether it would have an effect on other outcomes (than emotional recovery), such as psychological distress or physiological measures, as these has not been tested so far. There is therefore a need for a social sharing study, with verbal emotion expression, that would apply the instructions from the writing task, to *multiple* sharing sessions, and measuring outcomes similar to those measured in expressive writing research. The present study addresses these issues.

Coronary Heart Disease

Cardiovascular Disease (CVD) is currently the world's major cause of death, killing 17 million people a year (World Health Organisation; WHO, 2011). In Ireland 10,000 people die each year from CVD (33% of all deaths), half of whom die due to a heart attack (Irish Heart Foundation, 2012). CVD is a general term to describe a variety of cardiovascular disorders such as Coronary Heart Disease (CHD), heart failure, stroke, and peripheral artery disease. These cardiac conditions have similar aetiologies, but differ in their manifestations, treatment, and prognosis; therefore they are often treated separately in research. The present study is focused on CHD, and this chapter defines CHD before considering its relationship to psychosocial factors. Numerous demographic, clinical and behavioural characteristics have been associated with the higher risk of development of CHD and recovery from cardiac events.

4.1. Definition and Aetiology

CHD is a narrowing of the small blood vessels that supply blood and oxygen to the heart, leading to insufficient oxygenated blood flow (ischemia), and heart tissue hypoxia (reduced oxygen content). This causes chest pain (angina), shortness of breath, and eventually, a heart attack (myocardial infarction; MI) (Goldman & Braunwald, 1998). The next sections describe clinical manifestations of CHD, its aetiology and risk factors.

4.1.1. Stable angina.

The most common manifestation of CHD is chronic and predictable chest pain called stable angina (*angina pectoris*). Other symptoms include a feeling of pressure and back pain. The

chest pain goes away after a rest or can be treated with medication. Stable angina usually occurs after physical exertion or in emotional situations, which leads to sympathetic activation of the cardiovascular system (explained in detail in the *Cardiovascular Physiology* section of this chapter) causing elevations in blood pressure. An increase in oxygen requirement that occurs at that time cannot be fulfilled due to the atherosclerotic luminal narrowing (i.e., the thickening of the artery wall) in the coronary vessels.

4.1.2. Unstable angina.

Another manifestation of CHD is unstable angina (*Crescendo Angina*). It is an acute, unpredictable pain, not easily relieved with medicine. An episode can occur at rest and lasts longer than stable angina; the pain becomes more severe with each attack. The cause of unstable angina is similar to that of a heart attack (*myocardial infarction*; MI), namely a blood clot (thrombus or its smaller version – embolus) plugging a coronary vessel. This condition requires medical attention as it is a warning sign that a heart attack might occur.

4.1.3. Myocardial infarction.

An MI is permanent damage of heart muscle caused by a sudden blockage of a coronary artery by a thrombus/embolus, and the damage to the heart may lead to it stop contracting (*Cardiac Arrest*). In some cases an MI may be a cause of sudden cardiac death (SCD).

4.1.4. Aetiology: biological and behavioural risk factors.

The next section briefly outlines the main biological, behavioural, and psychological risk factors for CHD. Later parts of the chapter focus on cardiovascular physiology and on psychological factors and their physiology. The major CHD risk factors, defined by the Framingham Heart Study are hypertension, cigarette smoking, high serum cholesterol, low

levels of high-density lipoprotein (HDL) cholesterol, and diabetes mellitus. Other risk factors, mentioned in several reviews, are family history of CHD (Friedlander et al., 1998), physical unfitness and inactivity (P. T. Williams, 2001), and excessive alcohol intake (Roerecke & Rehm, 2010; a meta-analysis). Research also stresses the importance of obesity in cardiovascular disease aetiology and prognosis. Bogers et al.'s (2007) meta-analysis reviewed a combined sample of 302,296 participants and concluded that overweight and obesity accounted for a 16% increased risk of CHD after adjustments for age, gender, physical activity, smoking, blood pressure and cholesterol levels.

There are common physiological processes underlying all the above risk factors. An increase in blood lipid and glucose levels, often generated by unhealthy diet, and chronic cardiovascular exertion (i.e., elevated blood pressure and heart rate) wear out the heart and blood vessels. Genetic and gender predispositions to CHD are also associated to individual proneness to increased blood lipids/lipoproteins or glucose levels (Lecerf & de Lorgeril, 2011). Finally, individuals who are physically inactive may not be able to burn unnecessary lipids and sugar, or “exercise” the heart by systematically elevating the cardiac indexes in a healthy way.

4.1.5. Cardiovascular physiology.

From a physiological perspective, assessing the risk of CHD in healthy people, or the risk of adverse cardiac events in CHD patients, involves monitoring a person's blood pressure (BP), heart rate (HR) and heart rate variability (HRV); blood lipids/lipoproteins levels (fatty acids and cholesterol), and blood glucose. This section commences with an outline of the role of the Autonomic Nervous System (ANS) in cardiovascular regulation.

The ANS, one of two branches of the Peripheral Nervous System, coordinates many bodily processes, such as body temperature, digestion and cardiovascular system. The ANS's

antagonistic two branches, Sympathetic Nervous System (SNS) and Parasympathetic Nervous System (PNS), complement and balance each other. Activation and inhibitory processes keep the body in a homeostatic state. The SNS is responsible for a stress (“fight or flight”) response and cardiac activation. It increases respiration rate, sweating, blood pressure and heart rate, and coordinates hormonal response responsible for releasing glucose and lipids into the blood stream. It is “switched on” during emotional and physical arousal to ensure sufficient energy levels. The role of SNS is to constantly antagonise the PNS to prevent exhaustion. The PNS “relaxes” the muscles, decreases cardiac indicators, activating those systems that were inactive during the arousal phase (e.g., digestion). The balance between sympathetic and parasympathetic branch is vital for normal functioning, but can be compromised by either prolonged sympathetic activation (e.g., chronic stress, physical exhaustion) or insufficient parasympathetic function (Stauss, 2003).

4.1.5.1. Hypothalamo-pituitary-adrenal (HPA) axis.

During a stress reaction, a corticotrophin-releasing hormone (CRH) is secreted by hypothalamus, stimulating the pituitary gland to release adrenocorticotropin hormone (ACTH). ACTH in turn stimulates the adrenal glands, which are responsible for cortisol secretion. High levels of cortisol down-regulate the hippocampus, hypothalamus, and pituitary inhibiting further release of CRH and ACTH (G. E. Miller, Chen, & Zhou, 2007).

The role of cortisol in the body is mixed; further to its regulatory effect of the metabolic system (glucose storage and exploitation), it is also involved in the regulation of body’s inflammatory response (Sapolsky, Romero, & Munck, 2000), and it takes part in cognitive (learning and memory) and emotional processes.

Both elevated and abnormally low cortisol levels have been linked to stress (Heim, Ehlert, & Hellhammer, 2000; Sternberg, Chrousos, Wilder, & Gold, 1992).

4.1.5.2. Blood pressure.

One of the indexes that rise during the sympathetic activation phase is blood pressure (BP). Pressure measured during heart contractions (systole) is Systolic Blood Pressure, whereas one measured during relaxation of the heart is Diastolic Blood Pressure. Whether it is during exercise or other physical activity, or caused by an emotional episode, occasional elevations in BP are normal. When BP rises above the normal threshold ($>140/90$ mmHg) and this is not balanced out by the parasympathetic response, a person is diagnosed with hypertension. Prolonged hypertension exerts the heart and blood vessels, and makes a person 2 to 3-fold more prone to CHD (Kannel, 1996). One of the cardiac indexes that measures the functioning of the PNS is heart rate variability, which is outlined in the next section.

4.1.5.3. Heart rate and heart rate variability.

Heart rate variability (HRV) is the variation of the time period between consecutive heart beats and it reflects the modulation of the heart rate by both the SNS and PNS. This interplay between the two branches of the nervous system mirrors the adaptation to physiological demands brought by either physical or psychological conditions.

Although increased HR has been linked to CHD and negative prognosis (Palatini, Casiglia, Julius, & Pessina, 1999), recent studies focus on heart rate variability (HRV) as a measure of a sympathetic-parasympathetic balance. A compromise of this balance is a serious CHD risk factor (Stauss, 2003); HRV has been linked to CVD and poor cardiac prognosis in CHD patients (J.F. Thayer, Yamamoto, & Brosschot, 2010, a review).

The two domains in which HRV can be measured are time (e.g., standard deviation of the interbeat intervals (IBI), standard deviation of R to R intervals (SDNN), the root mean square successive differences (RMSSD), and HRV triangular index) and frequency [low frequencies

(LF; 0.04–0.15 Hz), and high frequencies (HF; 0.15–0.40 Hz)] (Bilchick & Berger, 2006).

HRV is typically measured over a 24-hour period (using the Holter method), however short recordings have also been studied (Prinsloo et al., 2011).

HRV is closely related to psychological stress (Julian F Thayer & Lane, 2007), as well as other psychological constructs. For example effective emotion regulation is associated with increases in HRV (e.g., Appelhans & Luecken, 2006; Julian F Thayer & Brosschot, 2005) and better performance during cognitive tasks (Hansen, Johnsen, Sollers III, Stenvik, & Thayer, 2004; Hansen, Johnsen, & Thayer, 2003). It has been observed that emotion expression via expressive writing has beneficial physiological effects that indicate decreased sympathetic arousal as evidenced in changes in HRV (McGuire, et al., 2005; described in section 2.3.1.1.).

4.2. Treatment of CHD, Prognosis, and Secondary Prevention

There are several ways of treating CHD (Yusuf, Cairns, Camm, Fallen, & Gersh, 2008). The main goals in both treatment and secondary prevention are: hypertension control, blood lipid and blood glucose levels management, and diabetes control. These are reached with the use of medications such as anticoagulants/antiplatelet agents (for blood thinning), statins (cholesterol synthesis inhibitors, reducing serum total and LDL cholesterol), beta-blockers (for decreasing heart rate, lowering blood pressure and reducing oxygen use by the heart); nitrates (for dilating the coronary arteries, stopping chest pain and improving blood supply to the heart), calcium-channel blockers (relaxing coronary arteries and all arteries in the body, reducing the workload for the heart and lowering blood pressure), and ACE inhibitors, diuretics or other medications to lower blood pressure. Beside medications, many patients undergo medical interventions such as Percutaneous Coronary Interventions (PCI's). The main PCI's are coronary angioplasty (Balloon Percutaneous Transluminal Coronary

Angioplasty; PTCA) and coronary stenting. The most invasive methods of treating CHD are surgical procedures such as coronary artery bypass surgery (CABG) and other types of heart surgery.

4.2.1. CHD prognosis, secondary prevention, and cardiac rehabilitation.

If CHD is detected relatively early, the damage to the heart is easier to treat. Prognosis is the worst for patients after a heart attack – their risk of CHD-related death is 7.6- fold (2.5- to 3.2-fold for angina patients) compared to healthy people, whereas all-cause mortality risk is 3.7-fold (1.7- to 2.2-fold for angina patients) compared to healthy individuals (Sigurdsson, Sigfússon, Agnarsson, Sigvaldason, & Thorgeirsson, 1995).

On top of the medications mentioned above, which prevent subsequent adverse cardiac events, the main methods of secondary prevention in CHD are:

- a) Smoking cessation
- b) Weight control
- c) Promoting physical exercise

All the above can be addressed through Cardiac Rehabilitation (CR) programmes, which are recommended for patients after and MI, CABG or PCI and for some patients with angina, stable chronic heart failure or peripheral arterial disease (Leon et al., 2005). These programmes are based on education and support from a multidisciplinary team of professionals (e.g., Physiotherapist, Clinical or Health Psychologist, Dietician, Cardiac Nurse, Fitness Instructors, Health Promotion Officer, Occupational Therapist). The main areas of CR are physical exercise, weight management, smoking cessation, lipid and BP management, reducing excessive alcohol intake, stress management, and psychological counselling.

Besides the obvious physiological benefits of exercise programmes, the main goals of CR are to give the patient a sense of control over the illness, explain and facilitate lifestyle changes, and help deal with psychological and social consequences of CHD (e.g. anxiety, depression, relationship changes, and occupational issues). They are reached through the following methods:

- Group psychoeducation (managing illness cognitions and cardiac misconceptions),
- Relaxation and stress management, counselling, cognitive therapy, psycho-sexual counselling , anger management (treating anxiety, depression, managing negative affect),
- Cognitive behavioural therapy (CBT), behavioural smoking-cessation programs (changing lifestyle behaviours).

Overall, cardiac rehabilitation has been found effective in improving health in cardiac patients (Clark, Hartling, Vandermeer, & McAlister, 2005; Oldridge, Guyatt, Fischer, & Rimm, 1988), although is still underused, with low referral and participation rates (Mampuya, 2012). Exercise-based CR has been linked to reduced all-cause and cardiac mortality, decreases in cholesterol level and systolic blood pressure; it also positively affected HRQoL and smoking cessation (Lawler, Filion, & Eisenberg, 2011; R. S. Taylor et al., 2004). Psychosocial components of CR also have been independently tested for their effectiveness. For example, psychoeducational programmes were found to reduce cardiac mortality by 34%, and subsequent MIs by 29%, and were beneficial in relation to lowering blood pressure, decreasing cholesterol level, and positively affected lifestyle changes (smoking cessation, weight loss, diet change, and increasing physical activity)(Dusseldorp, van Elderen, Maes, Meulman, & Kraaij, 1999), although a recent meta-analysis suggested that mental health treatments are better suited for decreasing psychological distress and should be combined

with exercise programmes for greater benefits for post-MI patients (Rutledge, Redwine, Linke, & Mills, 2013). No significant differences in effectiveness have been found between centre-based and home-based CR programmes, suggesting that the type of programme should be individually matched to the patients' needs and preferences (Dalal, Zawada, Jolly, Moxham, & Taylor, 2010).

In Ireland, a typical CR programme has three phases (Delaney et al., 2006):

- Phase I: acute treatment of patients admitted to the intensive care unit and the in-hospital stage in recovery (testing, medications and education on diagnosis and risk factors, as well as discharge advice),
- Phase II: a period of convalescence for the patient
- Phase III: a 4-12 week outpatient programme (exercise, psychological counselling, education classes, nutrition education). It is recommended to start within three months post-discharge and lasts 6-8 weeks.

At the hospital where the main study took place, patients receive discharge advice and are informed of the Phase III CR that they would be invited to within several months, whereas during Phase II, psychological help is offered those patients who are clinically depressed as diagnosed by a psychologist during hospital stay. Of note, these patients were not included in the main study.

4.3. Negative Affect, Personality Traits, and Emotional and Social Inhibition as Risk Factors in CHD

The contribution of psychological factors in the development of CHD and in an adverse prognosis after a cardiac event has been documented (L. Kubzansky, Kawachi, Weiss, & Sparrow, 1998; L. D. Kubzansky & I. Kawachi, 2000; Rozanski, Blumenthal, &

Kaplan, 1999). The main factors are negative affect (especially depression, anxiety and anger), personality traits, and emotional and social inhibition (S. Pedersen & Denollet, 2003, a review).

4.3.1. Type A behaviour pattern, anger and hostility.

Individual differences have long been considered important in the development and prognosis of physiological illnesses. Dunbar (1947) proposed that specific personalities may make people prone to different disorders. In 1974 Friedman and Rosenman introduced Type A (“coronary prone”) behaviour pattern (TABP) and therefore illustrated the role of personality in CHD aetiology. People with this pattern of behaviour are competitive, achievement oriented, aggressive, hostile, impatient, overly responsible, often under pressure of time. Physical attributes of TABP include facial tenseness, teeth clenching, and explosive speech. The association between TABP and CHD was confirmed in meta-analyses (Booth-Kewley & Friedman, 1987; Matthews, 1988). The most recent meta-analysis (Myrtek, 2001) found that the overall effect size of the impact of TABP on CHD was rather small ($r = .009$), and researchers were encouraged to search for other psychological factors in cardiovascular disease. Subsequent research examined anger and hostility, two traits connected to TABP and often linked to CHD development and progression. Friedman, Hall, and Harris (1985) found that Type A men who were low in expressiveness (they were suppressors) were more unhealthy and aggressive, whereas, highly expressive Type A's were comparatively healthy and popular. Since then focus also has been on the role of negative affect and its suppression in CHD aetiology and prognosis.

4.3.1.1. Anger and hostility.

Anger may be regarded as an emotion, therefore comprising cognitive, physiological and behavioural components (Kassinove & Sukhodolsky, 1995). There is however a distinction between emotion anger and trait anger, the latter being a predisposition to frequent and intensive feeling of emotion anger (Williams et al., 2000), a distinction similar to the one between negative affect and negative affectivity. Trait anger is one of the three compounds of hostility (Eckhardt & Deffenbacher, 1995), although both of these terms are sometimes used interchangeably (Spielberger, Krasner, & Solomon, 1988), whereas emotion anger is a state triggered by hostility (e.g. Schum, et al., 2003). Frequent episodes of anger have been associated with CHD (Chida & Steptoe, 2009). Trait anger is positively associated with LDL/HDL ratio and triglyceride levels (Johnson, Collier, Nazzaro, & Gilbert, 1992). It also has been linked to both elevated systolic and elevated diastolic blood pressure (Markovitz, Matthews, Wing, Kuller, & Meilahn, 1991; Schum, et al., 2003), increased cardiovascular reactivity (Durel et al., 1989), the risk of CHD (Johnston, 2002) and the risk of MI (Gallo & Matthews, 2003; Rozanski, et al., 1999; T. W. Smith & Ruiz, 2002).

In Smith et al.'s review (T. W. Smith, Glazer, Ruiz, & Gallo, 2004) hostility is portrayed as a belief in others' negative motives, devaluation of their worth and an attitude towards them filled with aggression. In general, hostility is a construct comprising three components: cognitive (negative beliefs about other people including mistrust and cynicism), affective (a predisposition to experience anger) and behavioural (aggressiveness – the tendency to harm other people) (Barefoot, 1992; Lipkus & Barefoot, 1994; T. Q. Miller, Smith, Turner, Guijarro, & Hallet, 1996). Hostility has been linked both to the onset of CHD and to adverse prognosis (Whooley & Wong, 2011), as well as general mortality (Howard S Friedman, 1992; T. Q. Miller, et al., 1996; Rozanski, et al., 1999), although null findings have also been reported (Suls, Wan, & Costa, 1995, a meta-analysis).

A meta analysis, examining the link between anger/hostility and CHD (Chida & Steptoe, 2009), showed a significant overall hazard ratios of 1.19 in a healthy population and 1.23 in clinical samples, confirming the link between anger/hostility and CHD development and prognosis. An association was also found between anger/hostility and CHD mortality in clinical samples (1.18).

4.3.1.2. Anger expression style.

The discussion on anger expression types started over half a century ago when a distinction was presented between anger-out (directing anger at other people) and anger-in (overt expression of anger directed at oneself)(Funkenstein, King, & Drolette, 1954). Of note, later accounts used the term “anger-in” interchangeably with anger suppression (e.g. Haynes, Levine, Scotch, Feinleib, & Kannel, 1978; Spielberger, et al., 1985). Both ends of anger expression, whether it is expression or suppression of anger, have been linked to CHD (Kubzansky & Kawachi, 2000).

Anger suppression has been associated with increased blood pressure (Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985; Dimsdale et al., 1986; Everson, Goldberg, Kaplan, Julkunen, & Salonen, 1998) and atherosclerosis (Dembroski, et al., 1985). The Framingham Heart Study also reported that suppressed anger predicted CHD in men and women (Williams, et al., 2000). Anger-out was linked to increased blood pressure (e.g., Suchday & Larkin, 2001), and the risk of CHD, angina, MI, and SCD (Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996). Schum et al.’s meta analysis (Schum, et al., 2003) also found a small negative relationship between overt expression of anger and diastolic blood pressure ($r = -.072$).

4.3.2. Anxiety and depression.

The three main groups of negative emotions have been associated with cardiovascular disease are anger, anxiety and depression. Anxiety is a combination of emotions like fear, worry, uneasiness often accompanied by physical symptoms like palpitations, shortness of breath, and chest pain. Anxiety places people at risk of developing CHD and predicts a variety of adverse outcomes in patients after a MI (Barger & Sydeman, 2005; Kubzansky, Cole, Kawachi, Vokonas, & Sparrow, 2006). Epidemiological studies show that even when factors like cholesterol level, blood pressure or smoking are being controlled for, depression and anxiety still predict CHD morbidity and mortality (Kubzansky & Kawachi, 2000; Rozanski, et al., 1999; Rugulies, 2002). Roest et al.'s meta-analysis concluded that anxiety is an independent risk factor in development of CHD, as well as cardiac mortality (Roest, Martens, Denollet, & de Jonge, 2010).

Depression is a state of mixed negative emotions and behaviour like sadness, anhedonia (loss of interest/pleasure), tiredness/fatigue, difficulty concentrating, low self-esteem and many others, and is an established risk factor both for the development of CHD (Frasure-Smith & Lespérance, 2006; Rugulies, 2002, a meta-analysis) and for negative outcomes after an initial cardiac event (Frasure-Smith & Lespérance, 2006). It has also been found to predict cardiac mortality (Barth, Schumacher, & Herrmann-Lingen, 2004, a meta-analysis; Meijer et al., 2011). This effect of depression on cardiac health has been proposed to be mediated by a number of mechanisms, both behavioural ones (e.g., smoking, physical inactivity, isolation) and biological ones (e.g., through physiological effects of stress such as sympathetic activation or suppression of immune function)(Whooley & Wong, 2013). In cardiac patients, depression seems to resemble poor adjustment to life post-MI rather than clinical depression, therefore may often go unnoticed (Hare, Toukhsati, Johansson, & Jaarsma, 2013).

A number of mechanisms have been proposed to account for the relationship between anxiety, depression and CHD; current research implicates altered neuroendocrine functioning (Carney et al., 1995), decreased immunity (increase in C-reactive protein markers; Miller, Freedland, Duntley, & Carney, 2005) and behaviours such as smoking, alcohol consumption, lack of exercise and medication non-adherence (T. Smith & Gallo, 2001; Suls & Sanders, 1989).

4.3.3. Type D (“distressed”) personality.

Type D (outlined in Chapter 1, section 1.4.2.) describes people who, in general, suppress negative affect (anger, anxiety, sadness) at the same time being more prone than others to experiencing it. Currently Type D personality is one of the most researched psychological factors in CVD. It is comprised of two separate constructs, negative affectivity (similar to neuroticism) and social inhibition (inhibition of affect and behaviour in a social setting). Numerous studies have linked this personality type to the progression of various cardiovascular disorders (e.g. J. Denollet & D. Brutsaert, 1998), response to conventional treatment (e.g. J Denollet, et al., 2006; J. Denollet, et al., 2000), negative prognosis in CHD patients (Denollet & D. L. Brutsaert, 1998; Denollet, et al., 2000), cardiac mortality (e.g. Denollet, et al., 1995; J. Denollet, et al., 1996), or impaired health and depression in heart failure patients (A.A. Schiffer, et al., 2005). More recent studies suggest an association between Type D and maladaptive illness perceptions (Williams, O'Connor, et al., 2011 a), unhealthy lifestyle (Gilmour & Williams, 2012; Svansdottir, van den Broek, et al., 2012), as well as poor adherence (Williams, O'Connor, et al., 2011 b), maladaptation to stress (Howard, et al., 2011), and increased health care utilisation (Michal, et al., 2011). Separately from SI, NA has been linked to angina pectoris and other CHD-related complaints, although it is unrelated to objective cardiac health indices (Watson &

Pennebaker, 1989). This implies that some people may complain about their health because of high NA even though their health is not worse than low-NA people. Another theory suggests that NA is a secondary trait that occurs in the presence of an illness, rather than NA causing the illness (Watson & Pennebaker, 1989). However chronic NA makes people more prone to experience increased stress (H S Friedman, 2000; Suls, et al., 1998) and stress has been linked to the development and progression of CHD (e.g., Iso et al., 2002). The link between SI and CVD is similar to that of emotion inhibition and CHD, as outlined in the previous chapter. Additionally, socially inhibited people are often isolated and such isolation is associated with poor CHD prognosis (e.g., Carpeggiani, et al., 2005). Decrease in social support has also been linked to cardiac mortality (Bucher, 1994; Frasure-Smith, et al., 2000). It has been suggested that diabetes patients with Type D personality were more depressed, lonely, anhedonic and anxious, and had less social support while having experienced more stressful life events than non Type D patients (Nefs, et al., 2012). A more recent study found a relationship between Type D and emotionally triggered stress cardiomyopathy, suggesting a possible effect of Type D on bioreactivity to emotional stress (Compare, et al., 2013). The effects of Type D personality on various outcomes have been analysed in four meta-analyses (Grande, et al., 2012; O'Dell, et al., 2011; J. Reich & Schatzberg, 2010; Versteeg, et al., 2012). The first meta-analysis that analysed the impact of Type D personality on physical health was conducted by Reich and Schatzberg (2010). After analysing 6 studies, the authors concluded that Type D personality predicted poorer medical outcome (OR = 3.76), namely a 376% increase in the odds of a poor medical outcome compared to patients without Type D personality.

Another meta-analysis was conducted by the “Tilburg group” themselves (Versteeg et. al., 2012) and included ten studies. Separate analyses were conducted for physical and for mental health outcomes, to avoid heterogeneity of studies. The authors concluded that Type D was

associated both with impaired physical health (OR = 1.94, 95% CI: 1.49 – 2.52; $p < .001$) and mental health (OR = 2.55, 95% CI: 1.57 – 4.16, $p < .001$).

A meta-analysis conducted by American researchers independent from the Tilburg group supports the cardio-toxic character of type D personality (O'Dell, et al., 2011). Authors analysed 15 separate studies that tested patients with either CHD, heart failure, MI or other cardiovascular disorders, or who underwent a coronary intervention (e.g. PCI, CABG). All studies either used one Denollet's type D measures (e.g. DS-16, DS-14) or assessed NA and SI separately, but only included high-NA and high-SI patients in the Type D group. Included studies tested one of the following outcomes: major adverse cardiac event (MACE; e.g. cardiac death, MI or cardiac surgery; 2,903 patients), health related quality of life (HRQOL; 1,263 patients) or cardiovascular disease biochemical markers (305 patients). Type D was found to predict both MACE (OR = 3.16, $p < .001$) and decreases in HRQOL ($d = 0.69$, $Z = 9.47$, $p < .001$).

Finally, Grande et al. (2012) analysed twelve studies on patients with cardiovascular diseases and observed significant associations between Type D and mortality and nonfatal myocardial infarction (adjusted OR = 2.28, 95% CI: 1.43 – 3.62), adjusted HR = 2.24, 95% CI: 1.37 – 3.66.

4.4. Chapter Summary

Cardiovascular Disease (CVD) is currently the world's major cause of death (WHO, 2011), accounting for 33% of all deaths in Ireland (Irish Heart Foundation, 2012). Assessing the risk of CHD includes monitoring a person's blood pressure, heart rate and heart rate variability, with the latter gaining more popularity in the recent years (J.F. Thayer, et al., 2010, a review). From a physiological perspective, there should be a balance between sympathetic and parasympathetic branches of the nervous system; however, due to prolonged

psychological or physical stress (requiring chronic activation of the sympathetic branch) or the weakening of the parasympathetic branch, this balance is not achieved (Stauss, 2003).

Whereas indexes such as BP and HR measure the sympathetic functioning, HRV gives separate information about both branches, as well as about their interaction.

Prolonged hypertension exerts the heart and blood vessels, and makes a person 2 to 3-fold more prone to CHD. Although both increased BP (Kannel, 1996) and HR (Palatini, et al., 1999) has been linked to CHD, more recent studies stress the fact that decreased in HRV have been linked to CVD and poor cardiac prognosis in CHD patients (J.F. Thayer, et al., 2010, a review).

From the psychological perspective, HRV is inversely related to psychological stress (Thayer and Lane, 2007), and positively associated with effective emotion regulation (Appelhans & Luecken, 2006; Julian F Thayer & Brosschot, 2005) and better performance during cognitive tasks (Hansen, et al., 2004; Hansen, et al., 2003). Furthermore, expressive writing has been found to decrease sympathetic arousal as evidenced in changes in HRV (McGuire, et al., 2005).

Beside the physiological correlates of CHD, there is a number of psychological constructs that have been associated with it, for example (L. Kubzansky, et al., 1998; L. D. Kubzansky & I. Kawachi, 2000; Rozanski, et al., 1999), the main factors being negative affect (especially depression, anxiety and anger), personality traits, and emotional and social inhibition (S. Pedersen & Denollet, 2003, a review).

Combining predisposition for negative affect with social and emotion inhibition, a construct of Type D personality was introduced (J. Denollet, et al., 1996), which later became an established predictor of poor medical outcome (Reich & Schatzberg, 2010), impaired physical health and mental health (Versteeg et al., 2012), and cardiac mortality and nonfatal myocardial infarction (Grande et al., 2012).

Study Rationale and the Pilot Study

The present chapter consolidates the theories and main findings of the previous chapters and offers a conceptual framework for the main study procedure. The second part of this chapter outlines the feasibility study, its method, results and discussion.

5.1. Study Rationale

Pennebaker's inhibition theory, according to which prolonged emotion inhibition has a negative impact on the physiology (and therefore health) has been investigated in numerous studies on various participant samples (e.g., healthy, somatic disease, psychological disorder; Smith, 1998). Research testing the effects of suppression on physiology confirmed this (Mauss & Gross, 2004a). Subsequent emotion expression interventions were designed to address the problem of adverse effects inhibition had on health (e.g., J W Pennebaker & S K Beall, 1986). Later, it has been established that expressive writing benefits some, but not all, and the benefits range from better psychological or somatic health to improvements in grades (Frattaroli, 2006). Importantly, in expressive writing research, instructions specifically asked participants to discuss the deepest thoughts and feelings relating to the event that they were describing. Indeed, experimental conditions in which participants shared both facts *and* emotions about the traumatic event (compared to *just facts* or *just emotions*) were the most beneficial for psychological and physiological health (e.g., Pennebaker & Bell, 1986).

Parallel to the developing research on expressive writing, Rime and his colleagues investigated a common social phenomenon - social sharing (e.g., Rime, et al., 1991a), however subsequent research (whether based on retrospective, diary, or laboratory methods)

was unable to find positive effects of sharing on emotional recovery from a traumatic event (B. Rimé, 2009).

The discrepancy between the inhibition theory and expressive writing research on one end, and the findings of studies on social sharing on the other, has not been examined in literature. The content of social sharing, especially the extent of emotion expression during repeated sharing episodes, has not been thoroughly investigated, while this would be crucial for understanding the null findings of sharing. Although Rime conducted a study where social sharing and emotion expression were induced, it is not clear from the article whether participants in the emotion expression condition were asked to extensively share the facts *and* emotions relating to the traumatic event, or *just emotions* (Rime & Zech, 2005). In expressive writing, however, the instructions were very clear and either allowed discussing the facts, or the facts *and* emotions relating to the event (e.g., Pennebaker et al., 1988). A previously mentioned study comparing “facts and emotions” expression with “just facts” and with “just emotions” showed the greatest benefits of the former condition (e.g., Pennebaker & Bell, 1986). This suggests that social sharing must include both emotion expression *and* factual sharing, for the benefits to occur.

Another weakness of Rime and Zech’s study is that only one sharing session was conducted, whereas expressive writing was proved to work better the more sessions were applied (Frattaroli, 2006). Furthermore, similar outcomes and similar follow-up periods to those in expressive writing research should be applied in social sharing research, to better understand the discrepancy between effects.

Additional evidence for the fact that social sharing may not elicit emotion expression comes from the fact that if a majority (80 – 96%) of people engage in social sharing (e.g., Rime, 2009) this implies that (at least some) inhibited individuals (e.g., with Type D or Alexithymia)

also engage in social sharing, based on the fact that prevalence of Alexithymia ranges between 13% (Salminen, et al., 1999) and 18% (Parker, et al., 1989) and prevalence of Type D personality – between 23-27% (Hausteiner, et al., 2010) and 38.5% (L. Williams, et al., 2008); depending on the country.

Inhibited individuals, by definition, would not express their emotions to the extent other people would; therefore, there is a certain group of people who engage in social sharing, however, they do so without engaging in emotion expression.

Additionally, there is still no information on the extent of emotion expression that non-inhibited people engage in during social sharing.

There is therefore a need to investigate the effects on health of two types of social sharing – sharing with the added instruction for emotion expression and sharing without it (“natural social sharing”). The introduction of sharing with emotion expression serves an additional purpose of investigating the differences in the amount of emotion expression between those two types of sharing.

5.2. Choosing a CHD Sample

CHD is currently one of the greatest killers in the western civilisation.

People with Type D personality not only are more prone to cardiovascular disorders (which is both confirmed by Pennebaker’s inhibition theory and Denollet’s research on Type D), but prognosis for those people who already have cardiovascular disease is worse if they are Type Ds, than if they are not. Meta-analytic reports showed associations between Type D mortality and nonfatal MI (Grande et al., 2012).

Analogically, trait Alexithymia, although conceptually different from Type D personality, has been linked with worse disease prognosis mainly through psychosocial pathways, such as

proneness to depression/anxiety and unhealthy behaviours. Similarly, Alexithymia has been associated with hypertension (Grabe, et al., 2010), poor psychological health among CHD patients (Valkamo, et al., 2001), depression and anxiety (Bogdanova, et al., 2010; Hendryx, et al., 1991; Lumley, et al., 1996; Marchesi, et al., 2000), as well as psychological stress (McIntosh, et al., 2014). Some suggest there is a psychosocial link between Alexithymia and cardiovascular disease (Peters & Lumley, 2007; Valkamo, et al., 2001). Moderating effects of Type D personality and Alexithymia on the relationship between emotion expression and psychological outcomes are outlined in section 5.3.

Not much is known about emotion expression in CHD samples, although the studies that exist suggest possible benefits of expressive writing for CHD patients (e.g., Hevey et al., 2012; Willmott, Harris, & Gellaitry, 2011). There is therefore a gap in literature that would be filled by conducting additional emotion expression interventions for CHD patients.

5.2.1. Psychological outcomes in cardiac patients.

To better understand psychological functioning of cardiac patients before and after the social sharing intervention, similar outcomes should be tested to those investigated in previous research with cardiac samples (such as anxiety, depression, general distress, perceived stress and positive and negative affect), as negative affect has been associated with poor CHD prognosis (Barger & Sydeman, 2005; Kubzansky, et al., 2006), CHD morbidity and mortality (Kubzansky & Kawachi, 2000; Roest, et al., 2010; Rozanski, et al., 1999; Rugulies, 2002), as well as with negative outcomes after an initial cardiac event (Frasure-Smith & Lespérance, 2006).

5.2.2. Physiological outcomes.

In cardiac samples, three physiological indexes usually investigated by health psychology researchers are blood pressure, heart rate, and recently, heart rate variability.

Expressive writing studies which tested such physiological outcomes found better physiological functioning resulting from emotion expression (McGuire, et al., 2005; J W Pennebaker & S K Beall, 1986; J W Pennebaker, et al., 1988). Previous studies suggested that HRV decreases during affective states; however, effective emotion regulation has been associated with better physiological functioning (J F Thayer & Siegle, 2002). This may suggest that reliving (re-experiencing) negative emotions, but not expressing them (as in the natural sharing group) may lead to lower HRV.

Additionally, there is a link between inhibition and physiology, where emotion inhibition acts as a stressor and if used continuously it has adverse effects on the body's ability to cope (Brosschot & Thayer, 1998), leading to decreases in vagal tone. When the vagal tone is low, HRV is low, whereas BP and HR increase, as they cannot be balanced with the parasympathetic system.

5.3. Moderating Effects of Type D and Alexithymia

Whereas Alexithymia (O'Connor & Ashley, 2008; Paez, et al., 1999; Solano, et al., 2003) and Type D personality (D. Hevey, et al., 2012), has been found to moderate the effects of expressive writing, repressive coping have not (Baikie, 2008; Lumley, et al., 2002). As Type D personality is an established factor in CHD prognosis, and Alexithymia have been linked to CVD and to psychological distress, testing moderation effects of those two constructs on the impact the sharing intervention has on distress in a post-MI/PCI sample may add to existing literature.

5.4. Mediating Effects of Two Types of Emotion Regulation

Sloan and Marx (2004b) discuss three mediators of the effects of emotion expression. First, it was proposed that the effects on physical health may be mediated by disinhibition, which leads to decreases in BP and HR, and increases in HRV, and therefore have a positive impact on cardiac prognosis. For psychological outcomes, the benefits are likely to occur through either emotional habituation, or cognitive change (i.e., increase in reappraisal and decrease in rumination)(J W Pennebaker, 1997a; J W Pennebaker & S K Beall, 1986; J. Smyth, et al., 2001). Whereas the process of emotional habituation is the most easily tested via an analysis of narrative (linking emotion words use to psychological distress measures), the cognitive change can either be tested with analysis the use of cognitive words (Pennebaker, et al., 1997; Shaw, et al., 2006) or investigating whether changes in reappraisal and rumination impact on distress (e.g., Gortner, et al., 2006).

5.5. The Conceptual Model

Psychology is such a type of science where causal relationships are sometimes difficult to determine, as different psychological variables affect each other simultaneously. For example, although it has been established that rumination may generate more distress, the reverse is also true (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). For the purpose of this study, a simplified model is being tested here (*Fig. 6*). For practical reasons, in the tested model, reappraisal and rumination are treated solely as mediators of the intervention's effects on psychological outcomes, whereas distress as an outcome. Measuring outcome reappraisal and outcome rumination would not address the main question this study is investigating – whether a social sharing intervention with emotion expression has a positive impact on distress and physiology. Similarly, it is clear that psychological distress and physiology affect

each other; however, the present study tests the hypothesis of disinhibition – a direct effect of emotion expression on physiology through counteracting inhibition’s effect on physiology.

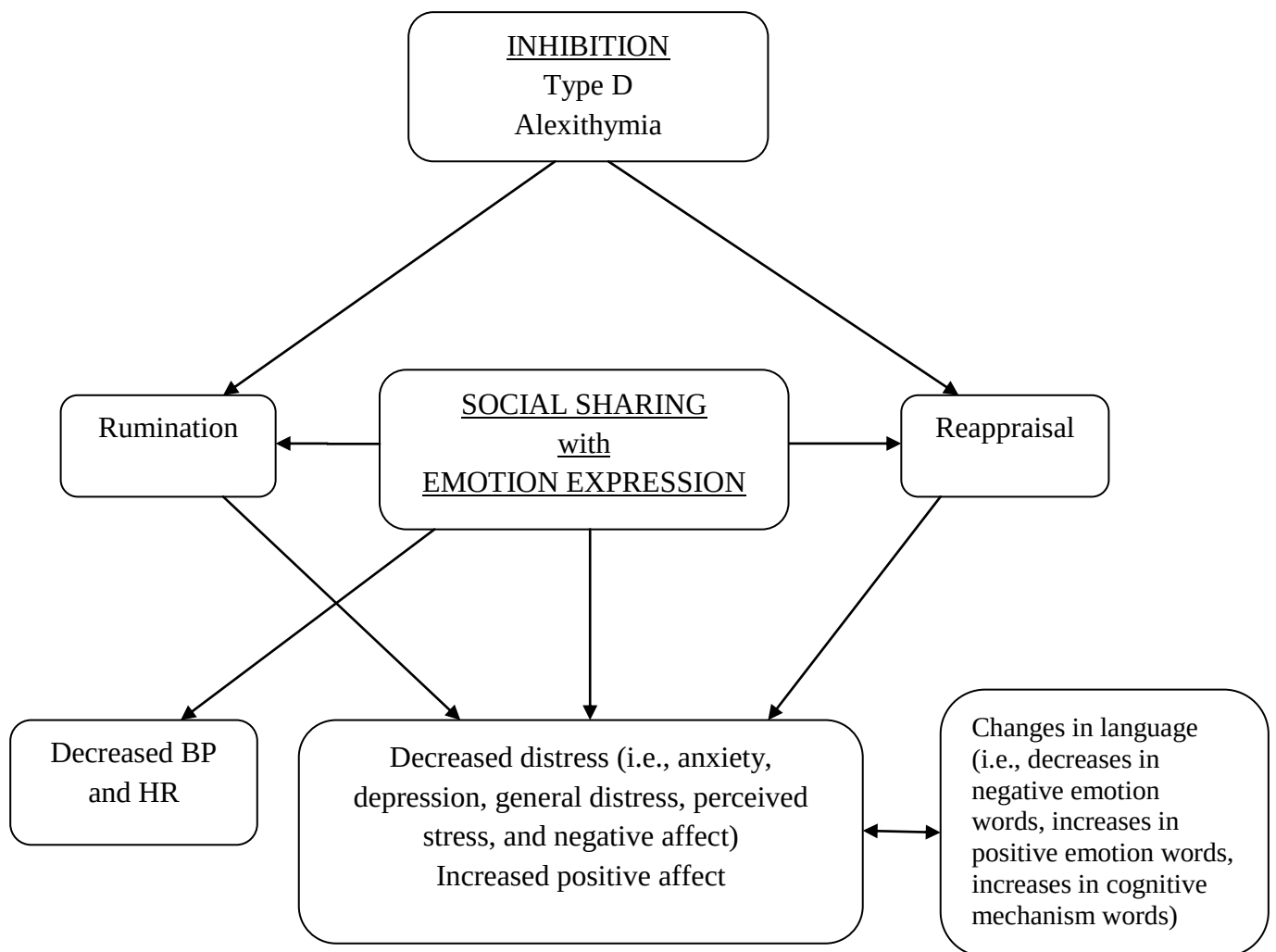


Fig. 6. The Simplified Model (Tested)

5.6. Feasibility Study

A feasibility study was conducted to test several aspects of a social sharing intervention with emotion expression and a social sharing intervention without an emotional expression aspect.

The primary aim of this feasibility study was to inform the main study design. This was not a pilot study, as it did not apply an RCT method (e.g., randomisation and the use of a control condition)(for a review, see: Arain, Campbell, Cooper, & Lancaster, 2010); therefore, no hypotheses regarding potential effects of social sharing on outcome variables were tested. The feasibility study answered questions regarding participants' ability and willingness to complete the questionnaires, the timing of the meetings, instructions' clarity, and participants' ability and willingness to engage in social sharing with or without emotion expression with the researcher (a stranger). The following questions were investigated:

1. Is the questionnaire pack an acceptable size for participants?
2. Do participants understand the instructions and follow the protocol, both during assessments and during sharing sessions?
3. Is the course of three sharing sessions an acceptable scope for participants?
4. Are participants willing to engage in social sharing with a researcher?
5. Are participants willing to express their emotions during sharing sessions?

A secondary aim of this feasibility study was to explore potential changes in outcome measures (i.e., distress and affect) and to explore associations between changes in language use and changes in affect and distress.

The questionnaire battery for the feasibility study (see section 5.8.2.) includes:

- 1) Background measures informing of the baseline character of the sample,

a) Personality:

- Traits related to sociability and cooperativeness: extraversion, agreeableness, and social inhibition,
- Traits related to emotionality: negative affectivity,
- Traits related to emotion inhibition: Alexithymia subscales (difficulty identifying feelings, difficulty describing feelings, and externally oriented thinking)
- Personality constructs related to emotion inhibition: Type D and Alexithymia,

b) Demographic data;

2) Outcome measures:

- a) Distress: anxiety, depression, general distress, perceived stress, and positive affect.

No specific hypotheses were proposed, however the following questions were tested:

1. Are changes in psychological variables associated with language use patterns or with between-session changes in language use?
2. Are changes in affect or distress variables associated with changes in emotion regulation variables?

5.7. Design

The design of the study was based upon Pennebaker's expressive writing paradigm and Rime's social sharing theory (see sections 2.3. and 3.1., respectively). Two types of disclosure were tested: social sharing of facts and emotions ("emotion expression", EE) surrounding a stressful experience, and social sharing without specific instructions to express emotions ("natural sharing", NS). Each disclosure episode was conducted on a one-to-one basis with a researcher. The researcher allowed the participant to choose a topic of disclosure.

Each session had a conversational character rather than being a participant's monologue. The researcher often asked for elaborations or clarifications regarding the topic of participant's disclosure.

5.8. Method

Before commencement of the pilot study, an ethical approval was granted by the School of Psychology, Trinity College Dublin (Appendix 1).

Fourteen participants were randomly selected from the Trinity College Participant Panel list.

This list comprises former students and/or former research participants of the college.

Inclusion criteria were fluency in the English language and Dublin residency. Of those who could be contacted, all people agreed to participate in this pilot study.

5.8.1. Procedure.

During the initial phone conversation participants were informed about the general study requirements, namely filling out the questionnaires and meeting the researcher three times within three weeks to discuss stressful negative life events they experienced or are experiencing. After the conversation, the consent form, the questionnaires and a stamped addressed envelope were posted to the participants. Upon receiving the completed questionnaires together with a signed consent form, the researcher telephoned every participant again in order to schedule the first interview meeting. The second meeting took place a week after the first one and the final meeting a week after that. Most meetings were held in the participants' homes, but one participant from the "natural sharing" group was seen in the college. During each meeting participants were asked to talk about the most upsetting events that happened to them in the past or are happening now. They were informed that they were free to choose the topic they wanted to discuss and that if there was anything they did

not wish to discuss they were not obliged to do so. Each meeting was set to last approximately 40 minutes. This was to ensure that participants had sufficient time to develop their stories and to disclose emotional material. After the third meeting participants received the same questionnaire pack, without the personality measures and the demographic questionnaire.

5.8.2. Measures.

The questionnaire pack comprised the standardised psychological scales listed below.

5.8.2.1. Mini-International Personality Item Pool.

Mini- International Personality Item Pool (Mini IPIP; Donnellan, Oswald, Baird, & Lucas, 2006). This 20-item scale is a short form of the 50-item International Personality Item Pool—Five-Factor Model measure (IPIP FFM; Goldberg, 1999). It comprises five subscales: neuroticism (N), extraversion (E), agreeableness (A), openness (O), and conscientiousness (C), with four items for each subscale. The measure uses a five point Likert scale (1-5), therefore scores range between 4 and 20 for each subscale. For the purposes of the present study only two subscales were used: Extraversion (e.g., “*Am the life of the party*”) and Agreeableness (e.g., “*Sympathize with others’ feelings*”), as they allowed for measuring traits such as sociability (extraversion) and cooperativeness (agreeableness), which is beneficial for the general understanding of the character of the sample in a study testing social sharing, as these traits illustrate how people relate to others in social situations. These scales serve a purpose of describing the sample. High scores on extraversion reflect that a person is extrovert (sociable and outgoing), whereas high scores on agreeableness scale indicate that one is cooperative and preoccupied with others’ needs.

The Neuroticism scale was excluded as the same trait is measured by the Type D scale (see below). The Openness (measuring openness to experience) and Conscientiousness (measuring self discipline and order) scales were not included in the final questionnaire pack, as neither of the traits were of relevance to the study aims.

The scale has acceptable internal consistency, values for all scales are well above .60 (Cooper, Smillie, & Corr, 2010), a very good test–retest reliability, convergent, discriminant, and criterion-related validity (comparable to other measures of the Big Five)(Donnellan, et al., 2006).

5.8.2.2. Type D Scale (DS 14; Johan Denollet, 2005).

This 14 item measure comprises two separate subscales, Negative Affectivity (NA; e.g. “*I often make a fuss about unimportant things*”; “*I often feel unhappy*”), which is similar to neuroticism, and Social Inhibition (SI, “*I make contact easily when I meet people*”, “*I often talk to strangers*”). It uses a 4 point Likert scale, and scores range from 0 to 28 for each subscale. High scores indicate high negative affectivity and high social inhibition. Type D is diagnosed when a person scores 10 or higher on both scales.

Both subscales are internally consistent (Cronbach’s alphas are .88 for NA, and .86 for SI) and stable (test-retest correlation: $r = .72$ and $.82$)(J. Denollet, 2005). Convergent validity has also been demonstrated: NA correlated with neuroticism ($r = .68$) and SI was negatively associated with extraversion ($r = -.59/- .65$) (Johan Denollet, 2005). Cut off points for SI are: 0 for “very low”, 1-3: “low”, 4-7: “below average”, 8-11: “average”, 12-15: “above average”, 16-21: “high”, and 22-28: “very high”. Cut off points for NA for males are: 0 for “very low”, 1: “low”, 2-3: “below average”, 4-6: “average”, 7-10: “above average”, 11-16: “high”, 17-28: “very high”. For females: 0 for “very low”, 1-2: “low”, 3-5: “below average”, 6-8: “average”, 9-12: “above average”, 13-18: “high”, and 19-28: “very high”.

5.8.2.3. Toronto Alexithymia Scale (TAS 20; Bagby, Parker, & Taylor, 1994; G. Taylor, R. Bagby, & J. Parker, 1992).

This 20-item self-report measure of Alexithymia has three subscales. The first two scales, Difficulty Identifying Feelings (7 items, e.g., “*I am often confused about what emotion I am feeling*”) and Difficulty Describing Feelings (5 items; e.g., “*It is difficult for me to find the right words for my feelings*”) are used to measure difficulty identifying and describing emotions. The third subscale, Externally-Oriented Thinking, is used to measure the tendency of individuals to focus their attention externally (8 items; “*I have physical sensations that even doctors do not understand*”). The scale uses a 5-point Likert scale, with total scores falling between 20 and 100. Cut-off scores set for this scale are: 51 or less: non-Alexithymia; 52-60: possible Alexithymia; 61 and more: Alexithymia. The scale’s internal consistency has been previously demonstrated (Cronbach’s alpha between .78 and .83), as well as test-retest reliability (.74) (Kooiman, Spinhoven, & Trijsburg, 2002). Previous research also shows adequate convergent validity (Parker, Taylor, & Bagby, 2003).

5.8.2.4. Emotion Regulation Questionnaire (ERQ; Gross & John, 2003).

This 10-item measure of emotion regulation comprises two subscales: Reappraisal (e.g., “*I control my emotions by changing the way I think about the situation I’m in*”) and Suppression (e.g., “*I control my emotions by not expressing them*”). The measure uses a 7-point Likert scale, and scores range between 6 and 42 for Reappraisal scale, and 4 and 28 for Suppression scale. High scores indicate a person who tends to apply reappraisal in a stressful situation (R), and/or to suppress their emotions (S). This scale is used as it measures two constructs conceptually linked to the study model.

The ERQ has acceptable internal consistency (Cronbach’s alpha above .70 for both scales), and adequate test–retest reliability ($r = .69$), and good convergent validity against the COPE

and mood regulation measures, and good discriminant validity against personality measures and impulse control measures (Gross & John, 2003).

5.8.2.5. Emotion Control Questionnaire (ECQ; Roger & Najarian, 1989).

This is a 56-item true-or-false measure of emotion control. The questionnaire has four 14-item subscales: Emotional Inhibition, Aggression Control, Benign Control, and Rehearsal (Rumination). The scale has good internal consistency and test-retest reliability with Cronbach's alphas for all the scales ranging between 0.71 and .80 (Roger & Najarian, 1989). For the purposes of this study only two of these scales were used: Emotional Inhibition (e.g., *"I seldom show how I feel about things"*) and Rehearsal (e.g., *"I often find myself thinking over and over about things that have made me angry"*), as both emotion inhibition and rumination are conceptually linked to the study model.

Aggression control measures suppression of aggressive behaviour (e.g., *"If someone pushed me I would push back"*), not suppression of emotion anger and therefore it was not relevant to this study. Benign control scale measures the level of impulsivity and similarly was not relevant to this focus of this study.

5.8.2.6. Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983).

This is a 14-item scale measuring anxiety (HADS-A; 7 items; e.g., *"I feel tense of wound up"*), depression (HADS-D; 7 items; e.g., *"I still enjoy the things I used to enjoy"*), and general distress (total score; Cosco, Doyle, Ward, & McGee, 2012; M. Dunbar, Ford, Hunt, & Der, 2000). The scores are measured on a 4-point Likert scale (0-3). The maximum score is 21 for each scale. The cut-off for both scales is 11: equal and higher scores indicate clinical anxiety/depression, scores between 8-10 suggest their presence, and scores equal or below 7 for each subscale are considered normal. The HADS has good internal consistency (all

Cronbach's alphas above .68 across different samples) and test-retest reliability ($r > .80$) of both the two-factorial and unifactorial structure (total score; e.g., Bjelland, Dahl, Haug, & Neckelmann, 2002).

5.8.2.7. Positive and Negative Affect Scale (I PANAS Short Form; Kercher, 1992).

This 10-item mood scale comprises two subscales – Positive Affect (e.g., “*inspired*”) and Negative Affect (e.g., “*hostile*”), and uses a 5-point Likert scale (1 – never to 5 – always). The time referent used in this study was the last month. The scales internal consistency is very good (Cronbach's alphas above .86), test-retest reliability $r = .79$ (PA) and $r = .81$ (NA)(David Watson, Clark, & Tellegen, 1988) The PANAS also has good convergent and discriminant validity (David Watson, et al., 1988).

5.8.2.8. Perceived Stress Scale (PSS; (Cohen, Kamarck, & Mermelstein, 1983).

This 10 item scale measures a person's own perception of stress, especially its unpredictability and uncontrollability (e.g., “In the last month how often have you been upset because of something that happened unexpectedly?”, “In the last month how often have you felt that things were going your way?”). It uses a 5 point Likert scale (0-4), and the scores range from 0 to 40. Internal consistency of the scale is very good (Cronbach's alphas above .84) and there is good evidence of concurrent validity versus depressive symptomatology ($\geq .65$)(Cohen, et al., 1983). Test-retest reliability varies across samples (correlations ranging between .55 and .85), which however can be a result of an unstable character of the construct of perceived stress, contrary to a stable character of e.g., personality traits (Cohen, et al., 1983).

5.8.2.9. Demographic Detail Questionnaire.

Demographic details were recorded for each participant with the use of a self-constructed questionnaire (Appendix 2). Questions asked for participants' age, marital status, education level, and work status (e.g., full time or retired), as well as about health (e.g., a type of cardiac event experienced), and medical treatment received (e.g., stents). Participants in this feasibility study (a healthy sample) were instructed to leave the questions blank if they did not apply to them (i.e., questions asking about cardiac health and treatment).

5.8.3. Observation and post-intervention interviews' data.

During each sharing session the researcher observed participants' behaviour and made notes about their ability and willingness to engage in social sharing and emotion expression. At the end of the study each participant was asked whether the study instructions were understandable and easy to follow, how they found the intervention in terms of discussing private stressful experiences and their emotions with a stranger, whether the amount of questionnaires they were asked to complete was acceptable for them, and what they thought about the intervention in general. The researcher reminded participants that they took part in a pilot study and that the main study method depends on their feedback.

5.9. Analysis

The study feasibility was tested through observation data, participation rate, and content analysis of post-intervention interviews. Quantitative analysis of questionnaires' data answered questions regarding potential post-intervention distress in participants. Statistical significance was set at $p < .05$ for all analyses.

5.9.1. Content analysis of post-intervention interviews and observation data.

To answer the questions regarding the study's feasibility, a content analysis was conducted of post-intervention interviews with participants. In the content analysis of post-intervention interviews participants' opinions and thoughts about the study (i.e., size of the questionnaire pack, the timing and amount of the meetings, instructions' clarity) were gathered.

Additionally, potential attrition and missing (or incomplete) questionnaires gave insight into participants' willingness to participate in the study. Observation of participants' behaviour during each interview answered the question about participants' willingness to engage in social sharing with a researcher and to express their emotions.

5.9.2. Changes in psychological variables.

A quantitative analysis, using a series of Wilcoxon sign rank tests, was conducted to investigate whether there were significant changes in psychological variables between Time₁ and Time₂.

5.9.3. Linguistic analysis.

To answer questions regarding trends in language use patterns during social sharing, and potential associations between language and psychological variables, a correlational analysis was conducted of associations between changes in language variables and changes in psychological variables, using non-parametric Spearman's rank-order correlation test.

Word categories tested were: positive emotion, *negative emotion*, *anxiety*, *anger*, *sadness*, *cognitive mechanisms*, *cause*, and *insight words*. Mean percentages of each category words were calculated for each sharing session (M_1 , M_2 , and M_3) using the LIWC 2007 software (Pennebaker, Chung, Ireland, Gonzales, & Booth, 2007). Emotion and cognitive mechanism word categories have been previously linked with the effects of emotion expression (e.g.,

Pennebaker, Mehl, & Niederhoffer, 2003). Furthermore, the increased use of emotion words is consistent with the emotion habituation model of emotion expression effects, whereas the increased use of cognitive words gives support for the cognitive model.

LIWC analyses each line of written text and categorises each word present in the text by comparing it to all the words present in LIWC's extensive dictionary file, which has evolved with each version of LIWC. LIWC subsequently code words found in the text into "content words" (e.g., nouns, regular verbs, adjectives, adverbs) or "style words" (e.g., pronouns, prepositions, articles), and it provides information about psychological processes. For example, it counts the frequency of positive emotion words (e.g., *happy, love, nice*) and negative emotion (e.g., *sad, frustrated, hurt*,) words. In addition it measures words that signify cognitive processing (e.g., causal words such as *because* or *cause*, and insight words such as *think* or *consider*). High levels of correlations have been reported between LIWC's coding of words and ratings made by external judges (Tausczik & Pennebaker, 2010).

5.10. Results

One participant was disqualified due to morbid alcohol dependency; 13 participants completed the study. Eleven were assigned to the emotion expression (EE) condition, and two to the sharing of facts (NS) condition. This imbalance arose, as very early into the study it was observed by the researcher that all participants found it easy to talk about negative experiences alone, but some had difficulties in describing the negative emotions associated to these events. Due to this fact, it was important for the researcher to conduct as many emotion expression sessions as possible, in order to test how participants would react to being asked to engage in emotion sharing, as well as sharing of negative experiences. This information was critically important to determining the feasibility and process of the main intervention.

Results are therefore based on the group of eleven participants from the emotion expression group.

5.10.1. Participants' characteristics.

The mean age of participants in the EE group ($n = 11$) was 56 years ($SD = 8.85$, Range: 27). In this group two participants were male (18.2%), and nine were female (81.8%). Table 2 presents demographic characteristics of participants in this group. Participants' agreeableness and extraversion levels were average, whereas neuroticism was borderline between average and high. Average total Alexithymia (TAS) score was low, as were scores on individual TAS scales. There were no cases of Alexithymia in this group. Negative Affectivity was above average, social inhibition was average (Table 3). There were 4 (36.4%) cases of Type D personality in this sample.

Table 2
Demographic Characteristics of EE Group Participants

Variable	N	%
<i>Marital status</i>		
Married	8	72.7
Separated	2	18.2
Widowed	1	9.1
<i>Education</i>		
Second level	4	36.4
Third level	7	63.6
<i>Work status</i>		
Full time	2	18.2
Part time	6	54.5
Retired	1	9.1
Unemployed	2	18.2
<i>Health</i>		
Healthy	8	72.7
Chronic disease (CHD, Arthritis)	3	27.3

Note. N: number of participants; %: percentage in the whole sample

Table 3
Baseline Personality Characteristics in the EE Group

<i>Variable</i>	<i>M (SD)</i>
Extraversion	12.27 (2.83)
Agreeableness	15.91 (3.56)
TAS	35.64 (9.14)
Difficulty Identifying Feelings	11.18 (3.25)
Difficulty Describing Feelings	9.09 (3.39)
Externally Oriented Thinking	15.36 (4.01)
NA	11.91 (5.59)
SI	8.64 (6.10)

Note. *M*: mean; *SD*: standard deviation; TAS: total Alexithymia score; NA: negative affectivity; SI: social inhibition.

5.10.2. Study procedure feasibility.

All thirteen participants engaged in social sharing and (when in the EE group) expressed their emotions when they were asked to, which indicated that the procedure was understandable and easy to follow. All participants but one completed all three meetings, and the one that completed only two was unavailable for the third meeting due to travel. One participant did not return baseline questionnaire pack due to time restraints on his part, but later completed all three meetings and returned post-intervention questionnaires.

5.10.2.1. Observation and post-intervention interviews.

The content analysis of post-intervention interviews with participants revealed that all participants were happy to have participated in the intervention; they found it easy and understandable, and an interesting experience. For seven participants the intervention was an opportunity to discuss negative experiences that were either previously shared only to a small extent or not shared at all. Two participants reported that they found the amount of questionnaires too large, but this did not affect their completion.

5.10.2.2. Changes in psychological variables.

The question of whether the intervention was distressing for participants was answered by analysing the data from the questionnaire packs, and comparing baseline to post-intervention. Medians and interquartile ranges for distress and affect variables are presented in Tables 4 and 5. Results of Wilcoxon signed rank tests revealed that depression and general distress (total HADS score) decreased significantly from pre- to post- intervention (Table 4). Effect sizes were calculated by dividing the Z statistic by the square root of the number of observations over two time points (Pallant, 2007).

Table 4
Medians and Interquartile Ranges for Distress Variables Pre- and Post-Intervention

	Anxiety	Anxiety ₂	Depression	Depression ₂	General Distress	General Distress ₂
<i>Mdn</i>	7.00	6.00	5.00	3.00	13.00	9.00
<i>IQR</i>	4.00	4.00	4.00	3.00	6.00	6.00

Note. Mdn: median; IQR: interquartile range

Table 5
Medians and Interquartile Ranges for Perceived Stress and Positive Affect Pre- and Post-Intervention

	Perceived Stress	Perceived Stress ₂	Positive Affect	Positive Affect ₂
<i>Mdn</i>	24.00	25.00	18.00	20.00
<i>IQR</i>	9.00	10.00	5.00	5.00

Note. Mdn: median; IQR: interquartile range

Table 6
Results of Wilcoxon Test for Distress and Affect

	Anxiety	Depression	General Distress	Perceived Stress	Positive Affect
Z	-1.94	-2.04	-2.15	-0.81	-0.68
p	.052	.042	.032	.420	.500
ES	-0.38	-0.40	-0.42	-0.16	-0.13

Note. Mdn: median; IQR: interquartile range

5.10.3. Linguistic analysis - association between changes in language use and changes in distress and affect.

Changes in distress and affect variables were correlated with changes in language use between interview 1 and interview 3 using Spearman's correlation (Table 7).

Increased use of anger words was found to be associated with decreases in anxiety, depression and general distress, as well as with increased use of cause words, which also correlated with decreases in anxiety. These findings suggest that participants who expressed more anger over the course of the intervention, as well as more cause words, experienced a decline in psychological distress.

Table 7
Changes in Anger Words and Changes in Distress

Variable	1	2	3	4
1. Anger words change	---			
2. Cause words change	.573*	---		
3. Anxiety change	-.671*	-.543*	---	
4. Depression change	-.612*	-.061	.566*	---
5. General Distress change	-.711**	-.355	.943**	.745**

* $p < .05$, ** $p < .01$ (1-tailed)

5.11. Discussion

This feasibility study tested the procedure designed for the main study and confirmed its feasibility. It additionally provided some data regarding the dynamics between psychological and language variables, which is further tested in the main study.

5.11.1. Study procedure feasibility.

The feasibility of the method design was confirmed - people engaged in social sharing and expressed emotions following instructions, which indicated that the procedure was understandable and uncomplicated. None of the participants withdrew from the study; however, this might have been due to the special character of this sample. As participants were members of the college participant panel, they may have been more motivated, or felt obligated to complete the study.

5.11.2. Observation and post-intervention interviews.

Participation in this social sharing intervention, which required expressing negative emotions from most of them, was found to be an interesting experience. The intervention gave most participants an opportunity to uncover emotions they never shared with others or to discuss life events that have never been discussed.

5.11.3. Changes in psychological variables.

The finding of significant decreases in depression and general distress may not suggest that the intervention itself was beneficial (as no comparison has been made to a control group), but it indicates that this design is safe for participants and it does not induce distress. Whether these declines in distress are due to social sharing with emotion expression is further tested in

the main study. Previous research found positive effects of disclosure (albeit written disclosure) on psychological health (e.g., Sloan & Marx, 2004a; Zakowski et al., 2004).

5.11.4. Associations between changes in language use and changes in psychological variables.

Results of correlation analysis suggested that decreases in psychological distress are linked to increases in anger words use from pre- to post-intervention. This is in line with previous research suggesting that psychological health benefits are associated with moderate expression of negative emotion words use, rather than excessive or low expression of negative emotions (Pennebaker, et al., 1997). However, as the present observation was made for anger words category only, it is plausible that there is a link between anger expression and distress, as previous research suggested that anger directed inward underlies depressive symptoms (e.g., Clay, Anderson, & Dixon, 1993). This is further investigated in the main study.

5.11.5. Conclusions.

This study confirmed the feasibility of a social sharing intervention with or without emotion expression and suggested that there are potential positive effects of such an intervention on psychological indices. Interesting observations were also made regarding associations between language use patterns and changes in psychological variables. The main study will compare two types of social sharing (sharing with emotion expression vs. sharing of facts only) with a control group and add a follow-up assessment to determine long-term effects.

5.11.6. Limitations.

Participants in this feasibility study were members of a college participant panel, therefore they were highly motivated to complete the study. This level of motivation can not be generalised to other populations and non-compliance and drop outs are expected when other populations are used. It is possible that future social sharing studies will discover that it is not an easy task for people to socially share their emotions with a stranger, especially if sample involved in sharing is a chronically ill sample, or sample of participants who just experienced a serious life-threatening (traumatic) event.

Main Study Hypotheses, Research Questions and Method

The present chapter outlines the study aims, hypotheses and research questions, and the study method.

6.1. Study Aims

This study had four aims:

- 1) The first aim is to compare the psychological and physiological effects of two social sharing interventions - sharing of facts and emotions, and natural sharing, with a usual care (no sharing intervention) group.
- 2) The second aim is to investigate potential moderators and mediators of the effects of sharing on distress and affect.
- 3) The third aim is to investigate the differences between both social sharing groups in language use, with a focus on emotion, cognitive and social word categories, as well as in physiological indices measured during each sharing episode (HRV; and HR_f - heart rate, as measured by finger pulse).
- 4) The fourth aim is to investigate the associations between overall language use and outcome measures.

6.2. Research Hypotheses and Research Questions

This section presents the hypotheses and (if applicable) study questions relating to each of the four study aims.

6.2.1. Comparison of the psychological and physiological effects of two social sharing interventions (Aim 1).

Generally, it is hypothesized that relative to natural social sharing, or to no sharing, social sharing with emotion expression will have a positive impact on psychological and physiological health, as well as decreasing levels of rumination and increasing levels of reappraisal.

With regards to the first study aim, the study is designed to test a number of specific research hypotheses.

6.2.1.1. Effects on psychological outcomes.

Hypothesis_{1a}: Compared to baseline levels, social sharing with emotion expression will decrease anxiety compared to natural sharing and no sharing at time 2 (immediately post-intervention) and time 3 (follow-up).

Hypothesis_{1b}: Social sharing with emotion expression will decrease depression compared to natural sharing and no sharing at time 2 (immediately post-intervention) and time 3 (follow-up)..

Hypothesis_{1c}: Social sharing with emotion expression will decrease general distress compared to natural sharing and no sharing at time 2 (immediately post-intervention) and time 3 (follow-up).

Hypothesis_{1d}: Social sharing with emotion expression will decrease negative affect compared to natural sharing and no sharing at time 2 (immediately post-intervention) and time 3 (follow-up).

Hypothesis_{1e}: Social sharing with emotion expression will increase positive affect compared to natural sharing and no sharing at time 2 (immediately post-intervention) and time 3 (follow-up).

Hypothesis_{1f}: Social sharing with emotion expression will decrease perceived stress compared to natural sharing and no sharing at time 2 (immediately post-intervention) and time 3 (follow-up).

6.2.1.2. Effects on physiological outcomes.

Changes are expected in systolic and diastolic BP, as well as HR post-intervention (Time 2) and at the follow-up (Time 3).

Hypothesis_{2a}: Social sharing with emotion expression will decrease patients' systolic BP compared to natural sharing and no sharing.

Hypothesis_{2b}: Social sharing with emotion expression will have decrease patients' diastolic BP compared to natural sharing and no sharing.

Hypothesis_{2c}: Social sharing with emotion expression will have decrease patients' HR compared to natural sharing and no sharing.

6.2.2. Investigating the potential moderators and mediators of the effects of sharing on distress and affect (Aim 2).

Type D personality and Alexithymia are expected to moderate the effects of the interventions on the outcomes of distress and affect at time 2 and 3. In addition, pre- to post-intervention changes in rumination and reappraisal are expected to mediate the effects of social sharing on distress and affect at time 2 and 3 (see Chapter 5, sections 5.3. and 5.4.).

The following hypotheses relate to the second aim of the study:

Hypothesis_{3a}: Type D personality will moderate the direct effect of group on psychological outcomes at Time 2 (post-intervention) and Time 3 (3 months follow-up),

Hypothesis_{3b}: Alexithymia will moderate the direct effect of group on psychological outcomes at Time 2 (post-intervention) and Time 3 (3 months follow-up),

Hypothesis_{3c}: Pre- to post- intervention decreases in rumination will mediate the effect of group on distress and affect at Time 2 (post-intervention) and Time 3 (3 months follow-up),

Hypothesis_{3d}: Pre- to post- intervention increases in reappraisal will mediate the effects of group on distress and affect at Time 2 (post-intervention) and Time 3 (3 months follow-up),

Hypothesis_{3e}: Type D personality will moderate the indirect effect of group on psychological outcomes at Time 2 (post-intervention) and Time 3 (3 months follow-up),

Hypothesis_{3f}: Alexithymia will moderate the indirect effect of group on psychological outcomes at Time 2 (post-intervention) and Time 3 (3 months follow-up)

6.2.3. Investigating the differences between both social sharing groups in language use, and in physiology, during sharing (Aim 3).

The following hypotheses predict the differences between sharing groups (social sharing with emotion expression vs. natural sharing) in language use (with a focus on emotion and cognitive word categories associated with psychological outcomes (see Chapter 2, section 2.4.), and in physiological indices measured during each sharing episode (HRV and HR_f).

6.2.3.1. Differences in language use between EE and NS groups.

Hypothesis_{4a}: The overall use of emotion words and cognitive words (averaged across all three meetings) will be greater in the emotion expression group

Hypothesis_{4b}: The use of emotion words and cognitive words will increase over the course of the intervention more as a result of social sharing with emotion expression than of factual sharing.

6.2.3.2. Physiological changes during sharing episodes.

During sharing episodes HR_f (heart rate measured by finger pulse) and HRV were assessed to test the hypothesis that emotion expression has positive effects on physiology in terms of decreasing physiological arousal deriving from inhibition. This process, the disinhibition, is reflected in the following hypotheses.

Hypothesis_{5a}: During each sharing interview, mean HR_f will be lower in the EE group than in the NS group (as an effect of disinhibition).

Hypothesis_{5b}: During each sharing interview HRV will be higher in the EE group than in the NS group (as an effect of disinhibition).

6.2.4. Investigating the associations between overall language use and outcome measures (Aim 4).

Previous research found the associations between language use during emotion expression and psychological outcomes (Tausczik & Pennebaker, 2010). Although the role of negative emotion words in predicting outcomes is not clear, more evidence suggests that an overtime decline in those words is more beneficial, than an increase (Pennebaker, et al., 1997).

The following hypotheses predict associations between overall language use and post-intervention and follow-up outcome measures.

Hypothesis_{6a}: Decreases in the use of negative emotion words, over the course of the intervention, will be associated with decreases in distress and increases in positive affect.

Hypothesis_{6b}: Increases in the use of positive emotion words over the course of the intervention, will be associated with greater decreases in distress and increases in positive affect.

Hypothesis_{6c}: Increases in the use of cognitive words over the course of the intervention, will be associated with greater decreases in distress and increases in positive affect.

6.3. Method

This section outlines the study method.

6.3.1. Overview.

This study brings together methods of both expressive writing research and social sharing research. The mode of expression is verbal with a listener; however, the content of disclosure is similar to the writing task method, where the expression of emotions and facts around a negative life event is compared to expression of facts only. In the emotion expression (EE) group participants engage in sharing and are asked about their emotions relating to the event they are describing. In the other sharing group (“natural sharing”, NS group), participants are *not* asked to suppress their emotions and to only discuss facts, instead, they engage in sharing in a way most natural to them, without specific instructions.

The sharing modes are compared with the control (no sharing) condition. This group is a “usual care” group that, after discharge, typically waits 3-6 months for CR without being contacted by the hospital. Such a control condition was chosen in order to test whether an additional social sharing intervention for post-MI/PCI patients awaiting CR would be beneficial in lowering distress and physiological indexes. Comparing experimental conditions to a “usual care” group, who however was also invited to the hospital for assessment at similar time points, would satisfy the study aim, controlling for extraneous variables related to a hospital setting (i.e., visiting the hospital where one spent several days after their cardiac event, meeting cardiac nurses, inquiring about CR, perceived social support) and the effects of time on psychological distress and positive affect post-event.

Participants came from a group of outpatients in a cardiology department. The study lasted for 36 months, during which time 101 patients completed at least one assessment and 85 completed the whole intervention. The follow-up period was 3 months after intervention

completion and 63 participants completed the follow-up assessment. The detailed study procedure, the randomisation process, and analysis design are outlined below.

6.3.2. The philosophical stance and the RCT method.

The age of enlightenment saw rationalism being replaced by empiricism; experience superseded deduction. In the 20th century, however, this modernist and determinist approach to science was challenged by post-modernist views. Rather than accepting objective reality, post-positivists acknowledged the imperfection of human intellect and its ability to capture the truth; hypothesis verification was replaced by theory falsification - disproving the theory, as opposed to proving it. As Karl Popper suggested, only falsifying a theory generates certainty. Despite this, the main goal in both scientific approaches remained the same – to study cause and effect of phenomena, to predict and generalise them, using standardised tools and ensuring an objective researcher role (although post-positivists noticed a possibility of experimenter's bias due to human imperfections).

A unified ontology of psychology has not been agreed upon; positivist “reality” is objective, material and easily measured. Psychological phenomena, when approached from this perspective, are usually viewed as states that are determined by underlying biological processes, which has led some scientists to believe human mind and human experience are nothing more than a series of neurobiological processes (e.g., Churchland, 1995). Although this reductionist approach may certainly add to the knowledge (e.g., neural and hormonal influence on psychological functioning), non-materialistic psychological phenomena should not be solely translated into biological or neurobiological processes, as such a simplification may reduce psychology to a paradigm of natural sciences, making the very science of psychology not worth pursuing (Yanchar & Hill, 2003). A post-positivist approach to psychology allows for a degree of uncertainty, as it views “reality” as what is most probable,

rather than objectively true. The post-positivist stance taken in the present thesis accounts for the existence of subjective human experience and of “mental life” (see Yanchar & Hill, 2003), as “probably real” components of psychology. Most importantly, however, the positivist preoccupation with what is “real” is replaced here by the interest in studied phenomena per se, even if their reality can not be objectively established.

From the epistemological point of view, positivist objective knowledge was replaced in post-positivism by knowledge that does not exist outside of human experience and is not “black or white”, as is the case in the positivist approach (e.g., Ryan, 2006). Furthermore, for post-positivists, knowledge that is subjective is of the same value as objective knowledge and all people are trusted to be capable of naming their own experience, therefore are valid sources of information. Finally, making inferences is not straightforward in post-positivist science; results may be interpreted in various ways and multiple explanations are often given, contrary to the positivist approach which aims to establish cause and effect and to make predictions, and which offers external knowledge with no scope for relativity.

Non-material mental processes investigated by psychologists (e.g., personality, emotions) have been measured using self-reports, thus relying on subjective experience of studied individuals, by many researchers so far, despite criticisms of participants’ lack of self-awareness or cultural differences. Although certainly valid and important, those criticisms do not make the reality “less real”, as “real” is no longer the main interest of the post-modernist psychology – studied phenomena are (Gergen, 2001). Data quantification has not been abandoned by post-positivists, as it allows for investigating patterns across cases, and for statistical analysis, however qualitative methods, which derive from the constructivist (and not positivist) paradigm, are also used by post-positivists, often as a combination with the quantitative ones. Qualitative analyses add depth to flat numerical data and account for the uniqueness of individuals studied.

6.3.2.1. A randomised controlled trial.

To test the present hypotheses a randomised controlled trial (RCT) was designed to test the effects of two types of social sharing (one with emotional expression and one without emotion expression, just facts) on psychological and physiological outcomes. RCT was a suitable method for testing the effects of an intervention as it allows causal inferences, it requires randomisation to avoid allocation bias, and it allows replication. An RCT made it possible to apply similar instructions to those used previously in expressive writing research and it provided answers about the effects of two social sharing interventions on psychological and physiological outcomes in a group of post-MI/PCI outpatients awaiting CR.

Following the post-positivist view on science, despite being a “gold standard”, in a social science a randomized controlled trial needs to be applied with an understanding that not all psychological phenomena can be easily measured; cause and effect, in psychology, is not always straightforward. For example, while high level of rumination can lead to increased distress, the opposite is also true. Therefore, in randomized trials, results become evidence only when it is decided by the experimenter that they support the case that is being investigated (Cartwright & Montuschi, 2014).

Controlling for extraneous variables may also be problematic in psychology. For example, although the study design required one group to engage in emotion expression, another group – in natural sharing, and the third group – no to engage in sharing, what happened outside the study setting could not be controlled for, which is where the principles of an RCT meet the reality of human existence. Furthermore, even the natural sharing group was not prevented from emotion expression (although not encouraged to do so), which muddles the conditions even further. All these comprise the practical side of conducting an RCT within a social science.

Despite this, RCTs offer general knowledge, which helps to understand the human experience and, most importantly, there is a general agreement within the psychological community that such studies have great value. While it is not possible to understand an individual without studying the individual, to understand more general concepts and behaviours (such as emotion inhibition, or social sharing), quantitative studies are both easy to implement and less time consuming than qualitative research.

6.3.2. Design and research setting.

The present study was a RCT with parallel design. To test the effects of two types of social sharing on outcome measures, two experimental groups were compared with a control group. The study was conducted in a hospital setting and each meeting was held in a room in the Cardiac Rehabilitation Department of the hospital.

6.3.3. Participants.

Patients with a recent major cardiac event (i.e., an MI and PCI, or PCI only), as diagnosed by a cardiologist, were recruited within 3 months post-discharge. None of the patients started Phase III CR before completing the intervention. Patients who were diagnosed as clinically depressed and therefore were receiving psychological help were excluded from the study. Participants were adult Irish residents, fluent English speakers, capable of understanding the study procedure and giving informed consent. Patients after a heart transplant or with heart failure, and patients with major co-morbidities (including psychological and neurological) were excluded from the study. These data were included in patients' hospital charts and therefore were accessible to the cardiac nurses involved in the recruitment process. All potential patients were approached by cardiac nurses, who informed patients about the study, and obtained consent, whereby participants agreed to be contacted

by the researcher within a month after hospital discharge. At this stage patients received general written information about the study, and what the involvement in the study might comprise. The researcher contacted each patient who agreed to be contacted approximately one month after initial contact by the cardiac nurses. A phone conversation with each patient took place after the randomisation process, which meant that participants were informed over the phone what their involvement will be if they agree to participate. At that time a first meeting with each participant was scheduled.

6.3.4. Randomisation procedure.

Before being contacted by the researcher potential participants were randomly assigned to one of the three study conditions (see below). The randomisation process had two phases. First, a research randomiser programme (Research Randomiser Form v4.0; Urbaniak & Plous, 2011) was used to generate a 100-item sequence of digits between 1 and 3: 1 corresponded with group A (emotion expression; EE), 2 with group B (natural sharing; NS), and 3 with (the control; CTRL) group C. Each participant had an equal possibility to be allocated to any of the three groups. When the string was generated, each number between 1 and 100 had a corresponding letter A, B or C. Order of allocation was established by shuffling the participant numbers using a list randomiser (random.org/lists/).

6.3.5. Sample size.

A sample size of 101 participants was recruited for the study. This was based on the G*Power 3.1 programme analysis (Faul, Erdfelder, Lang, & Buchner, 2007) and provided power of 0.80 to detect an effect size of $f = 0.15$ using a mixed-model ANOVA (three groups x three time points) as being statistically significant at the .05 level.

6.3.6. Assessment.

All participants were assessed three times. Sharing participants (groups EE and NS) were assessed at the first hospital meeting (pre-intervention), at the third meeting (post-intervention), and three months after the third meeting. Control participants were assessed over a similar time period as the intervention participants. They were required to meet the researcher in the hospital for each of the three assessments. The first two assessments were two weeks apart. The follow-up assessment took place approximately three months after the second one. It has been previously suggested that follow-ups of similar length were more feasible in investigating the effects of emotion expression on outcomes than shorter ones (Frattaroli, 2006).

6.3.7. Study groups.

The three study conditions were:

Experimental condition 1: Social sharing: Emotion expression group – EE group

Experimental condition 2: Social sharing: natural sharing group – NS group

Control condition: No sharing group – CTRL group

The intervention comprised three weekly meetings with each participant from both experimental groups. The meetings took between 30 minutes and one hour.

6.3.8. The Intervention.

The EE condition, sharing with emotion expression, required participants to discuss stressful life events and to name the emotions related to them. In this condition, instructions asked for both stressful life events (e.g., “*I would like to ask you to choose any stressful, negative event from your past that you would like to tell me about*”) and emotions/feelings experienced (e.g., “*Can you tell me exactly what you were feeling?*”).

The NS condition, natural sharing, required participants to freely discuss the stressful life events, without any additional instructions. Of note, participants in this group were not specifically asked to suppress emotions/feelings and were not stopped when they did. This was to ensure that the researcher didn't induce emotion suppression in patients, as that might have lead to higher stress levels, and greater sympathetic response in those patients (James J Gross & Levenson, 1997). Exemplar transcripts of two sessions – one from each intervention group – are attached (Appendix 3).

Participants from both experimental groups took part in 4 meetings each:

- 1) Meeting 1 – up to 1 hour and ten minutes duration – first psychological assessment, first interview, physiological assessment;
- 2) Meeting 2 – up to 30 minutes – second interview, physiological assessment;
- 3) Meeting 3 – up to 50 minutes – third interview, physiological assessment, second psychological assessment;
- 4) Meeting 4 – up to 30 minutes – third (follow-up) psychological assessment, physiological assessment, follow up interview and debriefing.

Control group participants took part in 3 meetings:

- 1) Meeting 1 - 30 minutes – first psychological assessment, first physiological assessment (blood pressure only);
- 2) Meeting 2 - 30 minutes – second psychological assessment, second physiological assessment (blood pressure only);
- 3) Meeting 3 - 30 minutes – third (follow-up) psychological assessment, physiological assessment (blood pressure only), and debriefing.

The process of assessment is presented in *Fig. 7*. The interviewer only asked questions and listened to participants' stories. The conversation was made as similar to everyday conversations people experience as it was possible, considering the unusual (hospital) setting and the fact that participants knew they were taking part in a research study. All the interviews were tape-recorded. The control group participants were not involved in social sharing, but were invited to the hospital to be assessed, at three time points. Both meetings with the control participants comprised of psychological assessment and BP measurement.

The study procedure involved all study participants attending hospital meetings with the researcher. The rationale for this was to avoid a situation in which control participants were the only ones not coming back to the hospital and not meeting with the researcher. This was proposed in order to control for an added element of being invited to the hospital shortly after an MI.

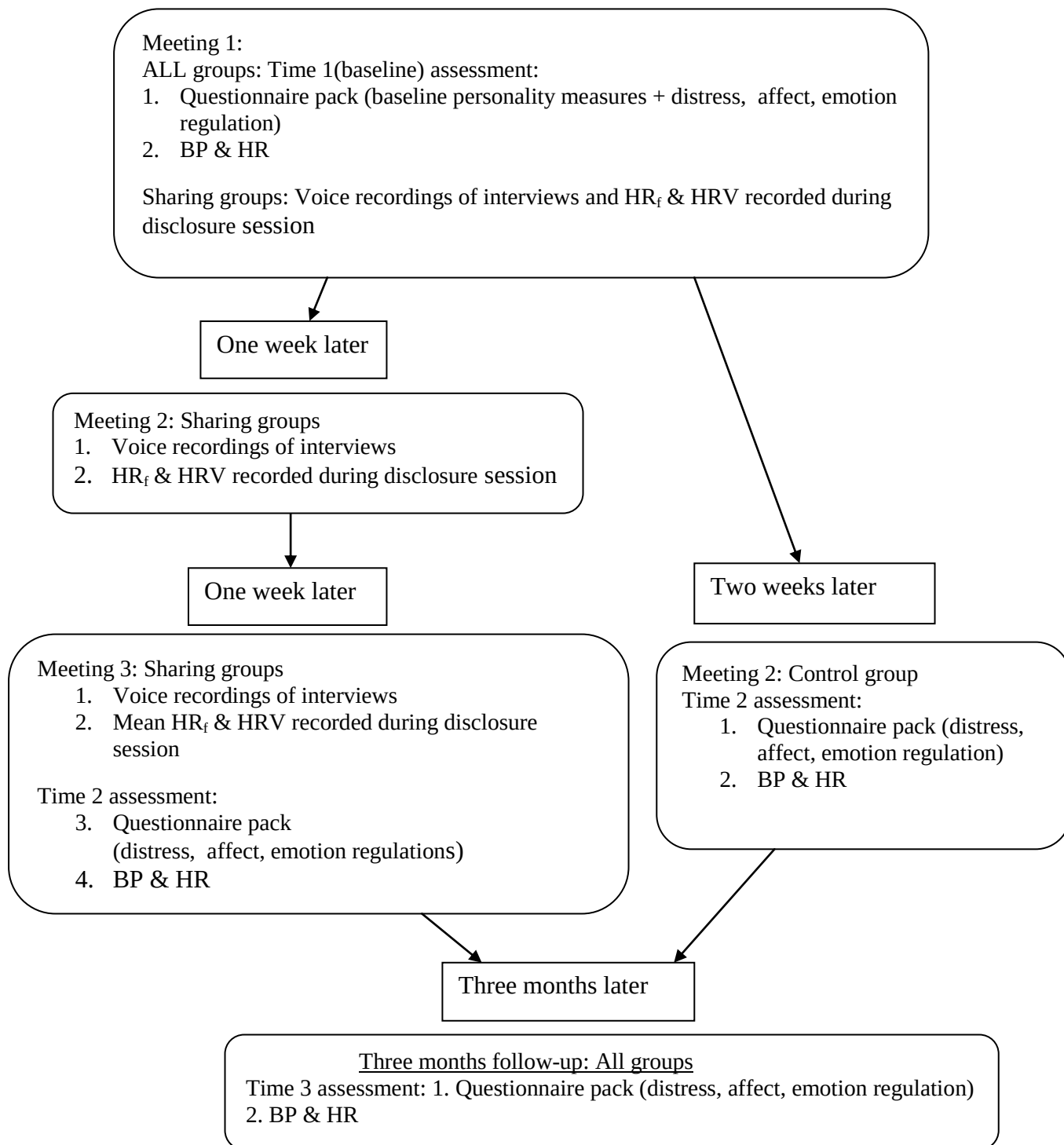


Fig. 7. Outline of Assessment of Participants during the Intervention.

The questionnaire battery for the main study (see section 5.8.2.) includes:

- 3) Background measures informing of the baseline character of the sample and not included in the main model that is being investigated;
 - a) Personality:
 - Traits related to sociability and cooperativeness: extraversion, agreeableness, and social inhibition,
 - Traits related to emotionality: negative affectivity,
 - Traits related to emotion inhibition: Alexithymia subscales (difficulty identifying feelings, difficulty describing feelings, externally oriented thinking)
 - b) Emotion regulation:
 - Strategies related to inhibition: suppression, emotion inhibition;
 - c) Demographic data;
 - Gender,
 - Marital status, education, and work status (to give an overall description of the sample),
 - d) Cardiac health data;
 - Type of cardiac event experienced (MI and PCI, or PCI only),
 - Time since cardiac event,
- 4) Baseline measures included in the model as moderators of the intervention effects,
 - a) Personality constructs related to emotion inhibition: Type D and Alexithymia,
- 5) Emotion regulation measures assessed pre- and post- intervention and included in the model as mediators of the intervention effects: reappraisal and rumination,
- 6) Outcome measures:
 - b) Distress: anxiety, depression, general distress, perceived stress and negative affect,

c) Positive affect;

As all the scales used in the main study were outlined previously (Chapter 5; section 5.3.2.), only present study reliabilities (based on “as treated” data) are presented below (Table 8).

Table 8
Cronbach's α for Personality Scales in the Present Sample

Scale	Cronbach's α
<i>Mini IPIP</i>	
Agreeableness	.40
Extraversion	.64
<i>TAS-20</i>	.78
DDF	.63
DIF	.77
EOT	.46
<i>DS-14</i>	.86
SI	.79
NA	.87
<i>ERQ</i>	.78
Suppression	.78
Reappraisal	.85
<i>ECQ</i>	.83
Rumination	.87
Emotion Inhibition	.71
<i>HADS total</i>	.87
Anxiety	.85
Depression	.77
<i>I-PANAS Short Form</i>	
Positive Affect	.79
Negative Affect	.76

6.3.8. Physiological assessment.

Physiological assessment comprised measuring blood pressure (BP) and brachial heart rate (HR) in all three groups.

For the two sharing groups, during each sharing episode, HR_f (heart rate measured by finger pulse) and heart rate variability (HRV) was continuously recorded.

The following HRV indexes were analysed: SDNN (a standard deviation of all normal RR intervals (measured between consecutive sinus beats), HRV triangular index, and RMSSD (the square root of the mean of the squared differences between successive NN intervals).

These are all time-domain measures of HRV suitable for short epochs (HRV recordings). The first two indexes reflect sympathetic and parasympathetic influence, whereas RMSSD is a measure of parasympathetic influence on HR_f.

6.3.8.1. Blood pressure.

At each meeting with the researcher, every participant (experimental and control) had their BP measured at three time points: at arrival, after a 10-minute rest period, and at the end of a meeting. BP was measured with the use of an electronic BP monitor (A&D UA-767 Plus Digital Blood Pressure Monitor). The monitor measures brachial BP and pulse. Brachial pulse is an indicator of heart rate and will be referred to HR_b in further chapters.

6.3.8.2. Heart rate and heart rate variability.

All experimental participants additionally had their HR_f and HRV monitored during each interview to assess the level of physiological arousal during the session. HR_f and HRV were measured with the *Vilistus Sensors* (Blood Volume Pulse sensors). Blood volume pulse

sensors record finger pulse, which is an indicator of heart rate. It will be referred to as HR_f in future chapters.

Data were recorded by *Vilistus Software* (v.2.5.8). One blood volume pulse sensor was put on participants' index finger of a *non-dominant hand* as the interview started and was taken off at the end of the interview. During this time participants were asked to lay their hand comfortably on the table. Each software recording was initiated at the exact time a voice recorder was switched on.

6.3.9. Post-intervention and 3-months follow-up assessment.

Post intervention, and follow-up assessment comprised psychological assessment using the following scales: DS-14, BEQ, ERQ, ECQ, HADS, I-PANAS-SF, and PPS. In addition, physiological assessment of patients' BP and HR were also recorded at these time points. During the follow-meeting each participant completed the last set of psychological questionnaires, and had their BP and HR measured. All participants were debriefed at this meeting - this included a thorough explanation of the study and anonymity reassurance (see debriefing sheet, Appendix 2).

6.4. Data Analysis

All results in this section are based on the “Intention to Treat” (ITT) analysis. To confirm the data patterns all analyses testing hypotheses were also run using “as treated” analysis – as no differences were found between the methods, the Results present the findings from the ITT analyses.

6.4.1. Type I and II errors.

For ITT analysis, statistical significance was set to $p < .01$ to control for a Type I error; however, due to novel character of this study, p values of $> .01$ but $< .05$ are followed up with *post-hoc* testing, to control for a possible Type II error. Post-hoc tests with Sidak adjustment are used, as Bonferroni adjustment was considered too conservative for a sample with a potential for a loss of power.

For “as treated” analysis (conditional process analysis and linguistic analysis) statistical significance was set to $p < .05$, due to limited sample size.

Similarly, due to exploratory character of HRV and HR_f analysis in both sharing groups, controlling for Type II error took precedence over controlling for Type I error, therefore despite multiple analyses probability was set to $p < .05$.

6.4.2. Data handling.

This section illustrates how the data prepared for analysis (missing data analysis) and how the violations of assumptions were handled.

6.4.2.1. Missing data.

Missing data was imputed using the regression method in SPSS, whereas outliers were transformed using the winsorising method (i.e., replacing the outlier with the nearest highest or lowest value from the non-outlier set).

6.4.2.2. Violations of normality, homogeneity of variance and sphericity.

In case of violations of normal distribution, for the purpose of descriptive statistics, means are reported in tables whenever means and medians have similar values. When comparing group

differences on baseline variables, non-parametric tests were computed and compared with one-way ANOVAs. If similar results were obtained, results of ANOVAs are presented. For the repeated measures analysis, in case of violations of normal distribution and homogeneity of variance assumptions, variables were log-transformed in order to make data suitable for a parametric analysis. When the assumption of sphericity was violated, Greenhouse-Geisser correction or Huynh-Feldt correction were applied to degrees of freedom. The choice between the two corrections is based on a recommendation to apply the Greenhouse-Geisser correction when the estimated epsilon falls below the value of 0.75, and the Huynh-Feldt correction when it exceeds 0.75.

6.4.3. Manipulation check.

As it was suggested in Chapter 3, there is no evidence that natural social sharing involves expressing emotions. For this reason natural social sharing was compared to social sharing with (an added instruction of) emotion expression. To test whether participants in the emotion expression group followed the instructions, linguistic analysis of the recordings of the interviews was conducted. Percentages of emotion words for each interview, for both intervention groups, were compared using a series of 2 x 3 (group by time) ANOVAs and, if applicable, *post-hoc* t-tests. Main effects of group for percentage of emotion words were expected to be observed suggesting greater use of emotion words in the EE group, as per study design.

6.4.4. Psychological and demographic categorical variables.

Chi-square tests were computed to test the associations between psychological categorical variables between the groups. The variables tested this way were Alexithymia, Type D personality, gender, marital status, education and work status.

Of note, Fisher's Exact test was used wherever expected values were too small and the assumptions of a Chi-Square test were violated.

6.4.5. Within and between-group effects.

To analyse the within- and between-groups differences in all psychological variables, over the three time points, 3 x 3 mixed-model ANOVAs were used with focus on group/time interactions to investigate how different forms of social sharing (emotion expression or facts disclosure, with comparison to each other and to the control group) impacted psychological functioning. Effect Sizes were reported using η^2 or Cohen's d (for between-groups design) and d_z (for within-groups design).

6.4.6. Mediators and moderators of effects.

A conditional process analysis (Hayes, 2013) was applied in order to test the moderators of the direct effect of the intervention on outcomes (i.e., anxiety, depression, general distress, perceived stress, negative affect, and positive affect), as well as to investigate the mediators and moderators of the indirect effect. The model was tested by analysing the "as treated" data using 5,000 bootstrapped bias-corrected resamples via the PROCESS macro for SPSS (v. 2.15; Hayes, 2016). The advantages of this method are that more than one mediator can be tested simultaneously, controlling for correlation between those mediators, and that Confidence Intervals are generated by a bootstrapping method, which does not assume

normality of data distribution. The bootstrapping method has been found superior to other resampling methods (Fan & Wang, 1996).

Analysis was based on the “as treated” complete data participants and the *CI*s level was set at 95%, to prevent Type II error that might occur due to a limited (“as treated”) sample size. For the same reason, near-significant results were followed-up, although interpreted with caution.

Two potential mediators were included in the model (*Fig. 8*): change in rumination and change in reappraisal, both calculated as a difference between Time 1 and Time 2 (i.e.,

$Rumination_{\text{chg}} = Rumination_2 - Rumination_1$; $Reappraisal_{\text{chg}} = Reappraisal_2 - Reappraisal_1$).

Two moderators also included in the model were Type D personality and Alexithymia. For the analysis, Type D was coded “1” for Type D and “0” for non-Type D; Alexithymia was

coded “2” for Alexithymia, “1” for possible Alexithymia, and “0” for non-Alexithymia, and

Group was coded “1” for Emotion Expression, “2” for Natural Sharing and “3” for Control.

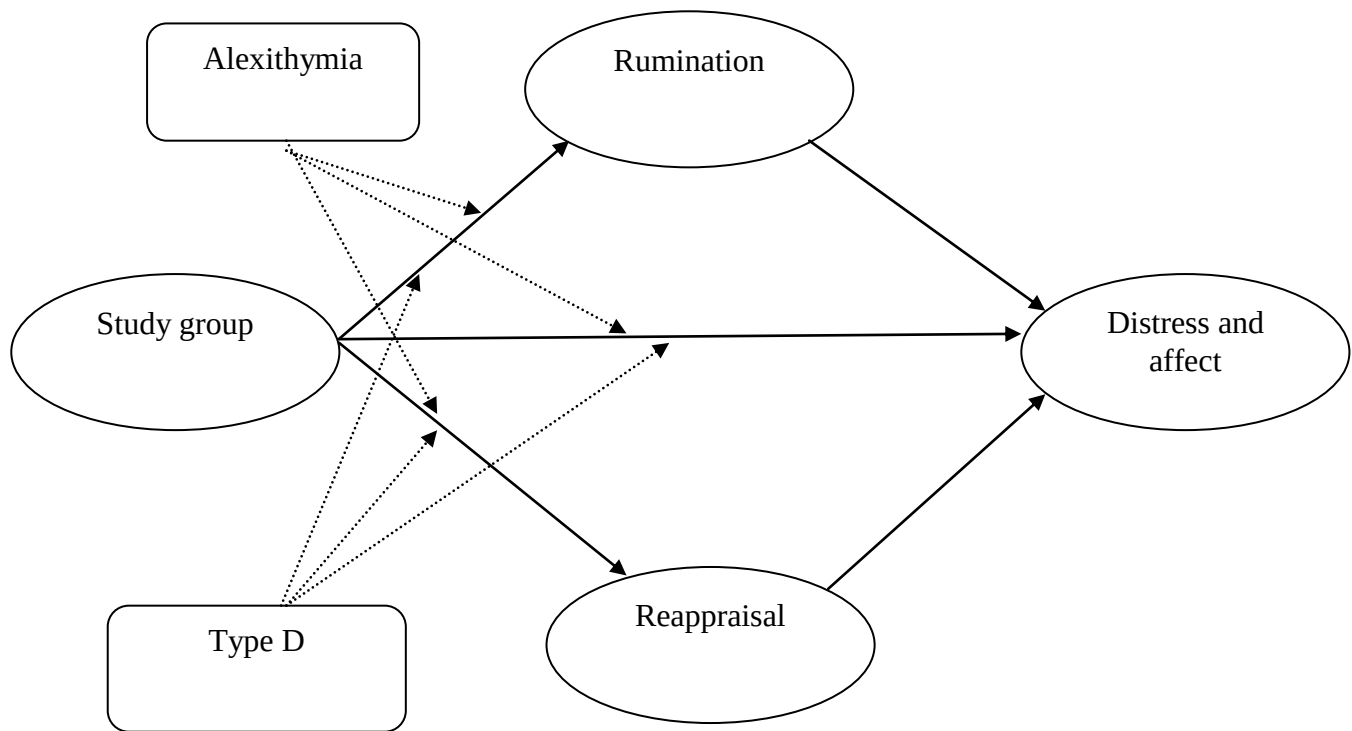


Fig. 8. Conditional Process Analysis with Rumination and Reappraisal as Mediators and Alexithymia and Type D as Moderators.

In the model, the following paths were tested:

- a) The effect of group on rumination change
- b) The effect of the group x moderator interaction on rumination change
- c) The effect of group on reappraisal change
- d) The effect of the group x moderator interaction on reappraisal change
- e) The direct effect of group on outcome
- f) Conditional (moderated) direct effect of group on outcome
- g) The indirect effect of group on outcome
- h) Conditional (moderated) indirect effect of group on outcome

The model was tested twelve times: twice (i.e., each outcome at Time 2 and Time 3) for each of the six outcomes (anxiety, depression, general distress, perceived stress, negative affect, positive affect). This way, two sets of information were obtained: 1) the moderating/mediating effects on post-intervention psychological outcomes, and 2) the mediating/moderating effects on psychological outcomes at follow-up.

6.4.7. Analysis of BP and HR.

Systolic and diastolic blood pressure were analysed separately: within- and between-subjects differences in systolic and in diastolic BP were compared. Three sets of data were analysed for all three groups (Time 1, Time 2, and follow-up Time 3). Each BP and HR data set contained two measurements (at arrival and after 10 minutes rest). Results from both measurements were averaged in order to achieve stable baseline.

A series of 3 x 3 (group by time) mixed ANOVAs were used to analyse the differences in systolic and in diastolic BP and HR within and between the intervention groups (baseline, post-intervention, and 3-months follow-up), and between time points.

6.4.8. Social sharing groups' data analysis.

A separate set of analyses were conducted for both sharing groups – linguistic analysis of recorded interviews and analysis of physiological indices recorded during interviews.

6.4.8.1. Linguistic analysis.

All the sharing sessions (interviews) were tape recorded and transcribed. The Word documents were analysed with the use of LIWC (2007) programme, described in Chapter 5, section 5.9.3. This analysis allowed for the between-group comparison, as well as the within group comparison across time, in relation to the language use. Furthermore, the association

between language use and psychological outcomes were investigated. Following analyses in previous research (e.g., Pennebaker, 1993) the focus of the analysis was a percentage of emotion words (positive, negative, anxiety, anger and sadness) and cognitive words (cognitive mechanisms, insight, and cause words).

As previously outlined (Chapter 5, section 5.9.3.), the use of emotion words, if increased, is consistent with the emotion habituation model of emotion expression effects, whereas the increased use of cognitive words gives support for the cognitive model. Within- and between-group differences in these percentages were analysed using a series of 2 x 3 mixed model ANOVAs.

6.4.8.2. Heart rate/heart rate variability.

HR_f and HRV were recorded continuously during each sharing episode. For analysis, fifteen minute epochs were extracted from each HR_f/HRV recording to make the data suitable for analysis. Mean HR_f and time domain indices of HRV were analysed and compared between the groups. Additionally, a series of Pearson correlations were conducted to test potential associations between the use of language and physiological measures during sharing episodes.

6.4.9. Flow chart.

Participants' flow chart in the present study is illustrated in *Fig. 9*.

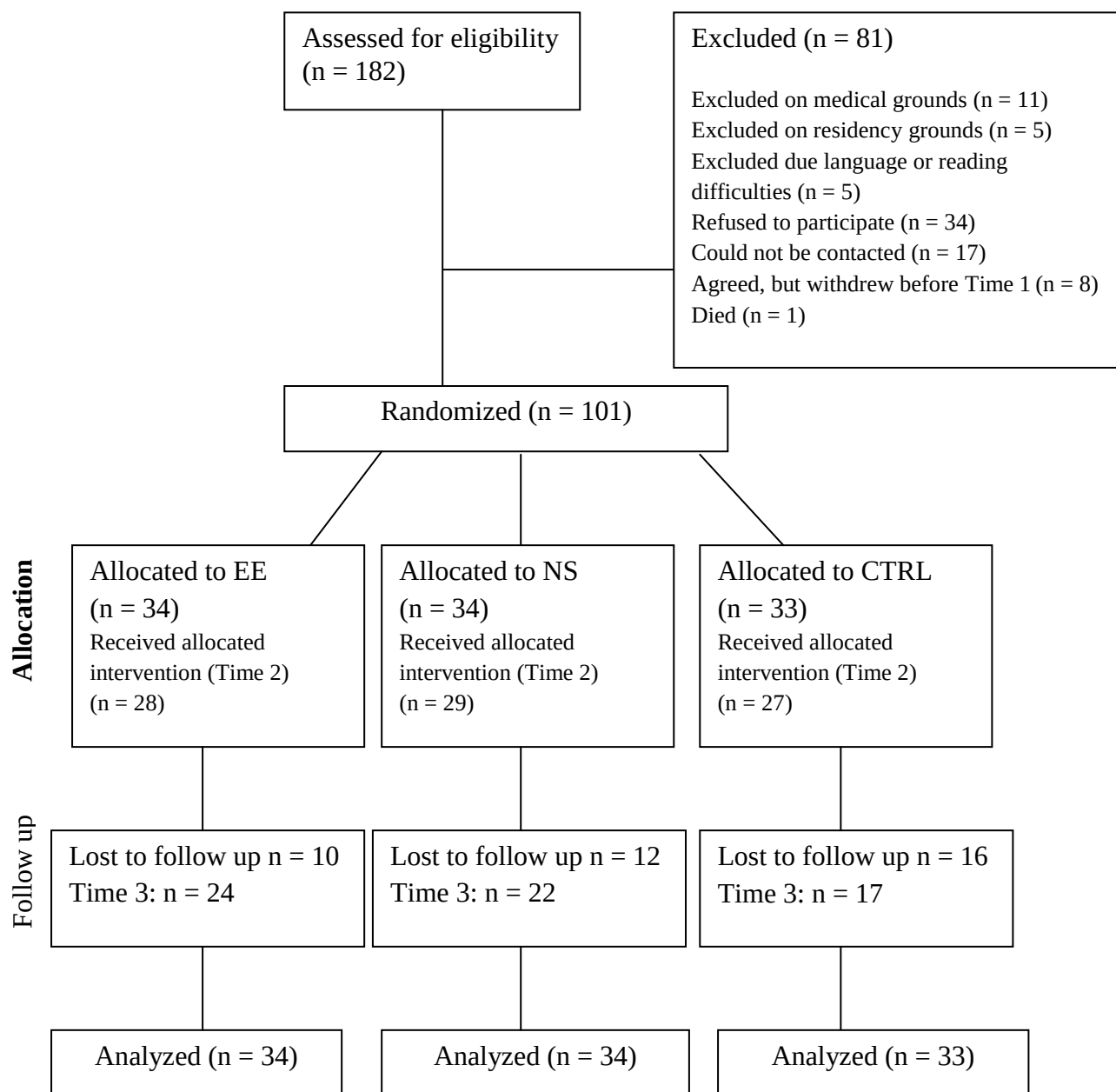


Fig. 9. Flow Chart

Results I

The Effects of Social Sharing with Emotion Expression on Psychological and Physiological Outcomes

The present chapter outlines the results of the analysis of the effects of the intervention on outcomes.

7.1. The Background Profile of the Sample

This section outlines the demographic, psychological and physiological characteristics of the sample at baseline.

7.1.1. Demographic statistics.

Data from thirty four participants in the emotion expression (EE) sharing group, thirty four in the just facts (NS) sharing group and thirty three in the control (CTRL) group were analysed. Participants' gender, MI incidence, history of depression, mean age and number of weeks since the cardiac event are presented in Tables 9 and 10. In each group majority of participants were male, had an MI and had no history of depression. They were in their late fifties to early sixties and commenced the study approximately 7 weeks after their cardiac event. In all groups, most participants were married, educated to a secondary level and either retired or in full time employment (Table 9). Results of Chi-Square tests (gender, MI prevalence), Fisher's Exact test (history of depression, marital and employment status, and education) and one-way ANOVAs (age and number of weeks since event) indicated that there were no baseline differences between the groups in these variables (Tables 9 – 11). Of

note, the Fisher's Exact test was used wherever expected values were too small and the assumptions of a Chi-Square test were violated.

Table 9
Gender, MI Prevalence, History of Depression per Group at Baseline

	EE	NS	CTRL	χ^2 (df)
Variable	N (%)	N (%)	N (%)	
Gender				3.95 ^a (2)
Male	28 (82.3)	26 (76.5)	31 (93.9)	
Female	6 (17.1)	8 (23.5)	2 (6.1)	
MI incidence				2.11 ^a (2)
MI + PCI	32 (91.4)	27 (79.4)	27 (81.8)	
PCI without MI	3 (8.6)	7 (20.6)	6 (18.2)	
History of Depression				1.47 ^b
Yes	5 (14.3)	5 (14.7)	2 (6.1)	
No	30 (85.7)	29 (85.3)	31 (93.9)	

Note. ^aPearson Chi-Square test results; ^bFisher's Exact test results
EE: emotion expression group; NS: natural sharing group; CTRL: control group;

Table 10
Descriptive Statistics for Demographic Variables by Group at Baseline

Variable	EE	NS	CTRL	F (2, 98)
	M (SD)	M (SD)	M (SD)	
Age	56.65 (9.76)	59.94 (8.36)	61.88 (9.14)	2.84
Weeks since event	6.77 (2.41)	7.93 (1.92)	7.03 (2.17)	2.66

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group;
F: the F statistic (ANOVA)

Table 11
Descriptive Statistics for Marital Status, Education Level, Employment Status by Group at Baseline

Variable	EE N (%)	NS N (%)	CTRL N (%)	Fisher's test
Marital Status				11.16
<i>Single</i>	4 (11.4)	2 (5.9)	1 (3.0)	
<i>Married</i>	26 (74.3)	26 (76.5)	25 (75.7)	
<i>Living Together</i>	1 (2.9)	3 (8.8)	0 (0)	
<i>Separated</i>	2 (5.7)	0 (0)	4 (12.1)	
<i>Divorced</i>	2 (5.7)	1 (2.9)	1 (3.0)	
<i>Widowed</i>	0 (0)	2 (5.9)	2 (6.1)	
Education				9.63
<i>Primary</i>	7 (20.0)	9 (26.5)	13 (39.4)	
<i>Second Level</i>	16 (45.8)	21 (61.8)	16 (48.5)	
<i>Third Level</i>	12 (34.3)	4 (11.8)	4 (12.1)	
Employment Status				6.03
<i>Full-time</i>	12 (34.3)	12 (35.3)	7 (21.2)	
<i>Part-time</i>	4 (11.4)	4 (11.8)	4 (12.1)	
<i>Retired</i>	13 (37.1)	16 (47.1)	17 (51.5)	
<i>Unemployed</i>	5 (14.3)	1 (2.9)	2 (6.1)	
<i>Other</i>	1 (2.9)	1 (2.9)	3 (9.1)	

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group;

7.1.2. Personality measures.

Table 12 presents prevalence of Alexithymia and Type D personality in each group at baseline. In each group between 30 – 40 % of participants were Type Ds, and between 15 – 30% had Alexithymia. There was no association between either Alexithymia categories (χ^2 (4, N = 101) = 2.51, p = .64) or Type D categories (χ^2 (2, N = 101) = 0.63, p = .73) and research group.

Table 12

Frequency of Alexithymia and Type D by Group at Baseline

	EE	NS	CRTL
Variable	N (%)	N (%)	N (%)
Alexithymia			
No	20 (58.8)	18 (52.9)	17 (51.5)
Possible	9 (26.5)	8 (23.5)	6 (18.2)
Yes	5 (14.7)	8 (23.5)	10 (30.3)
Type D			
Yes	11 (32.4)	14 (41.2)	13 (39.4)
No	23 (67.6)	20 (58.8)	20 (60.6)

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group;

People with Alexithymia were also more likely to have the Type D personality, although 19 individuals with Alexithymia or possible Alexithymia were not Type Ds and 11 Type Ds did not have Alexithymia (Table 13).

Table 13

Association between Alexithymia and Type D at Baseline

	Alexithymia	Possible Alexithymia	No Alexithymia	χ^2	<i>p</i>
Type D	17	10	11	20.52	< .001
No type D	6	13	44		

Note. χ^2 : the Chi-square statistic;

Descriptive statistics for extraversion, agreeableness, negative affectivity and social inhibition, as well as Alexithymia subscales are presented in Table 14. Results of one-way ANOVAs indicated that there were no significant differences between the groups in any of those variables at baseline.

Table 14

Descriptive Statistics for Personality Variables by Group at Baseline

Variable (range)	EE (n = 34) M (SD)	NS (n = 34) M (SD)	CTRL (n = 33) M (SD)	F (2, 98)	p	η^2
Extraversion (4-20)	13.44 (3.99)	14.00 (3.71)	13.15 (3.15)	0.48	.62	0.01
Agreeableness (4-20)	16.29 (2.44)	15.41 (2.76)	14.85 (2.64)	2.60	.08	0.05
Negative Affectivity (0-28)	11.50 (6.62)	12.97 (7.10)	11.97 (7.22)	0.39	.68	0.01
Social Inhibition (0-28)	8.29 (6.72)	8.65 (5.73)	9.70 (5.41)	0.50	.61	0.01
Difficulty Describing						
Feelings (5-25)	12.50 (3.96)	12.50 (4.84)	13.79 (4.78)	0.89	.41	0.02
Difficulty Identifying						
Feelings (7-35)	14.71 (6.25)	14.91 (6.18)	15.55 (6.43)	0.16	.85	0.003
Externally Oriented						
Thinking (8-40)	20.59 (5.47)	22.15 (5.30)	21.51 (4.61)	0.81	.45	0.02

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group; M: mean; SD: standard deviation; F: the F statistic (ANOVA); η^2 : effect size (eta-squared)

In general, participants had above average levels of extraversion, indicating that they were outgoing and enjoyed the company of others, and above average agreeableness, which suggested they were cooperative, sympathetic and kind. At the same time, participants' social inhibition was average, which indicated that, in general, the groups did not inhibit their emotions and behaviour during social interactions. Negative affectivity was high in all groups, indicating high levels of distress, which is to be expected after a serious cardiac event.

Scores of all three TAS subscales were average in each group, suggesting that in general, participants in each group had no problems identifying or describing their feelings and did not attribute their emotion related bodily sensations to the external causes.

7.1.3. Distress and affect.

Descriptive statistics of distress and affect variables are presented in Table 15. Despite violations of normality for HADS variables, due to means and medians being similar, the means are reported here. Furthermore, as non-parametric and parametric tests yielded the same results for those variables, results of one-way ANOVAs are reported here.

Participants in each group had low levels on all distress measures and above average level of positive affect. Results of a series of one-way ANOVAs indicated there were no between-group differences on these variables at baseline (Table 15), which was confirmed in an “as treated” analysis.

Table 15
Descriptive Statistics for Distress and Affect by Group at Baseline

Variable (range)	EE <i>M (SD)</i>	NS <i>M (SD)</i>	CTRL <i>M (SD)</i>	<i>F</i> (2, 98)	<i>p</i>	η^2
Anxiety (0-21)	6.65 (4.00)	6.35 (4.35)	6.21 (4.20)	0.10	.91	0.002
Depression (0-21)	4.26 (3.10)	3.56 (3.15)	3.76 (2.84)	0.49	.62	0.01
General Distress (0-42)	10.92 (6.33)	9.91 (6.51)	9.97 (6.33)	0.27	.77	0.01
Perceived Stress (0-40)	16.26 (7.83)	15.38 (8.31)	16.48 (8.33)	0.17	.84	0.003
Negative Affect (0-25)	11.23 (4.42)	10.44 (3.86)	10.73 (3.52)	0.34	.71	0.01
Positive Affect (0-25)	19.46 (3.60)	18.79 (3.91)	18.82 (4.32)	0.31	.74	0.01

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group; *M*: mean; *SD*: standard deviation; *F*: the F statistic (ANOVA); η^2 : effect size (eta-squared)

Participants were categorised to distressed and not distressed using cut-off point of 8 for anxiety and depression and 13 for general distress (Table 16). In each group, and overall, approximately one third of participants were distressed as measured by anxiety subscale and total HADS score, and 15% as measured with depression subscale.

Table 16
Frequency of Distress Categories by Group and Overall Sample

Variable		EE	NS	CTRL	Total sample
Anxiety	Low (< 8)	23 (68%)	20 (59%)	21 (64%)	64 (63.4%)
	High (> 8)	11 (32%)	14 (41%)	12 (36%)	37 (36.6%)
Depression	Low (< 8)	29 (85%)	29 (85%)	28 (85%)	86 (85.1%)
	High (> 8)	5 (15%)	5 (15%)	5 (15%)	15 (14.9%)
General Distress	Low (< 13)	24 (71%)	23 (68%)	21 (64%)	68 (67.3%)
	High (> 13)	10 (29%)	11 (32%)	12 (36%)	33 (32.7%)

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group;

7.1.4. Emotion regulation.

Table 17 presents mean scores in emotion inhibition, rumination, reappraisal and suppression. Participants in each group had average levels of emotion inhibition, suppression and rumination, and above average level of reappraisal. Of note, the assumption of normality was violated for Emotion Inhibition and Rumination; however, due to means and medians being similar, means are reported here. Results of non-parametric and parametric tests yielded the same results. No significant differences were observed between the groups in any of those variables at baseline. The “as treated” analysis confirmed this.

Table 17
Descriptive Statistics for Emotion Regulation by Group

Variable (range)	EE <i>M (SD)</i>	NS <i>M (SD)</i>	CTRL <i>M (SD)</i>	F (2, 98)	<i>p</i>	η^2
Emotion Inhibition (0-10)	4.15 (2.27)	4.50 (2.18)	4.75 (2.73)	0.55	.58	0.01
Rumination (0-10)	4.38 (3.45)	3.26 (3.31)	3.12 (2.67)	1.61	.21	0.03
Reappraisal (6-42)	27.97 (8.01)	26.32 (7.28)	27.09 (10.05)	0.32	.73	0.01
Suppression (4-28)	14.63 (6.19)	16.94 (5.48)	17.33 (6.25)	2.01	.14	0.04

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group; *M*: mean; *SD*: standard deviation; *F*: the F statistic (ANOVA); η^2 : effect size (eta-squared)

7.1.5. Blood pressure and heart rate.

Table 18 presents values of BP (systolic and diastolic) and HR (measured by brachial pulse) in each group at Time 1. Participants' BP and HR were normal in all three groups at baseline (i.e., below the cut-off of 140/90 that indicates hypertension). Results of one-way ANOVAs showed no baseline differences in BP or HR.

Table 18
BP and HR by Group at Baseline

Measurement	EE <i>M (SD)</i>	NS <i>M (SD)</i>	CTRL <i>M (SD)</i>	<i>F</i> (2, 98)	<i>p</i>	η^2
Systolic BP	127.52 (20.03)	131.05 (18.20)	131.62 (16.19)	0.50	.61	0.01
Diastolic BP	75.89 (9.27)	75.85 (9.15)	74.16 (11.27)	0.33	.72	0.01
HR	65.24 (9.75)	60.42 (8.86)	62.80 (9.56)	2.23	.11	0.04

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group; *M*: mean; *SD*: standard deviation; *F*: the F statistic (ANOVA); η^2 : effect size (eta-squared)

7.1.6. Comparison of baseline characteristics of completers vs non-completers.

A series of one-way ANOVAS were conducted separately for each study group to compare baseline measures of participants who attended Time 1 and Interview 1, but later withdrew from the study, and participants who completed the intervention. All results were non-significant (all *ps* < .01). No baseline differences were observed between completers and non-completers in the other two study groups.

7.1.7. Summary.

On average, participants in each group were their late fifties - early sixties, most of them were male, had an MI, entered the study less than two months after their cardiac event, and had no history of depression. Most were married, educated to a second level and either retired or in full time employment. In general, participants were high in negative affectivity, and although, on average, participants in each group were low in distress and above average on positive affect, a third of them were distressed and 15% had elevated level of depression.

Participants' above average levels of extraversion and agreeableness, and average level of social inhibition indicated that they were outgoing, cooperative and not afraid to express themselves in front of other people. This was confirmed by average levels of emotion inhibition, suppression and the TAS scales.

All groups were also likely to use the mechanism of reappraisal, rather than ruminate about their problems. A third of all participants were Type Ds, and less than a third had Alexithymia. Finally, participants' BP and HR were in the normal range in all three groups at baseline.

None of the baseline variables were different across the three groups.

7.2. Manipulation Check: Sharing Group Differences in Emotion Words Use

To test the differences between emotion words use between both sharing groups over the 3 assessments, a series of 2 x 3 ANOVAs were computed for each group of emotion words (i.e., positive emotion, negative emotion, anxiety, anger and sadness).

Results showed statistically significant group effects for negative emotion words [$F(1) = 6.51, p = .005, \eta^2 = .90$], anxiety words [$F(1) = 5.17, p = .01, \eta^2 = .07$] and anger words [$F(1) = 15.23, p < .001, \eta^2 = .19$], and marginal ones for positive emotion words [$F(1) = 4.09, p = .02, \eta^2 = .06$] and sadness words [$F(1) = 4.37, p = .02, \eta^2 = .06$]. On average, the EE group used more negative emotion words ($M_{EE} = 1.97$ vs $M_{NS} = 1.76$), more anxiety words ($M_{EE} = 0.55$ vs $M_{NS} = 0.44$), and more anger words ($M_{EE} = 0.52$ vs $M_{NS} = 0.37$); trends were observed towards greater use of sadness words ($M_{EE} = 0.29$ vs $M_{NS} = 0.23$), but lower use of positive emotion words ($M_{EE} = 2.14$ vs $M_{NS} = 2.33$) in the EE group compared with the NS group.

7.3. Repeated Measures Analysis

Before conducting a series of 3 x 3 mixed ANOVAs, all variables were log-transformed in order to make data suitable for a parametric analysis. Violations of normal distribution and homogeneity of variance assumptions occurred in Time 2 and Time 3 data.

7.3.1. Study Aim 1: Comparison of the effects of two social sharing interventions on psychological and physiological outcomes.

This section presents the results of testing the effects of the intervention on distress, affect, and BP and HR.

7.3.1.1. Changes in distress and affect.

Table 19 presents means for all distress and affect variables for each time point. Contrary to hypotheses 1_a to 1_f, no interaction effects of group x time on distress and affect were observed (Table 20).

Table 19

Distress and Affect Means (SD) by Group for Each Time Point

Variable (range)	Time	EE <i>M (SD)</i>	NS <i>M (SD)</i>	CTRL <i>M (SD)</i>
Anxiety (0-21)	1	6.81 (4.05)	6.35 (4.35)	6.21 (4.20)
	2	6.43 (3.36)	6.52 (3.28)	6.88 (3.95)
	3	6.09 (3.98)	5.34 (2.71)	5.78 (3.25)
Depression (0-21)	1	4.31 (3.08)	3.56 (3.16)	3.76 (2.84)
	2	4.21 (3.27)	3.62 (2.50)	3.77 (3.19)
	3	4.06 (3.56)	3.56 (2.44)	3.81 (2.34)
<i>General Distress</i> (0-42)	1	11.12 (6.35)	9.91 (6.51)	9.97 (6.33)
	2	10.64 (6.09)	10.14 (4.83)	10.65 (6.53)
	3	10.15 (7.20)	8.89 (4.43)	9.59 (4.94)
<i>Perceived Stress</i> (0-40)	1	16.43 (7.79)	15.38 (8.31)	16.49 (8.33)
	2	16.12 (6.57)	14.79 (8.52)	15.50 (7.29)
	3	14.51 (7.44)	12.79 (6.25)	14.24 (5.21)
<i>Negative Affect</i> (0-25)	1	11.35 (3.62)	10.38 (3.84)	10.70 (3.54)
	2	11.22 (3.02)	10.20 (3.67)	10.91 (3.68)
	3	9.69 (2.87)	8.60 (2.67)	10.28 (3.69)
<i>Positive Affect</i> (0-25)	1	19.49 (4.40)	18.79 (3.91)	18.82 (4.32)
	2	19.03 (3.33)	18.19 (4.10)	19.31 (2.79)
	3	19.99 (3.67)	19.52 (2.90)	19.86 (2.47)

Note. For ease of interpretation the raw mean scores are presented

EE: emotion expression group; NS: natural sharing group; CTRL: control group;
M: mean; SD: standard deviation;

Table 20
The Main Effect of Group x Time on Distress and Affect

	<i>df</i>	<i>F</i>	<i>p</i>	η^2
Anxiety	3.91*	0.48	.75	.01
Depression	4	0.09	.99	.002
General Distress	4	0.30	.88	.01
Perceived stress	3.88*	0.64	.63	.01
Negative affect	4	2.20	.07	.04
Positive affect	3.78*	1.31	.27	.03

Note. *Huynh-Feldt adjusted degrees of freedom
df: degrees of freedom; *F*: the F statistic (ANOVA); η^2 : effect size (eta-squared)

7.3.1.2. Changes in blood pressure and heart rate.

This section presents the tests of the hypotheses (2_a to 2_c) relating to social sharing with emotion expression effects on physiological measures. Due to violations of normality and homoscedasticity, all the BP and HR variables were log-transformed to make them suitable for parametric analyses. A 3 x 3 ANOVA was computed to test over time changes in systolic and diastolic BP and HR. Table 21 presents mean values for physiological measures for each group at each time point. For the ease of interpretation the values of BP and HR in tables and the graphs are based on untransformed data.

Table 21
Average BP and HR by Group over Time

Measurement	Time	EE <i>M (SD)</i>	NS <i>M (SD)</i>	CTRL <i>M (SD)</i>
Systolic BP	1	127.52 (20.03)	131.05 (18.20)	131.62 (16.19)
Systolic BP	2	120.41 (12.31)	124.60 (12.28)	131.32 (15.74)
Systolic BP	3	122.55 (10.10)	133.06 (11.72)	127.32 (5.44)
Diastolic BP	1	75.89 (9.27)	75.85 (9.15)	74.16 (11.27)
Diastolic BP	2	72.31 (8.57)	70.66 (5.88)	73.73 (9.99)
Diastolic BP	3	74.27 (4.21)	71.05 (4.44)	74.72 (5.28)
HR	1	65.24 (9.75)	60.42 (8.86)	62.80 (9.56)
HR	2	64.32 (8.78)	59.16 (7.14)	62.48 (6.91)
HR	3	67.63 (7.77)	58.24 (5.80)	66.05 (3.41)

Note. EE: emotion expression group; NS: natural sharing group; CTRL: control group; *M*: mean; *SD*: standard deviation;

A small and marginally significant group by time effect was observed for systolic BP [$F(3.70, 181.32) = 2.71$; $p = .035$, $\eta^2 = .05$] and for HR [$F(3.66, 179.49) = 3.06$; $p = .02$, $\eta^2 = .05$], but not for diastolic BP [$F(3.92, 191.85) = 1.94$; $p = .11$, $\eta^2 = .03$], suggesting possible between-group differences in time changes of systolic BP and HR. Of note, the Huynh-Feldt correction was applied to degrees of freedom.

A series of repeated measures ANOVAs were conducted separately for each group. Results showed that there was a significant time effect on systolic BP in the NS group [$F(2,66) = 5.67, p = .005, \eta^2 = 0.15$] and on HR in the CTRL group [$F(1.56, 49.82) = 5.74, p = .01, \eta^2 = 0.15$]. *Post hoc* tests showed that between the second and the third interview systolic BP increased for the NS group ($p = .003$), as did HR in the CTRL group ($p = .005$). Of note, a Greenhouse-Geisser correction was applied. No such time changes were observed for the EE group ($ps > .05$). This partially confirms hypotheses 2_a and 2_c. Although systolic BP and HR did not significantly decrease in the EE group, a possible protective effect of emotion expression observed.

Hypothesis 2_b of significant benefits of the intervention for diastolic BP was not confirmed.

7.3.1.3. Summary.

After testing the hypotheses related to the first study aim, which were predicting the benefits of the intervention for distress, affect, and BP and HR, it was found that although none of the distress and affect variables were affected by the intervention, systolic BP and HR in the EE group did not change in the present study, while in the other two groups those indices increased slightly. This may suggest potential benefits of social sharing with emotion expression on systolic BP and HR.

7.4. Mediators and Moderators of Effects (Aim 2)

The second aim of the study was to investigate possible moderators and mediators of the intervention effects on distress and affect (see Fig. 8, p.148). The analysis was conducted to investigate theoretically novel effects among individuals characterised by a tendency to inhibit their negative emotions, either deliberately (e.g., Type D personality), or due to

incapability to indentify and express them. Conservative views on testing mediation/moderation in the face of null main effects of the intervention (Baron & Kenny, 1986) were criticised for low power (e.g., Fritz & MacKinnon, 2007), as well as for the fact that the mediation effect, instead of being quantified, is logically inferred from the existence of the main effect (Hayes, 2009).

Conditional process analysis results are presented below. This analysis is based on the “as treated” data.

The following results tested hypotheses 3_a to 3_f.

7.4.1. Moderation analysis (Time 2; N = 81): the direct effect of group x moderator interaction on outcome.

At Time 2, neither Type D nor Alexithymia moderated the direct effect of the intervention (Tables 22 and 23), unsupportive of the hypotheses 3_a and 3_b.

Table 22
Direct Effect of Group on Outcome Variables at Time 2 Moderated by Type D

Outcome	β	$t(73)$	p	<i>LLCI</i>	<i>ULCI</i>
Anxiety	0.44	0.43	.67	- 1.57	2.44
Depression	- 0.81	- 0.91	.36	- 2.59	0.96
General Distress	- 0.47	- 0.28	.78	- 3.84	2.89
Perceived Stress	- 0.50	- 0.22	.83	- 5.09	4.08
Negative Affect	- 0.39	- 0.34	.73	- 2.70	1.91
Positive Affect	1.21	1.02	.31	- 1.17	3.59

Note. β = unstandardized beta weight; t = t-test statistic, *LLCI*: lower level 95% confidence interval; *ULCI* = upper level confidence intervals

Table 23

Direct Effect of Group on Outcome Variables at Time 2 Moderated by Alexithymia

Outcome	β	$t(73)$	p	<i>LLCI</i>	<i>ULCI</i>
Anxiety	0.35	0.57	.57	- 0.87	1.58
Depression	0.49	0.91	.37	- 0.59	1.58
General Distress	0.66	0.64	.52	- 1.40	2.71
Perceived Stress	1.10	0.78	.44	- 1.70	3.89
Negative Affect	0.15	0.21	.83	- 1.25	1.56
Positive Affect	- 0.12	- 0.17	.87	- 1.57	1.33

Note. β = unstandardized beta weight; t = t-test statistic, *LLCI*: lower level 95% confidence interval; *ULCI* = upper level confidence intervals

7.4.2. Mediation analysis (Time 2; N = 81).

The next two sections test the mediation of rumination change and reappraisal change of the intervention effects on psychological outcomes.

7.4.2.1. The effect of group and of the group x moderator interaction on rumination change.

Although group did not significantly predict rumination change [$\beta = - 0.28$, $t(75) = - 0.85$, $p = .40$, *CI*s: - 0.95, 0.38], the Type D x group interaction had an effect on the mediator [$\beta = 1.15$, $t(75) = 2.03$, $p = .045$, *CI*s: 0.23, 2.28]; decreases in rumination were the greatest among Type Ds in the EE group and the smallest among Type Ds in the control group.

Alexithymia did not moderate the group effect on the mediator [$\beta = 0.56$, $t(75) = 1.61$, $p = .11$, *CI*s: - 0.13, 1.25].

7.4.2.2. The indirect effect of the group $\times$ rumination change interaction on outcomes.

Rumination change predicted post-intervention anxiety, general distress and perceived stress, but not positive or negative affect (Table 24). The effect of rumination change on post-intervention depression was almost significant at $p = .05$. In all cases increases in rumination were associated with increases in distress. Further analysis showed that rumination change predicts these outcomes only for Type D participants who were also classified as being Alexithymics or possible Alexithymics (Tables 25-28).

These findings support hypothesis 3_c for post-intervention (Time 2) distress, but not for affect variables.

Table 24
The Effect of Rumination Change on Outcome Variables at Time 2

Outcome	β	$t(73)$	p	<i>LLCI</i>	<i>ULCI</i>
Anxiety	0.46	2.30	.02	0.06	0.86
Depression	0.35	1.96	.05	- 0.01	0.70
General Distress	0.75	2.22	.03	0.08	1.42
Perceived Stress	1.12	2.44	.02	0.21	2.03
Negative Affect	0.28	1.20	.23	- 0.18	0.73
Positive Affect	- 0.02	- 0.06	.95	- 0.49	0.46

Note. β = unstandardized beta weight; t = t-test statistic, *LLCI*: lower level 95% confidence interval; *ULCI* = upper level confidence intervals

Table 25

Conditional Indirect Effect of Group on Time 2 Anxiety at Values of the Moderators

Alexithymia	Type D	Effect	BootLLCI	BootULCI
No	No	- 0.13	- 0.63	0.10
No	Yes	0.40	- 0.03	1.58
Possible	No	0.04	- 0.25	0.60
Possible	Yes	0.57	0.07	1.60
Yes	No	0.25	- 0.19	1.39
Yes	Yes	0.78	0.07	1.91

Note. BootLLCI: bootstrapped lower level confidence intervals; BootULCI: bootstrapped upper level confidence intervals

Table 26

Conditional Indirect Effects of Group on Time 2 Distress at Values of the Moderators

Alexithymia	Type D	Effect	BootLLCI	BootULCI
No	No	- 0.21	- 1.15	0.16
No	Yes	0.65	- 0.07	2.54
Possible	No	0.06	- 0.46	0.89
Possible	Yes	0.92	0.07	2.62
Yes	No	0.40	- 0.38	2.05
Yes	Yes	1.26	0.08	3.17

Note. BootLLCI: bootstrapped lower level confidence intervals; BootULCI: bootstrapped upper level confidence intervals

Table 27
Conditional Indirect Effects of Group on Time 2 Perceived Stress at Values of the Moderators

Alexithymia	Type D	Effect	BootLLCI	BootULCI
No	No	- 0.32	- 1.51	0.27
No	Yes	0.97	- 0.06	3.49
Possible	No	0.09	- 0.63	1.15
Possible	Yes	1.38	0.31	3.85
Yes	No	0.60	- 0.52	2.88
Yes	Yes	1.89	0.38	4.48

Note. BootLLCI: bootstrapped lower level confidence intervals; BootULCI: bootstrapped upper level confidence intervals

Table 28
Conditional Indirect Effects of Group on Time 2 Depression at Values of the Moderators

Alexithymia	Type D	Effect	BootLLCI	BootULCI
No	No	- 0.10	- 0.52	0.08
No	Yes	0.30	- 0.02	1.26
Possible	No	0.03	- 0.24	0.38
Possible	Yes	0.43	0.05	1.30
Yes	No	0.19	- 0.17	0.98
Yes	Yes	0.59	0.06	1.52

Note. BootLLCI: bootstrapped lower level confidence intervals; BootULCI: bootstrapped upper level confidence intervals

7.4.2.3. The effect of group and the group x moderator interaction on reappraisal change.

Hypothesis 3_d was not supported in this study. Neither the group [$\beta = 0.64$, $t(75) = 0.47$, $p = .64$, $CIs: - 2.11, 3.40$] nor the group x moderator interaction [group x Type D: $\beta = 0.90$, $t(75) = 0.38$, $p = .70$, $CIs: - 3.78, 5.57$; group x Alexithymia: $\beta = -1.20$, $t(75) = - 0.83$, $p = .41$, $CIs: - 4.06, 1.67$] predicted changes in reappraisal, suggesting that changes in reappraisal did not mediate the effect of group on outcomes. Mediation of reappraisal change on the intervention effects at Time 3 was therefore not tested further.

7.4.3. Moderation analysis (Time 3; N = 59); the effect of the group x moderator interaction on outcomes.

Although Type D did not moderate the intervention effects on outcomes at follow-up (Table 29), Alexithymia moderated the effect of the intervention on perceived stress and negative affect (Table 30). Perceived stress and negative affect decreased the most in the EE group; however, only in participants who had Alexithymia, but not Type D personality (see Tables 31 and 32). This confirms hypothesis 3_b for Time 3 perceived stress and negative affect. Hypothesis 3_a for follow-up outcomes was not supported.

Table 29

Direct Effect of Group on Outcome Variables at Time 3 Moderated by Type D

Outcome	β	$t(51)$	p	<i>LLCI</i>	<i>ULCI</i>
Anxiety	- 0.41	- 0.27	.79	- 3.51	2.69
Depression	- 0.74	- 0.61	.54	- 3.16	1.68
General Distress	- 1.15	- 0.44	.66	- 6.37	4.06
Perceived Stress	- 4.10	- 1.31	.20	- 10.37	2.17
Negative Affect	- 2.11	- 1.37	.18	- 5.22	0.99
Positive Affect	- 1.20	- 0.92	.36	- 3.84	1.44

Note. β = unstandardized beta weight; t = t-test statistic, *LLCI*: lower level 95% confidence interval; *ULCI* = upper level confidence intervals

Table 30

Direct Effect of Group on Outcome Variables at Time 3 Moderated by Alexithymia

Outcome	β	$t(51)$	p	<i>LLCI</i>	<i>ULCI</i>
Anxiety	1.79	1.59	.12	- 0.46	4.03
Depression	1.71	1.95	.06	- 0.05	3.46
General Distress	3.49	1.85	.07	- 0.29	7.28
Perceived Stress	5.07	2.24	.03	0.52	9.62
Negative Affect	2.38	2.12	.04	0.13	4.64
Positive Affect	- 0.16	- 0.17	.87	- 2.07	1.75

Note. β = unstandardized beta weight; t = t-test statistic, *LLCI*: lower level 95% confidence interval; *ULCI* = upper level confidence intervals

Table 31

Conditional Direct Effect of Group on Time 3 Negative Affect at Values of the Moderators

Alexithymia	Type D	Effect	<i>t</i>	<i>p</i>	BootLLCI	BootULCI
No	No	- 0.09	- 0.11	.91	- 1.66	1.49
No	Yes	- 2.20	- 1.44	.16	- 5.28	0.88
Possible	No	1.12	1.38	.17	- 0.51	2.76
Possible	Yes	- 0.99	- 0.78	.43	- 3.52	1.54
Yes	No	2.91	2.10	.04	0.13	5.70
Yes	Yes	0.80	0.62	.54	- 1.78	3.38

Note. *t*: t-test statistic; BootLLCI: bootstrapped lower level confidence intervals; BootULCI: bootstrapped upper level confidence intervals

Table 32

Conditional Direct Effect of Group on Time 3 Perceived Stress at Values of the Moderators

Alexithymia	Type D	Effect	<i>t</i>	<i>p</i>	BootLLCI	BootULCI
No	No	- 0.28	- 0.18	.86	- 3.46	2.90
No	Yes	- 4.38	- 1.41	.16	- 10.60	1.83
Possible	No	2.30	1.39	.17	- 1.00	5.60
Possible	Yes	- 1.80	- 0.71	.48	- 6.91	3.31
Yes	No	6.11	2.18	.03	0.48	11.74
Yes	Yes	2.01	0.77	.44	- 3.20	7.22

Note. *t*: t-test statistic; BootLLCI: bootstrapped lower level confidence intervals; BootULCI: bootstrapped upper level confidence intervals

7.4.4. Mediation analysis (Time 3; N = 59); the indirect effect of group and of the group x rumination change interaction on outcomes.

At Time 3, rumination change did not predict any of the outcomes (Table 33) therefore hypothesis 3_c for Time 3 outcomes was not supported.

Table 33
The Effect of Rumination Change on Outcome Variables at Time 3

Outcome	β	$t(73)$	p	<i>LLCI</i>	<i>ULCI</i>
Anxiety	0.22	0.71	.48	- 0.41	0.85
Depression	0.37	1.50	.14	- 0.12	0.86
General Distress	0.59	1.12	.27	- 0.47	1.65
Perceived Stress	0.31	0.50	.62	- 0.96	1.58
Negative Affect	0.49	1.57	.12	- 0.14	1.12
Positive Affect	0.17	0.65	.52	- 0.36	0.71

Note. β = unstandardized beta weight; t = t-test statistic, *LLCI*: lower level 95% confidence interval; *ULCI* = upper level confidence intervals

7.4.5. Summary.

The analysis of the possible moderators and mediators of the intervention effects on distress and affect showed that Type D individuals in the EE group benefited the most in terms of decreases in rumination, which in turn predicted lower post-intervention anxiety, general distress and perceived stress, and a trend towards lower depression. There was therefore a Type D-moderated indirect (rumination change-mediated) effect of the intervention on those distress variables. Further analysis suggested that those effects were present only for those Type D participants who were also classified as Alexithymics or possible Alexithymics. At the 3-months follow-up, Alexithymia was found to moderate the direct effect of group on

perceived stress and negative affect, which decreased the most in the EE group; however, this effect was present only in participants who had Alexithymia, but were not Type Ds.

Changes in reappraisal did not mediate the intervention effects in this study.

These results should be treated with caution due to small sample size (“as treated” data) and multiple paths’ testing, both of which increase the possibility of Type I error.

7.5. Chapter Summary

On average, participants in the present study were high in negative affectivity, a third of them were distressed and 15% had an elevated level of depression. A third of them had Type D personality, and less than a third had Alexithymia. Their baseline BP and HR were in the normal range in all three groups.

No intervention effects were observed for any of the psychological variables; however, small increases in BP and HR were observed in the control group and the natural sharing group, but not in the emotion expression group. This may suggest potential benefits of social sharing with emotion expression on physiological indices.

The results of the mediation/moderation analysis suggested that, in the emotion expression group, Type D individuals, who were also high in Alexithymia, had lower post-intervention anxiety, general distress and perceived stress, and a trend towards lower depression, than participants in the other groups. These effects were mediated by pre- to post-intervention decreases in rumination. At the 3-months follow-up, participants in the emotion expression group, who were high in Alexithymia, but not Type D, had lower perceived stress and negative affect, than those in the other study groups. These results should however be treated with caution due the possibility of Type I error (small sample size and multiple paths’ testing).

Results II

Sharing Groups – Analysis of Language Use and Physiological Indices during Sharing Episodes

This chapter summarises the results of an ITT analysis of language use, as well as the ITT analysis of HR_f and HRV, and associations between those variables, addressing the third and the fourth study aims.

8.1. Differences between both sharing groups in language use, and in physiology during sharing (Aim 3)

This section investigates the hypotheses and questions relating to the third study aim – examination of the between-group differences in language use and physiology during sharing episodes. This analysis is based only on the data from the EE and NS groups.

8.1.1. Differences in language use between EE and NS groups.

This section illustrates the between-group difference in the use of emotion words and cognitive words. Data for each category are presented as a percentage of the total word use per session.

8.1.1.1. *Emotion words.*

Average use of emotion words at each meeting is presented in Table 34.

Section 7.2. (outlining the manipulation check) presented group differences in the overall use of emotion words.

Results showed that, on average, the EE group used more negative emotion words, anxiety words and anger words than the NS group. There were also marginally significant differences in the use of sadness words (group EE used more of them) and positive emotion words (NS group used more). This partially confirmed hypothesis 4_a for emotion words.

No group x time effects were observed for any word categories, therefore hypothesis 4_b for emotion words was not supported.

Table 34
Percentages of Emotion Words Used by Groups at Each Interview

Word Category	Group	$M_1 (SD_1)$	$M_2 (SD_2)$	$M_3 (SD_3)$
Affect	EE	4.07 (0.55)	3.99 (0.61)	4.11 (0.73)
	NS	4.11 (0.85)	3.66 (0.79)	4.25 (1.09)
Positive Emotion	EE	2.06 (0.43)	2.17 (0.55)	2.20 (0.46)
	NS	2.15 (0.59)	2.27 (0.58)	2.58 (0.73)
Negative Emotion	EE	2.08 (0.44)	1.96 (0.43)	1.88 (0.61)
	NS	1.97 (0.56)	1.54 (0.45)	1.77 (0.55)
Anxiety	EE	0.59 (0.30)	0.60 (0.35)	0.46 (0.28)
	NS	0.54 (0.35)	0.38 (0.28)	0.39 (0.29)
Anger	EE	0.54 (0.19)	0.49 (0.24)	0.51 (0.23)
	NS	0.38 (0.18)	0.35 (0.21)	0.39 (0.34)
Sadness	EE	0.26 (0.13)	0.27 (0.17)	0.34 (0.20)
	NS	0.23 (0.15)	0.19 (0.13)	0.28 (0.24)

Note. EE – emotion expression group, NS – Natural Sharing group; M_1 , M_2 , M_3 : mean word use at meeting 1, 2, or 3; SD_1 , SD_2 , SD_3 : standard deviation at meeting 1, 2, or 3.

8.1.1.2. Cognitive words.

Mean percentages of cognitive word use are presented in Table 35.

Results showed statistically significant group effects for all these categories [*cognitive mechanisms*: $F(1) = 8.53, p = .005, \eta^2 = .11$], *insight*: $F(1) = 13.45, p < .001, \eta^2 = .17$; *cause*: $F(1) = 19.09, p < .001, \eta^2 = .22$]. In line with hypothesis 4_a for cognitive words, participants in the EE group used more *cognitive mechanisms* words ($M_{EE} = 18.09$ vs $M_{NS} = 17.23$), more *insight* words ($M_{EE} = 2.22$ vs $M_{NS} = 1.84$) and more *cause* words ($M_{EE} = 1.36$ vs $M_{NS} = 1.04$) than participants in the NS group.

No group x time interaction effects were observed for any of the cognitive word categories, therefore hypothesis 4_b for cognitive words was not supported.

Table 35
Percentages of Cognitive Words Used by Groups over Time

Word Category	Group	$M_1 (SD_1)$	$M_2 (SD_2)$	$M_3 (SD_3)$
<i>Cognitive Mechanism</i>	EE	17.57 (1.21)	18.19 (1.49)	18.51 (1.74)
	NS	16.63 (1.83)	17.52 (1.31)	17.55 (1.57)
<i>Insight</i>	EE	2.12 (0.55)	2.20 (0.60)	2.34 (0.51)
	NS	1.87 (0.64)	1.91 (0.60)	1.75 (0.46)
<i>Cause</i>	EE	1.37 (0.33)	1.33 (0.44)	1.38 (0.48)
	NS	1.00 (0.33)	1.07 (0.35)	1.06 (0.42)

Note. EE – emotion expression group, NS – Natural Sharing group; M_1, M_2, M_3 : mean word use at meeting 1, 2, or 3; SD_1, SD_2, SD_3 : standard deviation at meeting 1, 2, or 3.

8.1.2. HR_f and HRV analysis.

This section presents the results of a between-group comparison of HR_f and HRV during sharing episodes. The following HRV indexes were analysed: SDNN (a standard deviation of all normal RR intervals (measured between consecutive sinus beats), HRV triangular index, and RMSSD (the square root of the mean of the squared differences between successive NN

intervals). Table 36 presents mean group values of these physiological measurements at each sharing interview. Results of 2 x 3 ANOVAs showed no group x time interaction effects for mean HR_f, or for SDNN, RMSSD, or HRV triangular index (all $ps > .05$), therefore hypotheses 5_a and 5_b were not supported.

Table 36
Descriptive Statistics for HR_f and HRV by EE and NS group over Time

Measurement	Group	$M_1 (SD_1)$	$M_2 (SD_2)$	$M_3 (SD_3)$
Mean HR _f	EE	81.41 (13.40)	85.33 (12.35)	84.51 (11.91)
	NS	76.18 (11.79)	77.29 (11.32)	75.43 (12.12)
SDNN	EE	98.18 (22.81)	99.15 (24.38)	96.05 (23.01)
	NS	91.71 (20.61)	100.42 (22.19)	92.68 (14.29)
RMSSD	EE	123.07 (14.69)*	122.97 (14.75)*	120.88 (8.25)*
	NS	126.61 (24.41)*	130.65 (15.69)*	124.29 (11.28)*
Triangular index	EE	21.48 (6.19)	23.18 (6.28)	21.52 (5.54)
	NS	20.10 (6.40)	21.34 (4.87)	18.84 (4.28)

Note. *Median (Interquartile Range); EE: emotion expression group; NS: natural sharing group; M : mean; SD : standard deviation; HR_f: heart rate, as measured by finger pulse; SDNN: standard deviation of normal RR intervals; RMSSD: square root of the mean of the squared differences between successive NN intervals

Despite null statistical results, raw scores suggested potential clinically relevant group differences in HRV. Using the recommended cut-off point to distinguish between low-HRV (< 20 units) and high-HRV (≥ 20 units) at each interview, the data were dichotomously categorised. Frequencies of participants with low versus high HRV, separately for each group and each interview, are presented in Table 37. Results of non-parametric analyses using Chi-squared tests showed a marginally significant association between group and HRV category at the third interview [$\chi^2(1, 68) = 4.77, p = .015$], suggesting that there were more low-HRV participants in the NS group than in the EE group.

Table 37

Low-HRV vs. High-HRV at Each Sharing Interview by Group

HRV		Interview 1	Interview 2	Interview 3
EE group	Low-HRV	16 (47.1%)	7 (20.6%)	13 (38.2%)
	High-HRV	18 (52.9%)	27 (79.4%)	21 (61.8%)
NS group	Low-HRV	18 (52.9%)	9 (26.5%)	22 (64.7%)
	High-HRV	16 (47.1%)	25 (73.5%)	12 (35.3%)

Note. EE: emotion expression group; NS: natural sharing group; HRV: heart rate variability

8.1.3. Summary.

The above sections illustrated the between-group differences in language use and physiological indexes during sharing.

In line with the hypotheses, the EE group used more emotion and cognitive words than the NS group, however no differences were observed between the groups in changes in language use.

With respect to physiology, hypotheses were not supported, however at the last interview there were more low-HRV participants in the NS group, than in the EE group, suggesting potential clinically relevant benefits of emotion expression on HRV.

8.2. Association between Changes in Language Use and Changes in Outcome Variables (Aim 4).

To test whether changes in language use were associated with improvements in psychological outcome variables, change scores ($\text{Interview}_3 - \text{Interview}_1$) were computed for each language category and correlated with short-term ($\text{Time}_2 - \text{Time}_1$) and long-term ($\text{Time}_3 - \text{Time}_1$) changes in distress and affect variables, using Pearson correlations, separately for

each sharing group. Correlation matrices for long-term effects are presented in Tables 38 - 41. Correlation matrices for short-term effects are presented Appendix 4.

8.2.1. Emotion words.

In line with the hypothesis 6_a, in the EE group, over-time decreases in the use of anxiety words were related to long-term decreases in anxiety. Contradicting the hypothesis, however, *increases* in the use of sad words were related to short-term and long-term increases in positive affect, as well as long-term decreases in depression, general distress and perceived stress, in the EE group. In other words, while a decrease in anxiety words' use was beneficial for psychological outcomes, increases in the use of sadness words were associated with benefits in the EE group. In the NS group, increase in the use of sadness words was indeed related to short-term and long-term decreases in positive affect, and increased use of negative emotion words in general was related to short-term increase in depression, in line with the hypothesis.

Changes in anger words' use were unrelated to distress or affect in both groups.

Supporting hypothesis 6_b, increases in the use of positive emotion words were associated with short-term increases in positive affect, however only in the EE group.

8.2.2. Cognitive words.

Supporting hypothesis 6_c, significant correlations with psychological outcomes were observed for cause words in the EE group: the increase in the use of cause words was related to long-term decrease in anxiety, depression, general distress and negative affect.

In the NS group, unexpectedly, increases in the use of each cognitive category words were related either to short-term decrease in positive affect or increase in depression and in general distress, contrary to what was hypothesised (see Appendix 4).

Table 38

Correlations between Changes in Emotion Words and Long-term Changes in Distress in the EE Group

Variable	1	2	3	4	5	6	7	8	9	10
1. Anxiety	---									
2. Depression	.585**	---								
3. General Distress	.897**	.884**	---							
4. Positive affect	-.315	-.425*	-.408*	---						
5. Negative affect	.430*	.556**	.547**	-.081	---					
6. Perceived stress	.477*	.434*	.508**	-.395*	.605**	---				
7. Positive emotion words	-.155	.055	-.052	.212	.298	.377	---			
8. Negative emotion words	-.062	-.094	-.075	.020	-.119	-.039	.323	---		
9. Anxiety words	.461*	.099	.321	.183	.152	.316	.153	.505**	---	
10. Anger words	.058	-.097	.002	-.234	-.192	-.100	.009	.682**	.228	---
11. Sad words	-.388	-.396*	-.442*	.407*	-.616**	-.546**	.192	.384*	.103	.087

* $p < .05$, ** $p < .01$ (one-tailed)

Table 39

Correlations between Changes in Emotion Words and Long-term Changes in Distress in the NS Group

Variable	1	2	3	4	5	6	7	8	9	10
1. Anxiety	---									
2. Depression	.106	---								
3. General Distress	.829**	.645**	---							
4. Positive affect	.358*	-.285	.115	---						
5. Negative affect	.007	.348	.201	-.159	---					
6. Perceived stress	.349	.682**	.584**	-.057	-.002	---				
7. Positive emotion words	.285	.220	.355	.124	-.235	-.139	---			
8. Negative emotion words	-.030	.072	.010	-.110	-.021	.055	.051	---		
9. Anxiety words	-.302	.021	-.249	-.123	-.137	.192	-.081	.440*	---	
10. Anger words	.188	-.132	.095	.025	-.160	-.232	.248	.548**	.036	---
11. Sad words	-.213	.237	-.063	-.467*	.218	-.032	.105	.160	-.108	-.062

* $p < .05$, ** $p < .01$ (one-tailed)

Table 40

Correlations between Changes in Cognitive Words and Long-term Changes in Distress in the EE Group

Variable	1	2	3	4	5	6	7	8
1. Anxiety	---							
2. Depression	.585**	---						
3. General Distress	.897**	.884**	---					
4. Positive affect	-.315	-.425*	-.408*	---				
5. Negative affect	.430*	.556**	.547**	-.081	---			
6. Perceived stress	.477*	.434*	.508**	-.395*	.605**	---		
7. Cognitive mechanisms words	.065	-.051	-.013	-.230	-.287	.037	---	
8. Insight words	-.259	-.364	-.359	-.165	-.115	-.039	.360*	---
9. Cause words	-.463*	-.395*	-.520*	-.226	-.636**	-.263	.353*	.175

* $p < .05$, ** $p < .01$ (one-tailed)

Table 41

Correlations between Changes in Cognitive Words and Long-term Changes in Distress in the NS Group

Variable	1	2	3	4	5	6	7	8
1. Anxiety	---							
2. Depression	.106	---						
3. General Distress	.829**	.645**	---					
4. Positive affect	.358*	-.285	.115	---				
5. Negative affect	.007	.348	.201	-.159	---			
6. Perceived stress	.349	.682**	.584**	-.057	-.002	---		
7. Cognitive mechanisms words	-.145	-.134	-.192	-.314	.110	-.165	---	
8. Insight words	-.164	.185	-.048	-.238	.250	-.081	.529**	---
9. Cause words	-.278	.074	-.201	-.459*	.206	.159	.472*	.103

* $p < .05$, ** $p < .01$ (one-tailed)

8.2.3. Summary.

There were a number of significant associations between changes in language use and changes in outcome variables.

The hypothesis regarding the benefits of an over-time decrease in the use of negative emotion words was only partially supported. While in the NS group, increase in the use of sadness words and general negative emotion words was indeed related to worse psychological functioning, mixed results were observed in the EE group. While increases in anxiety words predicted higher distress in that group, increased use of sadness words seemed beneficial, as it was related to decreases in distress and increases in positive affect, even 3 months post-intervention.

In line with the hypothesis, increases in positive emotion words were associated with positive affect, however only in the EE group. Similarly, only in the EE group did increased cause words' use predict a decline in distress, supporting the hypothesis. Unexpectedly, however, in the NS group increases in the use of cognitive words were related with more distress.

8.3. Chapter Summary

The results presented in this chapter confirmed the hypothesis that emotion expression participants would express more emotion words and more cognitive words than participants in the natural sharing group. Interestingly, however, the association between negative emotion words use and psychological outcome variables was not as straightforward as predicted. Increased use of sadness words and negative emotion words was linked to worse psychological functioning only in the natural sharing group. In the emotion expression group increased use of sadness words was related to decreases in distress and increases in positive affect, even 3 months post-intervention. At the same time, however, increases in anxiety words predicted higher distress in that group. Furthermore, confirming the hypothesis, increased use of positive emotion and cause words

predicted better psychological functioning, although these effects were only observed in the emotion expression group. In the natural sharing group increased use of cognitive words was associated with worse psychological functioning.

With respect to the analysis of physiological indices, although hypotheses were not supported, it was observed that at the last interview there were more low-HRV participants in the natural sharing group, than in the emotion expression group, suggesting potential clinically relevant benefits of emotion expression on HRV.

Qualitative Analysis of Narratives

This chapter presents the topics that were discussed by participants in both sharing group, and illustrates how different participants (Type Ds, Alexithymics, and others) dealt with the instruction to express their emotions. It also presents how deep and emotional some of the narratives were.

Qualitative analysis is based on data from 58 completed narratives. The remaining 10 participants either did not engage in sharing (i.e., withdrew after Time 1 assessment; $N = 2$) or only took part in one sharing session ($N = 8$). This qualitative analysis is exploratory and not included in the main study model.

9.1. General Overview

Table 42 presents typed of topics discussed by participants and their prevalence among the sample. One topic that was discussed by all participants was their MI (or CHD), although only in 24 cases this was the first topic discussed. Other most common topics participants started with were: father's death (7), unemployment (5), divorce/separation (3), child's drug addiction (2), mother's death (2), marital problems (2), and alcoholic father (2), as well as topics involved with child/grandchild's death/illness, alcoholic husband, mother's illness, own illness (other than CHD), childhood illness, accidents, being assaulted, and financial hardship.

Table 42

Topics Discussed by Participants in Both Sharing Groups

Topic discussed	N (%)
MI/Own CHD/stents	58 (100)
Father's death (1 in childhood)	23 (40%)
Mother's death (1 in childhood)	18 (31%)
Chronic disease/Serious illness	18 (31%)
Child's serious illness/disability/accident	16 (28%)
Sibling's death (1 in childhood)	15 (26%)
Spouse/parent/sibling's serious illness	15 (26%)
Financial hardship	15 (26%)
General worries about work/future	11 (19%)
Divorce/separation/	8 (14%)
Close one's death	8 (14%)
Being unemployed/Lost his job	8 (14%)
Marital problems	7 (12%)
Anger at current economic crisis/system	7 (12%)
Depression/anxiety/addiction	7 (12%)
Child's death (incl. miscarriage, N=2)	6 (10%)
Serious conflict with parent/child/sibling	6 (10%)
Accident at work	5 (9%)
Traumatic school experiences	4 (7%)
Physically assaulted by a stranger	4 (7%)
Child drug addict	4 (7%)
Victim of bullying	4 (7%)
Grandchild's death/serious illness	3 (5%)
Spouse's death	3 (5%)
Husband alcoholic	3 (5%)
Raising a grandchild	3 (5%)
House problems (flood, break-in)	3 (5%)
Legal matters	3 (5%)
Child sexually abused in childhood by a family member/child bullied	2 (3%)
Father alcoholic	2 (3%)
Family break-up in childhood/Living with abusive stepmother	2 (3%)
Life threatening disease in childhood	2 (3%)
Relationship break-up	2 (3%)
Being raped in childhood	1 (2%)
War	1 (2%)

Note. N: number of participants; %: percentage of the whole sample

The following overview of the narratives illustrates how participants described their cardiac event, how they initially reacted to it, and the process they went through during weeks following the event. It also shows that whether they had Type D/Alexithymia or not, participants often waited with seeing their doctor hours or days after the first symptoms occurred and most did not discuss their feelings regarding the event. Other life events that participants discussed are also presented in this section to show the depth people went into when sharing.

All the names have been changed and every name is unique.

9.2. Discussing the Recent Cardiac Event

When discussing their cardiac event, participants often described it with great amount of detail, whether they were Type Ds, or they had Alexithymia, or neither of those traits. Some waited hours or even days after the first symptoms, before visiting a doctor or going to the hospital. The first example is of Seamus, 34 year old male from the EE group:

“I got out of the shower and I rang a friend, and my exact words to my friend were “John, I’m having a heart attack”. But only in a messing way and John said “no, you haven’t cycled, you have to remember you haven’t cycled in two months, it’s just your lungs opening up, you’ll be grand”, and I said “yeah”(…) On the Sunday night, I was making a cup of tea actually for the wife and... I felt the arm starting to lock up again. (...) I sat down never did anything about it, and then obviously went to bed. Four o’clock in the morning... Monday morning 4 o’clock, I was awoken from the sleep. (...) I got up because the pain was unbearable... and what seemed to relieve the pain for me is if I put my hands over my head... I went back to bed, fell asleep, woke up at half seven, I had a wash and all... and I was on the way out to work and I felt the pain coming again and I put the cup down on the mantelpiece and I dropped onto the floor. Now, I came through within seconds and... I realised that I

was actually on the phone to my mom asking her to get me an ambulance even though my wife was downstairs.”

Similarly, Patrick (age 65, EE group; Type D and possible Alexithymia) also kept the symptoms to himself for days:

“I was going for a walk... where I normally walk and I started to have a bit of a pain, I said “it’s nothing”, walked on further and further, and I, to where I normally stop and I said “I’ll head back to the car” (...) I stopped ever so often had to pretend I was admiring the view to get me breath back. (...) The next day, fine. (...) Friday morning... I got straight up out of bed. And soon as I stood up... a severe pain radiated around from... the heart right around the left side and round the back... So I woke my wife up and I said, I think we better call the ambulance because I don’t feel great.”

Patrick did not express any emotions, whereas Seamus was additionally asked to express them and he said:

“I was panicked. (...) Panic, pure panic.”

Some participants tried to relieve the MI pain with painkillers, which seemed to work at the moment, however as the pain returned they realised it had to be more serious than they had thought. Sean (age 49; EE; Type D).

“On the Tuesday I woke up with pains in both arms and I thought it was just the way I had been sleeping and I didn’t take any more notice of it. It passed with a couple of pain killers. And then on the Friday the same pain came and this was at half 9 at night so I knew it wasn’t anything to do with sleeping, and then pain came across the chest. I was on my own in the house at the time for about 10 mins and then when the wife came in with the daughter I asked her to ring an ambulance.”

Sean later said:

“I was nervous, a little how can I put it... apprehensive. (...) Afraid. Basically afraid of what was happening, and knowing what was happening”

Cian (age not recorded; EE; Type D and possible Alexithymia) would not let his family know he was having a heart attack until he was sure the situation was serious:

I never told my missus. As I said to you, I never told her. Until the last minute. Until was lying on the stretcher down... like in, in the, I was downstairs wasn't I. (...) I wouldn't, I even told them not to ring her. See that's what I'm saying when I was, when I was lying on the stretcher, with the ambulance fellas they, the, putting all the stuff on you, the drips and all that. Your man said “you have to ring your family”. And I said “no listen, just do what you have to do and I'll ring her when it's finished”. “No” he said, “you have to ring”. (...) I still wouldn't tell her until I knew exactly. (...) I wouldn't... put pressure on her, by stressing her by saying.”

When asked about his emotions at that stage he said:

“Emotion-wise... I wouldn't have been... I was thinking about it alright but it was never, I wasn't, I was a little bit was worried”

There were those who sought help almost immediately, as they recognised the seriousness of the symptoms. Peter (age 49; NS) said:

“I felt a kind of a bit tight in my chest. And I thought it might be indigestion from kind of eating. ... I went outside to get a bit of air, it didn't really make any difference and I went into the bedroom. I didn't say anything to Ann, she was sitting there watching the telly and then I just lay down... oh no that doesn't feel good you know so I sat back up again. And then I started getting pins and needles down one arm and then down the other... It wasn't getting any worse it was like there was a belt

about that thick underneath my ribcage just being tightened but not unbearable ... just a little bit uncomfortable and so I went in to the bathroom, my first thought would be I'm either having a heart attack or a stroke and I went in to look at my face... but my face was white... And the sweat running down my face and so I just went in and said it to her. "Look I'm going to go to the hospital, I might drive or maybe you drive me" She said "no call an ambulance"“

The experience of an MI was described as very painful by Kevin (age not recorded; EE)

“I knew I had horrendous pain but I still didn't know because I don't know what to expect from a heart attack. I thought I was a goner. I thought I won't come out of this. I thought if I do there's certain things I will do (...) For a while, for a certain amount of time, I thought, okay, I'm ready. But then I said, no hold on, I'm not ready, I want to live a bit longer. This happens now, whether it was 2 minutes, 1 minute, 10 seconds, I don't know because all this goes through ... initially I thought well if it's to be it's to be and then I said, no, no I want to be around. I want to live a bit longer and it started coming into my mind 'Now I need to fight this'. But initially it was kind of I'll just lie back and whatever happens happens.”

He also admitted that he was afraid at that moment:

“I realised then and I was then panicking. (...) Initially kind of fear, a small bit of fear”.

Other participants from the EE group expressed their disappointment and annoyance, as well as helplessness. Frank (age 50; EE; possible Alexithymia) said:

“I had over 90% blockage. And I was so disappointed and annoyed and... A little bit afraid, but I was more disappointed. I don't think the reality had hit in of what had actually happened. But I felt disappointed in myself because I had done so much training, and I felt this can't happen to me, and how am I going to do this in the future. I didn't think about dying, I didn't think about anything. I just thought this is really something that shouldn't

happen to me because I've exercised, I've... I watch more or less what I eat. And why? Why? You know, why has it happened to me."

Declan (61 years old, EE) felt he lost control over what was happening to him:

"I was out of control... and a... that's... that was probably a stressful situation as well... when you are out of control... I'm sitting on that aeroplane... I knew what was happening but I felt I can't do anything about it, you know... So that was the emotion of um... of a... helplessness."

Participants in the NS group often described their state at that time as "shock", however they rarely specified their negative emotions. For example Mary (65 years old; NS; Type D and Alexithymia) said:

"I really got a shock, it was a shock to the system about the stents. Cause I was always, I always kept fit and eat eh sensibly, or as healthy as you can, you know and I walked everywhere and exercised and generally okay you know, looked after myself."

Some participants did not tell anyone that they were not feeling well. Seamus hid the situation from his wife, he said:

"I didn't tell the wife, but that was more because I didn't want her to worry about... and that's why I say I bottle up everything and try not to."

Rob (71 years old; EE; Type D) for example did not want to upset his wife and disrupt her day out.

My wife, I didn't want to upset her ... I just told her I was going for an angiogram because she had her sister up from Australia and they were out. ...so I didn't want to give them a shock..."

Participants were also describing their hospital experience. Mark (50 years old; EE; Type D and Alexithymia recalled being reassured by the doctor:

“The doctor came in and he was great to talk to me, he was saying to me “look it, don’t worry about it, you will be okay”

Pat (NS; age not recorded) described how he found out that he was having a heart attack:

They had done the ECG and the print-out was here. It was quite, as you know, it prints out the sheets of paper and I said to the technician, I said to him “Well, what does it say?”, “You’re having a heart attack”. So that was that. So then the nurse came and she said “Who is your next of kin?”. Now that that didn’t give me a second heart attack is another matter”.

Some participants expressed discontent about not being well informed by hospital staff. They were under the impression that there was a rule in that particular hospital of not telling MI patients that they are having (or had) a heart attack, to protect them from distress. This was not well received by some participants. Jim (EE; age not recorded) said that not knowing worried him:

“So you see I was worrying, of course I was worrying a bit but I don’t suppose I show it. I wasn’t given any direct information like to the extent that I had any sort of blockages or anything. But the mere fact that I was getting an angiogram I thought well there must be some little thing that may require looking at. I really was naive. (...) No information passed to me at all until I took to walking Sunday down the corridor (...) So you see I was worrying, of course I was worrying a bit but, I don’t suppose I show it. I didn’t show it too well I was laughing and the nurses were like “you look a healthy specimen” (...) And I’m like “yes I don’t have a clue why I’m here”. (...) And they’re not answering me, they are not saying “you’re here because, you know, such and such”. And they don’t do that anyway, but nobody not even the doctor didn’t come down. One of his understudies only came

to me the Monday morning just explained to me where I would be going for the echo sounding. It would probably be in the afternoon doing the angiogram and he didn't say "stent". He didn't use the word "stent", nobody used the word "stent" that wasn't mentioned whatsoever. Heart attack wasn't mentioned nothing was mentioned."

Patrick found out about his having a heart attack after he had the stents put in:

"Nobody told me they were putting them [stents] in, I thought they were putting dye in and I was getting severe pressure, the pain was coming back. So I was trying to sit up and he was saying "lie back down"... and it was only when I got up to the ward, the family came round me and the doctor came in. He says "he's after having what we call a widow-maker"'"

Chris (age 71 EE, possible Alexithymia) described being taken in for the stenting procedure without being told about this beforehand:

"I was having my lunch on the Thursday and when they stood in the doorway with a wheelchair and the nurse says come on, are you ready? And I said, ready for what? My blood pressure must have gone out through the roof, my heart rate went up and down like, the stress immediately came back again because I didn't know what to expect.

Dave also noticed this initial withdrawal of information among hospital staff, but believed it was for his own good:

"My son was sitting outside, I could see him through the door (...) somebody opened the door... what she said to me at one stage, um... I didn't know she had spoken to him, um... "I've I've spoken to your son, Alan" she said, and I didn't know she knew his name was Alan (...) I says "Jesus Christ, I must be bad if they're speaking to my family" (...) I noticed one

thing about them all in this hospital , they'd never come out and say you had a heart attack, they lead you into it, so that they don't frighten you (...) "you had a bit of a procedure", or something, (...) "do you do you know what's happening here", I said "yeah I think so", and and um... it took him a long time to tell me, you know "it's actually a heart attack", so they did that very gently, and when I went up to the ward, again, the nurse that came to see me she says "do you know why you're up here" (...) they do it in a very nice way the way they tell you, they don't say "you're having a heart attack", so you don't get a fright"

The stenting procedure turned out to be painful for some participants and the pain during the procedure was compared to the pain during the heart attack. Chris not only experienced severe pain, but he had a cardiac arrest as a result of it.

(...) They wheeled me down into the, into the um, what do you call it, theatre (...) I got onto this bench, and there was a screen here and your man poked his head around the corner and he said: "now, John we're going to do this, you'll feel a little sensation" (...) And I believe, now from that point on I can describe pain, I've had pain in my life but this was off, off the planet. (...) I knew I had all these sticky things on me but I had a big patch down my left-hand side and one up here. And I said [to] the nurse "excuse me what's these?" ...and she said "has nobody told you?" And I said, "no" (...) "We had to shock you". And I said, "what for?" And they said the very last stent they put in apparently unblocked the artery to such an extent that the blood rushed into my heart so fast that my blood pressure went up to 300. My heart rate, sorry, my heart rate went up to 300. "And in a sense" she said "your heart stopped. So we had to shock you"."

When describing their emotions after their MI, participants often said they were annoyed. Steve (age 66; EE; Type D and Alexithymia) said:

“I’m annoyed that this happened. Probably probably yeah. Unconsciously. I’m not happy about my heart. I always known that there was something there. And I feel... (...) there’s probably even a shock (...) change in whole... attitude (...) I was... near it [death] (...) it broke my emotions a bit (...) silly things that you’d be getting uptight about... they don’t matter anymore.”

Steve described being verbally more aggressive towards his family:

“I’m never sick in my life you know what I mean right, but in the last month... the the survival instinct is taking over from... you know the enjoyment things you do in life you know what I mean, so when something hits you... it changes your attitude... ...you’re trying to get back... but I mean it’s my heart and now it’s (...) I wouldn’t cause any hassle, but I’m kind of... answering back, I don’t mean to do it”.

Feelings of resignation, regret and disappointment were also expressed by some participants. Rob said:

“The one big emotion that I felt during this time was that I have no chance to do anything now. All the things I wanted to do, all the things I should have done I may not have a chance to do it. Because I knew that the position I was in, in relation to mortality, was that I might not make it. And it was a terrible feeling of shock and regret, a package of emotions that was going through me at that time (...) first of all I didn’t make the will. (...) I hadn’t had a chance to say goodbye. (...) I was thinking about death. I was thinking about lack of control. I was thinking I can’t get back to where I was and therefore I promised myself if I got back to where I was, I would change completely might outlook on life. But I didn’t realise that I would have a whole draining of emotions, the stress came in and the depression came in, shock I suppose everything came in.”

Some participants felt they were not coping very well with what happened to them. Siobhan (46 years old; NS) felt that her CHD caused such a decline in confidence in her that she was prevented from doing what she loved:

“Lately I’ve definitely felt as if I couldn’t cope with the silliest things. Even down to the fact that you know last Sunday I couldn’t, I was doing like a Sunday lunch and cooking is my hobby. I mean I absolutely love cooking and baking and even doing a Sunday lunch I found stressful, whereas I could normally have done that in my sleep, you know so it’s I feel these are emotions that I’m feeling. I just feel less confident, more frustrated with myself.”

Kieran (54 years old, EE) and Connor (age unknown; EE) were also frustrated with themselves.

Kieran said:

“I honestly thought I was going to bounce back quicker, being as fit as I was I honestly thought I was going to...”

Connor felt he was coping well at the beginning, however new chest pains slowed him back down:

“The first two weeks [after leaving the hospital] I was great (...) if you had said to me “climb the side of the house”, I’d have climbed the side of the house. That’s the way I felt. I could take on the world, basically. (...) I felt like I could do anything. And then I got a pain in my chest. I ended up back in casualty, and that knocked the confidence now... I was thinking “it’s starting again””

Mark said he still sometimes feels anger and finds it difficult to relax:

“I still get angry and I still get bad pains in me chest like as the doctor says I have to take it easy, if not I will have a bad heart attack and it’s just that sometimes I do get angry and I would be sitting there and [wife] looks and me and she says “Mark, relax””

Frank also expressed annoyance over suddenly being restricted physically:

“I get annoyed and disappointed that this has happened, because it now restricts me, because all I want to do is go on the bike, go for a swim, go, go, go, because I enjoy it. And the thought of not being able to keep up with my friends, I hope it doesn't stop me from doing it. Because that was part of the attraction was to be with my friends.”

Ann (51 years old; EE; Type D) said she is so afraid of having another heart attack that she now sleeps downstairs instead of her upstairs bedroom:

“I was scared ... I moved from upstairs to downstairs cause I wanted to be near the door ... I thought I'd be quicker for someone to come in to me. (...) The other night I woke up with a bit of a fluttering so I said to to the, when I was up having the injection, I said to him "I'm having a little bit of fluttering, would that be because my blood is low?", so they said it could be but the doctor, my own doctor had also told me that being the stent in, um... it's a foreign body in the thing and um... I have a clot too in the heart now as well, so they said between all those it does take time to settle down.”

9.3. Coping with the Cardiac Event

Some participants seemed to cope well with their cardiac event. They admit that they now notice the importance of healthy diet and try to live a healthier life style. Cathrin (58 years old; EE; Type D and Alexithymia) described the discrepancy between what she knows know and is teaching her children, and what she learned and observed at her own home during childhood:

“I never know the importance of diet, life style... cholesterol is not just a number anymore... my mortality then really really came up a big issue (...) we might be a little bit healthier because we understand not to smoke, and we understand more about cholesterol, and we understand diet... my mother used to just think “keep away from salt” and that's it... I never really knew what cholesterol

was... all my children know about cholesterol and how important it is and what numbers are... I didn't... and that's all I can teach my children as well... just to be aware of heart disease..."

Similarly, Peter changed his cooking habits and is more focused on a healthy diet:

"I haven't eaten a bag of crisps since it happened (...) I never had breakfast so now every morning I have a fibre bowl of All-Bran or something like that with the tablets in the morning. ... eating a lot healthier stuff. I am conscious that I'm cooking my own food vegetables every day but you know the other thing before it happened I use to always eat a banana and apple everyday anyway at work and I would always drink orange juice."

At the same time, some participants express their worries regarding not being able to change everything about their lifestyle. Paul (58 years old; EE) said although he's fairly active, his working patterns and leisure habits might stand in the way of full recovery:

"I'm fairly active regarding my health. I mean I go for walks and I'm an active person and I played tennis two and three times a week. I haven't played a lot this year but would generally be an active person physically. So I don't have a huge concern about that. I'd say that probably my lifestyle as regards ... I work hard and I play hard and that would be a concern. That's the only concern I would have".

Pat expressed his worries over having to wait several more weeks for cardiac rehab. He said that even teaching him which exercises he could do at home, would have been helpful:

"So I'm playing indoor bowls and pitch and putt, that's three days out of the week you know. So I'm doing my gardening and everything else. So I have never got breathless, so I'm still waiting for the exercise. (...) I was giving out last week because I wasn't asked to do the exercise before now, you know, which is a long time. I mean if I was a person that wasn't going out, or wanted to go out, I would be sitting at home for Tom months and that would be even worse. You'd get another heart

attack. So I know they're busy and you can only take so many ... they could have given a card, do ... start off and do these exercises or whatever."

Mortality was on the mind of many participants, however some went as far as contemplate what life without them would have been like, had they died. Seamus described his fear over what their family's life would have been without him:

"I suppose it's just obviously when you started bringing up things, not saying you put anything in my mind, but it's just (...) even when I was speaking to you I could feel myself getting emotional.(...) I could feel sort of something building up there, because it's like anything, you think the worst case scenario, what could have happened, and if I had died, and then what would have happened to [wife] and the kids.(...)"

Unlike Seamus, Frank understood that even after his death life would have kept going. He described having a surreal experience of watching his own children together in a room and realised that whether he was dead or alive they would spend time together in such a way. This seemed to have brought him a sense of continuity:

"There is sometimes a little bit of concern that it's going to come back again or you kind of get the feeling of, phew, that was a close one, it could have been a lot worse, but if it was I wouldn't know anything about it because I'd be gone. (...) And, funny, last night it's a weird ... not a weird, it was a strange thing, I was sitting on the couch and I was looking at my son who is 18 and my daughter who is 22 and they were having a conversation but they were enjoying each other's company and I enjoy when I see them enjoying their company and what actually crossed my mind, which is a weird thing, was if I had died, this is what they would be doing. I hope it's what they would be doing. If I was gone, if I wasn't here, and I was ... like they were here where we are, I was where the door is looking at them, and I said now this could be me looking down from wherever and this is what would be going

on if I had passed away... In fact I'm getting more emotional about it now thinking about it than I did yesterday."

Martin described the process he went through from before the MI to weeks after it. Although he went from shock to sadness and depression, he was eventually able to reach an understanding of what happened to him.

"I'm more relaxed. I'm happier... even though I had the, the heart attack ... I was definitely sad and depressed about it. (...) But I have... to look ... to the future, see what's out there... those two days was a low you know, it was emotional. And you know, I, I don't want to go back to that... to that situation. And... the shock or denial I was in, like there was, I felt there was nothing wrong with me, I thought I'm invincible, not in that sense that I can't be killed ... Nothing can hurt, but obviously I realised that I can be hurt and I was hurting and I blocked it out."

Sandra (46 years old; EE; Alexithymia) decided that acceptance of the new limitations would give her happiness:

"I'm getting rid of the worry and if I can take, take the step back and admit you can't, I just can't do it anymore, then... I think I'll be back to myself."

Finally, some participants' healing process brought gratitude over having been granted a second chance, as well as hope for the future. Fergus (65 years old; EE) put it this way:

"I'd say initially I was upbeat, but say in the last number of weeks I'm a bit more conscious, of uh... my condition, probably more aware you know all the... things are dialling down getting back to normality and what... I'm back doing the things I was doing I was spending the time doing, a little bit more aware now, what's inside me now, the physical side of my body. (...) I have a faith and I believe that, my time wasn't up or... maybe I was put back for... to do something better (...) I think you have to

keep moving on (...) I know what happened to me, I know the the real story, I have it all now and pictured it, and I just see, touch wood, that I was given time.”

Similar gratitude was expressed by Kieran:

“The fact that I’m still here... I felt lucky... that I was still around really... that I had number one I had one heart attack on the bike, and then my heart had stopped so I felt that I was lucky to be you know back here and...”

9.4. Discussing Other Difficult Life Experiences

Participants also discussed other difficult life events. One such topic was a parent’s death. Fergus recalled never attending his father’s funeral, as he was a child then:

“When my father died times were different, we weren’t going to church, we we were taken away, I heard him dying in the room and uh I knew he was dying, we were taken around to an aunt, the aunt would look after us, we weren’t let go to church, at the funeral we were upstairs, we were taken home back to her house right away, and so we weren’t part of the funeral. Which today you are. I would bring my kids into... to kiss their grandfather goodbye... But not then, not those days no. The young kids, they were sheltered.”

A common reflection among some participants who talked about their father’s death was related to not having been close to the father when he was still alive. Emotion of regret was expressed by Tom (49 years old; EE; Type D):

“A lot of guilt to be honest, because I was never close to him. (...) Regret about not being closer than we were.”

Similar thoughts were expressed by Frank:

“And maybe I didn't make as big an effort with him when he was alive, and you only realise that, even though I did, but sometimes you think maybe I should have gone the extra”.

Kristin (64 years old, NS) talked about her mother's death. Although her mother had advanced metastatic cancer, Kristin did not know about it and was shocked when she lost her mother so suddenly:

“[I] let myself in, straight up the stairs, and she was in bed. But she was alive. She was breathing. Okay called an ambulance straight away, they came immediately.... they had her in, in the emergency for, what seemed like ages but it really wasn't and then they called me in, and they said, “this is a very sick, very sick lady, she's had a brain haemorrhage. Now we've take x-rays”, and he had the x-rays up on the wall. He said “she has major tumour in her stomach, major tumour in your lungs, you can see them there?” And I could see them. ... “the cancer is now gone to her brain”... I was shocked you know. .. “but I didn't know she had cancer”. I said, “she didn't know she had cancer”. .. so he said “well, I'm very sorry” he said... “we're going to recommend “do not resuscitate””. ... So it was very peaceful for her. It was heart breaking for me as her only child.”

Some participants experienced their child's death. John (age 57; EE) and his wife decided against aborting their unborn baby, who was discovered to have a genetic disease:

“We were given 3 options... to carry out the test... but they said that was fairly high risk. Because... by doing it, it, it could result in aborting the baby... the other possibly was to have an abortion, which could be arranged... even though it wouldn't be arranged here. .. and then the third one was to just eh go through the pregnancy and let it run it's natural course and see what develops, but with the distinct possibility that when the baby was born she would need micro-surgery and lots of attention and care and whatever. Okay so we um... went home and thought about that for a long time and we just decided... you know we've had 2 children, if she's going to be ill, she's going to be ill, then we'll

look after her, and that's our child. .. But then on a follow-up scan... something else showed up, and they immediately took my wife in because the baby was in distress... she had to have an, an emergency... C-section and then Mary was there, she lived for 2 days and died."

When asked about his own emotions regarding his daughter's death, John said:

"I felt obviously a certain amount of anger. Eh and again um, I felt very confused. Because um, no matter how intelligent you are. Or maybe it's not even a case of intelligence but no matter how aware you are of how life works with children. Um you're saying to yourself, you know when you have your first child you're hoping everything goes well and whatever else, and then you have your second child and that goes well so when you have to, you're having a third one and something catastrophic goes wrong, it's very hard to understand that. Because you're saying, what did we do wrong or right or what, how did that happen all of a sudden?"

Martha (75 years old, NS; Alexithymia) also described how she felt after she lost her child:

"You're never the same again... you're not. No. No. So that's... that's... that's was about the worst thing that ever happened to me... It was horrible... Because you go to bed... and... you you dream... and you think... what's happened... is a dream... and you wake up... and then you realise it's... this is real... and you jump out of the bed (...)Probably should've gone for counselling... Probably should've. Yeah probably should have. But it was it was so severe... you know it it it was just... it was an incredible burden..."

Dave added that the mourning is never over, after losing a child:

"When [he] died... birthdays and funeral... um... that's that... those times you know bring back memories you know what I mean his his date of birth or date date when he died and that sort of thing".

Jim and his wife experienced multiple miscarriages and two stillborn children, before they adopted two children. He talked about the experience and his emotions, but he also stresses the importance of support, which they never received after having lost their children:

“We lost children a lot of children. Two full term babies and about five miscarriages and yes I kicked a bit of furniture around and that is probably the one time in my life that I was aggressive. (...) It was way back in the ‘60s and I’m afraid we weren’t given a lot of information or respect. (...) Two or three weeks prior to the time she was due they said the baby was dead in her womb. They never gave us a reason as to why it would have been dead. (...) I was in at the birth (...) seen it, my wife didn’t, she wasn’t given it a hold. We weren’t given an opportunity to christen it. We weren’t given an opportunity to bury it. I paid two and six, that’s old Irish money, for it to be buried in an angel’s plot. (...) There was nobody in my family or hers, her mother had died, to really advise us I suppose to point us in any direction. So we just let it all happen and even to this day we’ve taken a few trips to [place of burial] all right, there’s a special commemorative day once a year. And we’ve gone there just probably stand on the wall... the name isn’t even on the wall (...) I suppose you are disappointed because you’re young married people and you’re looking forward to having a child. And suddenly there is no child I suppose after the third or fourth miscarriage we nearly start saying to ourselves it is never going to happen”.

Connor said that his daughter’s drug addiction was the worst burden in his life. It lasted many years and he constantly felt the fear over her life:

“She done terrible things you know ... I definitely don’t want to talk about half the things that she did, but she did do terrible things, and she’s a good looking girl as well. (...) There was absolutely no... reason for her... to go down that road, but she’s getting there now. (...) I opened the bedroom door and she was on the floor and she had a syringe in her arm and she was on her knees and her head was on the floor and I thought she was dead, ... so I went back down and I said to the wife “I think she’s dead up there”... we went back up and we picked her up and when I picked her up her eyes

opened, so... The next morning she knew nothing about it you know. (...) Well it's hard because no parent should bury their own child you know, so that was always the fear and it was a fear ... because every time the police knocked on the door we thought this is it ... (...) But then after a while you kind of get immune to it, ... because you are expecting it, ...it's coming down the line".

Richard's (68 years old; NS; Type D) grown-up daughter was sexually abused in childhood by a family member, who used to be fairly close to:

"I confronted him about it and he didn't deny it or he didn't do either really. (...) I didn't do anything about it. I gave him time to consider it. (...) But then he died. (...) I know this man was very fond of me, the fact that this came out in the open within the family it was very hard on him, I know that and probably caused him to die. He died before he really should (...) There was never any acceptance. It was never accepted, we didn't speak and then he died. (...) I would have missed his friendship, I would have missed the things we did together. Like we used to have great sing songs and he'd organise a sing song party in my home town."

Some participants discussed marital problems, such as infidelity or spouse's chronic illness. Mary said this about her husband's infidelity with her sister:

"When I heard about him and my sister ... when I think back ... the stress it must have caused me inside my body. (...) It was her husband that caught them... And they stood there, he came over, my husband was home that day and she was there, she was staying with me for a week, it was me she would come to when anything happened between her and her husband. (...) I still, I don't think I'll ever forgive him for what he's done. He doesn't, understand or doesn't realise."

Ann on the other hand had to cope with her husband's disability after illness. At first she was spending a great amount of time at hospitals with her husband, after which period she had to deal with his mental disability:

"Your life wasn't your own it was just constantly at the hospital... they had to bring him down to theatre [operation room] every second morning... to take the infection out (...) He can remember who won the grand national years ago, but his... short term memory... can be lacking... I would do up a list of shopping and I'd say "go down and get them", in the supermarket, and he'd go down... and he might phone me when I was in work, "you know the vegetables you wanted and the fruit on that list?", "yeah", "well do you need them?", "yes, that's why they were on the list", "all right I have to go back down again".

She said she constantly felt angry with her husband. Additionally, Ann lost her sister recently and when she was asked about her emotions in relation to that, she said:

"[I felt] anger that we couldn't get her a transplant, I wanted somebody else to die so she could have her transplant... which is terrible but... I wanted..."

Cathrin was discussing the time when she was a victim of workplace bullying:

"It was complete and utter bullying, but because I was an adult in my age I didn't even think about that, you know that never enter my psyche that this is what's going on there, people gang up against you and make you feel uncomfortable."

She tried to describe the understanding she had at that time of the emotions she was experiencing. As she has Alexithymia, she was at first misinterpreting her emotions for physical problems:

“One day I came home, I actually took painkillers and I said it’s not pain that I have I shouldn’t be taking painkillers to get rid of that, I went back to the doctor again, and I was saying... explained about taking the painkillers but I’d sort of known that I done something wrong because I haven’t got a pain, it’s butterflies, and I... um... it was another doctor and she said “here I will give you these for anxiety” (...) I still don’t know what I was feeling... it must’ve been a whole lot of different things... I actually still can’t describe it... and I and I couldn’t even describe it to the doctor... but I just... you know it was horrible, it was actually anxiety (...) but I didn’t know what anxiety was (...) I don’t know how people describe it you now... fight or flight whatever it is... you know the heart my heart used to pound... it probably was fear...”

Annmarie (52 years old; NS) recalled mother’s depression and having to cope with her suicide attempts when she was only a teenager:

“I remember when I was about fourteen, she used to talk about killing herself and that kind of thing you know. One day I came in and I caught her, she was putting the scissors into the plug. (...) Another day I was coming into the house and she just threw a basin out the window and shattered the glass all over the place and you know she couldn’t help it like, she was just crying out for help. (...) [another day] She said I’m not well Mary; I’m after taking all the tablets. I ran to the press and there they were all, the bottles were emptied, but my father had been told like leave the names of them and all. So that you know what she had been on and I ran up the fields to my father, we had no phone in the house, he was making hay up the fields and I ran up frantic. I said to him “dad mum’s after taking the tablets, all the tablets””

Michael (66 years old; NS) recalled teachers physically abusing children at school:

“Some of them were lay teachers, some of them were Christian Brothers but there was only one particular Christian Brother that used to hammer me but like I think like he used to hammer other people as well, like it wasn’t, it’s just I used to get everything on the left hand because I wrote with me left hand. (...) I remember one young lad he didn’t like his homework and he made him tear it up and set it on fire in the metal bin, you know the metal waste bins. And then they’d pick it up like he got a few burns on his hands but at that time no one could say anything about them.”

Aaron (61 years old; NS; Type D and Alexithymia) indirectly described anger over having been made redundant:

“I couldn’t believe that someone could do that to me. It was a situation where I was after doing everything I could to hold onto my job, and he actually kept two fellas there that I knew weren’t as good workers as me, hadn’t done anything for him, and the reason he gave... he said well you have a better chance of getting a job than they will. Like, I had a wife a child at home and mortgage to pay, like I still had... he kept two, you know, and to my mind, that happened to me about ten years ago, and it’s still as if it happened to me yesterday. That was one of the main things and that really upset me.... I told him to go and fuck himself, and I can’t believe that you’d do that to me all this time. I actually went out crying, you know.”

Expressing his emotions in relation to his chronic skin condition (dermatitis), Seamus said:

“It’d be more anxiety because it’d be the fear of going out and someone going “Jesus, look at you”, or like I mean I had that, whereas I went out and I seen a friend who I didn’t see for a long time, and the first thing they went was “good god, what the hell?”, and that was their only reaction, good god what the hell, like. And then that’s just to feel, and it puts your heart sideways.”

All participants were asked whether they disclosed their problems and worries to other people. Tom said he preferred not to show emotions:

“I tend to work things out myself, keep things bottled up, not bottled up but I don’t get overly upset about things. I don’t show... my wife says I don’t show any emotion I’m almost too calm”.

Fergus said he confides in his wife, however keeps worries from the rest of the family:

“My wife knew what happened, I... haven’t told my sons, and I haven’t told any of my family, only my wife, I would say to my wife “don’t don’t mention it to... this is this is between us”, because I never want to worry people, like they all have their own families, they’ve all their own little cares in this world, you get enough problems...”

Jim said he was able to express his emotions if he’s asked about them, however in everyday life he does not discuss his feelings:

“I will tell you now, you’re asking about my emotions, I’m very good with... I wouldn’t call it good I suppose, I don’t wear it on my sleeve... I would be honest with you I don’t, and that is no disrespect to any situation that is in my life. It is just how all of our family is, believe it or not. My three sisters and my brother and me, we are very similar.”

Martin does not share with his family, although he sometimes confides in older friends.

“I don’t talk to anybody. I don’t talk to eh close family. Never say anything to brothers or sisters how I, I’m feeling. I won’t tell my parents. Um, my close friends I wouldn’t tell them. I do have one or two people that I do open up to. And, but, they would be... older. They would be say, say my, my mother’s generation, but not my mother, but not related to me either.”

Some participants not only do not express their emotions verbally, but they try to hide them from others. Sandra said she hides her emotions under make-up and clothes:

“...the first thing that happens to me is my appetite goes and you lose weight. So people kind of notice then. So now I feel when something does hit me, if I do take it bad, people know straight away. So you have to keep a cover up”.

Rob discussed suppressing his true emotions and pretending he was happy, when he was actually depressed:

“I was putting on a face, now don't get me wrong it was seeing me and my family in a group situation the things that families do. I would be very happy cracking jokes and looking at things and all the rest of it, but inside I was never... if I could avoid that situation I would have.”

When asked why they did not share with others, the most common answer was that they did not want to burden them (mostly spouses and family) with their own problems. When asked about discussing his war experience with anyone, Tom answered:

“Certain things you'd talk to your comrades in the army about, because they know what you're going through and it'd be a shared experience. (...) They'd understand, you'd show your emotions to a certain degree, but you would try and sort things out yourself. But occasionally some guys did commit suicide while we were there and that kind of played on your mind more than anything else. Like that could easily happen to anyone, you know, under that kind of situation. It just gets too much.”

When asked whether he discussed it with his wife he said:

“No she (wife) doesn't know any of that. I think she has an inkling of some of it but not specific details or anything like that. I've always been the type that just ... keeps it to myself if I can (...) because I wouldn't want her to worry”.

Kieran said he would not tell his wife about his health worries, as she might get upset:

“I don’t want to upset her, it would hurt her, ... I wouldn’t... pass those feelings along to her... she might feel guilty or something like that, ... I’m dealing with those emotions on my own to be honest with you (...)

The same point of view was presented by Adam (55 years old; NS; possible Alexithymia)...

“I don’t really want to worry her too much because she would be a worrier. She would be worrying about things so I don’t really want to, I wouldn’t tell her any lies but the less said the better”

...and by Aaron:

“No, all that stuff I keep inside, yeah. I don’t like giving her any burden. (...) I don’t want to give them anything to worry about, like you know.”

After losing his child, John believed his sharing would worry his family, and he had to support them at this time:

“You have to be extremely supportive. Um you, you have to hide, hide your feelings a lot. Because of the children as well. Because she [wife] was sick.”

Peter did not tell anyone he had relationship problems, as he did not want to talk about his partner behind her back, to people who know her:

“[on relationship problems] I didn’t say it to family only to friends.... I felt that saying that to family, because they would be closer to Anne, almost like belittling her kind of, might be putting her in a different light to them that would be a bit unfair you know.”

Some participants believed that other people would not want to listen to their problems. Martha recalled how unease she felt when her friends were burdening her with their health problems, therefore she did not discuss hers with them:

“No. I didn’t say [talk about MI]... I didn’t say it to anybody... I don’t know [why]... maybe I should’ve said something... but my friends... there’s always something wrong with them and you couldn’t listen to it...”

Ross (50 years old; NS; possible Alexithymia) believed that people are tired of his constant worries over his health:

“I think people have seen enough of me. Of of the last few months... of the heart attack. My own family. And I don’t know them to know anything, no. So I just try work through it myself.”

Frank had very similar thoughts on this matter:

“It was something that subconsciously if I had said she might have got upset you know so I didn’t even think it was relevant. She might say ‘Oh for God’s sake, it was 8 weeks ago, get over it’. Possibly or you feel that they might go ‘Oh for God’s sake, here he goes again’. Not necessarily, but I just didn’t think it was relevant. I didn’t actually, say, make a conscious decision to say “well I’m not going to tell my wife”, I just didn’t think of telling my wife.”

John chose to hide his thoughts and emotions related to his recent cardiac arrest, as he also believed people do not listen to other people anymore:

“I genuinely do not discuss it with anybody. Um for the simple reason that most people don’t like talking about it. About the, first of all you know a heart attack. And you know, then, you know, then a cardiac arrest. I find generally speaking that eh most people obviously live in fear of having a heart attack and, and the consequences of it. (...) Probably best to stay quiet, and say nothing because

people aren't listening anymore. (...) People, people you know just like moaning or complaining all the time, whatever, and I sensed that. So I... kept a lot of it inside me."

At the same time John said that he would not discuss losing his child with other men, whereas when talking to women he felt their support and understanding.

"If you're speaking to a woman you will get complete understanding and attention, if you speak to a man, the man will... generally in my experience not want to talk about it or discuss it. Women on the other hand... will talk to you about it. And ask questions. And be understanding and open and inquisitive, and want to know the baby's name. Men on the other hand, just draw a blank and will change the subject in the blink of an eye. That's an ancient man thing. That has to be an ancient man thing where I mean if you're talking about emotions."

Sandra did not tell her husband how nervous she was after having her heart attack, as she did not want to be regarded as a weak person by him:

"I would tell [daughter] I'm nervous going back to work... but I wouldn't say it to [husband]. Because I wouldn't class myself as a weak person".

There were also participants who believed talking about problems and emotions with loved ones was helpful. Cathrin said she discussed the bullying situation with her husband. She said:

"I tell him about it and tell him "oh I'm so annoyed at them" and he'd be angry with them, he himself would be angry and giving out... and that helped because... I felt well I've got somebody that I knew was completely hundred percent ... with me, and could see it clearly and understood".

Others said they would have appreciated having someone other than their spouses/family to talk to about their problems and worries. Seamus said:

“We’re men, we don’t really think of... we try and block it up... or out... And it’s not only that, like I mean there’s ... there’s nowhere that you can actually go and sit down with people and all... like... like you can, you can go and pay for it, but I mean... (...) In life there’s nowhere. Right, in school you used to have counsellors, but they’ve done away with all them now. (...) All those education cuts, so there’s nowhere, like. Again, like you don’t feel... in school, when I was in school, I didn’t think I needed any... (...) To go and speak to someone because I didn’t have a bad life, I didn’t feel like I had a bad life. But nowadays like there’s nowhere like unless you go and pay”.

Mary felt she had no one to talk to anymore:

“I don’t have any friend right now. Well, see, what happened was, there used to be, there was about four, five of us and as I said one moved down to [name of town], the other lady died, that left three of us and now in the last two years, eh the other two have fallen out.”

Simon (54 years old; NS), who worked with children and teenagers from pathological communities, and had faced the death of some of them, revealed he never had a chance to discuss these experiences or his emotions with anyone:

“There was no one to talk to about it... just you know... one of you found one of your kids dead in the basement on a Thursday night, you come in to work on a Friday morning, you just got on with it, and there was no... um... dedicated teams within the organisation to come and help you deal with your trauma and your stress or... there was certainly no staff support”.

9.5. Chapter Summary

As can be seen from the above examples, most participants in both groups did not express their emotions unless they were specifically asked to. When prompted to disclose them, they were able to do so whether they had Type D/Alexithymia or not, although in some cases more time was needed to identify a particular emotion.

Post-MI patients also tended not to tell anyone that they were feeling unwell at the time of their heart attack, which resulted in delayed hospital admissions in some cases. At the hospital they would have preferred to be better informed as to what exactly they were experiencing and what procedures they would have to go through. In the first weeks after leaving the hospital they often felt strong emotions of annoyance or anxiety, especially when they were also experiencing chest pain. Eventually, participants were able to understand the meaning of the situation from the mortality perspective, and feelings of gratitude and hope were expressed by some.

When discussing experiences other than their cardiac event, participants often went into much detail and their narratives were often very emotional, however, in general, they said that they did not share these emotions with other people, as they wished to protect them from worry. Some said that other people do not want to be confided in, whereas others did not feel they had anyone to discuss their problems with.

Discussion

This is the first longitudinal study to test the effects of social sharing intervention with an added component of emotion expression on psychological and physiological outcomes in post-MI patients. It is also the first study investigating associations between language use and physiology during sharing interviews. Specifically, the first aim of this study was to test the effects of an emotion expression intervention on psychological distress, affect, as well as heart rate and blood pressure. It was hypothesized that social sharing with emotion expression would be more beneficial than natural sharing or the usual care condition. This is based on the hypothesis that during social sharing with emotion expression, more negative emotions would be expressed than during natural sharing.

The second aim was to investigate the moderating effects of Type D personality and Alexithymia on the intervention effect on psychological outcomes, as well as to test whether changes in rumination and reappraisal mediated these effects.

The third aim of this study was to investigate the differences between both social sharing groups in physiological indexes measured during sharing episodes.

The fourth aim was testing the potential associations between overall language use and psychological outcome measures.

The first part of this chapter summarises the findings of the feasibility study. This is followed by the discussion of the main study's findings, as well as implications and limitations of the study.

10.1. Feasibility Study

The main study procedure was tested for feasibility on a group of healthy volunteers. Results of this study were discussed in Chapter 5 (section 5.11). It was observed that all participants engaged in social sharing and expressed emotions as per instructions. They regarded the study an interesting experience and no attrition from the study was observed. Interestingly, most participants admitted that taking part in the intervention gave them an opportunity to disclose thoughts and emotions they never disclosed before to anyone; of note, such perceived benefits of emotion expression were reported previously (Frattaroli, 2006; Zech & Rimé, 2005). From this perspective the procedure was assessed positively – as a tool for addressing undisclosed negative experiences and emotions.

Despite inducing negative emotion expression, the intervention did not induce distress, contrary to previous findings which suggested that short-term distress may be a result of (written) emotion expression (Joshua M Smyth, 1998). In fact, distress measures such as depression, general distress and anxiety were reduced from baseline to post-intervention, supporting previous research in the area of emotion expression (D. M. Sloan & B. P. Marx, 2004a; Zakowski, Ramati, Morton, Johnson, & Flanigan, 2004). However, given the very small sample size such findings need to be interpreted with caution.

The results of the linguistic analysis showed that increased use of anger words was associated with increased use of cause words, and both of these word categories were related to decreases in distress. The small sample size should be borne in mind in considering these effects. Although increased cognitive word use and increased (yet moderate) negative words use has been associated with better outcomes (Pennebaker, et al., 1997), studies have not investigated the effects of increased anger words use on health or distress. This is discussed further in section 10.3.

In summary, the study confirmed the feasibility of the intervention procedure and explored several trends in psychological and language variables that are followed-up in the main study. Of note, the feasibility study is limited in that participants were members of the college participant panel and their levels of motivation, and therefore compliance to instructions, were high and attrition rate was zero. The small sample size precludes any conclusions regarding inferential analyses. As it is discussed below, this was not the case with the main study sample of post-MI outpatients.

10.2. The Main Study

This section of the chapter addresses the first and the second study aim. The baseline characteristics of the study sample, and results are discussed below. Although no statistically significant intervention effects on psychological distress or affect were found, there were some possible short-term effects for the low number of individuals with Type D personality mediated by changes in rumination, as well as long-term effects for the low number of individuals with Alexithymia. Of note, the additional analyses were conducted to examine if theoretically novel effects could be found for individuals characterised by difficulties in emotional expression. These mediation and moderation analyses were conducted despite the null main effects, against the conservative causal steps approach (Baron & Kenny, 1986), which has been criticised (e.g., Fritz & MacKinnon, 2007; Hayes, 2009). The more recent approach allows for direct testing of the mediation/moderation effects, which is not based on the existence of main effects (Hayes, 2009). Physiological benefits of social sharing were also observed in the whole sample.

10.2.1. The Sample.

Baseline assessment showed no group differences on any measures. On average, participants in each group were their late fifties - early sixties, most of them were male, had an MI, entered the study less

than two months after their cardiac event, and had no history of depression. Most were married, educated to a second level and either retired or in full time employment. In general, participants were high in negative affectivity, and although low in distress and above average on positive affect, a third of them were distressed and 15% had elevated level of depression, which is in line with previous research on distress prevalence in post-MI patients (Hotz, Cazabon, O'Farrell, & Robbins, 1991). Participants had average level of social inhibition indicating that, in general, they were not afraid to express themselves in front of other people. This was supported by average levels of emotion inhibition, suppression and the TAS subscales. Although mean values on distress and inhibition do not indicate this, a third of all participants were Type Ds, and less than a third had Alexithymia. All groups were also likely to use the mechanism of reappraisal, rather than ruminate about their problems. Finally, participants' BP and HR_b were in the normal range in all three groups at baseline.

10.2.2. The effects of the intervention on psychological and physiological outcomes.

Mediators and moderators of the effects.

The emotion expression participants expressed more negative emotion words (*anxiety*, *anger* and *sadness* words) than the natural sharing group, which shows that, at least in the post-MI/PCI sample, natural social sharing involves disclosing significantly less negative emotion words than social sharing with induced emotion expression. In combination with other findings outlined in this chapter, this is an important observation, as it may explain why research on social sharing was unable to find sufficient proof of the benefits of social sharing, whereas positive effects of expressive writing have consistently been observed.

10.2.2.1. Short-term benefits for Type D participants with medium or high scores on Alexithymia, mediated by rumination change.

Social sharing with emotion expression showed potential for benefit for the low number of individuals with Type D in terms of lowering post-intervention (short-term) distress and these effects were mediated by decreases in rumination, in line with previous research (Lepore, Ragan, & Jones, 2000). The Type D subgroup that experienced a decrease in rumination had lower levels of anxiety, distress, and perceived stress, and a trend towards lower depression immediately after the intervention. However, no effect was found for positive or negative affect. This effect was present only for those Type Ds that also had medium or high scores in Alexithymia; consequently the overall clinical meaningfulness and generalisability of the findings based on some measures showing an effect for a low number of participants must be borne in mind. Type D participants low in Alexithymia, or those with high Alexithymia, but not Type D, did not benefit from the intervention with regards to post-intervention distress. This finding is in line with inhibition theory (Pennebaker, 1985), according to which people who naturally inhibit their emotions are more prone to adverse effects of their inhibition, and therefore would benefit more from emotion expression. Furthermore, the findings are in line with findings of the previous study on cardiac patients (D. Hevey, et al., 2012), where moderating effects of Type D personality were also observed.

The fact that, in the low number of Type D individuals, rumination mediated the effects of emotion expression on psychological outcomes is in line with previous studies, where decreases in rumination mediated the benefits of expressive writing in inhibited individuals (Gortner, Rude, & Pennebaker; 2006). Similarly, rumination was found to positively correlate with distress in bereaved mothers high in inhibition (e.g., Lepore et al 1996).

Reappraisal did not mediate any of the intervention effects, contrary to what was hypothesised.

Research on expressive writing suggested that cognitive reappraisal and the search for meaning are the mediators between writing and its benefits (Pennebaker & Francis, 1996; Pennebaker, et al., 1997). This suggests that the mere talking (writing) about the negative life event generates cognitive processing that, in turn, positively affects physical and psychological health. A possible explanation for the null mediation effects of reappraisal in this study, especially after contrasting this result with the results of linguistic analysis (see section 10.2.3.), is that operationalising reappraisal with the use of the ERQ subscale (Gross & John, 2003) may not have detected more subtle changes in cognitive processing that underline the effects of emotion expression interventions. It seems that reappraisal as measured with Gross and John's scale addresses a more stable personality trait that is not sensitive to interventions such as this. Of note, Pennebaker and his colleagues measured cognitive processing with linguistic analysis of narratives (i.e., the use of *cognitive mechanism* words). Future research should use a different scale, for example the Cognitive Emotion Regulation Scale (CERS; Garnefski & Kraaij, 2007), which may be more sensitive to changes in processing.

10.2.2.2. Long-term benefits for people with Alexithymia, who were not Type D.

When outcomes during the 3-month follow-up were investigated, Alexithymia was found to moderate the direct effect of the intervention on negative affect and perceived stress, showing that the low number of participants with Alexithymia (but not Type D personality) benefited from emotion expression in terms of decreases in negative affect and perceived stress. This finding is in line with previous findings that showed benefits of expressive writing for people with Alexithymia (e.g., Paez, Velasco, & Gonzalez, 1999), such that participants with Alexithymia who wrote about traumatic events, and emotions related to them, had lower negative affect (as measured by PANAS) at the 2-months follow-up. Other studies confirmed the benefits of expressive writing on psychological health in people with Alexithymia (Baikie, 2003; Solano et al, 2003). The fact that the

long-term effects were not found for participants who also had Type D personality contradicts previous findings, where Type Ds benefited at 3-months follow up (Hevey et al., 2012), although in Hevey et al.'s study different outcomes were investigated. A possible explanation is that lack of power to detect such effects; in addition, Type D participants, who (unlike individuals with Alexithymia) are usually aware of their negative affect, may have already suppressed new negative emotions during the time leading to follow-up. This would suggest that the conscious tendency to suppress negative emotions may not be affected by the present intervention, or such sharing interventions would have to be more frequent, in case of Type D individuals, to prevent new negative emotions from being suppressed. People with Alexithymia, on the other hand, whose emotion suppression derives from an inability, rather than unwillingness to share, may have gained insight in their emotional life as a result of the intervention, which may have helped them cope better with negative emotions post-intervention. This explanation is quite tentative as the results emerge from a subset of the intervention group on a small number of the measures. Additional research to prospectively test such a hypothesis is required.

The fact that rumination did not mediate the effects of the intervention at the 3-months follow-up is most likely due to the loss of power related to the small sample and participant dropout; however, other possibilities can also be considered. The post-intervention benefits that were mediated by rumination were found only for Type D individuals, which is consistent with the fact that the NA subscale of Type D has been found to be associated with rumination (Svansdottir et al., 2012). The same mediation may not occur for individuals with Alexithymia, as it has been observed that Alexithymia is separate from rumination (Olivier Luminet, Rimé, Bagby, & Taylor, 2004; O Luminet, Zech, et al., 2000).

10.2.2.3. Effects on physiological measures in the general post-MI/PCI sample.

Early studies using the writing task found decreases in BP as a result of emotion expression (Pennebaker & Beall, 1986). For example, McGuire et al. observed long-term benefits of disclosure on both systolic and diastolic BP in a hypertensive sample (McGuire, et al., 2005). Although no statistically significant changes in any of the physiological measures were observed in the emotion expression group, in both other groups small negative effects were found on systolic BP and HR; however, these changes were not clinically significant and the effects were marginal, which must also be considered in light of the small sample size. If the pattern noted here was to be replicated in a study with a larger sample, this may suggest a potential small protective effect of social sharing with emotion expression on physiology. Pennebaker suggested that those positive effects on physiology are a result of disinhibition – a physiological balancing of emotion expression on the sympathetic activation (previously generated by inhibition). In other words, targeting inhibition may have a decreasing effect on the sympathetic arousal, therefore on the physiological indexes.

As noted above, additional research reporting similar or stronger findings with larger samples on the effects of the intervention on physiology may support the biological impact of writing. Written expression may alleviate prolonged physiological arousal that is an effect of chronic inhibition (Pennebaker, 1989, 1993) and the memories for the negative life experiences can alter the psychophysiological systems (Van der Kolk, 1994). Additionally, assimilation of the traumatic memory decreases rumination and hyperreactivity associated with it, attenuating physiological responses. From a clinical perspective, however, all BP values in this study are within the normal range (below 140/90)(WHO; 2003). This is most likely due to the fact that cardiac patients not only take BP-lowering medicine, but also engage in lifestyle and diet change, which have been associated with significant decreases in BP (Dickinson et al., 2006). It would not be possible for ethical reasons

to conduct an emotion expression intervention in hypertensive patients who are not at the same time treated with BP-lowering medications, which is one of the shortcomings of psychological research on medical samples.

10.2.2.4. Effects on psychological measures in the general post-MI/PCI sample.

Previous research found positive effects of written emotion expression on psychological health (Frattaroli, 2006), although social sharing interventions did not replicate these findings (Zech & Rimé, 2005). In the present study, no effects on psychological measures were observed in the whole sample. Similar null results were obtained before, in expressive writing research, for chronic disease samples (J. E. Broderick, A. A. Stone, J. M. Smyth, & A. T. Kaell, 2004; A. H. Harris, C. E. Thoresen, K. Humphreys, & J. Faul, 2005), therefore these findings are not isolated.

This intervention may not have been suitable for a general sample of post-MI cardiac patients in terms of lowering psychological distress. The only two previous (written) expression interventions in cardiac samples (Willmott, Harris, & Gellaitry, 2011)(D. Hevey, et al., 2012) did not report any effects of writing on psychological distress, despite positive effects on physical health or quality of life. To date, the majority of interventions (implemented in standard cardiac rehabilitation programmes) for those patients focus on physical exercise and health behaviour change, with an addition of psychotherapy for clinically depressed patients (Rutledge, et al., 2013). The most prevalent therapy offered to post-MI patients with depression is Cognitive Behaviour Therapy (CBT), which has been found to effectively reduce depressive symptoms. In the Enhancing Recovery in Coronary Heart Disease (ENRICHD) trial such benefits has been observed within six months in depressed patients and those and low in social support compared to usual-care group (Berkman et al. 2003). In less severely depressed patients psychotherapy in combination with self-management had been proved effective (Cuijpers et al. 2009; Whooley 2006). In another trial, the Secondary

Prevention in Uppsala Primary Health Care project (SUPRIM), one year long CBT with stress management was offered to cardiac patients who recently had a cardiac event. In comparison to usual care group, those patients had a 41% lower rate of recurrent cardiovascular events during a mean of 7.8 years of follow-up (Gulliksson et al. 2011). This shows that successful treatments for depression are complex and time consuming.

Another possibility for the null effects of the intervention on distress is that control participants in fact engaged in spontaneous sharing during the two assessment meetings they attended. Although the intervention procedure did not account for this, control participants often talked with the researcher about their recent MI and hospital stay. The researcher did not ask questions or encourage patients to share more; however, it was also regarded unethical to prevent participants from talking about their health. This tendency to share in the control group supports Rime's theory that after a stressful life event people engage in social sharing (e.g., Rime, 2009).

The subgroup analyses suggest that a small subset of patients characterised by specific patterns of emotion inhibition (based on Type D and Alexithymia) reported benefits on some of the self-report measures. It is possible that these patients may have benefitted as they most likely would not have engaged in emotion expression outside the study setting. However, given the small sample size, such findings remain tentative

Additionally, post-MI/PCI usual care does not include patients coming to the hospital for at least three (and usually more than six) months post-discharge (based on the procedure used in the hospital where current participants were recruited). In this study control patients visited the hospital within two months post-MI and by being there they often took a chance to inquire (with a cardiac nurse) about their upcoming cardiac rehabilitation programme. Patients therefore gained information and they may have felt acknowledged or "looked after" by the health care system, which are important

additions to usual care that predict better psychological wellbeing in those patients (Hildingh, Fridlund, & Baigi, 2008; Kristofferzon, Löfmark, & Carlsson, 2005).

10.2.3. Language use, physiology, and associations between language use and psychological outcomes.

The sharing groups were compared with regards to their language use at each sharing interview, changes in language use, and physiological indices during sharing.

Previous findings suggested that increases in cognitive words and decreases in negative emotion words over the course of an emotion expression intervention was beneficial for psychological outcomes (Pennebaker, et al., 1997). In the present study, the purpose of analysing group differences in language use was to test whether these types of language changes would occur in both social sharing conditions, or just in the emotion expression condition.

10.2.3.1. Sharing groups' differences in language use.

This section discusses the differences in language use between the two social sharing groups, as well as the associations that were observed between language use and psychological outcomes.

1) Emotion words

On average, emotion expression participants expressed more anger, anxiety and negative emotion words, showing that asking participants to express their emotions resulted in their increase, relative to natural sharing. Natural sharing participants on average used more positive emotion words, which may be explained by a general tendency some people have to deal with negative emotional arousal (possibly generated by factual sharing in this group) by using positive emotions (L. Fredrickson & Levenson, 1998; Tugade & Fredrickson, 2004). This coping mechanism may not have occurred in the

EE group simply because those participants were sharing more negative emotions instead. Additional research is required to test this hypothesis.

Mixed results were observed with regards to the association between the emotion words use and psychological outcomes, in line with previous research that was inconclusive, suggesting either the benefits (Pennebaker, 1993), or possible adverse effects of the use of negative emotion words (Pennebaker, et al., 1997). Although, in this study these discrepancies can be explained, as different effects depended on different types of negative emotion word categories used.

For example, the increase in the sadness words was associated with improved psychological health at 3 months follow-up only in the EE group, whereas in the NS group it led to a decrease in positive affect. Expressing sadness may alleviate the need to ruminate about it and therefore decrease distress, especially when combined with the finding (albeit only within the small Type D subgroup) that decreased rumination predicted decreases in depression and distress. Previous research has found that sadness is the most ruminated about negative emotion; furthermore, other emotions (which are less ruminated about) are disclosed quicker after a negative event than sadness (B Rimé, et al., 1992). It is therefore plausible that a social sharing intervention that induces emotion expression prompts people to discuss negative emotions previously undisclosed (such as sadness). Sharing undisclosed material has been associated with better effects of emotion expression interventions (Frattaroli, 2006).

Contrary to the above, increased use of anxiety words were associated with increases in anxiety at 3 months follow-up; however, this effect was only visible in the EE group, presumably due to the fact that, overall, the EE group expressed more anxiety words than the NS group. This shows that although expression of sadness can be beneficial, expression of anxiety is not. A mediation model has been proposed in which anxiety generates depression and distress through rumination (Starr &

Davila, 2012), suggesting that the more anxiety is expressed the more people ruminate about it, although more evidence is needed to support this model.

This observation, and the fact that the EE group indeed used more anxiety overall, may explain why HR in the EE group was higher than in the NS group at each assessment, including the follow-up. The fact however that at the same time HRV was slightly higher in the EE group than in the NS group may derive from the fact that the EE group engaged in effective emotion regulation (cognitive processing), which has been found to have benefits for HRV (Julian F Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). In other words, although the negative effects of expression of anxiety were visible in HR indexes of the EE group, the vagal tone (HRV) was positively influenced by successful emotion regulation.

Indeed there are differences between anxiety, anger, and sadness. Anger prompts people to “fight”, whereas sadness does not induce behavioural changes, as “there is nothing to be done” in sadness (Schwartz, Weinberger, & Singer, 1981). In that sense, those two emotions do not initiate escape. Anxiety induces a “flight” response and also mobilise the body’s resources, similar to anger, however the purpose here is a constant escape. People who feel anxiety wish to avoid negative things happening. Talking about one’s anxiety simply reminds people of their worries and the need to escape. Currently, the most effective ways to address anxiety are CBT (Cuijpers et al., 2014), acceptance therapy and mindfulness (Vøllestad, Nielsen, & Nielsen, 2012). The mere act of expressing anxiety will not alleviate anxiety.

The fact that increased use of negative emotion words (general category) was linked to immediate negative mood in the NS group only, with a smaller overall use of those words, may be due to the fact that perhaps a certain threshold needs to be reached for negative emotion expression to be beneficial, and the NS group simply did not express enough of those words.

Finally, increases in the use of positive emotion words were associated with immediate increases in positive mood in the EE group, but not in the NS group, which is surprising when combined with the fact that the NS group, overall, used more positive emotion words than the EE group. Additionally, previous studies linked positive emotion word use to better psychological health (e.g., David Hevey & Wilczkiewicz, 2014; Pennebaker, et al., 1997). However, as proposed above, the greater use of positive emotion words in the NS group may have been a way of coping with increased negative emotional arousal (L. Fredrickson & Levenson, 1998; Tugade & Fredrickson, 2004) in the NS group.

2) *Cognitive mechanisms word use.*

Participants in the EE group shared more cognitive words than the NS participants. While the emotion expression intervention was designed to increase emotion words only, increased overall use of cognitive words occurred as a result. EE group used more *cognitive mechanisms* words, more *insight* words and more *cause* words than participants in the NS group. This suggests that social sharing with emotion expression induces cognitive processing of disclosed experiences more, than natural social sharing, concurrent with Pennebaker's theory (Pennebaker & Francis, 1996).

The fact that among the EE participants, increases in the use of cause words were associated with long-term decreases in anxiety, depression, general distress and negative affect is in line with previous research (e.g., Pennebaker, et al., 1997). Increases in cause words indicate increases in cognitive processes, which have been linked to better psychological health after an emotion expression intervention (e.g. Pennebaker, et al., 2007), which however has not been confirmed by previously outlined analysis of mediation effects of reappraisal change (section 10.2.2.1). It is therefore possible that the way reappraisal was operationalised in this study was not successful in addressing the second study aim.

Although cognitive words predicted psychological benefits in the EE group, unexpectedly, increased use of cognitive words was associated with increases in distress in the NS group. Similar findings were observed before by Pennebaker and Francis (1996), although in that study participants who experienced negative effects of cognitive words use were control participants writing about trivial topics. The authors then suggested that writing about trivial topics, and at the same time attempting to seek explanations, may be detrimental. They also proposed that without an opportunity to analyse meaningful material, such need for seeking insight may be maladaptive. In the present study however, participants in the NS group shared meaningful experiences, therefore trying to search for meaning should have had positive effects on their psychological health. The reason that did not occur may lie in the fact that without emotional processing sharing is incomplete, even when cognitive processing takes place (Sloan & Marx, 2004a). This implies that when people socially share their life experiences they should express their feelings related to those events, as without this, not only they may not benefit, but they may feel worse after sharing.

Research associated expression of insights with the cognitive adaptation model, whereas expression of negative emotions with the exposure-habituating model (Shim, et al., 2011), whereas Sloan and Marx (2004a) suggested that it is a mixture of processes, that is responsible for the benefits of emotion expression, rather than one model. The present study findings confirm this hypothesis.

10.2.4. Group differences in physiological measures: HR_f and HRV analysis.

Heart rate (HR_f) and three HRV indexes (SDNN, RMSSD, and HRV triangular index) were analysed in this study and results showed no statistically significant group by time effects in those measures. Despite this, however, the means in HRV triangular index at each interview indicated possible clinically important effects, as at the third interview the HRV in the NS group decreased to a level, which according to research is related to adverse prognosis for cardiac patients (Hayano et al., 1999),

suggesting that natural social sharing, without sufficient emotion expression may have adverse effects on physiology. However, this is a very tentative finding as the sample size was relatively small in the context of the number of effects being tested for; the clinical meaningfulness of the finding requires replication in larger samples. The EE group showed better vagal tone (i.e., better sympathetic-parasympathetic balance), at least at the third study meeting, partially confirming the hypothesis of positive changes in HRV in the EE group.

The most plausible explanation for the null effects on physiological indexes is that in cardiac patients, physiological functioning is usually monitored by doctors, and, if needed, altered with medications. As it is currently not possible, due to ethical reasons, to conduct an intervention with cardiac patients who would not at be medicated for measures such as BP and HR, results of these analyses should be looked at with caution.

10.3. Implications

This section considers implications of the present study for theory and future research, as well as for cardiac care.

10.3.1. Implications for theory.

In his inhibition theory Pennebaker (1989) proposes that unfronted negative experience leads to more stress and he suggests that emotion expression alleviates this constant physiological arousal, positively impacting physical wellbeing. Research on expressive writing found health benefits of emotion expression for individuals who inhibit their emotions (e.g., Pennebaker, 2012). The present study adds both to the inhibition theory and to expressive writing research, as it suggests that emotion expression may have some psychological benefits for sub-groups of emotionally inhibited post-MI/PCI individuals. Notwithstanding the small sample size, the finding that only Type D

individuals and those with Alexithymia may have benefited on some measures from the intervention suggests that there may be a need for emotion expression interventions among people who either inhibit, or are incapable of expressing their negative emotions. This finding is in line with Pennebaker's claim that those with difficulties in emotional expression (e.g., Type D and Alexithymia) may require more structured interventions or supports to help them express negative emotions (J W Pennebaker & S K Beall, 1986). Although the present study's findings are only suggestive, they point to the direction for future research and suggest that more knowledge is to be gained from replicating the present procedure. Furthermore, the intervention's impact on the specific cognitive processing (reappraisal) mechanisms that Pennebaker argues produce beneficial effects (Pennebaker & Francis, 1996) requires additional research.

Additionally, the present study suggests that social sharing with emotion expression may have a small positive physiological effect for post-MI/PCI patients. Further investigation of this finding is also recommended, especially as it is not known what accounted for small decreases changes in systolic BP or HR in the other two conditions in this study. Of note, Pennebaker's early research on emotion expression reported specific physiological changes (Pennebaker, et al., 1987) that were related to emotional disinhibition, which he interpreted as reflecting a direct effect of emotional expression on physiological functioning. His later work, however, moved away the direct effect model as he focused on cognitive processing and reappraisal variables that mediated the effects (James W Pennebaker, 1997); the present study found a physiological effect for the social sharing group and future research should test if Pennebaker's hypothesised cognitive processing mediates such an effect.

Rime on the other hand suggests that most people share with others about their negative life experiences (e.g., 1998); however, this natural social sharing does not have an effect on emotional recovery (2009). An implication of this study's findings is the fact that participants asked to simply

discuss negative life events, without an added instruction to discuss emotions, did not seem to share their emotions in the discussion to the same extent as the emotional expression group. Rime's research on social sharing has focused on only asking participants to discuss life events and the present study adds to this research by comparing the effect of such discussion with a specific instruction to express emotions. The social sharing paradigm may not be fully capturing the emotional aspect of the experiences if participants are not explicitly asked to consider their emotional responses to the events.

Combining the findings of research on expressive writing and social sharing research generates two main conclusions: 1) that emotion expression has a potential for health and psychological benefits, especially for inhibited individuals, and 2) that people naturally share their life experiences with others. Utilising this combined knowledge should prompt researchers into applying Pennebaker's method into the social sharing paradigm.

Another important implication of this study relates to Denollet's research on Type D personality. Denollet (1996) proposes that the reason for emotion inhibition in Type D individuals is that they do not feel secure enough in a social situation, fear rejection and the lack of understanding, or perhaps they are embarrassed to reveal their innermost feelings. In the present study, however, it has been observed that if those people are given an opportunity to express their negative emotions, not only are they capable of doing so, but they may also benefit from it in relation to some psychological variables, if only short-term, more than non-Type Ds. Interestingly, in the present study a decline in rumination mediated the beneficial effects for the Type D patients. This offers tentative support to the idea that rumination may be responsible for at least some detrimental effects of having Type D personality – additional research is required to further test this hypothesis. If lowering rumination in Type Ds is associated with lower distress, other interventions that target rumination (e.g., mindfulness training) might prove beneficial for Type D individuals. Indeed, a recent study found a

decline in negative affectivity and social inhibition (although not Type D caseness) in participants who engaged in mindfulness (Nyklíček, van Beugen, & Denollet, 2013).

With regards to the theoretical framework applied to the present study, the findings provoke certain amendments to the main study model (*Fig. 10*).

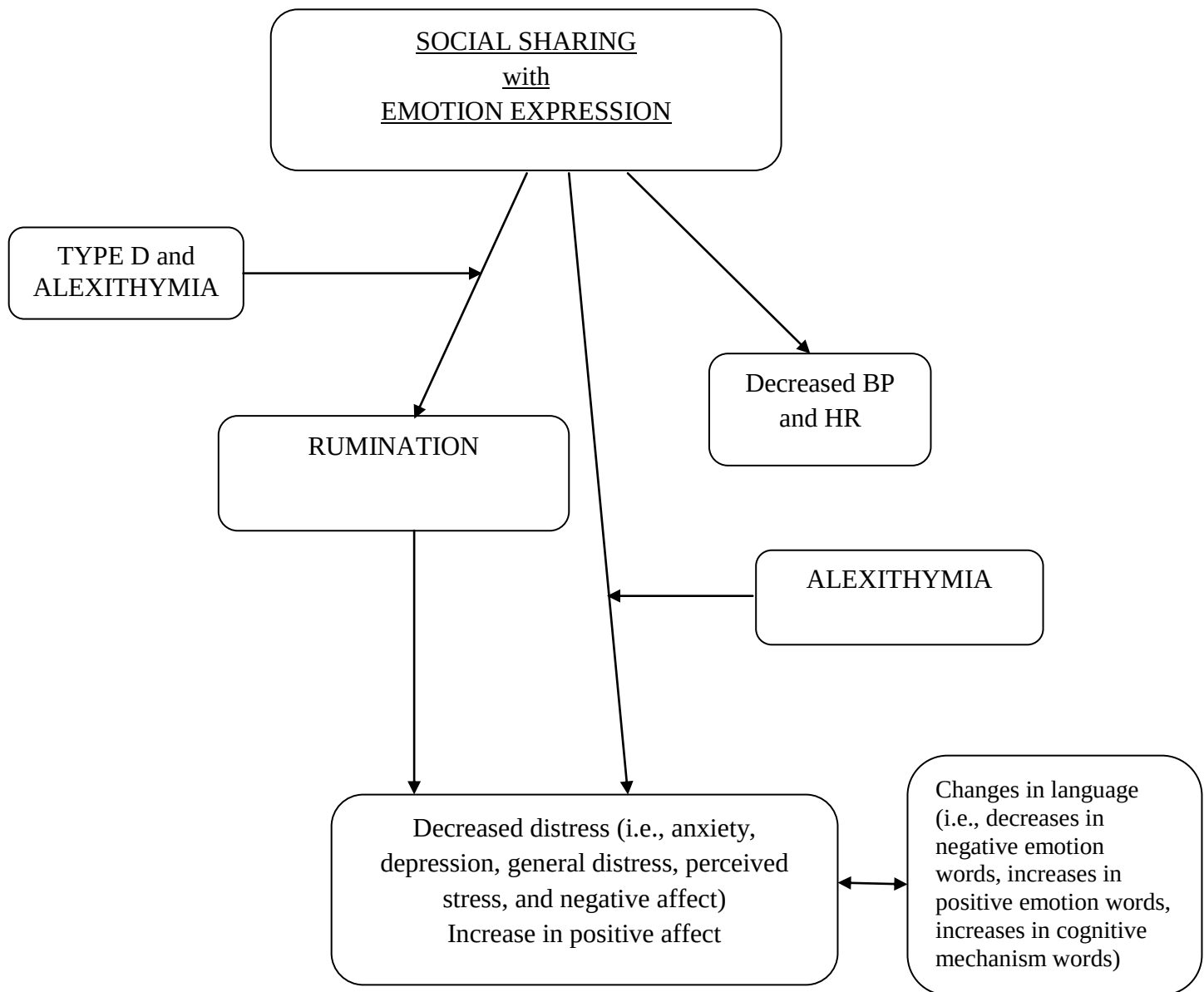


Fig.10. The Emergent Model Based on the Findings

The new model presents the pathways observed in the present study. Social sharing with emotion expression has a potential to decrease distress in Type D individuals whose rumination decreases as a result of the intervention. At the same time, the intervention potentially benefits people with Alexithymia with regards to changes in distress and affect, as well as it may have a positive impact on physiological functioning in general post-MI/PCI samples, despite their level of emotion inhibition. The benefits of the intervention for people with Alexithymia are not mediated by rumination, as is the case for Type D individuals.

As no mediating effect of reappraisal was observed in this study, reappraisal has been removed from the model. Changes in language use throughout the intervention kept their place in the model as correlates of post-intervention (and follow-up) distress.

10.3.2. Implications for future research.

Future research should investigate the effectiveness of emotion expression interventions in people with Type D personality or Alexithymia (or other groups of emotion inhibiting individuals), as they may benefit more from those types of interventions. If, however, studies are conducted on general samples, at least a comparison between the effects on participants low or high in emotion inhibition should be made. Furthermore, researchers should investigate whether applying Pennebaker's method to social sharing research (i.e., explicitly asking sharing participants to express their negative emotions during their disclosure) would have benefits for psychological and physiological functioning, and whether rumination or cognitive reappraisal would mediate these effects. With regards to the mediating factors of the intervention – outcome relationship, researchers might design interventions that would target rumination and test whether decreases in rumination lead to other health benefits in the participants. Furthermore, existing interventions, such as mindfulness interventions, could additionally investigate the rumination's potential to mediate the effects of

mindfulness and its outcomes. Additionally, future research should search for potential mediators of the effects of emotion expression on psychological outcomes in people with Alexithymia; especially rumination should be tested for the potential mediation of such effects, however with greater samples than one used in the present study.

Previous research has been inconclusive as to the effects of emotional language use on outcomes. Findings of this study may suggest that these inconsistencies derive from the fact that expression of different negative emotions may have different impact on psychological functioning. For example, in the present study the more participants talked about their anxiety the more distressed they became; however, discussing sadness was associated with a decrease in psychological distress. One difference between sadness and anxiety is that sadness does not induce any behavioural changes (Schwartz, et al., 1981), while anxiety induces a “flight” response and mobilises the body’s resources for a constant escape. Research on the effectiveness of emotion expression should also investigate into the differences in expression of different emotions, i.e., to compare expression of anxiety only with expression of sadness, to test how those different types of disclosure affect psychological and physiological health. Perhaps more importantly, future emotion expression interventions should concentrate on inducing expression of anger and sadness, but not anxiety, as it seems anxiety is better reduced with other forms of psychological support (e.g., CBT), than emotion expression interventions.

In clinical settings, social-sharing-psychoeducational interventions for patients and their spouses/loved ones might be tested for feasibility. In those interventions families should be informed of the characteristics of Type D personality and instructed how best to induce emotion expression via social sharing interactions in Type D individuals.

10.3.3. Implications for post-MI care and rehabilitation.

The results of this study suggest that while general post-MI/PCI samples may not need, or benefit from, emotion expression interventions via social sharing, patients with high levels of inhibition (e.g., Type D personality or Alexithymia) may indeed benefit from those interventions. Incorporating such an intervention in CR programmes, especially for Type D patients (or other inhibited individuals) would be advisable. Alternatively, as most post-MI patients are provided with leaflets regarding general medical information and instruction to implement health behaviours, perhaps they should also be offered information about the benefits of social sharing with emotion expression. Families should also be instructed to provide an opportunity for the patient to express their negative emotions such as sadness and anger, however in case of anxiety – other techniques (such as relaxation) might be more appropriate. Finally, taking into account the importance of lowering rumination levels in post-MI/PCI patients, psychological practise for cardiac patients could target its interventions specifically to lower rumination in those patients.

10.4. Limitations

This section outlines the study limitations.

10.4.1. Methodological limitations.

The study procedure involved all study participants attending hospital meetings with the researcher and this may have influenced the subsequent decline in distress, in order to control for an added element of being invited to the hospital shortly after an MI; however this precise factor may have acted as a buffer for all patients to decrease their psychological distress. Although it still does not support the emotion expression intervention itself, it gives important information of what is needed

by those patients, in terms of immediate post-MI care. It has been previously suggested that factors such as access to information and feeling “cared for” by the health system benefits post-MI patients (Hildingh, et al., 2008; Kristofferzon, et al., 2005).

Another weakness is that little is known about how the control participants coped after their MI, compared to the sharing groups. Information such as the number of GP visits, changes in prescribed medicines (both qualitative and quantitative) and changes in perceived social support would help establish whether the control group had, or was seeking, more help from others (whether medical or emotional), than the other groups. This is an important implication for future research, to gather more information about participants’ coping mechanisms outside of the study setting, to be able to answer the question why the control group improved too.

In line with the post-positivist stance taken, the present study uses self-report measures to assess psychological variables. However, the use of self-report scales may be a limitation of this study. Although currently the most often applied method in psychological research, it invites potential bias to the results, through the effects of social desirability or lack of self-awareness (for a review see McDonald, 2008). Other methods such as informant reports or behaviour observation are much more complex and time consuming, therefore self-report scales were the most efficient way to measure the constructs introduced in this study.

Finally, a weakness of this study’s procedure is that cardiac patients not only take BP-lowering medicine, but also engage in lifestyle and diet change, which have been associated with significant decreases in BP. As it is not possible, for ethical reasons, to conduct an emotion expression intervention in post-MI/PCI patients who are not being treated with BP-lowering medications, caution is advised when making conclusions from the results of physiological analyses; which is one of the shortcomings of psychological research on medical samples.

10.4.1.1. Attrition.

The ITT analysis was based on a representative sample (e.g., studies that used the writing task method used similar sample sizes: Greenberg et al., 1996; total $N = 97$; $n_1 = 34$, $n_2 = 32$, $n_3 = 31$); however the attrition rate (especially in the control group) resulted in the “as treated” analysis being underpowered. This limits the ability of the study to test the effects of receiving the intervention on outcome measures. The same limitation applies to mediation and moderation analysis, the findings of which, although interesting, are reduced to small subgroups of participants rendering generalisation impossible. Replications of this study with greater sample sizes are required. Additionally, there was one statistically significant difference between participants who attended the first study meeting, but withdrew afterwards and participants who completed the intervention. In the EE group the four participants who resigned after Interview 1 were significantly higher on negative expressivity than the rest of participants in this group. The study may therefore have limited power to detect an effect on negative expressivity.

10.4.1.2. Type I and Type II errors.

As it the case with most complex research, multiple comparisons increase the probability of making the Type I error. Here, a commonly applied Bonferroni correction was however rejected due to a novel character of this study and to prevent the Type II error. It was decided that the best balance could be achieved by applying the probability level of .01, although (as noted where applicable) for some analyses (exploratory) the .05 level was retained..

10.4.1.3. Single experimenter/interviewer.

Another weakness of the study procedure relates to the fact that it was conducted by one experimenter/interviewer, who also analysed and interpreted the results. This could have been a source of bias due to potential experimenter effects such as subjectivity and fatigue.

10.5. Summary and Conclusion

The most important conclusion from this study is that it suggested that social sharing with emotion expression may be beneficial for people with Type D personality and/or Alexithymia, in terms of decreasing distress. Whereas the benefits are directly due to emotion expression and are visible at 3-months follow-up for Alexithymics, in case of Type D individuals they are short-term and subject to changes in rumination, perhaps due to the fact that Type Ds are prone to rumination as a result of negative affectivity, whereas individuals with Alexithymia are not able to recognise their negative affect. Such results are consistent with the inhibition theory and previous research, however having been detected in small sub-groups of participants, these effects can not be generalised onto a wider cardiac population. No psychological benefits were observed for the whole study sample in this study. This may be due to the fact that most participants were not naturally inhibiting their negative emotions and perhaps engaged in social sharing outside of the study setting. Alternatively, it is plausible that the null effects derived from the study being underpowered. More studies are needed to further test the impact of social sharing with an added instruction for expression of negative emotions (following Pennebaker's method), especially in individuals who naturally inhibit their negative affect (e.g., Type Ds, Alexithymics). Additionally, as the present study also found small positive effects of the intervention on physiology in post-MI/PCI patients (albeit not clinically significant), future studies should incorporate such physiological measures in their method to investigate this further.

Confirming previous findings on language use during written emotion expression, cognitive words use was linked to better outcomes, however only in the emotion expression condition. Similarly, expression of sadness and positive emotion words was linked with benefits. In contrast however, increased use of anxiety words predicted future anxiety, which suggests that increased expression of anxiety intensifies this emotion, rather than reduces it. These effects were not observed in the natural sharing condition, suggesting that a certain threshold must be reached in the amount of emotions expressed for the benefits to occur. An interesting future research direction would be to compare emotion expression interventions that are focused on different negative emotions, in order to test whether indeed expression of anxiety has negative, while expression of sadness – positive effects on psychological functioning.

Implications of the present study also include implementing psychoeducation programmes for emotionally inhibited cardiac patients and their families, during which they would learn how to communicate their negative emotions and avoid accumulation of negative affect. Rumination is an emotion regulation technique which should specifically be targeted by interventions addressed at cardiac patients, especially those who inhibit their negative emotions.

Future studies, whether those focused on emotion expression per se, or on other types of interventions (e.g., mindfulness), should test the mediating role of rumination in the intervention's effects on outcome. Furthermore, future studies should investigate potential mediators for the emotion expression effects on psychological outcomes in participants with Alexithymia, however adequate sample sizes ought to be applied to avoid the Type II error.

Several methodological shortcomings may have played a role in the present study's results. First, as all participants visited the hospital to meet with the researcher, a bias might have occurred deriving

from the fact that true “usual care” does not involve patients to visit the hospital for months after the cardiac event. Furthermore, the knowledge about how the control group participants coped with their MI/PCI is missing, which knowledge might help to answer why the control group’s distress levels decreased in a similar fashion as the other groups’. Additionally, self-report scales have been used for assessment, inviting bias deriving from social desirability or lack of self awareness. Finally, there is no control (nor would it be ethically justified) over the amount and type of medications used by the participants, which are bound to have an impact their physiological functioning.

The study was underpowered due to a high attrition rate, although this has been amended with the application of the ITT analysis. Future studies might replicate the methodology of this study with reasonably greater sample sizes.

To avoid Type I error, the probability level was set to .01; a Bonferroni correction was rejected due to a novel character of this study and to prevent the Type II error.

The study was weakened by the fact that it was conducted by one experimenter/interviewer, who also analysed and interpreted the results, subjecting the results too bias due to potential experimenter effects such as subjectivity and fatigue.

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Appendix 1 - Ethical Approval Letters



COLÁISTE NA TRÍONÓIDE, BAILE ÁTHA CLIATH
Ollscoil Átha Cliath

TRINITY COLLEGE DUBLIN
The University of Dublin

14th June 2011

F.A.O. Ewa Wilczkiewicz

School of Psychology Research Ethics Committee

Dear Ewa,

Following receipt of amendments as prescribed by the Committee, I am pleased to inform you that your application entitled "Emotional expression among cardiac patients" has been approved by the School of Psychology Research Ethics Committee.

Yours sincerely,

SCHOOL OF PSYCHOLOGY
Aras an Phairsaigh
Trinity College
Dublin 2

Dr. Tim Trimble
Chair
School of Psychology Research Ethics Committee

Scoil na Siceolaíochta,
Dámh na nEolaíochtaí Sóisialta agus Daonna,
Áras an Phairsaigh, Coláiste na Tríonóide,
Baile Átha Cliath 2, Éire

School of Psychology,
Faculty of Arts, Humanities and Social Sciences,
Áras an Phairsaigh, Trinity College,
Dublin 2, Ireland

T 353 (0)1 896 1886
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www.psychology.tcd.ie

Ethics (Medical Research) Committee - Beaumont Hospital Notification of ERC/IRB Approval

Principal Investigator: Dr. Brendan McAdam

REC reference: 11/05

Protocol Title: Social Sharing among cardiac patients: impact of emotion expression on psychological and physiological functioning – a randomised, controlled trial

Ethics Committee Meeting Date: 11th February 2011

Final Approval Date: 21st April 2011

From: Ethics (Medical Research) Committee - Beaumont Hospital, Beaumont, Dublin 9

Document and Date	Documents Reviewed Date Reviewed	Approved
Application Form, V2, signed B. McAdam, 31/3/11	21/4/11	Yes
Study Protocol, V2	21/4/11	Yes
Letter of Invitation (Flyer), V2, 27/2/11	21/4/11	Yes
Information Leaflet, V2, 27/2/11	21/4/11	Yes
Consent Form, V1, 15/1/11	21/4/11	Yes
Debriefing Sheet (last meeting), V2, 27/2/11	21/4/11	Yes
<u>Interview Prompts</u>		
'Facts' V1, 2009	21/4/11	Yes
'Facts+Emotions' V1, 2009	21/4/11	Yes
<u>Questionnaires / Outcome Measures / Assessments (11):</u>		
Demographic Details	21/4/11	Yes
Lifestyle	21/4/11	Yes
Emotion Control	21/4/11	Yes
HADS	21/4/11	Yes
Mini IPIP	21/4/11	Yes
Emotion Regulation	21/4/11	Yes
I-PANAS Positive Effect	21/4/11	Yes
Perceived Stress Scale	21/4/11	Yes
DS-Scale	21/4/11	Yes
TAS-20	21/4/11	Yes
Berkeley Expressivity Questionnaire	21/4/11	Yes

Cert of Insurance,
Trinity College Dublin,
7/10/10

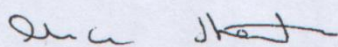
21/4/11

Noted

Curriculum Vitae
D. Hevey
B. McAdam

21/4/11
On File

Noted
On File



Professor Alice Stanton
ERC/IRB Convenor
Approval # 1, dated 21st April 2011

Feasibility Study Ethics Approval

ethics application  Ethics - IMPORTANT 

 **Kevin Thomas** <kthomas@tcd.ie> 21/04/2009   

to me 

Dear Ewa,

Following receipt of specified amendments, I am pleased to inform you that your application entitled "Social sharing and its health benefits" has been approved by the School of Psychology Research Ethics Committee.

Yours sincerely,

Dr. Kevin Thomas
School of Psychology
University of Dublin
Trinity College
Dublin 2
Ireland

People **Kevin**

Appendix 2 – Study protocol

Participant Information Leaflet

Protocol Title:

Emotions, social sharing and health

Principal Investigator's Name: Dr. Brendan McAdam

Principal Investigator's Title: Consultant Cardiologist

Telephone No. of Principal Investigator: 01 – 809 3366

You are being invited to take part in a clinical research study carried out at Beaumont Hospital. Before you decide whether or not you wish to take part, you should read the information provided below carefully and if you wish discuss it with your family, friends or GP. Take time to ask questions – do not feel rushed or under any obligation to make a hasty judgement. You should clearly understand the risks and benefits of participating in this study so that you can make a decision that is right for you – this process is known as Informed Consent.

You are not obliged to take part in this study and a decision not to participate will have no effect on your future care.

You may change your mind at any time (before the start of the study or even after you have started the study) for whatever reason without having to justify your decision and without any negative impact on the care you will receive from the medical staff.

WHY IS THIS STUDY BEING DONE?

This study is looking at patients' feelings, how they express (or show) them, at their mood and their stress levels. The purpose of this research study is to find a link between stress, feelings and health.

WHO IS ORGANISING AND FUNDING THIS STUDY?

This study is being organised by Dr. McAdam, Consultant Cardiologist, in Beaumont Hospital together with Dr. David Hevey and Ms. Eva Wilczkiewicz in the School of Psychology in Trinity College Dublin.

HOW WILL IT BE CARRIED OUT?

The study will start in July 2011 and potential participants will be contacted between July 2011 and December 2012. Potential participants are patients who have had a heart attack or cardiac surgery, and they will be contacted by phone by Ms. Eva Wilczkiewicz 3 weeks after leaving the hospital.

Participation in this study involves:

- 1) Filling in questionnaires at three time points;
- 2) Measurement of your blood pressure, heart rate and galvanic skin response in Beaumont Hospital at three time points. Galvanic skin response is a measure of skin electrical activity, which may change depending on one's stress levels.
- 3) Three Interviews with Ms. Eva Wilczkiewicz (some participants only)

WHAT WILL HAPPEN TO ME IF I AGREE TO TAKE PART?

If you agree to take part you will be randomly allocated to one of three groups.

Group One and Two:

Questionnaires:

You'll be given a series of questionnaires to fill in at home and post back to the researcher, Ms. Eva Wilczkiewicz. You will be provided with the stamped, addressed envelope. If you require assistance, the researcher is happy to meet you at the hospital to go through the questionnaires with you.

Filling in the questionnaires will take approximately 30 minutes. The questionnaires ask about your health, emotions, stress levels, and mood.

Interviews:

You will then be invited to come to Beaumont Hospital three times, over three weeks to be interviewed by Ms. Eva Wilczkiewicz. Each interview will last approximately 40 minutes. You will be asked to talk about stress in your life and your feelings. Interviews will be recorded.

The difference between the groups are the type of questions you will be asked. One group will focus on describing the stress in their lives whereas the other group will also talk about their feelings.

Blood Pressure / Heart Rate/Galvanic Skin Response:

Your blood pressure will be measured at the beginning and at the end of each interview, and your heart rate and galvanic skin response will be recorded during each interview. (Please note that you may take part in the interviews without these physical assessments, should you prefer this)

Last Interview:

Immediately after the last interview, you will be asked to complete a series of questionnaires, therefore the last meeting will take approximately 60 minutes of your time.

Three Months after the last interview:

Three months later, you will be invited to the hospital again, to complete a series of questionnaires and to have your blood pressure measured and your heart rate recorded. This will take approximately 30 minutes.

Group Three:

Questionnaires:

If you're in group three, you will also be given a number of questionnaires to fill in at home and post back to the researcher.

You will then be invited to come to Beaumont Hospital three times, over three months: 1) At the first meeting you blood pressure will be measured, and heart rate and galvanic skin response recorded. This will take up to 15 minutes of your time. 2) and 3) About 2 weeks later, and again 3 months later, you will again be asked to meet the researcher in the hospital to fill in some more questionnaires, and have blood pressure measured and heart rate and galvanic skin response recorded again. These two meetings will last up to 30 minutes each.

BENEFITS:***Group One and Two:***

Interviewing patients about stress and feelings is a quite a new idea and there is no guarantee of its benefits; however studies to date have showed this may be beneficial for psychological and physiological health.

Group Three:

Patients in Group Two may receive important information on changes in their physiological state over time since discharge (changes in blood pressure and heart rate).

RISKS:

Taking part in this study may be inconvenient. It involves attending the hospital for extra visits and also completing some questionnaires at home.

Group One and Two:

There is always a risk the interviews may make you feel upset or anxious.

All groups:

There is a risk that completing questionnaires about your mood, feelings and stress levels may make you feel upset or anxious.

WHAT IF SOMETHING GOES WRONG AS A RESULT OF MY PARTICIPATION IN THIS STUDY?

If you're distressed during your meeting with the researcher, the meeting will stop. You will then be referred to a clinical psychologist as soon as it's possible.

If you're distressed while completing the questionnaires at home, you should immediately contact Ms Wilczkiewicz, or her supervisor Dr Hevey (on the numbers provided below), and they will refer you to a clinical psychologist.

WILL THERE BE ANY ADDITIONAL COSTS INVOLVED?

All additional expenses (such as bus tickets) will be reimbursed.

CONFIDENTIALITY ISSUES

While there will probably be publications as a result of this study, your name will not be used. Only group results will be published.

All data will be coded. Every participant will have their own reference number.

Real names will only be accessible to the Principal Investigator, Dr Brendan MacAdam, the researcher, Ms Eva Wilczkiewicz, and her supervisor, Dr David Hevey.

Data collected in the Beaumont Hospital will be transferred to Trinity College, and stored there for a minimum of 10 years, in Dr Hevey's office or in the designated data storage space in the School of Psychology. *Your name will not appear on any of the questionnaires in Dr Hevey's office.*

IF YOU REQUIRE FURTHER INFORMATION

If you have any further questions about the study or if you wish to withdraw from the study you may do so without justifying your decision and your future treatment will not be affected.

For additional information now or any future time please contact:

Name **Dr Brendan MacAdam**

Address Cardiology Department, Beaumont Hospital

Phone No 01 – 809 3366

Name **Dr David Hevey**

Address School of Psychology, Trinity College Dublin, Dublin 2

Phone No 01 – 8962406

Name **Eva Wilczkiewicz**

Address School of Psychology, Trinity College Dublin, Dublin 2

Phone No 086 1270152

Emotions, social sharing and health.

Research Participant Consent Form

You are invited to participate in a research study about emotions and social sharing.

You will be asked to complete a series of psychological questionnaires and it will take you no more than 30 minutes to complete it.

After the assessment you will be asked to participate in a novel psychological intervention.

The intervention comprises of 3 weekly meetings, each lasting approximately 40 minutes. On each meeting you will be asked to talk with the researcher about stressful events in your life and about your emotional life. The conversations will be recorded.

At the beginning of every meeting, physiological assessment might take place, subject to your agreement. If you consent to this, your blood pressure will be measured at the beginning and at the end of each meeting, and your heart rate will be recorded during it.

After the intervention you will again be asked to complete some of the same questionnaires from the first assessment and it will take you no more than 20 minutes to complete it.

Physiological assessment (blood pressure, heart rate) might also take place at these times, subject to your agreement.

Your participation is completely voluntary. You have the right to withdraw from the study at any time.

Your signature indicates that you have read the information sheet and this consent form, had an opportunity to ask any questions about your participation in this research and voluntarily consent to participate. You will receive a copy of this form for your records.

Name of Participant (please print): _____

Signature

Date

Debriefing Sheet

Emotions, social sharing and health.

Thank you for taking part in our study.

People face difficult situations in life and often react with emotions such as anxiety, anger, and sadness. These emotions may affect their health if they are not talked about.

Research shows that most of us talk with others about stressful life events (we call that social sharing). We usually describe what happened to us (discuss a situation), but rarely say how we felt or still feel about this. But is this enough, or should we also talk about our feelings every time we mention our life stresses to others? This study is trying to answer this question.

If, for whatever reason, you decide that you no longer want your responses to be part of this study, then please contact Eva Wilczkiewicz (see details below) to have your data removed from the study and destroyed.

Please remember that all data collected in this study will be analysed, and only averaged results will be reported in any future publications. You will remain anonymous.

If as a result of this study you feel upset or distressed, please contact Eva Wilczkiewicz or her supervisor Dr David Hevey on the numbers provided below, and you will be referred to a clinical psychologist that will be able to help you.

Thank you again for helping us with this study. Your contribution to this study is very valuable and very much appreciated.

If you are interested to know the results of the study, or if you have any further questions about any aspect of this study, then please feel free to contact Eva Wilczkiewicz: Ph. 0861270152, email: schirmee@tcd.ie; or her supervisor: Dr David Hevey, School of Psychology, Trinity College Dublin, ph. 01 896 2406 or Dr Brendan MacAdam, Department of Cardiology, Beaumont Hospital, phone 01 809 3366.

School of Psychology
Trinity College Dublin

Cardiology Department
Beaumont Hospital

Demographic Details

Name _____

Gender: Male ☐ Female ☐

Age _____

Today's date: _____/_____/_____

1. Please state your marital status (*please tick*):Single ☐ Married ☐ Living together ☐Separated ☐ Divorced ☐ Widowed ☐**2. Please state your education (*please tick highest level completed*):**☐ Primary ☐ Completed 2nd level ☐ Third level**3. Please state your work status:**☐ Full-time ☐ Part-time ☐ Retired ☐ Unemployed☐ Other (please explain) _____**4. Health**Do you have coronary heart disease? ☐ Yes ☐ NoDo you have any other ongoing diseases or health problems? ☐ Yes ☐ No

If yes please state what disease _____

Have you had a major coronary event (such as a heart attack)? ☐ Yes ☐ No

If yes, please state what it was _____ and how long ago _____

Have you has a cardiac surgery (such as bypass surgery, a stent)? ☐ Yes ☐ No

If yes, please state what it was _____ and how long ago _____

Have you ever been diagnosed with depression or an anxiety disorder? ☐ Yes ☐ No

If yes, please state what it was _____ and how long ago _____

Below are a number of statements that people often use to describe themselves. Read each one and **circle the appropriate number** next to that statement to indicate your answer. There are no right or wrong answers: Your own impression is the only thing that matters

	false	rather false	neutral	rather true	true
1) I make contact easily when I meet people	0	1	2	3	4
2) I often make a fuss about unimportant things	0	1	2	3	4
3) I often talk to strangers	0	1	2	3	4
4) I often feel unhappy	0	1	2	3	4
5) I am often irritated	0	1	2	3	4
6) I often feel inhibited in social interactions	0	1	2	3	4
7) I take a gloomy view of things	0	1	2	3	4
8) I find it hard to start a conversation	0	1	2	3	4
9) I am often in a bad mood	0	1	2	3	4
10) I am a closed kind of person	0	1	2	3	4
11) I would rather keep people at a distance	0	1	2	3	4
12) I often find myself worrying about something	0	1	2	3	4
13) I am often down in the dumps	0	1	2	3	4
14) When socializing, I don't find the right things to talk about	0	1	2	3	4

Emotion Control Questionnaire

Listed below are a number of statements concerning emotions and feelings. Read

each item and decide whether the statement is **true** or **false** as it pertains to you personally.

1. When someone upsets me, I try to hide my feelings.....True ☐ False ☐
2. I remember things that upset me or make me angry for a long time afterwards.....True ☐ False ☐
3. People find it difficult to tell whether I'm excited about something or not.....True ☐ False ☐
4. I find it difficult to comfort people who have been upset.....True ☐ False ☐
5. I generally don't bear a grudge – when something is over, it's over,
and I don't think about it again. True ☐ False ☐
6. When something upsets me I prefer to talk to someone about it then to bottle it up.....True ☐ False ☐
7. I get “worked up” just thinking about things that have upset me in the past.....True ☐ False ☐
8. If I receive bad news in front of others I usually try to hide how I feel.....True ☐ False ☐
9. I seldom show how I feel about things.....True ☐ False ☐
10. I often find myself thinking over and over about things that have made me angry.....True ☐ False ☐
11. If I'm pleasantly surprised, I show immediately how pleased I am.....True ☐ False ☐
12. If I get angry or upset I usually say how I feel.....True ☐ False ☐
13. I can usually settle things quickly and be friendly again after an argument.....True ☐ False ☐
14. I don't feel embarrassed about expressing my feelings.....True ☐ False ☐
15. I never forget people making me angry or upset, even about small things.....True ☐ False ☐
16. I find it hard to get thoughts about things that have upset me out of my mind.....True ☐ False ☐
17. I often daydream about situation where I'm getting my own back at people.....True ☐ False ☐
18. If I see something that frightens or upsets me,
the image of it stays in my mind for a long time afterwards.....True ☐ False ☐
19. If I lose out of something, I get over it quickly.....True ☐ False ☐
20. I usually manage to remain outwardly calm, even though I may be churned up inside.....True ☐ False ☐

HADS

This questionnaire is designed to help us know how you feel. Read each item and **tick** the reply which comes closest to how you have been feeling **in the past week**.

I feel tense or wound up

- Most of the time..... ☐
 A lot of the time..... ☐
 Time to time, occasionally.... ☐
 Not at all ☐

I still enjoy the things I used to enjoy:

- Definitely as much..... ☐
 Not quite so much..... ☐
 Only a little..... ☐
 Hardly at all..... ☐

I get a sort of frightened feeling as if something awful is about to happen:

- Very definitely and very badly... ☐
 Yes, but not too badly ☐
 A little, but it doesn't worry me.. ☐
 Not at all ☐

I can laugh and see the funny side of things

- As much as I always could..... ☐
 Not quite so much now..... ☐
 Definitely not so much now..... ☐
 Not at all ☐

Worrying thoughts go through my mind:

- A great deal of the time ☐
 A lot of the time..... ☐
 Sometimes, but not too often.. ☐
 Only occasionally ☐

I feel cheerful:

- Not at all ☐
 Not often..... ☐
 Sometimes..... ☐
 Most of the time ☐

I can sit at ease and feel relaxed:

- Definitely ☐
 Usually ☐
 Not often ☐
 Not at all ☐

I feel as if I am slowed down:

- Nearly all the time..... ☐
 Very often ☐
 Sometimes ☐
 Not at all ☐

I get a sort of frightened feeling like "butterflies" in the stomach:

- Very definitely and very badly ☐
 Yes, but not too badly ☐
 A little, but it doesn't worry me..... ☐
 Not at all ☐

I can enjoy a good book, radio or TV programme:

- Often ☐
 Sometimes..... ☐
 Not often..... ☐
 Very seldom..... ☐

I feel restless as if I have to be on the move:

- Very much indeed ☐
 Quite a lot ☐
 Not very much..... ☐
 Not at all..... ☐

I look forward with enjoyment to things:

- As much as ever I did..... ☐
 Rather less than I used to..... ☐
 Definitely less than I used to..... ☐
 Hardly at all..... ☐

I get sudden feelings of panic:

- Very often indeed ☐
 Quite often ☐
 Not very often ☐
 Not at all ☐

I have lost interest in my appearance:

- Definitely ☐
 I don't take as much care as I should ☐
 I may not take quite as much care..... ☐
 I take just as much care as ever ☐

Emotion Regulation Questionnaire

For each statement below, please indicate your agreement or disagreement.

Do so by circling the appropriate number from the following rating scale:

	1	2	3	4	5	6	7
	strongly disagree	neutral				strongly agree	
1. When I want to feel more <i>positive</i> emotion (such as joy or amusement), I <i>change what I'm thinking about</i> .	1	2	3	4	5	6	7
2. I keep my emotions to myself.	1	2	3	4	5	6	7
3. When I want to feel less <i>negative</i> emotion (such as sadness or anger), I <i>change what I'm thinking about</i> .	1	2	3	4	5	6	7
4. When I am feeling <i>positive</i> emotions, I am careful not to express them.	1	2	3	4	5	6	7
5. When I'm faced with a stressful situation, I make myself <i>think about it</i> in a way that helps me stay calm.	1	2	3	4	5	6	7
6. I control my emotions by <i>not expressing them</i> .	1	2	3	4	5	6	7
7. When I want to feel more <i>positive</i> emotion, I <i>change the way I'm thinking</i> about the situation..	1	2	3	4	5	6	7
8. I control my emotions by <i>changing the way I think</i> about the situation I'm in.	1	2	3	4	5	6	7
9. When I am feeling <i>negative</i> emotions, I make sure not to express them.	1	2	3	4	5	6	7
10. When I want to feel less <i>negative</i> emotion, I <i>change the way I'm thinking</i> about the situation.	1	2	3	4	5	6	7

Perceived Stress Scale

Instructions: The questions in this scale ask you about your feelings and thoughts during the last month. In each case, please indicate with a **circle** how often you felt or thought a certain way.

	never	almost never	sometimes	fairly often	very often
1. In the last month, how often have you been upset because of something that happened unexpectedly?	0	1	2	3	4
2. In the last month, how often have you felt that you were unable to control the important things in your life?	0	1	2	3	4
3. In the last month, how often have you felt nervous and "stressed"?	0	1	2	3	4
4. In the last month, how often have you felt confident about your ability to handle your personal problems?	0	1	2	3	4
5. In the last month, how often have you felt that things were going your way?	0	1	2	3	4
6. In the last month, how often have you found that you could not cope with all the things that you had to do?	0	1	2	3	4
7. In the last month, how often have you been able to control irritations in your life?	0	1	2	3	4
8. In the last month, how often have you felt that you were on top of things?	0	1	2	3	4
9. In the last month, how often have you been angered because of things that were outside of your control?	0	1	2	3	4
10. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	0	1	2	3	4

The International Positive and Negative Affect Schedule Short Form (I-PANAS-SF)

Thinking about yourself and how you normally feel, to what extent do you generally feel:

Upset	(never) 1 2 3 4 5 (always)
Hostile	(never) 1 2 3 4 5 (always)
Alert	(never) 1 2 3 4 5 (always)
Ashamed	(never) 1 2 3 4 5 (always)
Inspired	(never) 1 2 3 4 5 (always)
Nervous	(never) 1 2 3 4 5 (always)
Determined	(never) 1 2 3 4 5 (always)
Attentive	(never) 1 2 3 4 5 (always)
Afraid	(never) 1 2 3 4 5 (always)
Active	(never) 1 2 3 4 5 (always)

Appendix 3 - Sample interviews

3.1. Emotion expression group

Participant: male, age: 35

Interviewer: So, as I said to you before, I would like to talk to you a little bit about difficult situations that you experienced in life. We will only talk about what you want to talk about and if I ask you a question that you don't want to answer then we're not going to go there. We're not going to talk about it, okay?

Participant: Okay.

Now, can you pick one situation that was the particularly difficult and negative?

Well, the time when I had mostly felt stressed was when I first got this dermatitis on my face.

Okay. When was that?

2008.

2008. So, six years ago?

Yeah. But it's still there, and it's only in the last two years where it's properly cleared... to where I could actually physically, sort of, feel a bit better about it. But other than...I mean the heart attack, I know I didn't think I was stressed, I don't, I still don't think I'm stressed, but that would be...it's only when I was talking to people and when, say, the GP said are you stressed, and I said the only time I ever felt stressed was the dermatitis on the face because it got to the stage where it was split and I was actually left with cuts and...

Oh, I know what you mean, yeah.

.... I couldn't go out, I couldn't socialise with people. So, that was it. Six years, running then say for the last...up to 2011, 2012. So it's been ongoing.

Yeah. How did you first notice it? Was it just very sudden or did it...?

It came on very sudden, literally, to be honest with you. I actually thought it was from a hot towel shave I got at a wedding, because it came on literally two weeks after getting a hot towel shave and I thought maybe it was something to do with that. But the dermatologist and all, they can't pinpoint that it is or that it was.

So they don't know?

They don't know.

Okay. Did you go straight to a doctor when you noticed it?

I went to doctors, I went to dermatologists, I went to then...I was admitted to the dermatology department in the Mater Hospital, and again, it's just...they can't pinpoint what actually was the cause of it, but it was two weeks

after, say, the hot towel shave. So that's... I thought it had something do with that; they don't know, they can't say it did or didn't.

Do you remember, when it first occurred, do you remember how you felt about it, in terms of emotions?

Well, definitely, there was definitely, there was a sadness because again I didn't know what was happening. It was a constant stinging sensation in the face. So, it was obviously stress as well. But again, as I said, I didn't know what it was. I was put onto medication; nothing was clearing it up. Then you had other people saying...then you were reading up...you had other people say try this, try that, try...and nothing was ever clearing it up.

Nothing?

So really again a lot of stress built up for me. And at the time, well, that was 2008, I was working, but 2009, the company that I was with went bust, so I was out of work for a year. But when I did finally get back to work I went into the food industry, and having that on my face didn't help either. So again...

I know. So, sadness...

Yeah, it'd be more anxiety because it'd be the fear of going out and someone going "Jesus, look at you", or like I mean I had that, whereas I went out and I seen a friend who I didn't see for a long time, and the first thing they went was "good god, what the hell?", and that was their only reaction, good god what the hell, like. And then that's just to feel, and it puts your heart sideways, really.

I know. Did you talk to... you were married then, weren't you? Okay, so did you discuss this with your wife then in terms of how it made you feel inside?

No, no. See I...people would be able say...to tell how I feel, but I don't express it great. I wouldn't talk about my feelings.

That's okay, yeah, I understand.

If they looked at...I try and stay happy, but I know if I was stressed, I just, they wouldn't know.

Especially your wife, probably, she would know anyway, yeah?

Yeah, I suppose she went through everything with me. She went through, say, me going in and out of the hospital, in and out, and paying for doctors' surgeries, and then as well as that I had lost my job, so again that would have brought on more worries. But I think even through the whole time of saying how am I going to make the next mortgage payment, I was more worried about what was on my face than... than making a mortgage payment.

Yeah. You said you were out of work for a year?

A year.

Okay. At that time, as you've just said now, you were more actually worried about the skin condition than...

Dermatitis, the skin condition.

So the doctors they didn't know what it was, you weren't sure what it was, and you were worried about that?

At the time, yeah. Because I was just given cream after cream and then nothing worked, I was given steroids.

Steroid tablets – nothing worked. Then I was put onto a cream that was costing me a hundred and fifty five euro a month; that didn't work.

That didn't work...

So again, it was just all building, the stress.

So how many years did it last for before they actually helped you?

It's only in the last two years, really, that it's sort of... It's only in the last two...now it's still coming and going, like I mean it's there now, but again it's not nearly as bad as what it was. I mean we got cracks here, cracks there where it'd actually physically bleed.

Would it be painful, physically painful?

Very, very painful...the only time I actually got a bit of relief is by shaving because it was like it was cutting, it was taking the badness out of it, or something.

Yeah. And what happened? Because you said the last two years actually were a little bit better.

They tried out plenty of creams and then they finally diagnosed it as this seborrheic dermatitis or something, which is the strongest dermatitis you can have. But I don't, like again I don't know, because the creams they had me on it obviously over time it finally kicked in and sort of calmed it down. Now it's still there and I do get outbreaks, but nothing to the extent I was getting.... It's still ongoing, no, it's not completely gone. It did flare up just after I got the heart attack again, pretty bad where it's like two weeks ago it started coming on the way you could actually feel the pain coming back into it. And again I went back over all the notes that I would have been given and it does clearly state that it can flare up due to a change in health.

Okay, yeah.

So again obviously having a heart attack is a certain change in health.

Tell me about your heart attack, because you said that it started two days before you ended up in the hospital. So tell me exactly what happened.

Okay. I was out for a cycle. I was only literally 4k into the cycle.

Recreational?

Yeah. I went out for a cycle. I was 4k into the cycle and...now, the cycle was primarily uphill, but I couldn't get a breath and so I stopped and I pulled over and I was ten minutes standing there, well, on the side, I was sitting down in a bus shelter and thinking this never happened before. I didn't have any pain prior to it. Like people keep saying did you feel any pain prior; I didn't.

You didn't? Okay.

I literally got on the bike, cycled 4k and that's when...as I said, I couldn't get a breath, so what I did was I got back on the bike and I continued, but I turned around to go home. So all in all it was 15k I cycled, and I was...When I got home I didn't say anything to the wife, 'cause again I didn't...now what I did do was I went for a shower and that's when it first hit me. The pains in the chest came and my two arms started locking up, and that was about 25 minutes. But what I did do, I got out of the shower and I ran a friend, and my exact words to my friends was, Robbie, I'm having a heart attack. But only in a messing way and Robbie said no, you haven't cycled, you have to remember you haven't cycled in two months it's just your lungs opening up, you'll be grand, and I said yeah. Again about 25 minutes it came and went. So, at 34 I didn't think I was having a heart attack. Left it, never said anything to the wife. On the Sunday, so that was a Saturday, on the Sunday...

That was Saturday? Okay.

That was a Saturday. On the Sunday night, I was making a cup of tea actually for the wife and...

Did you feel a little bit better by Saturday?

I was fine.

Okay.

I was fine. Literally, once that 25 minutes that was...

That was it?

There was nothing, that's why. So Sunday night I was making a cup of tea and I felt the arm starting to lock up again. So, I didn't get chest pains on the Sunday night, but the arms locked up. I said to her there my arm is locking up, and I just happened to say this happened to me yesterday but...and she says, but what is it, and I said it's just my arm's locking up. And that was 15 minutes came and went, because it came and went I sat down never did anything about it, and then obviously went to bed. Four o'clock in the morning...

Monday?

Monday morning, Sunday night, Monday morning 4 o'clock, I was awoken from the sleep. Sinead said I was tossing and turning in bed from 3 o'clock. But 4 o'clock I got up because the pain was unbearable, I couldn't do it. I went downstairs, chest, arms locked up, and I sat on the chair and what seemed to relieve the pain for me is if I put my hands over my head and I seen...I don't know whether it was blood flowing away from or blood flowing in or something, but that relieved the pain in the arm, so I just lifted the arms over my head.

Okay.

It went; that was 15 minutes. I went back to bed, fell asleep, woke up at half seven, I had a wash and all, got my cup of tea and I was on the way out to work and I felt the pain coming again and I put the cup down on the mantelpiece and I dropped onto the floor. Now, I came through within seconds and when I was...I realised that I was actually on the phone to my mam asking her to get me an ambulance even though my wife was downstairs; you always ring your mammy. But I literally then shouted down to Sinead, Sinead came running up the stairs and she rang the ambulance and that was it.

And Saturday when you were cycling, were you alone?

Yes.

By yourself, okay. And that was the first when it happened?

That was the first, yeah.

Do you remember how you were feeling – again, emotionally, back then?

I was fine. See, that's why people are saying are you stressed, and then again it's down to, I don't know, I don't think I am, I could be.

Okay, yeah, but even... okay, I understand, but even when, you know, when suddenly you were not feeling well and you had to stop and you couldn't breathe?

Oh sorry, at that stage?

Yeah.

I was panicked...

Okay, yeah.

...because again, as I said, I didn't know what was happening, I just knew that...Right, it was more, how would you...? It was like if – do you ever get wind where you [*panting sound*] that's like it was. It just felt as much as you tried to breathe in it was like nothing was happening, and that's what it was. And again, so it did then sort of ease and that's when I decided to get back on the bike and keep going. But again, as I say...

But you went home, anyway?

Yes, I went home, didn't realise what it was.

And then when you were taking the shower, again it came, yes, the pain?

That's when the actual pain... like the breathing was fine, but the pain in the chest it was like as if someone put a ton ball on your chest. It was like just a cursed feeling in your chest.

And how did you feel then?

Panic, pure panic.

Again more panic, yeah.

Because even though, as I said, I didn't tell the wife, but that was more because I didn't want her to worry about... and that's why I say I bottle up everything and try not to. But, again...

No, I understand, yeah.

For certain things it's all a bit...like happiness, yeah, you can pass that on to people, but when I am worried inside I bottle it up myself and I won't...I try not to... And I mean we've three kids, so I don't want to worry her in relation to it.

I know. And Sunday night, again more panic?

It was less panic on Sunday night because it was just the arms locking up, there was no chest pains, there was...So it was less panic, even, it was more anxious to see like what is our anxiety, anxiety, like what is it, like not knowing, not knowing what was actually going on.

And then Monday morning?

Again, that's when the chest pains came again, so...

And then when you collapsed that was the worst?

That was the... yeah, that was...even though it was only literally 10 or 15 minutes, but it obviously came along a lot stronger and that's why I collapsed. So obviously it was telling me for the whole weekend that something was going on that I needed to check it, which I didn't.

So, you got the ambulance and do you remember all this time from the moment when you collapsed and you managed to call your wife and you got the ambulance and all the way to hospital...?

I remember everything.

Okay. But... and do you remember how you were feeling all this time?

Yeah. Again because I obviously when the paramedic hooked me up to the ECU or whatever, he did say there was something with the heart, so that obviously brought on more, well obviously this is what was happening.

But again, as I said, I didn't think that I was taking a heart attack. And even in the hospital when I came in the

doctor who checked me out she actually said to me, she said what was the pain like. I said well it was like a crushing pain in the chest and then the two arms were locking up and she said to me no, your arms couldn't...her exact words was your arms weren't locking up, are you sure it wasn't pins and needles? And I said no, because I'm probably the only person in Ireland that will actually...I sit on my hands to get pins and needles 'cause I like them, I don't know why. But I like the feeling of pins and needles, and I said no, it's definitely not pins and needles.

It's something else, yeah.

I know what it...I know what pins and needles are. It was actually physically where my...even though and like that you could sort of feeling your fingers closing, it was trying to lock your hands, so.

And at which point were you the most ... panicked... anxious?

About twenty past ten when I had to ring the wife and tell her I actually had a heart attack.

Oh, that was the worst?

To actually physically ring her, because I knew she was gonna go...but, like even though they...

Was she at work?

No. She doesn't work, she...so when the doctor, when they sent me around for the chest x-ray and the doctor came back, and she said you had a heart attack and I said, okay. That's what I said – okay, it's happened, we'll move on, we'll get over...and they said look, we're admitting you in, and that's when I went... I have to ring her. But as they told me in the hospital that it was only a mild heart attack *[laughter]* that's not what the GP told me, and that's why...and to now I'm very cautious because obviously the GP said the heart attack I had the survival rate is not very high.

Okay.

So, again it's all a fear of what's going to happen next.

How did you tell her? How did she react, do you remember, when you called her?

She broke down in tears.

And how did that make you feel?

Emotional. Yeah, because like anything, you're supposed to be the breadwinner, I'm supposed to be the one that protects her, but...and then.

I know, I know. So that was sad for you to see her like...? Yeah. Now, how many days were you here?

Five.

Five days, okay. And when you went back after...so after the hospital, you went back home. The first couple of weeks, how was it for you? Was it difficult, or going back to...?

It's still difficult because... I mean obviously I have to...they tell you to do walking and they tell you to do...The first week out obviously I had the stress of...again then Sinead "be careful; don't do this, don't lift any...don't help out, don't...", and you're just trying to...you want to still do what you did with your children prior to.

Of course.

Then you had my own mam and dad, my own sister, her mam and dad, and they're all panicking over you, so it gives you a little bit of anxiety to...I just want to get on with it, I just want to...

But you took it easy anyway?

I took it easy. Well, I won't say I took it very easy in my terms, like I was back in the following week because I took an angina attack then.

A week after?

Week after. As the doctor said what were you doing? I said well I was walking. and they just said you could have overdid whereas an angina attack is your heart telling you to stop. So I've...to now, it's seven weeks, is it? Seven weeks I've put on 5 kilos in weight,

Because you're not doing much, yeah?

And that's obviously because, again, I've gone down from cycling 350k to just...

Obviously, yeah.

...walking a little, but I'm focusing a lot more on food. I seem to be eating a lot of fruit, whereas this whole last week now that I have to go without fruit. So it's like, one, they're telling you to eat this – like the food charts you have it tells you five a day, eat this, eat this, so you're trying to focus on it, but then you're being told on the other hand, don't eat that and don't eat that, so.

I know.

But I was terrible for drinking fizzy drinks, especially the Lucozade sport, I used to drink about seven bottles of that a day. Now I haven't touched any of that, so all I'm drinking now is water.

That's good, that'd good. You said a week after the heart attack you had the angina attack?

Well the week after I got out, so it was probably two weeks after the actual initial heart attack, so a week after I got out I came back in here because...

How did that angina happen? So you were walking...

I was walking and basically...

Alone?

Yes. Now I wasn't doing...it was only literally a two-mile walk or something. It's just the arm started locking up again and because I was leaving it as usual, as men do, you leave things, but the wife said no, she said ring the hospital, and when I rang them I told them and she just said come back in.

But they didn't keep you in, they just...did they?

They didn't keep me, they were going to keep me in because...but then they...obviously whoever came down from the specialist team they looked over and they said no, no, look he's all right, it was just an angina attack, so sent me home.

Do you remember how it felt this angina attack now? Because obviously it was after your heart attack, so it had to be emotional again or difficult or stressful?

Yeah...

How did you feel when it happened?

Panic.

More panic, yeah, yeah.

And I guess it's more panic because of obviously the fear of what you're going to put on Sinead, on my wife and the kids like. Again, the kids are young, like my oldest is seven, so she's not 100% sure what a heart attack is. Like she knows right, Daddy's very sick, but...our Daddy was very sick, but not the extent of where I was...again, as I said, when I was in hospital I was told it was a minor heart attack. It was only when I went to my own GP when I got out of hospital like, her exact words was – it's nice to see the 34 year old who took the widow maker. And I went, sorry? And she says you had what we term, what we doctors call the widow-maker. And I said, what do you mean? And she said you had a lethal heart...or a severe heart attack. And I said but they told me in hospital I only had a minor heart attack, and she says they won't tell you because of the stress it could bring in on you and you might...it might...if you're really stressed it could cause you to have another one. So that's why they don't you. So I said well, why do they let you tell me, do they [25:44]. And she said no, unfortunately we have to indicate and tell you that this is...

Serious.

...serious.

How...okay, so in the hospital they tried to calm you down with telling you that it was mild, and then she said that it was actually quite severe, and how did you... how did that make you feel when she said that, because that was a new information for you, right?

Well, obviously in the hospital and even to the time I was leaving like I always had...the nurse would always come in. I was in CCU, I was in a private room in CCU for the first three days and you'd have a nurse popping in every few seconds, and one of the nurses who always came in, and this is coming from my wife and mother, she said there's something more serious 'cause she even said that to Sinead, there's something more serious because the nurses are always coming in and they're always sitting there and they're always saying how are you feeling, you do know what's after happening? And to me I was just saying yeah, I had a heart attack, we'll get over it, we'll move on. But as Sinead's mam, Sinead's mam read more inference, there's something more there because they wouldn't be spending, like...

That much time.

...because she has friends who've had heart attacks, had mild heart attacks and nothing. They've come in and they've been hooked up and they've been sent home. So she was reading more into, whereas I didn't, again as I said I didn't read much into it, so.

3.2. Just facts group

Participant: male, age: 70

Interviewer: As I said, I would like to talk to you about difficult situations that happened to you. If I ask a question that you don't want to answer we don't have to talk about that, okay? First if you could pick something that was particularly difficult...

Participant: I was assaulted one night in work. That was a stressful weekend, alright, you know.

Okay. What happened?

The police came and took a fella off the bus and when they...I went off, I had to go around the block here from [name] Street, [name] Street, back around [name] Street, your man was at the stop. He jumped on, and I had to scream then when he just caught me here, you know.

Was he waiting for you?

He was, yeah. Well he was waiting for somebody, but I was the first one there, so.

Okay. And before that, he was on your bus?

Yes.

And the police took him?

Took him off.

What was he doing?

He was roaring...there's was two old ladies sitting down the back of the bus, and he was roaring and shouting at them and you know, giving them a lot of hassle and that, you know. So there was two guards passing by and I just said to them, would you ever, you know.

Okay. So they took him, but then they released him?

They must have released him, yeah. And that was stressful. I was out of work for four weeks 'til my face died now.

You don't have a scar or anything. Did you have surgery or something? Was it bad?

Yeah. No, no – now here.

Oh, that's it?

No, no, I have that there since I was born, yeah, the mark on my neck, yeah. That's like a mole or, it's been there now since I was that height. [Laughter].... no, no, no scar.

Was it very bad?

It was yeah. When my...my eye was closed and it out here, you know. But that was about the most stressful, that's the only time now I ever had a problem like that, you know, thank god, the first time in thirty-four year driving buses, you know.

That was the only time that it happened, something like that, at work?

The only time, yes, at work.

So tell me, you were sitting on your bus...

I was only after putting in the bus at stop... take people on. He came in behind him, yeah.

Okay. And immediately after that, did you...what did you do? Did people call an ambulance or did you...what was the next?

My first reaction was just to...the bus was fairly packed, so just to get the people home. So I just, your man, he jumped off and ran, so I just told the inspector on the radio what was after happening, and he said to me well, if you want to stay there and we'll get the guards. But the guards is only after being here, I said, they took him off. And he said ah well, they'll have all the details and all. So whether they picked him up then later on, I

don't know, but that's...we don't worry about that after that, you know, that's left up to the guards and that. So, I just carried on, and dropped the people home and then I went straight back to the garage then.

But you had...?

I went home, but I had to go up to my own doctor then the next morning and get my eye sorted out and made sure it was okay and...

But it turned out to be fine, right?

Oh it did, yes, yeah, thank god, yeah.

So how long, you said that you didn't go back to work for a few days or...?

It was about four weeks.

Four weeks?

Before the whole lot went back down, you know.

What did your wife say when you came home with this, with the eye? Or were you not married back then?

I was, yeah. Well one of people on the bus lived next door to her sister, and of course she knocked in and told her sister what was after happening, and then she rang...

Your wife?

To warn her now, you know, not to get a fright when I came home, you know, so.

Did you tell her though? Did you tell her everything that happened?

Oh I told her exactly what was after happening, yes, yes.

That's pretty bad, that must be...yeah. So okay, so that was the first thing that came to your mind when I asked you about stress. Did anything else ever happen in your life, you know, even with relationships with other people or, you know, people dying, people being sick, anything that you find...?

I took a...I was always great friends with my dad, and that was a stressful time now when he died, you know.

When was that?

That's fifteen year ago.

Fifteen? How did he die?

He had cancer of the stomach.

Oh, he had cancer, okay. So was he sick for a longer period of time?

He was indeed. He was sick over the years. It started off he had tuberculosis. He was a bricklayer on the building, and then days you didn't get time off it rained or anything, you just worked on and on. So that's where it started, and it all went into the stomach then, you know.

So, but you knew about him being sick?

Oh I knew, yes, yes.

So was it just, I suppose, gradually...?

It was stressful looking at him and not being able to do anything about it, you know, that's the way I felt.

Did you visit him a lot while he was sick? Did you see him?

Yes, yeah.

And the last, I suppose, the last couple of months, what did it look like? Was he in the hospital? Was he at home?

He was home.

He was at home, okay.

He was after being in the hospital and my mother brought him home. So they...we looked after him there, you know sort of. But...

Were you there or did you just...?

No, I wasn't there no. I wasn't there when he died.

So you got a phone call. Who called you, your mum or your sibling?

Ah the garage called me, actually, I was working at the time, and they told me that he had died an hour beforehand, you know, two hours, you know. They weren't expecting him to, he just...the doctor, the nurses and all were with him and they said oh no, he's not....you know.

Did they send another driver to release you and you went?

Ah they did, yes, yeah.

You went to your mother's home?

That's right, yeah.

And who's organising everything after your father died, the funeral. Was it you or was it other people?

No. I have an older sister.

Okay.

Two older sisters, and they looked after all that.

Right, yeah.

They organised everything.

Okay. I'm curious, obviously, your family and your wife and...do you have children?

I do indeed.

Obviously all of them knew about this situation because they were involved. But would you ever talk to other people, like friends or colleagues about...did you tell them what happened and that your father died? You know, sort of talk about this with other people?

Well in them days, like if somebody died we'll say they die, yes, that's it. That's it. So you look after all that yourself. But regards of going out and talking to anybody else about it, no, that wasn't the done thing then, you know, it was just you had the funeral and everything was looked after.

And you move on? Yeah.

Exactly, you move on, that's that, yeah.

And how were things in the first weeks and months after his death, you know, in terms of, even your mum?

I missed him greatly, you know. He was one of these people, no matter what happened he'd say to you, sit down there, now it's like this. But he'd never raise his hand or anything to you, you know. He'd talk through everything with you. He had a great way, you know.

Yeah?

Oh yes, yeah.

That's amazing. That's really very, very good, yeah. What about your mother then?

[Laughter]. Now my mother was a different story altogether. If you said something, she'd turn around and give you a smack or, you know.

She was more...?

More aggressive, sort of, you know, yeah. How the two of them got, now, together, because my father was totally different. He was more mellow and his attitude was well you don't have to strike them at all; you sit them down and talk to them. But she didn't believe in that at all!

Your mother's still alive?

No she's not, no. She died of kidney failure when she died.

When was that?

Ten year ago.

Ten years? Okay. So how was that for you... so you said kidney failure, was that a sudden thing or something also that she knew...?

She was in the hospital for a couple of days.

Okay. Did she expect that it would turn out this way or did you think that...?

No, we didn't. They thought that everything was working. She took a bad turn anyway, and that was it then.

And how were you...were you there in the hospital when it happened?

No, no.

How were you informed? Did something call you, the hospital or...?

The hospital called.

The hospital called, yeah.

But then again it's the funeral and then you move on, you know.

I suppose that's the way it is in life.

Ah well that's the way, not like it is now. Funerals now are major.

Yeah, but still that was ten years ago, I suppose, things are different now. Tell me about, we have just a few minutes left, I suppose.

I'm okay, relax, take it easy!

Take me about your heart attack

My heart attack, ooh now. I'm one of these guys who...to be honest, I have a brother-in-law, and he only lives across the road from me and he'll come over some days and he'll say to me, oh I'm having an awful day. You know? He's uptight over this, that, and the other, you know, meaningless things. Now to me, my misses will tell you my favourite word is choice. I have a choice: I can either feel good or I can feel bad. You know, all the time I'm going around the house and doing bits and pieces here and there and I'll say well, what's the point in having a bad day if you can have a good day.

If you can have...yeah.

Because if you have a bad...if you say you're going to have a bad day, that's, you're ruining the...

Obviously, yes.

Straightaway you've a problem. So that's been my attitude now for as long as I can remember. Or if you can do something for somebody, I'll do it, you know that sort of a way. But then that's the way it goes with me.

But tell me what happened? So that was six weeks ago?

Six weeks ago I was after being...I have a sister-in-law who lives in [town name] and we were after being out there with her that day, and I left about 7 o'clock in the evening. I drove all the way home, I love driving, but anyway, drove all the way home and the next thing then, I had a cup of tea, and I watched something on the television, I don't remember what it was, but we went to bed as per usual. This is on the Thursday evening. But at quarter to four on Friday morning, I'm sitting in bed and I have a pain in my chest, and I don't know, now I would never wish the pain to come back again, it was killing me, as though someone was just trying to tear your insides out and...

Okay, it was that bad?

And in the palms of my hands, this hand and this hand here, the pain in the palm of my hand was turning my stomach and everything, you know? So I said to her, she must have said are you okay and I said no, I have sort of a pain in my chest, I said, I think it's indigestion. So I said I'm going down to get a glass of milk. So I went down, got a glass of milk and drank it, and came back up to bed, and I must have fell back asleep because a quarter to eight I woke up and the pain was back. The milk seemed to ease the pain of it, you know. So anyway, when I...I still had the pains in the palms of my hands and the pain in my chest, and she said I'll tell you what we'll do, your doctor is there at 9 o'clock, he's only up the road, which he is, and we'll go into him and we'll get this sorted, cause she said...so in the meantime she was after ringing my daughter, who only lives in [town name] which is only five minutes away. She comes over, we all jump into the care and we're off to the doctor.

Your GP?

The GP, yes. So he's supposed to be there at 9 o'clock, but now he's after changing his times he doesn't start 'til half nine. So this is ten past nine, so I'm after seeing him going in, you see, when I was pulling into the car park I seen him going in and I went...there's a chemist next door and I went into the girls in the chemist next door and I said to them would you...and one of them said come in, sit here, sit down, get him a glass of water quick, you know. "Are you on your own?" I said no, the daughter's in the car there and misses is in the car. So they came in behind me, and she said he's there and he says you can go up to him, the door will be open. So when I went up, he took one look at me, I think he looked at the misses, she looked worse than what I did, and he said to me hold on. He rang an ambulance.

Oh right, yeah, he did.

So the ambulance man came and they gave me a tablet to put under my tongue, and then they carried me down the stairs and put me in the ambulance and brought me up here. They ran into the A+E with me in the trolley,

and when I was pushed into the cubicle the nurse said, no, no, no, he's not to go in there, he said, go up. They brought me up into this other ward at the back, and there was two nurses and a doctor there, and the doctor said do you know what a heart attack is, [patient's name]? And I said I've heard of them, but I've never had one. Well, he said, [patients name], we're having one now. He said here, did the ambulance man give you anything? I said they put something under my tongue, well he said there's another one, put that there, he said, it'll ease some of the pain. And they brought me straight in, and Dr [name] was there and he gave me two balloons and six stents. The main artery on my heart was blocked.

But that same day when you...?

The same morning, yeah, they just lifted me off the ambulance trolley, put me on the other one and they...that was about a quarter to ten I was getting the stents put in.

The stents, yeah.

Now I was still dressed like this, all I had was no shirt on me, still had my shoes, socks, trousers – just lifted me off the trolley and they worked on me there that day. And then they told me you have to come back now and get...we have to bring you back on Monday now and give you another three stents. So they gave me three stents and two balloons on that morning. He said to me your main artery, and he was showing it to me on the television as well what he was doing, so he said...so in the meantime Dr [name], who was a very good friend of mine, you know, he came in and he said to me the only thing we can't understand, he says, is why you had a heart attack, your blood pressure is normal, you don't smoke, you don't drink – only on occasion, you go to a wedding or that, that's the only time you have a few, you know. He said to me...like at home now they're all weight conscious, sort of, and they all drink low milk, low butter, you know, all the sort of thing. So, we're putting it down to age and wear and tear. Now he said there was a little bit of damage front of the back of your heart, but he says the medication will sort that out for you. But he said other than that, he said, we don't know what you're doing, can't find it, he says.

How long were you in the hospital for?

I was in it Friday, Saturday, Sunday. They brought me back down on Monday and gave me three more stents then on Monday. And the only thing about it was that when they gave me the three stents on Monday when they brought me back, my heart started racing and they couldn't get it to stop, and my blood pressure was up, it was 180/120 or something, he said. The machine will blow up, he said, if it keeps going up. So he said we were going to let you know go home today, but he said now you're not going home, you're staying where you are. So then they gave me this two hour...they put me on a drip for three hours, and then they put me on a drip for

twenty-two hours, and I was to go down then if that didn't work, I was to fast from 12 o'clock and they were bringing me back down to do that electro whatever they do, electro-cardio something.... Electric shock, to bring my heart back down to normal.

Oh right, yeah.

So then during the night when I went up the nurse came in at 4 o'clock to give me my tablet, and she said to me we had a little miracle during the night, when, she said, everything is spot on, so she said you can have your breakfast now, she said everything is perfect. So the best news then was on the Tuesday, he said to me, well you can go home now.

You can go home.

Yeah.

And okay, and how has it been? So, it's only fresh, it's only a few weeks.

Yes, yes.

How has it been since?

Well the hardest thing is doing housework. That's the hardest part of it.

You means in terms of not getting to...?

Not putting a nail in the wall, not going out cutting the grass, not cutting the hedging, do you know? The usual things when the sun starts shining you go out in the garden you do bits and pieces. "Where are you going? Are you going for a walk?" Yeah. "Well we're all going with you". You know [laughter]. I'm well looked after, I have to say that, well looked after. Now he told me not to drive the car, which I haven't driven the car now since...

But you did today, did you?

The day before I got the stents.

Did you drive today?

No, no, because it's not taxed anyway, because it was supposed to be taxed the end of last month, and the son said sure there's no point in taxing it, he said, if it's going to be lying there for four or five weeks, da, leave it there, he said, just tax it from the time that you go back to it, which will be probably I'll get it taxed tomorrow then and I'll be driving around then in it tomorrow, because the daughter now is bringing me here, there and everywhere, you know.

That's good.

Yeah. And Dr [name] was asking me last week, he was saying did you drive up this morning? I said no, no, I said the daughter's, says I, since I got the first stents, I said, the daughter is bringing me here, there and everywhere. And he said well, I said I'm going to get the car taxed, I said, next week and I said I'll be back driving myself, you know. And he said if I was you now, he said, I'd have her over everyday... that as long as I can, he said, leave the car, they can look after you. You know, he's only a doctor for saying it to me.

Exactly, yeah.

But that's the story with my stents and my heart attack. At the end of it all, everybody was saying no, he couldn't have a heart attack.

Oh, they didn't believe it?

They just wouldn't believe it, you know.

I know.

So, that's the story. But sure thank god now I feel great.

Appendix 4 - Correlations between changes in language use and short-term changes in distress (Time₂ – Time₁)

Table 43

Correlations between Changes in Emotion Words and Changes in Distress in the EE Group

Variable	1	2	3	4	5	6	7	8	9	10
1. Anxiety 2-1	---									
2. Depression 2-1	.119	---								
3. Distress 2-1	.781**	.659**	---							
4. Positive affect 2-1	-.402*	-.244	-.470**	---						
5. Negative affect 2-1	.168	.166	.129	-.159	---					
6. Perceived stress 2-1	.447**	.494**	.532**	-.149	.593**	---				
7. Positive emotion words 3-1	-.182	.248	-.001	.394*	.178	.310	---			
7. Negative emotion words 3-1	.104	.057	.101	.263	-.033	.144	.323	---		
9. Anxiety words 3-1	.005	-.314	-.125	-.029	-.230	-.242	.153	.505**	---	
10. Anger words 3-1	.229	.159	.247	.145	-.156	.158	.009	.682**	.228	---
11. Sad words 3-1	-.182	-.340	-.286	.399*	-.050	-.134	.192	.384*	.103	.087

Table 44

Correlations between Changes in Cognitive Words and Changes in Distress in EE Group

Variable	1	2	3	4	5	6	7	8
1. Anxiety 2-1	---							
2. Depression 2-1	.119	---						
3. Distress 2-1	.781**	.659**	---					
4. Positive affect 2-1	-.402*	-.244	-.470**	---				
5. Negative affect 2-1	.168	.166	.129	-.159	---			
6. Perceived stress 2-1	.447**	.494**	.532**	-.149	.593**	---		
7. Cognitive mechanisms words 3-1	.129	-.132	.049	-.199	.092	-.209	---	
8. Insight words 3-1	.222	.071	.243	.142	.001	.012	.360*	---
9. Cause words 3-1	-.052	-.270	-.196	-.099	.143	-.149	.353*	.175

Table 45

Correlations between Changes in Emotion Words and Changes in Distress in the NS Group

Variable	1	2	3	4	5	6	7	8	9	10
1. Anxiety 2-1	---									
2. Depression 2-1	.366 [*]	---								
3. Distress 2-1	.858 ^{**}	.791 ^{**}	---							
4. Positive affect 2-1	.117	-.101	.021	---						
5. Negative affect 2-1	.110	-.017	.063	-.085	---					
6. Perceived stress 2-1	.124	.107	.141	.022	-.108	---				
7. Positive emotion words 3-1	.532 ^{**}	.255	.537 ^{**}	.000	-.341	-.261	---			
7. Negative emotion words 3-1	.027	.374 [*]	.244	.123	.103	-.070	.051	---		
9. Anxiety words 3-1	-.153	.087	-.058	.010	-.122	-.256	-.081	.440 [*]	---	
10. Anger words 3-1	.089	.077	.110	.201	-.176	.061	.248	.548 ^{**}	.036	---
11. Sad words 3-1	-.236	.171	-.067	-.394 [*]	.010	-.172	.105	.160	-.108	-.062

Table 46

Correlations between Changes in Cognitive Word Use and Changes in Distress in NS Group

Variable	1	2	3	4	5	6	7	8
1. Anxiety 2-1	---							
2. Depression 2-1	.366 [*]	---						
3. Distress 2-1	.858 ^{**}	.791 ^{**}	---					
4. Positive affect 2-1	.117	-.101	.021	---				
5. Negative affect 2-1	.110	-.017	.063	-.085	---			
6. Perceived stress 2-1	.124	.107	.141	.022	-.108	---		
7. Cognitive mechanisms words 3-1	.074	.207	.177	-.488 ^{**}	.124	.064	---	
8. Insight words 3-1	.277	.508 ^{**}	.504 ^{**}	-.233	-.003	.099	.529 ^{**}	---
9. Cause words 3-1	-.142	.125	-.027	-.397 [*]	.018	-.129	.472 [*]	.103