Studies on the social and economic patterning of health behaviours in childhood and adolescence.

A thesis submitted in fulfilment of the requirements for the degree of PhD

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Submitted under the Department of Sociology,

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Trinity College Dublin, The University of Dublin

2025

Declaration of the Author

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Declaration of co-authorship

This signed declaration describes the contribution of the candidate and the co-author to each of the papers in this thesis to identify the candidate's independent contribution to each work.

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Paper one: The role of family, school, and neighbourhood in explaining inequalities in physical activity trajectories between age 9 and 18.

Olivia McEvoy is the first author of this paper. This paper was co-authored with Prof. Richard Layte and was published in Social Science and Medicine – Population Health in September 2022. Olivia McEvoy compiled the literature review, developed the theoretical framework and conducted the analyses. Prof. Richard Layte provided advice and feedback on the theoretical framework crucially introducing Annette Lareau's theories on parenting(Lareau, 2017), and the statistical analyses, specifically the decision to use linear spline multi-level models(L. D. Howe et al., 2016). He assisted in interpreting the findings and with the editing of the different drafts of the paper.

Paper two: Bringing the Group Back In: Social Class and Resistance in Adolescent Smoking.

Olivia McEvoy is the first author of this paper. This paper was co-authored with Prof Richard Layte and was published in Sociology of health and Illness in October 2024. Olivia McEvoy compiled the literature review, developed the theoretical framework,

conducted the statistical analyses and wrote the original drafts of the paper. Prof Richard Layte advised on the theoretical framework, specifically for Granovetter's social network theory (Granovetter, 1973) and research on peer selection versus peer socialisation, statistical analyses, and provided advice and feedback on the findings and multiple drafts of the paper.

Paper three: Social Exclusion and Health Behaviours: Insights from a Longitudinal Study of Irish Young People.

Olivia McEvoy is the first author of this paper. This paper was co-authored with Prof Richard Layte and is, at the time of writing, under review in European Sociological Review. Olivia McEvoy compiled the literature review, the theoretical framework, conducted the analyses and wrote the original drafts of the paper. Prof. Richard Layte provided advice and feedback on the theoretical framework specifically by introducing the literature on 'fundamental causes' (Phelan et al., 2010) and carried out part of the statistical analyses that involved the initial separation of classes, and gave feedback on the findings and multiple drafts of the paper.

Paper four: Does Family Economic Strain increase the likelihood of Child Smoking? An extension of the Family Stress Model using Longitudinal population-based data and the Great Recession in Ireland.

Olivia McEvoy is the first author of this paper. This paper was co-authored with Prof Richard Layte. Olivia McEvoy developed the theoretical framework, compiled the literature review, conducted the analyses and wrote the original drafts of the paper. The paper is based on a methodology that Prof Richard Layte developed for considering the relationship between family economic strain and educational achievement. Prof Richard

Layte also aided in the development of the theoretical model drawing on his expertise in psycho-social processes and provided feedback on the findings and drafts of the paper.

Statement by the co-author:

I hereby confirm that the doctoral candidate's described contribution to the work where I am

listed as co-author is correct, and I consent to including the work in the named dissertation.

Signed: 

Date: 30th September 2024

Summary

The social patterning of harmful health behaviours (HHBs) is a major determinant of the social gradient in non-communicable diseases, and health generally. For this reason, more research is needed to understand *why* HHBs vary according to the social and economic position (SEP) of individuals in this structured manner. As middle-childhood and adolescence is recognised as a 'critical' period for the formation of HHBs the data used for this thesis cover the period from age 9 to age 20.

Previous research has often adopted an *individualist* paradigm that focuses on providing 'bottom-up' explanations for health inequalities i.e. how individual choices and decisions aggregate to produce inequalities. In comparison, a *structural* paradigm considers the role of 'top-down' processes. According to this paradigm, HHBs are a response to conditions imposed by social structures and must be understood in their social and economic context. Drawing on longitudinal data from the Growing Up in Ireland (GUI) '98 Cohort, this thesis sets out to contribute to both perspectives.

The first paper examines the role of aspects of the young person's social environment in shaping their physical activity (PA) trajectories over a nine-year period, while controlling for established individual-level influences. We used multi-level linear spline modelling to estimate PA trajectories by SEP and compared trajectories of children from the least and most advantaged families. We then observed the reduction in this SEP differential with the addition of groups of variables (to our models) representing different aspects of the child's social environment – family, neighbourhood and school. Family-level processes, namely role modelling and gatekeeping of PA, were the most effective in explaining the

social patterning of PA followed by neighbourhood and school-level processes. This paper highlighted the important role of parents in shaping SEP differentials in PA.

In the second paper, we theorised how family SEP in childhood might lead to smoking in late adolescence. Using path analysis, we established mediators of this relationship, and similar to paper one, we distinguished between individual-level and structural-level explanations. Our results indicate that there is no pathway via the variables that represent our individual-level explanations. There are, however, statistically significant mediating pathways between childhood SEP and smoking via the variables that represent our structural explanations.

The role of the third paper was to identify HHBs that are likely to co-occur, and whether young people with similar patterns of behaviours have distinctive risk profiles. Using latent class analysis, we identified the clusters of HHBs: lifestyle behaviours (poor diet, low physical activity and high screentime) tend to occur together and appear to be relatively independent of substance use behaviours (tobacco smoking and underage drinking). Distinct clusters suggest distinct underlying processes, which we tested using multi-nominal logistic regression. According to our results, the experience of persistent poverty is associated with a higher risk of unhealthy lifestyle behaviours and appears to promote the development of 'oppositional values', though only in a minority of cases. Where persistent poverty is accompanied by oppositional values, the probability of unhealthy behaviours and substance use rises significantly.

The final paper examines why family economic hardship and deprivation is associated with a higher probability of smoking status among adolescents. The paper uses the Great

Recession in Ireland as a ‘natural experiment’ and quasi-experimental methods to examine the explanatory value of the ‘Family Stress Model’. We first adjust for the non-random probability of experiencing the effects of recession, using propensity score matching based on family characteristics before the onset of recession in 2009 when the child was aged 9. Second, we test whether economic strain, as a result of the recession, increased the probability of smoking initiation by the time the adolescent was aged 17/8. Our results show that increased economic strain results in a significantly increased likelihood of being a smoker. Moreover, we demonstrated that poor parental mental health and poor child social-emotional outcomes mediate this relationship, as proposed by the Family Stress Model (FSM).

It could be argued that the central theoretical problem of sociology is the identification of the processes through which social structures shape individual behaviour, and the role of individual agency in this. This thesis teases out theoretical explanations for how social structures and agency interact to influence individual health behaviours that are tested empirically.

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List of Abbreviations

HHB – Harmful Health Behaviours

SEP – Social and Economic Position

NCD – Non-communicable Diseases

SDH – Social Determinants of Health

WHO – World Health Organisation

GUI – Growing Up in Ireland

PA -Physical Activity

SEM – Structural Equation Modelling

GSEM – Generalised Structural Equation Modelling

LCA – Latent Class Analysis

FSM – Family Stress Model

IHME - Institute for Health Metrics and Evaluation

RCTs - Randomized Controlled Trials

PPS - Probability Proportionate to Size

BMI - Body Mass Index

TIPI - Ten Item Personality Inventory

ISCED - International Standard Classification of Education

PCG - Primary Caregiver

T1, T2, T3 - Timepoint 1, Timepoint 2, Timepoint 3

ANOVA - Analysis of Variance

SE - Standard Error

GS – Godin Shephard (Leisure Time Questionnaire)

ME - Maternal Education (Differential)

MICE - Multiple Imputation by Chained Equations

TIPI - Ten Item Personality Inventory

EDS - Everyday Discrimination Scale

AIC - Akaike Information Criterion

BIC - Bayesian Information Criterion

FCT - Fundamental Cause Theory

RRR - Relative Risk Ratios

CI - Confidence Interval

LCG – Latent Class Group

HPA - Hypothalamic-Pituitary-Adrenal

CESD-8 - Centre for Epidemiological Studies Depression Scale (8-item)

SDQ - Strengths and Difficulties Questionnaire

OR - Odds Ratio

PSM – Propensity Score Matching

BMI - Body Mass Index

1 Introduction

Social Inequalities in Health

Non-communicable diseases (NCDs; e.g. heart disease, cancer, chronic respiratory disease) represent the leading cause of death globally (see fig. 1.1), with their contribution to all-cause mortality being largest in high-income countries, such as Ireland (see fig. 1.2). Despite their affluence, it is the extent of income inequality in these wealthy countries that contributes to ongoing health and social problems (Wilkinson & Pickett, 2009). It is estimated that cardiovascular diseases account for 18.9 million deaths annually, followed by cancers (9.57 million), chronic respiratory diseases (4.29 million) and the number of individuals afflicted is increasing overtime (WHO, 2020). NCDs are the largest threat to world health and generate considerable economic costs to society through the health and care systems and through lost productivity (Goodchild et al., 2018; Scarborough et al., 2011).

Figure 1.1. Cause of death globally, 2019. Source: IHME, Global Burden of Disease.

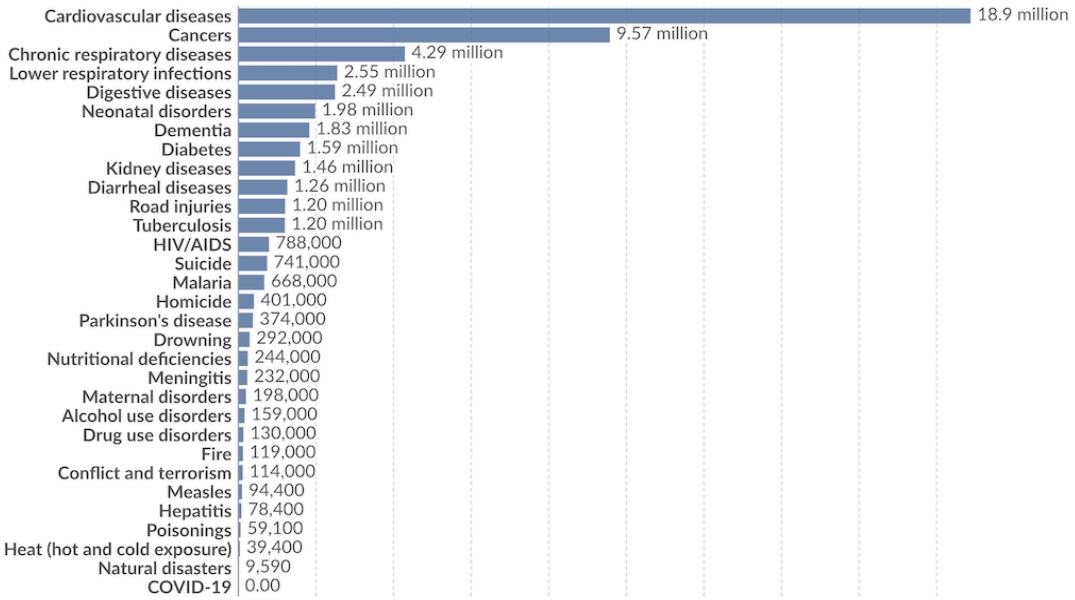
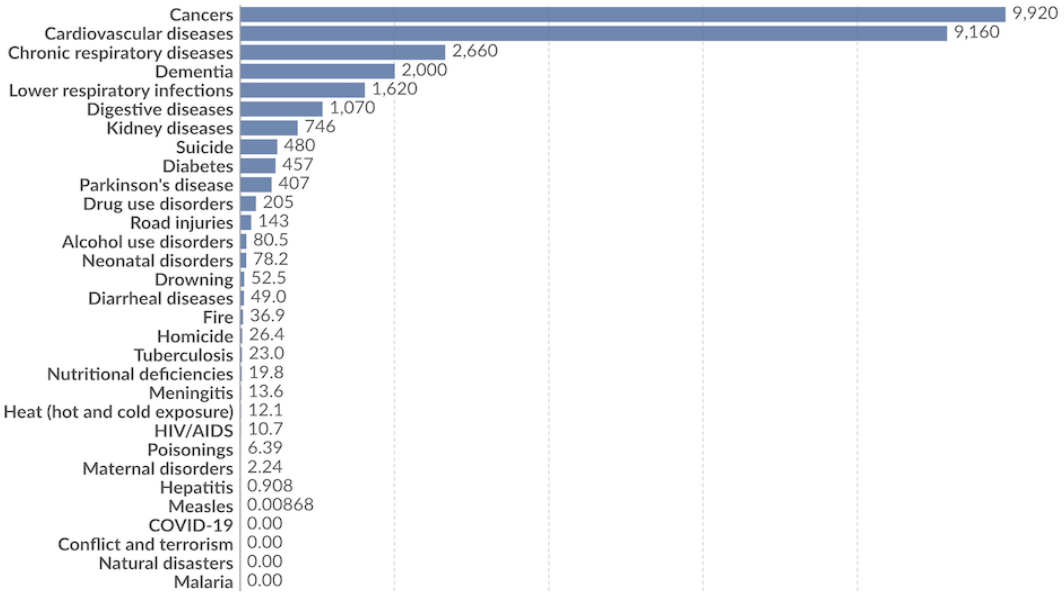


Figure 1.2. Cause of death Ireland, 2019. Source: IHME, Global Burden of Disease.



For some time (Macintyre, 1997; Marmot & Shipley, 1996), there has been no dispute about the fact that disadvantaged sections of society are more likely to suffer disease and early death. The social patterning of health, where individual's economic and social resources determine their risk of disease and mortality, is now one of the most consistent findings in epidemiological research. Although the nature of the relationship varies depending on the measure of socio-economic position used, the risk of disease and mortality consistently increases with disadvantage, and for this reason, the relationship has come to be known as the 'social gradient in health' (Stringhini et al., 2017; Tanaka et al., 2019). The social gradient in NCDs is particularly pronounced; measures of SEP are demonstrably powerful predictors of NCDs (Marmot & Bell, 2019). This gradient is persistent cross-nationally and when comparing time-periods in which the leading causes of death differ (Glymour et al., 2014). This gradient is evident as early as infancy and extends through childhood, adolescence and adulthood (Chen et al., 2006; Glymour et al., 2014).

SEP is typically characterized along three dimensions: education, occupational "status" and income, and major health inequalities prevail along all three dimensions. Although the dimensions are rationally interrelated (e.g. education affects both employment and income), it is not advisable to use these socioeconomic indicators interchangeably and without any serious consideration how these measures of social inequality may implicate different pathways likely to influence health (Bartley, 2022; Muntaner, 2013).

Each measure often constitutes a different set of health-relevant resources with fundamentally different effects on health. For example, parental and particularly maternal higher education is associated with greater health literacy and self-efficacy, both of which are influential in shaping the probability of children engaging in health-promoting behaviours (Prickett & Augustine, 2016). It is now well-established lower education is associated a less healthy diet and less physical activity; hence we adopt this measure for our second chapter that considers trajectories in physical activity by SEP overtime. Income, available to a family, can dissolve financial barriers to health promoting behaviours and healthcare. Income is often seen as the least problematic measure of socioeconomic position (Bartley, 2022), because of this straightforward meaning, with the only issue occurring being directionality or reverse causation, which has been largely demystified by several longitudinal studies which adjusted for health status at baseline. We utilise this measure in fourth chapter to represent how the experience of periodic or persistent poverty means individuals cannot afford the same lifestyle as their more advantaged counterparts, which can lead to social exclusion with implications for all members of a society even those not "excluded." However, we acknowledge the mounting evidence for income distribution rather than absolute income per capita as more

important for health inequality (Pickett & Wilkinson, 2015; Wilkinson & Pickett, 2009). Bartley (2022) suggestion to construct a "plausible narrative" or "chain of risk" between your chosen measure of SEP, to compliment her assertion that one measure is not superior to the others rather inequality of different kinds may influence health in different ways, is applied in our third chapter. We put forward the *plausible narrative* that certain occupational "status" can result in experiences of discrimination, which triggers the processes of social resistance that we test in this chapter. The basis for this narrative is also founded in previous links of occupational "prestige" to health behaviours, where individuals who wish to mark their status using various aspects lifestyle, such as dietary practices and attitudes toward alcohol and smoking etc. by restricting themselves to certain activities and attempting to associate only with others who adopt a similar form of lifestyle. Moreover, a cultural theory of the relationship between prestige and lifestyle makes more sense of the social distribution of smoking than a theory based on income, as smoking costs money but being a non-smoker can be more or less obligatory depending on your social environment (Bourdieu, 1992; Goldthorpe, 1997; Weber, 1946).

Having established this social patterning of health, epidemiological research turned to explanations for inequalities in poor health with a focus on identifying potential modifiable risk factors (Blane et al., 2013). One third of NCDs, in high-income countries, are attributable to four key harmful health behaviours (HHBs): smoking, excessive consumption of alcohol, poor diet and low levels of physical activity (Gavin, 2024; Költő et al., 2020; Morgan et al., 2008). With the exception of poor diet, the prevalence of behaviours such as smoking, underage drinking and physical in-activity is slowly decreasing. However, reductions in rates have been largest for more socio-economically advantaged individuals and this has led to widening disparities between socio-economic

groups. These differentials in health behaviours, between social and economic groups, clearly play a significant role in the formation of health inequalities. This begs the question as to why HHBs vary by SEP? Marmot (2018)has characterised this as the question of the ‘causes of the causes’ of health inequalities. He argues that less is known about how social stratification and social context influences individual’s health behaviours. This thesis aims to identify these ‘causes of causes’ of health inequalities.

Conceptual Framework

As early as 1948 René Sand, co-founder of WHO, raised concern about the 'social roots' of illness (Sand, 1952). However, this line of pursuit was temporarily hindered by meaningful technological advancements, in the form of vaccines and new medicines, that pivoted focus to curative care. The surge of community health initiatives in the 1970s established a renewed concern for the social roots of health. However, the dominance of strategies for privatisation, competitive markets and deregulation to enhance economic activity among western governments in the 1980s meant reduced public expenditure on public health and social security, derailing many such initiatives. For the decades following, literature on health promotion largely focused on the role of individual factors in shaping the distribution of risky health behaviours, and by extension, for explaining differentials across SEP groups (Cockerham, 2005; Marmot, 2018). Speculatively, as a result of the neo-liberal ideology of 'personal responsibility' (Brown & Baker, 2012). The emerging 'individualised health model' (Goldberg, 2012) explained the adoption of harmful health behaviours as resulting from poor information and individual psychological deficits (Delaney & Doyle, 2012; Khosravi et al., 2016; Lawrance & Rubinson, 1986; Richard Layte & Christopher T Whelan, 2009; Van De Ven et al., 2007). Sentiments that were echoed in the literature on criminal psychology in the 1990s, which considered harmful health behaviours and crime to be "gratifying" behaviours that seduces individuals with low self-control and an inability to conceive long-term consequences (Gottfredson & Hirschi, 1990; Pratt & Cullen, 2000).

The publication of the Black report in 1980 (Black et al., 1980) had a major, although delayed, impact on research on explanatory theories for social inequalities in health. The report inspired an intense period of empirical and conceptual research that prompted a

shift away from an individualised health model (Goldberg, 2012) to one concerned with 'force of circumstance' (Bartley, 2016), the Social Determinants of health (SDH) or 'structural model,' which has since dominated the field (Himmelstein et al., 2005; Lalonde, 1974; Marmot & Wilkinson, 2005; Smith, 1999). While crucial evidence emerged in support of the individualised health model, we argue that in focusing on the role of the individual, the important role that structural factors play is overlooked. This thesis considers the interplay of individual and structural explanations for inequalities in health behaviours.

Avoiding disease requires an *agentic* response to public health advice on how to protect against disease, or how to gain a health advantage, as described by the individualised health model. However, one's ability to benefit from public health knowledge relies on access to resources, such as money, knowledge, prestige, power, and beneficial social connections (Phelan et al., 2010). Access to such resources is structurally patterned by SEP in that access increases with social position. Drawing on Weber's (Weber, 1946) concept of 'lebenschancen' (life chances): individuals have agency to the extent that they can make 'life choices' but the options made available vary according to their degree of privilege (i.e. life chances). According to this perspective, the clustering of HHBs according to SEP is not the uncoordinated response of disconnected individuals but the patterned response of social groups to the constraints of their external environments (Cockerham, 2005). For this reason, focusing on individual-level risk factors, such as self-control, may lead to blaming individuals for phenomena that is outside their control (Glymour et al., 2014). David Gordon's 'alternative ten tips for health' parody a purely agent-centred approach to addressing health inequalities: '1. Don't be poor. If you are poor, try not to be poor for too long.' '2. Don't live in a deprived area. If you do, move.'

‘3. Don’t be disabled or have a disabled child.’ ‘4. Don’t work in a stressful low-paid manual job.’

The SDH are defined as the conditions in which people are born, grow, work, live, and age, as well as a wider set of forces and systems that shape health behaviours and outcomes(Marmot & Wilkinson, 2005). In an attempt for further specificity, Bartley (2016) condenses this expansive list of conditions into three conceptual categories: ‘materialist,’ ‘psycho-social’ and ‘cultural’ conditions. We adopt this three-part schema to help explain systematic patterning of HHBs by SEP in this thesis:

> The *materialist* explanation is concerned with the effect of material conditions, such as low income or insalubrious living environments, on health. It emphasises the importance of considering the direct effect of material conditions, as well as the intervening mechanisms that might link material deprivation and health. Higher income enables better access to the means to produce good health, including better access to health care and other healthy consumption. Research, to date, shows that the effect of income on health depends on the population studied, whether income shocks are temporary or permanent, and the period in the life-course in which income changes are experienced(Glymour et al., 2014).

> Bartley puts forward alternative determinants of health beyond material conditions. Her *psycho-social* explanation is concerned with the way people feel (e.g. stressed), and how these feelings may alter body chemistry, and variation in levels of social support to tackle periods of stress. Psychological stress may emerge because of prolonged exposure to conditions such as poor housing, low pay etc., especially where the possibilities to control the situation appear limited.

This explanation represents a shift in emphasis from absolute to relative standards in that it considers the direct psychosocial effects of deprivation as well as the consequences of inequalities(Macintyre, 1997).

Her *cultural* (or behavioural) explanation acknowledges that health behaviours are to some extent subject to individual autonomy, choice and decision-making (Blane et al., 2013) but that cultural context and social norms may also exert influence(Bourdieu, 1992). Other outcomes, such as social integration, may trump personal health goals especially where the health risk behaviour precedes the outcome, as is the case with HHBs and NCDs(Glymour et al., 2014). A simple focus on behaviours as a fundamental theory of health inequalities is problematic when it ignores the symbolic dimensions of HHBs that feed into why individuals in particular social groups adopt them(McCartney et al., 2013).

Fig. 2.1 below represents a summary of the SDH adapted from illustrations provided by Solar and Irwin (2010) and the Commission on the SDoH and the WHO(2008). Fig. 2.2 represents a re-working of fig. 2.1, where the chain of causes is altered. In fig. 2.2, we adopt the same conceptual framework provided by the SDH but consider how material, cultural, psycho-social and biological mechanisms influence inequalities in health behaviours, as opposed to health inequalities generally.

Figure 2.1: Social Determinants of Health Inequalities. Source: CSDH, WHO.

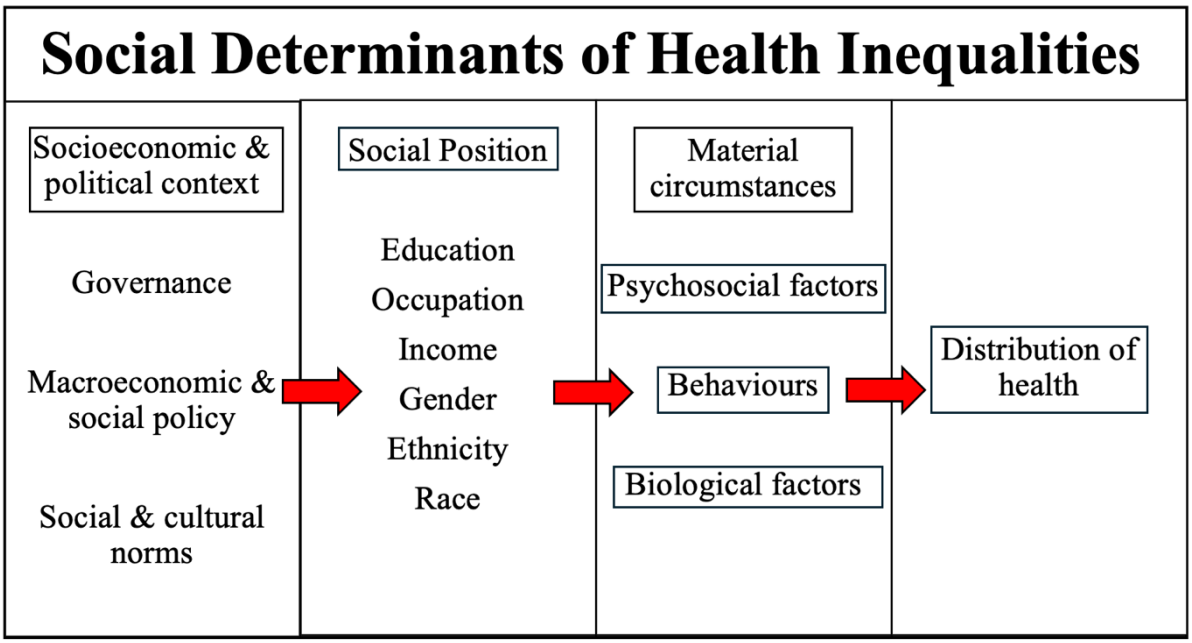
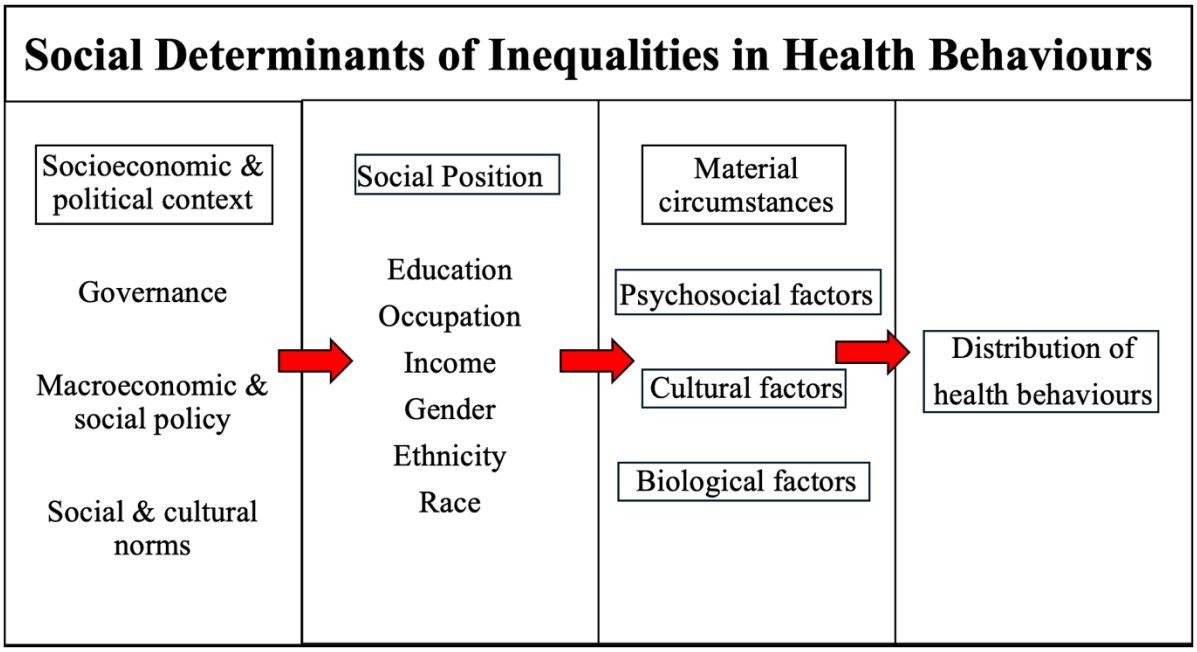


Figure 2.2. Social Determinants of Inequalities in Health Behaviours.



As demonstrated in fig. 2.2, we will investigate the pathways linking structural factors (education, occupation, income etc.) to HHBs using the conceptual tools outlined in this section.

Life-course Approach

Ben-Shlomo and colleagues (Kuh et al., 2003) defined the life-course approach as the study of long-term effects on later health of physical or social exposures during gestation, childhood, adolescence, young adulthood and later adult life. Their aim is to elucidate pathways, possibly biological, behavioural, psychosocial that operate across an individual's life-course to influence the development of disease risk(Ben-Shlomo & Kuh, 2002). This approach is built on the premise that various biological and social factors throughout life independently, cumulatively and interactively influence health and disease in adult life. A life-course approach to understanding health inequalities asks how much of the inequality observed in adult health is due to socially patterned earlier environmental exposures. It encompasses the study of NCDs that are occurring in adulthood but as result of exposure to risk factors earlier in the life-course, such as HHBs and the environmental exposures that led to engagement in HHBs in the first place.

One of the successes of life-course research is providing further clarity on when, in the life-course, intervention may be most effective. The relative importance of an exposure or process can vary with life-course stage (Blane et al., 2013). For example, although much of the adverse health effects of smoking accumulate over decades, smoking initiation typically occurs within a relatively narrow age band in adolescence (Glymour et al., 2014) making this a good target for preventative-style intervention.

There are three dominant life-course models of health, the first being 'chains of risk.' According to this model, there are sequences of linked exposures throughout the life-course that raise disease risk. Exposure to causal factors at earlier life stages form part of long term biological or psychological chains of risk. 'Accumulation of risk,' on the other

hand, is examining the extent to which the number, duration or severity of exposures are causing cumulative damage to body systems, especially as they age and become less able to repair damage(Kuh et al., 2003). Both these models face the methodological challenge that data covering the entirety of the life span, with variables available on the individual and their social context, is often not available.

The third model is the ‘critical or sensitive periods’ model. A critical period refers to a time ‘when intrinsic changes in the organisation of living systems or sub-systems towards increasing complexity, greater adaptivity and more efficient functioning occurs rapidly and may be most easily modified in a favourable or unfavourable direction’ i.e. when there is a limited time window in which an exposure can have adverse or protective effect on development and subsequent disease outcomes, even many years later(Ben-Shlomo & Kuh, 2002). Adolescence is known to be a ‘critical period’ for health behaviour formation(Alberga et al., 2012; Viner et al., 2015). For this reason, this thesis considers the pathways that link SEP and harmful health behaviours at this specific juncture in the life-course, whilst also adopting a temporal perspective that considers earlier risk factors pre-adolescence.

Adolescence is a period when major biological, psychological, and social changes occur, laying the foundations for adult life. It is a unique period of discovery and experimentation, during which health practices emerge or are discarded. There are large cross-national consistencies in the emergence of ‘problem-prone’ behaviour and ‘experimentation’ during this period (de Looze et al., 2015). Patterns of health-compromising behaviours established during adolescence tend to track into adulthood and are said to be 'crystallized' after a final stage of movement just prior to age 19 (van

Nieuwenhuijzen et al., 2009). For this reason, research exploring potential causal factors for persistent HHBs among adolescents is key if we are to eliminate related health disparities that are evident in adult populations.

The Research Gap

The first major research gap is neatly referred to by Marmot (2018) as 'the causes of the causes' of health inequalities. As outlined in the introduction of this thesis, less is known about why HHBs vary across SEP in a structured manner. This is a worthy research pursuit considering HHBs play a significant role in the formation NCDs, and inequalities in NCDs in high-income countries(Gavin, 2024; Költő et al., 2020; Morgan et al., 2008; Pampel et al., 2010a; Petrovic et al., 2018).

Studies in the fields of sociology, psychology, social epidemiology have empirically tested the processes that mediate the relationship between social position and health, some specifying and testing the social and cultural processes(Adler et al., 1994; Blane et al., 2013). In terms of health behaviours, although the social patterning has been widely demonstrated(Marmot & Wilkinson, 2005), there is much less research on *how* social and economic context exerts influence. Hitherto, a small number of researchers have unpacked the role of neighbourhoods(Belon et al., 2014; Carroll-Scott et al., 2013; Sharkey & Faber, 2014; Staatz et al., 2021), social networks(Guo et al., 2015; Haas & Schaefer, 2014), discrimination and resistance (Chen & Yang, 2014; Roni Factor et al., 2013) and early life factors(Layte et al., 2023). More research is required testing the intervening social and cultural mechanisms that link childhood disadvantage and engagement in HHBs in adolescence.

One strand of life-course research centres on identifying 'critical or sensitive' periods (see previous section 'life-course approach'). This strand calls for research that can pin-point when an intervention is most helpful or harmful, and at what level (individual, household, neighbourhood, school, social structure) it should take place(Blane et al., 2013).

Adolescence has been identified as a critical period for the formation of health behaviours(Clark et al., 2007), but it is still unclear when risk factors emerge that heighten the likelihood of engaging in HHBs, and the settings in which they occur, e.g. family, school or neighbourhood. This thesis will help to address this research gap.

Determining a causal relationship between any aspect of SEP and health has its challenges (e.g. feedback loops, non-linearities, interactions) that make it challenging to isolate exposures(Glymour et al., 2014). Randomized controlled trials (RCTs) are the gold standard for causal inference because they can rule out the possibility of reverse causation or confounding. For practical and ethical reasons, this is often not possible in research on health behaviours. However, natural experiments offer a promising alternative to identify causal relationships, using observational data, and present an opportunity to address this gap in the literature. In the context of this thesis, it was possible to leverage economic change at the national level to isolate the impact of the economic strain on health behaviours, and to test the pathways that mediate the relationship.

There have been various expressions of the culture of poverty (Lewis, 2017) and how it impacts behaviour among people in poverty. Previous research on health inequalities has theorised that ‘culture’ can influence health behaviours within social groups (Bartley, 2016; Durkheim, 2023; Murray, 1999) and suggests that sub-populations can create sub-cultures that have potentially negative implications for health outcomes. Moreover, cultural patterns, whether destructive or not, are often inter-generationally entrenched and resistant to remediation. There is little theoretical, and less empirical research on the processes through which culture influences health behaviours. In fig. 2.2. above, we

distinguish culture as its own determinant of inequalities in health behaviours, to illustrate our aim to address this gap in the literature.

Some public health initiatives to address health inequalities related to NCDs have been shown to disproportionately benefit the better-off within societies, thereby improving population health but with the unintended consequence of increasing inequalities(Bleich et al., 2012; Link & Phelan, 2005; Thomson et al., 2018). In the context of this thesis, evidence points to larger reductions among more socio-economically advantaged groups for HHBs(Hiscock et al., 2012; Lawlor et al., 2003). For this reason, there is a call for research that focuses specifically on how to improve health behaviours for socio-economic disadvantaged groups(Marmot, 2015). This research will shed light on this group's seeming immunity to public health intervention.

Aims and Objectives

The aim of this research is to establish some of the root causes of social inequalities in health behaviours i.e. the 'causes of causes' of health inequalities. The objectives of are five-fold:

1. Establish the social patterning of key harmful health behaviours (smoking, underage drinking, poor diet, physical in-activity and excessive screen-time) in the Irish context.
2. Utilise advances in social theory to draw out potential mechanisms through which SEP might determine level of engagement in harmful health behaviours.
3. Test empirically the comparative role of individual-level and structural-level explanations for the social patterning of harmful health behaviours.
4. Test empirically for distinctive risk profiles for individuals with similar patterns of engagement with harmful health behaviours.
5. Isolate the causal effects of economic circumstances on harmful health behaviours using a natural experiment that leverages national economic changes in the Irish context.

Structure of Thesis

The remainder of this thesis is structured as follows. The empirical part of the thesis (Chapter 2,3,4 & 5) is made up of four papers that shed light on inequalities in health behaviours.

The first paper sets out PA trajectories of young people from age 9 to 17/8 by maternal education level, drawing on data from the first, second and third waves of GUI. The chapter employs linear spline multi-level models to estimate these physical activity trajectories. Linear spline multi-level modelling is a useful approach for deriving individual summary measures of growth as it overcomes several data issues: co-linearity of repeated measures and bias due to missing data. Whilst adjusting for individual differences in personality, BMI and health status, we then empirically assessed the role of the child's family, school and neighbourhood environment in shaping these trajectories, using the difference method(Tyler J VanderWeele, 2016). Here the first model is simply a regression of the outcome Y (physical activity) on the exposure A (social position) and covariates C, and the coefficient is interpreted as the total effect of the exposure A on the outcome Y. The subsequent models include mediators, such as variables that represent family environment. If the exposure coefficient of the first regression (without the mediators) reduces in the subsequent regressions (with mediators), this is indicative of mediation i.e. the mediator explains some of the effect of the exposure on the outcome. The exposure coefficient itself (in the subsequent regressions) is taken as a new measure of the direct effect (DE) as it remains even when controlling for our chosen mediators(Tyler J VanderWeele, 2016).

In the second paper we redirect our focus to social gradients in smoking. We examine three theories about the processes through which SEP influences the probability of smoking and compare the effectiveness of individual-level theories concerned with personality and psychological traits and structural-level theories on social resistance and social networks. Also drawing on data from the first three waves of GUI, we utilised generalised structural equation modelling (GSEM) to test the hypothesised causal pathways through which family SEP at age 9 contributes to the probability of smoking at age 17/8. GSEM relaxes the constraints of structural equation models (SEM) to allow for our binary dependent variable (smoking status). Path analysis estimates the relationship between SEP and our mediating variables and the effect of the mediators on smoking probability. If both equations reveal significant effects, we have evidence of a statistically significant mediating pathway. Although path analysis cannot demonstrate causality (and significant results should be interpreted as correlational), using longitudinal data it is possible to test if the ordering among sets of variables from our hypothesised causal pathways is plausible.

The third paper considers multiple health behaviours: physical activity, poor diet, screentime, smoking and alcohol consumption. Using latent class analysis (LCA) we identify groups of individuals who share similar clusters of our chosen health behaviours. LCA is a data-driven approach, identifying the optimal subdivision of the data using empirical patterns rather than pre-existing theory. Once distinct clusters of our behaviours were identified, we used multi-nominal logistic regression to examine whether young people with similar patterns of behaviours have distinctive risk profiles. Specifically, we test theories on social resistance from paper two and established theories on social

exclusion and its effect on healthy consumption. Like papers one and two, this paper also employs the first three waves of GUI data.

The fourth and final paper focuses on estimating the causal effect of childhood family economic context on smoking initiation. Leveraging the experience of the Great Recession in Ireland, we adopted quasi-experimental methods to isolate the role of family economic strain from other often correlated socio-demographic factors that influence smoking probability in adolescence. Firstly, we used propensity score matching techniques to account for differential selection into our exposure (the recession). We then examined the effects of recession on smoking risk by comparing adolescent smoking risk for families with a similar likelihood of being exposed to the effects of the recession (the propensity score) but who vary in their actual experience of recession, using fixed-effects logistic regression models. Secondly, we examined whether the psycho-social consequences of economic strain, as hypothesized by the FSM, explained the differentials in smoking risk in adolescence that emerged as a consequence of the family's experience of the recession. As in the previous chapters, the study relied on data from the first three waves of GUI.

Chapter 6 provides an overview of the main findings for each empirical chapter, summarises their contribution to the field and concludes the thesis. Within each chapter, the limitations of the corresponding paper are outlined separately.

Data

The epistemological foundations of our strategy and analysis are deductive in that we use theory to generate testable hypotheses. As we use quantitative survey data, by default we adopt the ontological paradigm of objectivism, where social phenomenon confronts us as external factors that can be objectively measured.

This study used data from Growing Up in Ireland (GUI) data, a nationally representative cohort study of young people living in the Republic of Ireland. The GUI commenced when participants were aged nine, with 8,568 participants. Data collection for wave one took place between September 2007 and June 2008. Data was subsequently collected when children were aged 13 (between August 2011 and February 2012) with an 88% retention rate (n=7,525), aged 17/18 (between November 2015 and September 2016) with a 73% retention rate (n=6,216) and aged 20 (between August 2018 and June 2019) with an 61% retention rate (n= 5,190).

This longitudinal design, which involves interviewing the same participants on several occasions, is one of the key assets of this dataset. Longitudinal data tracks the development of the young person, meaning we can observe trajectories in our variables of interest over time and by socio-economic group. This enables us to locate the onset, or acceleration, of engagement in HHBs across childhood and adolescence, a critical period for these behaviours. Moreover, we can examine the effects of earlier exposures to risk factors for later development of HHBs, as contended by the life-course approach.

The sample was selected through a two-stage clustered sampling method within the Irish primary school system, with the school as the primary sampling unit and the age eligible

young people attending the school as secondary sampling units. Probability proportionate to size (PPS) sampling was used to select a representative sample of 910 primary schools (82% response rate) with 8,568 families (57% response rate) from these schools agreeing to participate. Interviews were carried out with young people and parents at home and with young people, teachers and principals at school, by trained interviewers.

The nature of the data collection strategy, whereby children are nested within schools and families, lends itself to addressing our research objectives that are concerned with identifying the determinants of health behaviours that exist in the young person's social environment. This, coupled with the multi-disciplinary coverage of the questionnaires, which survey family, school, and neighbourhood context, enables consideration for the child and their wider social context. Not only can we identify the risk factors that emerge and heighten the likelihood of engaging in HHBs but also the settings in which they occur, e.g. family, school or neighbourhood. Access to data from multiple settings, as well as the young person's individual characteristics allowed us to compare individual-level and structural-level influences on behaviour.

Although the study was not planned as such, baseline data collection occurred as the economic boom of the previous 12 years was coming to an end and just prior to the onset of the Great Recession in the first quarter of 2009. The second wave of data collection occurred at the trough of the same recession and wave three occurred as the economy was recovering strongly. As discussed in the previous section, in paper three of this thesis we exploit the fortuitous timing of data collection to carry out a quasi-experiment.

A limitation of the data was a lack of repeat observations across studies, making longitudinal analysis using repeated observations impossible in some cases. Levels of missing data across variables were low, on average, but non-response was a feature of all surveys. By using tracing procedures, it was possible to establish if inter-wave attrition is systematically associated with characteristics measured in our survey(Thornton et al., 2010). This has the potential to bias our analysis and for this reason, missing values were imputed for analysis, where necessary. The data were also reweighted for analyses to take account of the original sample error and subsequent attrition using a range of factors and a minimum distance algorithm. Moreover, when working with cohort studies it is important to consider both the period in which the study took place and the age of participants, to identify any possible external cohort effects e.g. advent of new technologies or growing omnipresence of fast food.

2 The role of family, school and neighbourhood in explaining inequalities in physical activity trajectories between age 9 and 18.

Introduction

Non-communicable diseases (NCDs) are the largest cause of mortality globally, accounting for 71% of all deaths, despite the common modifiable risk factors (tobacco and alcohol use, unhealthy diet and physical inactivity) being well-established (WHO, 2020). The inverse association between social and economic position (SEP) and NCDs, known as the social gradient in health, is one of the most robust findings in epidemiology. Research suggests that health behaviours are a major determinant of the social gradient in health, with robust evidence showing that these behaviours are also differentially distributed by SEP (Petrovic et al., 2018). Less is known about what Marmot (2018) has referred to as 'the causes of the causes' of health inequalities, that is why health behaviours vary across SEP in a structured manner. Models of behaviour change often focus on the role of knowledge, beliefs and intentions as the main determinants of poor health behaviours (Ogden, 2016), but there is a long-standing literature that argues that health behaviours must be understood in their social and economic context and viewed as a response to conditions imposed by social structures (House, 2002). It has also been argued that adult health behaviours need to be viewed through a life-course perspective, as the cumulative response of individuals and groups to circumstances in which they live (Lynch et al., 1997). In response, this paper focuses on social gradients in physical activity (PA), specifically the role of child environment in shaping the young person's PA trajectory from middle childhood to early adulthood (from age 9 to 17/18). Whilst adjusting for individual differences in personality BMI and health status, we empirically assess the role of the child's family, school and neighbourhood environment in shaping

PA over a crucial period of development. Social gradients in PA have been consistently found among adolescents (J Inchley et al., 2020; Inchley, Currie, Young, et al., 2016) and research has shown that there is a sharp decline in PA during adolescence between 11 and 15yrs (Dumith et al., 2011; Farooq et al., 2018; Rovio et al., 2018). Adolescence has been described as a critical period for health behaviour formation, with habits established during adolescence likely to continue into adulthood (Alberga et al., 2012).

Family-level Mechanisms

The family is a primary site of socialisation and both the behaviours of parents and the specific parenting practices that parents adopt can have an influence on children's levels of PA. Parental level of PA can act as a role model for their children, but parents also have an important 'gatekeeping' role by introducing their child to their own sporting interests. There is a proven link between having a parent that plays sport the likelihood that their child will play sport (Lunn, 2006; McMinn et al., 2008). Differences in parenting practices, across households, are also a potential pathway for the intergenerational transmission of health behaviours. Lareau (2018) ethnographic research demonstrated that high SEP parents are more likely to engage in what she calls 'concerted cultivation' by enrolling their children in various enrichment activities (e.g. learning an instrument, organised team sport), whereas lower SEP parents are more likely to adopt a strategy of 'natural growth,' where young people get more discretion and less of their free time is structured. Unstructured and unsupervised time is an opportunity for children to engage in an array of tempting sedentary pursuits that compete for young people's time and have been shown to lead to physically inactive lifestyles (Pearson et al., 2014).

School-level Mechanisms

Young people from different social and economic backgrounds are not randomly distributed across schools, and schools vary in terms of the type and amount of funding they have access to. Self-reported principal data, in the Irish context, demonstrates that some schools have access to better quality and a greater variety of school sporting facilities (Fahey et al., 2005; Woods et al., 2010; Woods et al., 2019). Variation in sport facilities may contribute to explaining the social gradients in PA, given the long-term relationship between attending a school with inadequate sports facilities and the likelihood of being physically inactive in adulthood(Black et al., 2019). Mode of transport to and from school may contribute to gradients in PA. Children with an active commute can increase their PA levels, and improve their fitness, without the need for planned sports or activities but little is known about the distribution of active commute across SEP groups (Heelan et al., 2005; Murtagh et al., 2016).

Neighbourhood-level Mechanisms

The emergence of socio-ecological models of health behaviours (Sallis et al., 2015) has led to an abundance of literature successfully demonstrating the extent to which a young person's physical environment impacts their level of PA (McGrath et al., 2015; Sterdt et al., 2014). A number of different dimensions of local area have been shown to impact on PA. Lower social and economic groups may be more likely to be confronted with these barriers to PA (Handy et al., 2002). This paper will focus on examining the role of neighbourhood safety and neighbourhood social and physical disorder, such as crime, public loitering, vandalism, graffiti and abandoned buildings, in reducing levels of PA,

particularly among low SEP female adolescents, as the perceived risk to physical safety may be heightened in this group (Dulin-Keita et al., 2013).

Parental Resources: Education & Income

The association of education with health behaviours is now well established with lower education associated with a greater prevalence of smoking, higher alcohol consumption, a less healthy diet and less physical activity (Cavelaars et al., 2000; Droomers et al., 1999; Gidlow et al., 2006). Parental and particularly maternal higher education is associated with greater health literacy and self-efficacy, both of which are influential in shaping the probability of children engaging in health-promoting behaviours (Prickett & Augustine, 2016). The financial resources available to a family may have a direct bearing on the participation of the young person in PA. Lower income and savings may directly restrict the ability of the family to pay for the young person's membership of sports clubs, buy sports equipment/clothing or provide transport to sports events. Previous research has shown a clear effect of family income on the level and type of PA that adolescents participate in and whether they are members of sports clubs (Kantomaa et al., 2007).

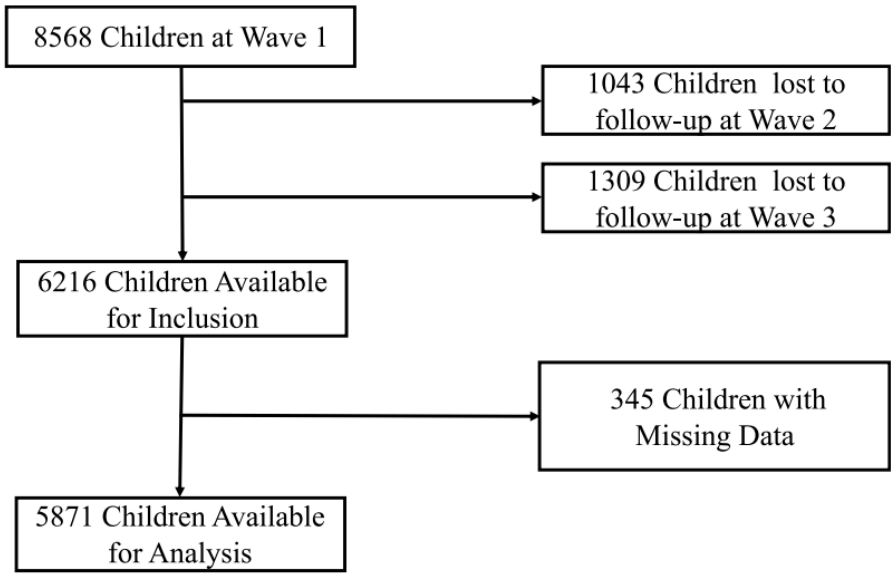
Material & Methods

Sample

(see Data section in the Introduction)

The loss of participants to follow-up raises concerns that the pattern of attrition may be selective and thus introduce systematic bias into our analyses. The data were reweighted (Murphy et al., 2018; Thornton et al., 2010) for analyses to take account of the original sample error, and subsequent attrition using a range of factors and a minimum distance algorithm. The large sample sizes, the probability samples and the calibration to ensure representativeness mean that the results can be generalised to the population. The final reweighted sample is representative of young people who were residing in Ireland at 9 years of age in 2006, and who continued to live in Ireland at 17/18 years of age in 2016. Analyses were restricted to participants who had available data at all three waves on our variables of interest. This reduced our sample to 5,871 (see Fig. 3).

Figure 3. Sample Exclusion Criteria and Final Case Base for Analysis.



Measures

Dependent variable

PA levels were calculated using the Godin-Shephard Leisure Time Exercise Questionnaire (GS) (Godin & Shephard, 1985) at all three timepoints. The primary caregiver (PCG) reported the number of days out of the previous 14 that the young person had engaged in 'hard' or 'light' exercise for at least 20 min at T1 & T2, and the young person (YP) reported at T3. Hard exercise was defined as 'enough to make him/her breathe heavily and make his/her heart faster'. Light exercise was defined as 'not hard enough to make him/her breathe heavily and make his/her heartbeat fast.' The total activity score is computed by weighting the frequency of hard and light exercise per week by values based on the intensity of the activity (e.g. "x3" points for frequency of light exercise). These values were established by the developers. The total score reflects the individual's level of physical activity and is calculated by summing up the weighted scores. For analyses, it was necessary to categorise the activity score into tertiles: 'low' 'moderate' and 'high,' where low levels of PA were scores <69. As a reference point the mean level of PA is 90.28 (43.22). The GS has demonstrated concurrent validity with measures of maximum oxygen intake and muscular endurance (Godin et al., 1986) and construct validity (Godin & Shephard, 1985; Zelener & Schneider, 2016) and test-retest reliability.

Individual-level control variables

The YP's personality was measured at T2 by PCG report using the Ten Item Personality Inventory (TIPI) and was assumed to be fixed across the three timepoints. TIPI consists of five subscales: agreeableness, extraversion, conscientiousness, emotional stability, and openness to experience(Gosling et al., 2003). The significant associations between personality and PA, specifically agreeableness conscientiousness and extraversion are evidenced elsewhere(Rhodes & Smith, 2006; Wilson & Dishman, 2015). The PCG

reported on whether or not the YP had a chronic illness (‘yes’/’no’) at all three timepoints. This variable was included to control for instances where participants could not be active for health reasons. BMI was recorded at all three timepoints by trained interviewers, who carried out height and weight measurements. Standard sex and age specific BMI cut-off points were used to classify participants as ‘normal weight,’ ‘overweight’ ‘obese’ or ‘missing’(Cole et al., 2000). BMI has a reciprocal relationship with PA, where physical inactivity can lead to increased BMI but increased BMI has also been shown to be an independent significant negative predictor of PA(Pietiläinen et al., 2008). The final control variable was reported by the PCG at T1 indicating whether the YP lives in an ‘urban’ or ‘rural’ area, rural was defined as ‘open country’ or ‘a village’ with a population of less than 1,500 persons(CSO, 2019). 31.4% of Ireland’s population live in a rural area, which is above average within the EU making the urban rural distribution an important feature of Irish life, especially for health behaviour research as rural adolescents were more likely to be obese than those in urban areas(Li et al., 2017).

Independent variable: maternal education

Maternal highest reported qualification was coded using the International Standard Classification of Education (Statistics) and collapsed into four groups: lower secondary (junior certificate) (ISCED 0–2), upper secondary (leaving certificate) (ISCED 3), post-secondary (diploma) (ISCED 4 & 5) and tertiary, i.e. bachelor’s degree or above (ISCED 6–8).

Income

The PCG reported on household income from each household member at all three timepoints. In order to make meaningful comparisons between households, on their income, household size and structure was taken into account by assigning a weight to each household member to create an ‘equivalised’ total disposable income (after tax and

welfare transfers). The first adult is assigned a weight of 1 with a weight of 0.66 allocated to all subsequent adults and 0.33 to each child (14 years or less). These weightings are identical to those used for the Irish official income poverty line (Roantree et al., 2021). Equivalised income was ranked and transformed into income quintiles with the addition of a category for missing income.

Family-level variables

The YP's attendance ('yes'/'no') at several activities was reported by the PCG at T1 and the YP at T2 & T3. Activities included: dance, music, drama, art, youth club, homework club, scouts, guides, and organised sport (with an instructor). With the exception of organised sport, the activities were combined into one 'enrichment activities' variable. The combination of these activities is used to measure the extent to which parents practise concerted cultivation (Lareau, 2018). Organised sport was kept separate so as to not artificially inflate the importance of concerted cultivation in explaining the relationship between maternal education and PA. Minutes on a normal weekday that the YP spent doing 'unstructured activities' such as watching TV, playing video games and on the computer was reported by the PCG at T1 and the YP at T2 & T3. These variables were combined. This variable is used to represent the degree to which parents practised accomplishment of natural growth, as demonstrated elsewhere(McCoy et al., 2012). PCG's also reported the regularity of their own exercise at T2. They classified themselves as 'very physically active,' 'fairly physically active,' 'not very physically active' or 'not at all physically active.'

School-level variables

School principals reported on the adequacy of the school's sporting faculties in primary (T1) and secondary (T2 & T3) school. They indicated whether they were 'poor,' 'fair,' 'good' or 'excellent,' when compared to other schools in Ireland. The categories were recoded to combine conceptually similar categories and resulted in two new categories: 'poor/fair' and 'good/excellent.' The PCG reported how their child usually travelled to primary (T1) and secondary (T2 & T3) school. There were six response categories: 'walks' 'public transport' 'school-bus' 'car' 'rides a bicycle' or 'other.' Walking or cycling was classified as active commuting, due to the small number of cyclists, both bus categories (public and school) were also combined and the 'other' category was excluded from analyses.

Neighbourhood-level variables

The PCG reported on their perceived safety of their neighbourhood at all three timepoints. Using a four-point scale from strongly agree to strongly disagree, they reported on whether: (1) it is safe to walk alone in this area after dark (2) it is safe for children to play outside during the day in this area (3) there are safe parks, playgrounds and play spaces in this area. Parental-perceived neighbourhood safety is significantly associated with encouraging outdoor physical activity (Kimbro et al., 2011; Kimbro & Schachter, 2011) and less screen time(Hunter et al., 2020). The PCG also reported on the quality of the neighbourhood. Using a four-point scale, they stated how common the following occurrences were: (1) rubbish and litter lying about (2) homes and gardens in bad condition (3) vandalism and deliberate damage to property (4) people being drunk or taking drugs in public. Lower SEP families were much more likely to report unfavourable physical conditions in their local neighbourhood(Williams et al., 2009).

Analysis strategy

First, we carried out descriptive statistics (frequencies and percentages, means and standard deviations) on our dependent and potential mediating variables by maternal education categories at each timepoint. Pearson's correlation coefficient, independent sample t-tests and one-way ANOVAs, where appropriate, were used to examine statistically significant differences across maternal education categories. Levene's statistic was used to determine that the assumption of the equality of variance was met, and if the ANOVAs were valid. Where the assumption of the equality of variance was not met Welch's F was used instead. Multi-level linear spline models(Laura D Howe et al., 2016), with appropriate study weights, were used to estimate PA trajectory differentials by maternal education level between ages 9 and 17/8. To account for the nesting of PA measures within individuals, each model includes a random intercept (μ0j) for each young person, which follows a bivariate normal distribution and measurement error is explicitly modelled overtime (e0ij). The random intercepts model for linear change in PA can be written as:

$$Yij = \beta0 + \beta1 \text{ x (age)}ij + \beta2 \text{ x (Maternal education)}j + \beta 3$$
$$\text{x (Maternal education)}i \text{ x (Age) } ij + \mu0j + e0ij$$

Here, β0, β1, β2 and β3 were 'fixed' coefficients, which represent the average intercept across the sample and the average slopes for age, maternal education plus the interaction of age and maternal education. μ0j is the 'random' coefficient, which represents the individual's deviation from the average intercept. e0ij represents the observation-level measurement error. We quantified the role of different groups of predictors in accounting for the maternal education differential by calculating the change in β1+β2+β3 with the

addition of each group of predictors to the model. Model 2 (baseline and controls) was our reference point. Theoretical justification was used to assign variables to each of the models (income, family, school and neighbourhood). Models were stratified by sex. All analyses were performed in Stata/SE 16. P-values less than 0.05 were considered statistically significant.

Results

Maternal education and PA

Fig. 4 (see graph below) demonstrates the clear social gradient by maternal education in levels of PA, measured using the GS scale. The differences in average levels of PA between the four maternal education groups were statistically significant ($p < 0.001$) at age 9, 13 and 17/18. The differential between the highest and lowest maternal education group is widest at age 13 (19.6) compared to age 9 (5.9) and age 17/18 (8.9). Fig. 4 also demonstrates the clear age gradient in levels of PA over the 10 year period, where activity levels were highest at age 9 (114.0), decreasing at age 13 (80.8) and lowest at age 17/18 (74.1). As an indicator into whether the significant differences in PA by maternal education groups are meaningful, we carried out a one-way ANOVA to compare group means. As expected, there were statistically significant difference in levels of PA between the different maternal education groups [$F(3,15258)=36.13$, $p<.001$]. This, however, represents a small effect (eta squared=0.01) as suggested by Cohen (1988) interpretation of effect size.

Maternal education and family, school and neighbourhood-level mediators

Table 1 (below) demonstrates the social patterning of our variables of interest. Families with low levels of maternal education were also likely to have low levels of income. The family-level mediating variables show a clear social patterning: young people whose mothers have lower levels of education report more adverse behaviours and outcomes at each timepoint. Lower levels of maternal education were associated with YP's non-participation in organised sport and non-attendance at other structured activities, and spending more time doing unstructured activities. Highly educated mothers also tended to be 'very' or 'fairly' physically active themselves, compared to 'not very physically active'

and ‘not physically active.’ School-level mediating variables do not follow a clear social patterning where low levels of maternal education were associated with more adverse behaviours and outcomes. In terms of commuting to school, participants whose mothers had a lower or higher secondary qualification education were more likely to be active commuters, a positive health behaviour, than participants whose mothers had tertiary education. However, participants whose mothers had low levels of education were still less likely to travel to school by car and more likely to get the bus, when compared to participants whose mothers had higher levels of education. There were no significant differences in the perceived adequacy of school sporting facilities across maternal education groups. The neighbourhood-level mediators, perceived neighbourhood disorder and perceived neighbourhood safety, were both significantly ($p < 0.001$) associated with maternal education. Young people whose mothers had lower levels of education being more likely to live in areas with higher levels of social disorder and lower levels of neighbourhood safety.

Figure 4. Levels of PA on the GS scale by maternal education categories.

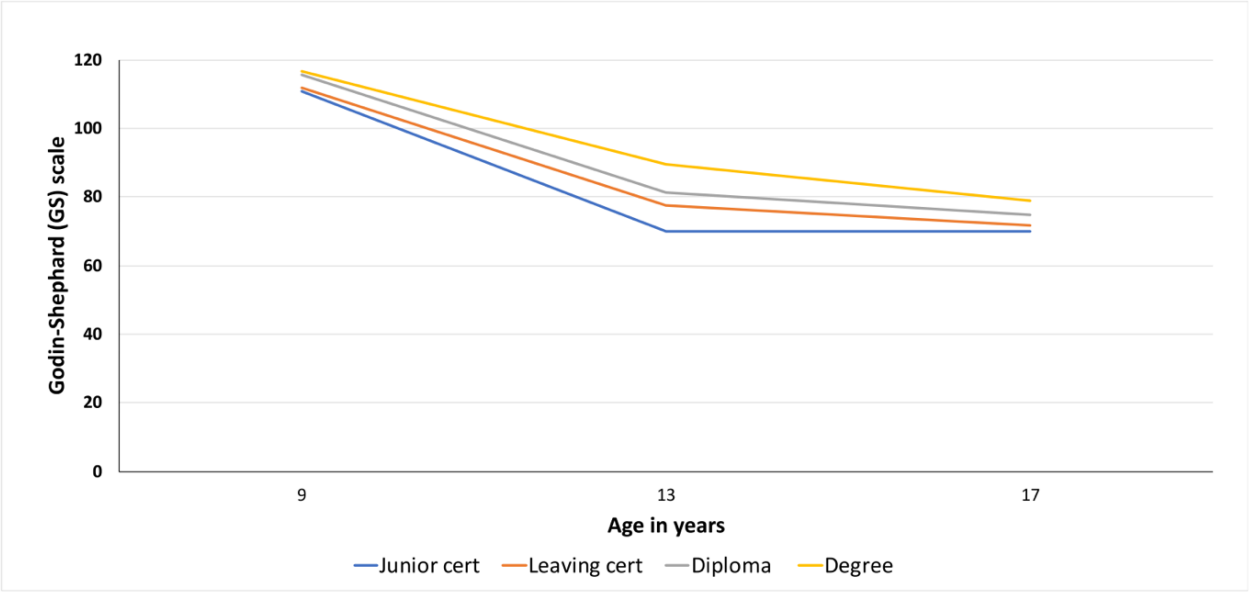


Table 1. Mediating variables by maternal education categories.

	Junior Cert. (N = 942)	Leaving Cert. (N = 1909)	Diploma (N = 1587)	Degree (N = 1778)	Total (N = 6216)	p-value
Income (age 9)						
lowest	259 (27.5%)	210 (11.0%)	119 (7.5%)	57 (3.2%)	645 (10.4%)	<0.001
2nd	240 (25.5%)	380 (19.9%)	212 (13.4%)	149 (8.4%)	981 (15.8%)	
3rd	181 (19.2%)	438 (22.9%)	316 (19.9%)	232 (13.0%)	1167 (18.8%)	
4th	128 (13.6%)	405 (21.2%)	430 (27.1%)	406 (22.8%)	1369 (22.0%)	
highest	71 (7.5%)	329 (17.2%)	398 (25.1%)	814 (45.8%)	1612 (25.9%)	
missing	63 (6.7%)	147 (7.7%)	112 (7.1%)	120 (6.7%)	442 (7.1%)	
Income (age 13)						
lowest	259 (29.2%)	311 (16.7%)	163 (10.6%)	93 (5.3%)	826 (13.7%)	<0.001
2nd	237 (26.7%)	320 (17.2%)	224 (14.5%)	139 (8.0%)	920 (15.2%)	
3rd	170 (19.2%)	381 (20.4%)	275 (17.8%)	223 (12.8%)	1049 (17.4%)	
4th	100 (11.3%)	427 (22.9%)	394 (25.5%)	409 (23.4%)	1330 (22.0%)	
highest	56 (6.3%)	288 (15.4%)	373 (24.2%)	768 (44.0%)	1485 (24.6%)	
missing	64 (7.2%)	138 (7.4%)	114 (7.4%)	113 (6.5%)	429 (7.1%)	
Income (age 17)						
lowest	270 (28.7%)	249 (13.0%)	149 (9.4%)	72 (4.0%)	740 (11.9%)	<0.001
2nd	228 (24.2%)	354 (18.5%)	229 (14.4%)	137 (7.7%)	948 (15.3%)	
3rd	164 (17.4%)	412 (21.6%)	290 (18.3%)	249 (14.0%)	1115 (17.9%)	
4th	116 (12.3%)	369 (19.3%)	345 (21.7%)	418 (23.5%)	1248 (20.1%)	
highest	64 (6.8%)	316 (16.6%)	394 (24.8%)	744 (41.8%)	1518 (24.4%)	
missing	100 (10.6%)	209 (10.9%)	180 (11.3%)	158 (8.9%)	647 (10.4%)	
Maternal PA						
Very physically active	178 (20.1%)	357 (19.1%)	316 (20.5%)	333 (19.1%)	1184 (19.6%)	<0.05
Fairly physically active	472 (53.3%)	1067 (57.2%)	850 (55.1%)	1028 (58.9%)	3417 (56.6%)	
Not very physically active	188 (21.2%)	383 (20.5%)	331 (21.5%)	332 (19.0%)	1234 (20.4%)	
Not at all physically active	48 (5.4%)	58 (3.1%)	46 (3.0%)	52 (3.0%)	204 (3.4%)	
Children's activities (age 9)						
0	430 (45.6%)	701 (36.7%)	536 (33.8%)	453 (25.5%)	2120 (34.1%)	<0.001
1	374 (39.7%)	930 (48.7%)	807 (50.9%)	1041 (58.5%)	3152 (50.7%)	
2+	138 (14.6%)	278 (14.6%)	244 (15.4%)	284 (16.0%)	944 (15.2%)	
Children's activities (age 13)						

0	378 (42.8%)	777 (41.8%)	617 (40.2%)	609 (35.0%)	2381 (39.5%)	<0.001
1	351 (39.7%)	739 (39.7%)	616 (40.1%)	744 (42.8%)	2450 (40.7%)	
2+	155 (17.5%)	345 (18.5%)	303 (19.7%)	387 (22.2%)	1190 (19.8%)	
Children's activities (age 17)						
0	605 (64.2%)	1194 (62.5%)	956 (60.2%)	997 (56.1%)	3752 (60.4%)	<0.001
1	247 (26.2%)	567 (29.7%)	491 (30.9%)	596 (33.5%)	1901 (30.6%)	
2+	90 (9.6%)	148 (7.8%)	140 (8.8%)	185 (10.4%)	563 (9.1%)	
SBSB (age 9)						
mean (SD)	205.4 (122.2)	186.2 (104.5)	173.6 (103.5)	154.4 (100.2)	176.8 (107.3)	<0.001
SBSB (age 13)						
mean (SD)	99.9 (63.8)	90.7 (57.2)	86.5 (60.7)	72.7 (52.8)	85.8 (58.6)	<0.001
SBSB (age 17)						
mean (SD)	271.8 (141.5)	251.7 (135.9)	229.6 (128.5)	210.8 (123.6)	236.7 (132.9)	<0.001
Organised Sport (age 9)						
No	320 (34.0%)	391 (20.5%)	270 (17.0%)	260 (14.6%)	1241 (20.0%)	<0.001
Yes	621 (66.0%)	1514 (79.5%)	1317 (83.0%)	1518 (85.4%)	4970 (80.0%)	
Organised Sport (age 13)						
No	267 (30.6%)	417 (22.5%)	282 (18.4%)	224 (13.0%)	1190 (19.9%)	<0.001
Yes	605 (69.4%)	1434 (77.5%)	1247 (81.6%)	1503 (87.0%)	4789 (80.1%)	
Organised Sport (age 17)						
No	494 (52.4%)	824 (43.2%)	628 (39.6%)	606 (34.1%)	2552 (41.1%)	<0.001
Yes	448 (47.6%)	1085 (56.8%)	959 (60.4%)	1172 (65.9%)	3664 (58.9%)	
Primary School Commute						
active commute	329 (35.2%)	545 (28.7%)	366 (23.3%)	427 (24.3%)	1667 (27.1%)	<0.001
bus	147 (15.7%)	278 (14.7%)	230 (14.7%)	241 (13.7%)	896 (14.6%)	
car	458 (49.0%)	1074 (56.6%)	973 (62.0%)	1090 (62.0%)	1667 (27.1%)	
Secondary School Commute						
active commute	246 (27.8%)	343 (18.5%)	264 (17.2%)	313 (18.2%)	1166 (19.4%)	<0.001
bus	282 (31.9%)	587 (31.6%)	480 (31.2%)	495 (28.8%)	1844 (30.7%)	
car	356 (40.3%)	927 (49.9%)	793 (51.6%)	912 (53.0%)	2988 (49.8%)	
Primary Sch. Sports Facilities						
Poor/Fair	392 (43.0%)	839 (45.4%)	661 (43.0%)	782 (45.9%)	2674 (44.5%)	0.255
Good/Excellent	520 (57.0%)	1011 (54.6%)	876 (57.0%)	923 (54.1%)	3330 (55.5%)	
Secondary Sch. Sports Facilities						
Poor/Fair	259 (31.1%)	569 (31.8%)	446 (30.2%)	480 (28.4%)	1754 (30.3%)	0.165

Good/Excellent	574 (68.9%)	1218 (68.2%)	1033 (69.8%)	1209 (71.6%)	4034 (69.7%)	
Disorder local Area (age 9)						
mean (SD)	2.0 (0.7)	1.8 (0.6)	1.8 (0.6)	1.7 (0.5)	1.8 (0.6)	<0.001
Disorder local Area (age 13)						
mean (SD)	1.8 (0.7)	1.7 (0.6)	1.6 (0.6)	1.6 (0.5)	1.7 (0.6)	<0.001
Safety local Area (age 17)						
mean (SD)	2.1 (0.8)	2.0 (0.7)	1.9 (0.6)	1.9 (0.6)	2.0 (0.7)	<0.001
Safety local Area (age 9)						
mean (SD)	2.8 (0.6)	2.9 (0.6)	2.9 (0.6)	3.0 (0.6)	2.9 (0.6)	<0.001
Safety local Area (age 13)						
mean (SD)	2.9 (0.7)	2.9 (0.6)	2.9 (0.6)	3.0 (0.6)	2.9 (0.6)	<0.001
Safety local Area (age 17)						
mean (SD)	2.8 (0.7)	2.9 (0.7)	2.9 (0.6)	2.9 (0.6)	2.9 (0.6)	<0.001

Multivariate analysis

Tables 2 and 3 (below) show the results of multi-level linear spline regression models predicting levels of PA for males and females respectively. In the base model, young people whose mothers have achieved lower or higher secondary education were significantly less likely to be PA than young people whose mothers who have achieved tertiary education. There were no statistically significant differences in levels of PA between young people whose mothers have a postsecondary level qualification (‘diploma’) and those whose mothers have tertiary education (‘degree’). In Model 2, the individual-level controls were added to the base model. In Model 3, adolescents whose families belong to the highest income quintile were significantly more likely to be physically active than adolescents whose families belong to all other income quintiles, with one exception for males whose household belongs to the second highest (‘4th’) income quintile. In Model 4, the family-level variables were associated with levels of PA in the expected direction. Adolescents whose mothers were ‘fairly’ or ‘not very’ or ‘not’ active (compared to ‘very’ active) were less physically active themselves. Participation in organised sport was significantly positively associated with levels of PA, and unstructured activities was significantly negatively associated with PA, for both males and females. Participation in enrichment activities had a significant positive effect on levels of PA for females and a nonsignificant negative effect on levels of PA for males. In Model 5 the school-level mediating variables were related to PA in the expected direction, where adolescents with access to poor/fair sports facilities do less PA than those with access to good/excellent sports facilities, and active commuting (compared to travelling by bus or car) was positively associated with levels of PA. In model 6, living in a disorderly neighbourhood was significantly negatively associated with levels of PA for females.

Unexpectedly, there was a small non-significant positive association between living in a disorderly neighbourhood and PA for males. Living in a safe neighbourhood was significantly positively associated with levels of PA for both males and females. The final model is fully-adjusted for individual, material, family, school and neighbourhood factors. For males, having a mother who completed higher secondary ('leaving cert.') compared to a tertiary education remains a significant negative predictor of PA at $p < 0.05$.

Table 2. Results from multi-level linear spline regression models predicting levels of PA (males).

	Model 1: Base	Model 2: Controls	Model 3: Income	Model 4: Family	Model 5: School	Model 6: Neighbourhood	Model 7: Fully Adjusted
	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)
Maternal Education Level							
Junior cert. (vs. degree)	− 8.59*** (2.24)	− 7.62*** (2.19)	-5.63* (2.24)	− 3.84 (2.14)	− 7.90*** (2.20)	− 7.50*** (2.20)	− 3.05 (2.19)
Leaving cert. (vs. degree)	− 7.55*** (2.09)	− 6.14** (2.05)	4.91* (2.06)	− 5.55** (1.99)	-6.25** (2.05	− 6.01** (2.05)	− 4.94*(2.00)
Higher diploma (vs. degree)	− 2.37 (2.43)	− 1.07 (2.38)	− 0.29 (2.38)	0.23 (2.31)	− 1.16 (2.38)	− 0.98 (2.38)	0.02 (2.31)
Age in years							
13 (vs. 9)	-27.19*** (2.15)	− 27.28*** (2.15)	− 27.48*** (2.15)	− 26.23*** (2.16)	− 27.07*** (2.16)	-27.27*** (2.15)	− 26.28*** (2.17)
17/18 (vs. 9)	-34.86*** (2.15)	− 35.19*** (2.15)	− 35.40*** (2.15)	− 31.56*** (2.17)	− 34.99*** (2.15)	-35.19*** (2.15)	− 31.72***(2.18)
Maternal Education#Age							
junior cert.#13	− 6.83* (2.82)	− 6.44* (2.82)	− 6.18* (2.82)	− 5.48 (2.83)	-6.19* (2.82)	-6.40* (2.82)	− 4.99 (2.83)
junior cert.#17/18	5.79* (2.83)	6.16* (2.83)	6.40* (2.83)	7.04* (2.84)	6.42* (2.83)	6.16* (2.83)	7.56** (2.84)
leaving cert.#13	− 2.91 (2.64)	− 3.06 (2.63)	− 2.86 (2.63)	-0.63 (2.64)	-2.79 (2.63)	− 3.20 (2.63)	− 0.29 (2.64)
leaving cert.#17/18	1.46 (2.63)	1.86 (2.63)	2.00 (2.63)	3.75 (2.64)	2.14 (2.63)	1.75 (2.63)	4.02 (2.64)
diploma#13	− 2.85 (3.07)	− 3.50 (3.07)	− 3.19 (3.07)	− 2.20 (3.08)	− 3.26 (3.07)	− 3.65 (3.07)	-1.80 (3.08)
diploma#17/18	0.64 (3.08)	0.42 (3.07)	0.51 (3.07)	0.52 (3.08)	0.69 (3.07)	0.30 (3.07)	0.71 (3.08)
Child Chronic Illness							
No (vs. yes)		− 5.93*** (1.39)	− 5.78*** (1.38)	− 4.62*** (1.35)	− 5.85*** (1.39)	− 5.84*** (1.39)	-4.37**(1.34)
Personality							
Agreeableness		-1.30** (0.46)	− 1.28** (0.46)	− 1.10** (0.43)	− 1.33** (0.46)	− 1.31** (0.46)	-1.06*(0.42)
Conscientiousness		0.83 (0.43)	0.86* (0.42)	0.24 (0.40)	0.84* (0.43)	0.83 (0.43)	0.30 (0.39)

Emotional Stability	1.97*** (0.40)	1.99*** (0.40)	1.53*** (0.37)	1.99*** (0.40)	1.98*** (0.40)	1.59***(0.37)
Extraversion	4.11*** (0.39)	4.06*** (0.39)	3.16*** (0.36)	4.11*** (0.39)	4.09*** (0.39)	3.15***(0.36)
Openness to Experience	− 0.04 (0.49)	0.08 (0.49)	0.42 (0.46)	− 0.02 (0.49)	− 0.02 (0.49)	0.56 (0.45)
Child BMI						
Overweight (vs. normal)	-3.46** (1.12)	− 3.50** (1.12)	− 2.70* (1.08)	− 3.50** (1.12)	− 3.50** (1.12)	− 2.68*(1.08)
Obese (vs. normal)	− 16.57*** (1.96)	− 16.64*** (1.95)	-14.27*** (1.89)	− 16.50*** (1.96)	− 16.61*** (1.96)	− 14.20***(1.89)
Missing (vs. normal)	− 7.88*** (2.39)	− 8.01*** (2.38)	− 6.72** (2.34)	-7.99*** (2.39)	− 7.94*** (2.39)	− 6.93**(2.34)
Region						
Rural (vs. urban)	− 4.58*** (0.96)	− 4.09*** (0.96)	− 5.60*** (0.89)	− 3.74*** (1.01)	− 4.43*** (0.99)	− 3.94***(0.96)
Income						
Lowest (vs. highest)		− 5.60*** (1.54)				− 3.17*(1.49)
2nd (vs. highest)		-4.50** (1.46)				− 2.08 (1.41)
3rd (vs. highest)		-6.18*** (1.42)				− 4.81***(1.37)
4th (vs. highest)		-1.00 (1.36)				0.25 (1.32)
Missing (vs. highest)		− 0.23 (1.80)				0.68 (1.75)
Maternal PA						
Fairly (vs. very PA)			− 1.91 (1.12)			− 1.61 (1.12)
Not very (vs. very PA)			− 4.73***(1.39)			− 4.36**(1.39)
Not (vs. very PA)			− 12.97***(2.48)			− 13.04***(2.48)
Enrichment Activities			− 0.07 (0.59)			-0.1 (0.59)
Organised Sport			20.10***(1.07)			20.04***(1.07)
Unstructured Activities			− 0.02***(0.00)			− 0.02***(0.00)
Sporting Facilities						

Excellent/Good (vs. poor/fair)					− 0.21 (0.93)		-0.25-0.89
School Commute							
Bus (vs. active commute)					− 3.77** (1.30)		-4.23***(1.25)
Car (vs. active commute)					− 1.38 (1.13)		-2.28*(1.09)
Perceived Disorder						0.39 (0.70)	0.79 (0.69)
Perceived Safety						2.13** (0.69)	1.68* (0.67)
Constant	122.36*** (1.71)	98.58*** (4.16)	99.54*** (4.17)	92.24*** (4.10)	99.62*** (4.21)	91.50*** (4.98)	86.59***(4.99)
Observations	8,494	8,494	8,494	8,494	8,494	8,494	8,494
Number of ID	2,915	2,915	2,915		2,915	2,915	2,915
Controls	YES	YES	YES	YES	YES	YES	YES

Standard errors in parentheses.

***p < 0.001, **p < 0.01, *p < 0.05.

Table 3. Results from multi-level linear spline regression models predicting levels of PA (females).

	Model 1: Base	Model 2: Controls	Model 3: Income	Model 4: Family	Model 5: School	Model 6: Neighbourhood	Model 7: Fully Adjusted
	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)	PA (s.e)
Maternal Education Level							
Junior cert. (vs. degree)	− 5.49* (2.24)	-2.82 (2.24)	− 0.69 (2.30)	1.68 (2.18)	− 2.81 (2.24)	−2.34 (2.24)	2.69 (2.24)
Leaving cert. (vs. degree)	− 4.33* (2.16)	− 3.11 (2.13)	− 1.40 (2.17)	− 0.95 (2.07)	− 3.19 (2.13)	−2.88 (2.13)	−0.04 (2.10)
Higher diploma (vs. degree)	0.24 (2.61)	1.54 (2.58)	3.01 (2.59)	2.65 (2.50)	1.58 (2.57)	1.90 (2.57)	3.67 (2.51)
Age in years							
13 (vs. 9)	− 27.74***(2.33)	− 28.04***(2.33)	27.99***(2.34)	− 27.67***(2.32)	− 27.60***(2.34)	− 28.31***(2.33)	− 27.56***(2.33)
17/18 (vs. 9)	− 40.66***(2.33)	− 40.87***(2.33)	-40.75***(2.33)	− 34.42***(2.33)	− 40.44**(2.33)	- 40.71***(2.33)	-34.19***(2.34)
Maternal Education#Age							
junior cert.#13	− 17.71*** (2.90)	− 17.55*** (2.90)	− 17.64*** (2.91)	− 17.09*** (2.88)	− 17.56*** (2.90)	− 17.14***(2.90)	− 16.91***(2.88)
junior cert.#17/18	− 7.76** (2.90)	− 7.73** (2.90)	− 7.87** (2.91)	− 6.88* (2.88)	− 7.74** (2.90)	− 7.37*(2.90)	− 6.75*(2.88)
leaving cert.#13	− 11.02*** (2.79)	− 10.83*** (2.79)	− 10.99*** (2.79)	− 9.99*** (2.77)	-10.64*** (2.79)	− 10.83*** (2.79)	− 9.94***(2.77)
leaving cert.#17/18	− 5.36 (2.78)	− 5.08 (2.78)	− 5.40 (2.79)	− 3.48 (2.76	− 4.89 (2.78)	− 4.74 (2.78)	− 3.31 (2.76)
diploma#13	− 14.08*** (3.36)	− 14.10*** (3.36)	− 14.41*** (3.36)	− 13.07*** (3.33)	− 14.10*** (3.36)	− 14.20*** (3.35)	− 13.36***(3.33)
diploma#17/18	− 10.90** (3.36)	− 11.13*** (3.36)	− 11.62*** (3.37)	− 9.74** (3.34)	− 11.08*** (3.36)	− 10.93** (3.36)	9.93**(3.34)
Child Chronic Illness							
No (vs. yes)		− 2.29 (1.61)	− 2.12 (1.61)	-2.47 (1.56)	− 2.17 (1.61)	− 2.01 (1.61)	-2.08 (1.56)
Personality							
Agreeableness		− 0.72 (0.44)	− 0.69 (0.44)	-0.62 (0.41)	− 0.74 (0.44)	− 0.74 (0.44)	−0.61 (0.41)

Conscientiousness	1.28** (0.41)	1.29** (0.41)	0.89* (0.38)	1.26** (0.41)	1.28** (0.41)	0.89*(0.38)
Emotional Stability	1.53*** (0.36)	1.52*** (0.36)	0.90** (0.34)	1.57*** (0.36)	1.41*** (0.36)	0.84*(0.34)
Extraversion	2.66*** (0.38)	2.57*** (0.38)	1.69*** (0.36)	2.65*** (0.38)	2.58*** (0.38)	1.65***(0.36)
Openness to Experience	1.23* (0.49)	1.37** (0.49)	1.40** (0.46)	1.25* (0.49)	1.36** (0.49)	1.61***(0.46)
Child BMI						
Overweight (vs. normal)	− 4.90*** (1.08)	− 4.70*** (1.08)	− 3.53*** (1.04)	-4.90*** (1.08)	− 4.73*** (1.08)	-3.34**(1.04)
Obese (vs. normal)	− 9.96*** (1.67)	− 9.40*** (1.67)	−7.92*** (1.61)	− 9.84*** (1.67)	− 9.85*** (1.67)	-7.48***(1.60)
Missing (vs. normal)	− 3.48 (2.14)	− 3.57 (2.14)	−1.64 (2.09)	-3.46 (2.14)	−3.14 (2.14)	−1.60 (2.09)
Region						
Rural (vs. urban)	− 5.45*** (0.95)	5.06*** (0.95)	−6.33*** (0.89)	− 4.62*** (0.99)	− 5.76*** (0.97)	− 5.36***(0.95)
Income						
Lowest (vs. highest)		-5.01** (1.55)				− 2.28 (1.50)
2nd (vs. highest)		− 6.65*** (1.51)				− 3.97**(1.46)
3rd (vs.highest)		− 4.76** (1.47)				− 3.09*(1.43)
4th (vs. highest)		-5.37*** (1.44)				-4.03**(1.40)
Missing (vs. highest)		0.25 (1.86)				2.42 (-1.81)
Maternal PA						
Fairly (vs. very PA)			− 2.39*(1.17)			− 1.99 (1.17)
Not very (vs. very PA)			4.99***(1.40)			− 4.28**(1.39)
Not (vs. very PA)			− 3.09 (2.58)			-2.46 (2.57)
Enrichment Activities			3.69***(0.56)			3.56***(0.56)
Organised Sport			16.35***(0.90)			16.04***(0.90)
Unstructured Activities			− 0.02***(0.0)			− 0.02***(0.00)

Sporting Facilities							
Excellent/Good (vs. poor/fair)					− 0.89 (0.89)		− 0.96 (0.86)
School Commute					− 3.96** (1.32)		− 3.92**(1.27)
Bus (vs. active commute)					− 1.14 (1.14)		−1.77 (1.10)
Car (vs. active commute)							
Perceived Disorder						1.45* (0.65)	−0.81 (0.64)
Perceived Safety						3.90*** (0.69)	2.74***(0.67)
Constant	111.28*** (1.81)	85.62*** (4.16)	87.43*** (4.18)	82.84*** (4.16)	86.74*** (4.24)	77.24*** (4.88)	78.42***(4.98)
Observations	8,962	8,962	8,962	8,962	8,962	8,962	8,962
Number of ID	3,071	3,071	3,071	3,071	3,071	3,071	3,071
Controls	YES	YES	YES	YES	YES	YES	YES

Standard errors in parentheses.

***p < 0.001, **p < 0.01, *p < 0.05.

Decomposition analysis

Table 4 (below) provides the average difference in PA levels between young people whose mothers have lower secondary education ('junior cert.') and young people whose mothers have tertiary education. This is referred to as the Maternal Education (ME) differential. Table 4 also provides the absolute reduction and the average proportionate reduction in PA with the addition of the each group of explanatory variables (income, family, school and neighbourhood), where model 2 ('controls') provides the baseline ME differential. The models have been ranked by the level of change. The average proportionate change represents the contribution of each group of variables to explaining the social patterning of PA. The addition of household income to the model reduced the ME differential by 24.1% for males and 14.7% for females, making it the second most effective model at explaining the social patterning of PA. The family model reduced the ME differential by 50.8% for males and 35.1% for females, making it the most effective model in explaining the social patterning of PA. In analysis (not shown) the unique contribution of each family-level variable was derived. Organised sport alone reduced the baseline ME differential by 43.6% for males and 25.3% for females, enrichment activities reduced the differential by 1.2% for males and 5.6% for females, unstructured activities reduced the differential by 18.6% for males and 14.8% for females and Maternal PA reduced the same differential by 2.8% for males and 2.5% for females. This analysis demonstrated that variables representing parenting practices (enrichment actives, organised sport and unstructured activities) were more effective in accounting for the gap in levels of PA between maternal education categories than the variables representing parents as role models (maternal PA). The school model did not contribute to the explanation of ME differentials in PA, indeed for males the ME differential increased in model 5 by 1.2%. There was a modest reduction for females of 0.1%. This may be due to

the reverse social patterning of the school commute variable, where families with low levels of maternal education were more likely to have an active commute (as outlined in the previous section). Adjusting for neighbourhood-level characteristics had a small impact on ME differential, reducing it by 1.5% for males and 5.2% for females. The fully adjusted model, including individual, family, school and neighbourhood characteristics reduced the ME differential by 61.8% for males and 43.3% for females (average of three timepoints). Our models were better able to account for the socially patterning of PA for males compared to females. Figs. 5.1 and 5.2 (see below) visually displays the reduction of the ME differential with the addition of explanatory variables at age 9, 13 and 17/18 separately.

Table 4. Average proportionate change in the SEP differential in levels of PA with the addition of explanatory variables between age 9 and 17/18.

	Males			Females		
	Average	Change	Change %	Average	Change	Change %
Model 1: Baseline	8.94	0.00	0.0%	13.98	0.00	0
Model 5: School	7.72	0.10	1.2%	11.25	0.01	0.1
Model 2: Controls	7.82	0.00	0.0%	11.24	0.00	0
Model 6: Neighbourhood	7.58	-0.14	-1.5%	10.51	-0.73	-5.2%
Model 3: Income	5.56	-2.16	-24.1%	9.19	-2.05	-14.7%
Model 4: Family	3.18	-4.54	-50.8%	6.34	-4.91	-35.1%
Model 7: Fully Adjusted	2.19	-5.53	-61.8%	5.20	-6.05	-43.3%

Figure 5.1. Estimated high/low educational differentials in levels of PA on the GS scale (males).

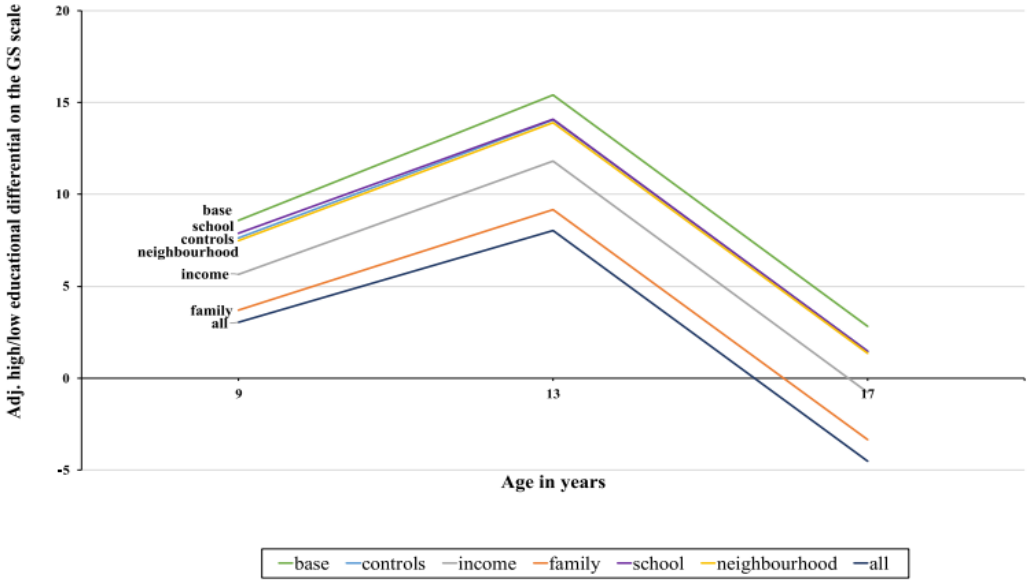
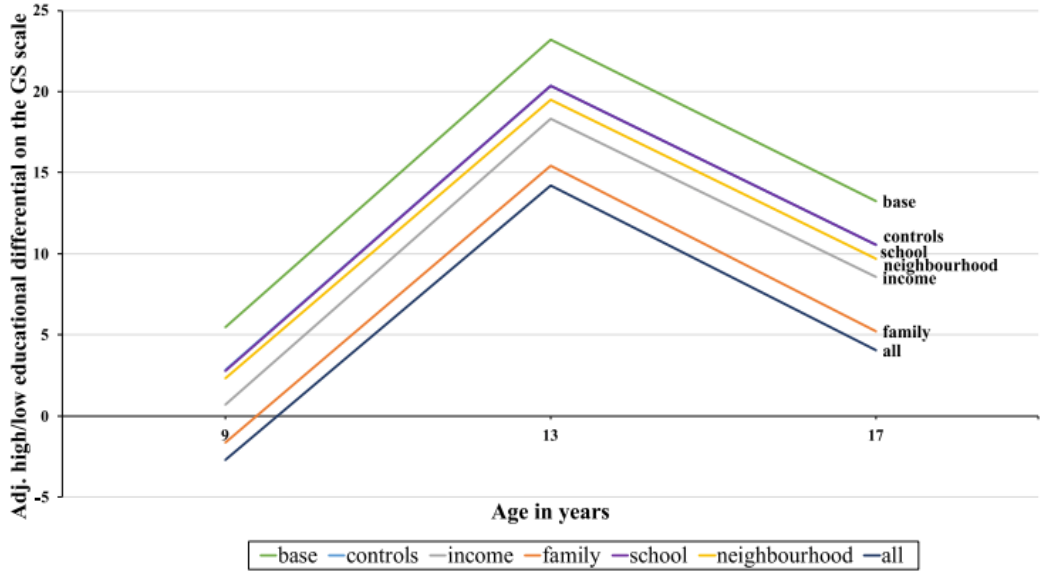


Figure 5.2. Estimated high/low educational differentials in levels of PA on the GS scale (females).



Conclusion

Physical activity is one of the common modifiable risk factors for the emergence of NCDs among low SEP groups. This paper aimed to understand the role of family, school and neighbourhood-level processes in accounting for the maternal education gradient in PA among adolescents between ages 9 and 18 using a large, nationally representative sample from the Republic of Ireland. Using a decomposition approach, we quantified the contribution of family, school and neighbourhood-level factors adjusting for individual-level personality, BMI, health status and household-level income.

Our data are from Ireland, but our descriptive findings support evidence from other national contexts that show a strong association between social and economic position and PA(J Inchley et al., 2020; Inchley, Currie, Young, et al., 2016), and a sharp decline in PA during adolescence(Dumith et al., 2011). Internationally there is variation in the age at which the decline in PA occurs during adolescence, but the literature suggests somewhere between 11 and 15yrs. Given the data points available to us, it was only possible to be certain that PA peaked before the age of 13 but may have been decreasing since age 9. Young people with lower educated mothers experienced steeper declines in PA than their more advantaged peers over the ten-year period of observation. We looked at the potential role of equivalised family income in mediating the relationship between maternal education and adolescent PA. Adjusting for income, our decomposition analysis accounted for 24.1% of the ME differential in levels of PA for males, and 14.7% for females. The ME differential was further reduced by the addition of family, school and neighbourhood level factors, suggesting that there were also cultural and social mechanisms through which maternal education influences young people's PA.

Family-level mechanisms

Family-level mechanisms accounted for the biggest reduction in the ME differential in PA for both males and females, they included: enrichment activities (youth club, dance, drama, music, organised sport etc.), unstructured activities (playing video games, time on the computer a, watching TV) and maternal levels of PA. Considered separately, parents enrolling their children in organised sport was the most effective family-level mechanism for explaining the social patterning of PA. The proportionate reduction was larger for males compared to females, which is in-line with Lareau's (2011) observation that her male participants were more likely to opt for organised sport (e.g. basketball, baseball, and soccer) as their primary enrichment activity, while female enrichment activities were more varied. Participation in organised sport has already been shown to account for both the age decline (Lunn et al., 2013; Lunn & Layte, 2008; Zick et al., 2007) and gender differences (Vilhjalmsson & Kristjansdottir, 2003) in levels of PA. Our findings indicate that participation in organised sport also contributes to the social patterning in levels of PA. Recent Irish qualitative research identified barriers to organised sport specific to low education families, including: not feeling welcome, expense and transportation(Tandon et al., 2021).

As shown elsewhere (McCoy et al., 2012), and in line with Lareau's theory of 'natural growth' (2017), we found that adolescents of lower educated mothers were more likely to partake in unstructured activities, and this contributed significantly to the social patterning of PA. This would support a 'displacement hypothesis' (Pearson et al., 2014) where engagement in sedentary activities displaces time that would have otherwise been spent being physically active.

This paper also investigated whether parents engaged in sport and PA acted as role-models for their children. We found that adjusting for maternal PA levels reduced the ME differential for both males and females, making it an important part of the intergenerational transmission
of inequalities in levels of PA in the home. However, our decomposition analysis revealed that controlling for parenting practices, compared to parental role-modelling, was more effective in accounting for the social patterning of PA.

School-level mechanisms

We did not find significant differences in the availability of school sporting facilities to young people in the different maternal education categories, which was unexpected given the qualitative reporting in the Irish context(Woods et al., 2010; Woods et al., 2019). Additionally, our analysis showed that active commute to school was more common among adolescents whose mothers had lower levels of education when compared to their more advantaged peers. This reverse social patterning has previously been documented in adolescents in Spain, Canada and Australia(Carlin et al., 1997; Chillón et al., 2010; Robertson-Wilson et al., 2008). Previous research, in the Irish context, reported that active commuting is more likely in urban settings(Murtagh et al., 2016), and so our results may be influenced by the high concentration of lower SEP families in urban areas in Ireland. The school environment did not contribute to the reduction in ME differential in levels of PA, but it is possible that other, unmeasured, school-level processes may have an influence on levels of PA that were not captured in this study. Moreover, our dependent variable (the GS scale) is the parent's estimate of their child's PA. Given this, it is perhaps not surprising that variation in the sports which parents organise is significant in our

models, whilst sport played in school, which parents were probably less likely to see, is not significant.

Neighbourhood-level mechanisms

Drawing on the socio-ecological models of health behaviours(Sallis et al., 2015), and answering the call for a focus on sub-groups within this area of research(Biddle & Mutrie, 2007), we argued that the association of higher education with neighbourhoods with safe spaces to exercise and low levels of crime, vandalism, litter etc. may account for some of the social patterning in PA. Adjusting for perceived level of neighbourhood safety and neighbourhood disorder produced a small reduction in the ME differential in levels of PA for males and females. The proportionate reduction was larger for females, compared to males, which suggests that neighbourhood safety and disorder is a bigger barrier to PA for lower SEP females. Our evaluation of the young person's physical environment was limited by the measures available in our chosen dataset. Adjusting for other features of neighbourhood environment could elevate the role of neighbourhood in explaining the social patterning of PA.

Strengths & Limitations

Research on the social distribution of PA is often based on small, nonrepresentative samples, partly because reliance on data from accelerometers, which is expensive and time consuming to collect. Although our dependent variable was based on parental report, the study provides

information on a nationally representative sample of young people covering an observation period from middle childhood to early adulthood. Combined with the wide variety of dimensions available, this enabled us to carry out a multi-dimensional longitudinal study. However, our use of a parent-reported PA is a clear limitation.

Accelerometer data are not subject to respondent bias or lapses in memory, or other limitations associated with self-report measures. However, the GS is an internationally validated instrument (Godin et al., 1986; Godin & Shephard, 1985; Zelener & Schneider, 2016). Although the absolute level of PA reported may not be as accurate as that derived from accelerometer data, it seems reasonable to assume that any biases involved are equal across groups defined by maternal level of education. The focus of this research was the relative differences between ME groups rather than the absolute levels of PA in each group.

The measure participation in organised sport, used as a predictor variable in this analysis, is based on a survey question which specifies whether the young person "play[s] sports with a coach or instructor or as part of an organised team." This specification is important to separate organised sport, which we argue has demonstrable social and economic barriers to participation, from other instances of PA. Potential barriers include the cost of membership fees and equipment, which prompted the addition of income as a control variable in the analysis. Other potential barriers include practical support from parents in the form of lifts to training sessions or emotional support at matches. These parenting practices are conceptualised by Lareau (2011) in her theory of 'concerted cultivation,' which we aim to test here. However, we acknowledge that the strength of evidence for this theory, as an explanation for inequalities in harmful health behaviours, would likely differ if another health behaviour was selected. For this reason, we separated out 'organised sport' from other enrichment activities, so as not to artificially inflate the importance of concerted cultivation in explaining the relationship between maternal education and PA. Organised sport has been used elsewhere in to make a meaningful contribution gender and age gradient in levels of PA, as outlined in this paper.

3 Bringing the Group Back in: Social Class and Resistance in Adolescent Smoking.

Introduction & Background

The finding that morbidity and mortality risk are inversely related to the socio-economic position of individuals, within countries, is one of the most consistent findings in sociological and epidemiological research(Stringhini et al., 2017). Although the nature of the relationship varies depending on the measure of socio-economic position used (i.e. income, education, occupation; henceforth SEP), the risk of poor health and mortality increases with disadvantage and, for this reason, the relationship has come to be known as the 'social gradient in health'(Tanaka et al., 2019). SEP does not influence health directly and researchers continue to test potential mechanisms through which it 'gets under the skin'(Blane et al., 2013). The patterning of health by social determinants (i.e. SEP) can be explained, in part, by differentials in harmful health behaviours by SEP. Health behaviours, such as smoking, have been shown to cluster according to SEP, and increase the risk of mortality from non-communicable diseases (NCDs) (Petrovic et al., 2018).

Smoking is the leading cause of preventable death globally and the single largest avoidable health risk(WHO, 2022). It also contributes to substantial health-related costs to society through smoking-attributable medical expenditures and lost productivity (Goodchild et al., 2018). Particular attention, in terms of policy intervention, is paid to youth smoking given that 93% of smokers take up smoking before they turn 24 (European Commission, 2024). Adolescence is a 'critical period' for the formation of risky health behaviours(Joanna Inchley et al., 2020; Viner et al., 2015). For this reason, research exploring potential causal factors for persistent smoking among adolescents is key if we are to eliminate health disparities related to smoking that is evident in adult populations.

Whilst absolute rates of smoking have been falling in recent decades(OECD, 2021; Staff et al., 2018), reductions have been larger among more socio-economically advantaged groups. The resulting widening socioeconomic gradient in smoking, as overall population rates decline, suggests that public health initiatives designed to reduce smoking rates have had little effect on the most disadvantaged members of society(Hiscock et al., 2012; Lawlor et al., 2003).

Less is known about *why* smoking varies across social groups in this structured manner, which is the focus of this paper. The dominant focus of social epidemiology on the immediate causes of disease has neglected to consider the larger social forces that expose individuals to these health behaviours in the first place, an observation initially highlighted in the Black report(1982).

Individual-level explanations for inequalities in smoking

For decades, literature on health promotion largely focused on the role of individual factors in shaping the probability of adopting risky health behaviours, and by extension for explaining differentials in behaviours across SEP groups(Cockerham, 2005; Marmot, 2018). These *individual-level explanations* come in three forms. First, the 'health knowledge' model views risky health behaviours as the outcome of poor information and argues that smoking is adopted or maintained because individuals do not know about the negative effects(Richard Layte & Christopher T Whelan, 2009). The 'rational choice' model, on the other hand, asserts that people can know about the risks involved, but still choose to smoke because they decide to trade off the current pleasure that can be derived from smoking against the more uncertain future health risks that may emerge, similar to 'hyperbolic discounting' (Ainslie & Haslam, 1992) or 'countervailing

mechanisms'(Phelan et al., 2010). This model prioritizes individual agency and the individual's evaluation of short and long term incentives, and acknowledges the long-standing tradition, within sociology and anthropology, that argues that behaviours that may otherwise be difficult to understand are rational within the particular contexts in which they occur. The third model, the 'psychological' model (Delaney & Doyle, 2012; Richard Layte & Christopher T Whelan, 2009) holds that individuals can know about the risks that a behaviour poses, and may even want to stop, but smoke nonetheless because of certain psychological factors. For example, low, or decreasing adolescent self-efficacy has been shown to predict smoking initiation (Lawrance & Rubinson, 1986; Van De Ven et al., 2007) largely through its negative influence on the young person's capacity not to smoke among smoking friends(Hiemstra et al., 2011), and there is a well-established literature on the role of low self-esteem in adolescent smoking initiation(Khosravi et al., 2016).

Structural-level explanations for inequalities in smoking

Whilst there is certainly evidence supporting these three models of adolescent smoking, it could be argued that in focusing on the role of individual deficits, biases and psychological traits, each misses the important role that structural factors play in the initiation and maintenance of smoking. In the early stages of cigarette diffusion there was a strong positive relationship between social advantage and smoking, but in the later stages of cigarette diffusion, the relationship reversed(Pampel, 2005). This changing relationship between SEP and smoking, overtime, suggests that social forces play a role in explaining the variation in smoking across social groups. More structurally orientated researchers have sought to unpack the processes through which social stratification and social context influence health behaviours at the individual-level(McCartney et al., 2013).

For example, Cockerham (2005)argued that the clustering of smoking according to SEP is not the uncoordinated response of disconnected individuals but the patterned response of social groups to the constraints of their external environments, drawing on Weber's (1946) concept of 'Lebenschancen' (life chances). Moreover, Bartley (2016)employs the work of Bourdieu (1989) to unpack the symbolic dimensions of smoking by arguing that smoking is a strategy adopted by individuals to display group membership, and distance themselves from other non-smoking groups. In this way, smoking acts as a consumption practice for identity and status. This work provides us with the broad sociological framework for the structural explanations put forward in this paper.

The first *structural-level explanation* put forward by this paper is based on social learning theory and social network theory(Bandura & Walters, 1977; Granovetter, 1973). Variation in smoking according to SEP suggests subcultural differences in the content of what is learned or reinforced within the young person's 'micro-system' i.e. parents, other household members and friends(Bronfenbrenner, 1977). Since smoking has been shown to be more prevalent among those with disadvantaged SEP, this would suggest that young people from disadvantaged backgrounds are exposed to more smoking in their social environment, compared to their peers from more advantaged backgrounds. Smoking has been shown to be more likely among parents who are younger, less educated and have more financial difficulties(Hitchman et al., 2014; Leonardi-Bee et al., 2011). The process through which this proximity may lead to smoking is, however, disputed. Social learning theory postulates that smokers learn to smoke by the same processes that non-smokers learn not to smoke, by the example of parents. Alternatively, according to Harris(1995, 1998, 2000), 'socialisation' processes occur when the young person inherits a peer

network and ‘shared culture’ from their parent’s network, which demands the same behaviours from the young person as it did from their parents i.e. smoking. Young people who have a high likelihood of smoking may seek out peers who already smoke, but it is also possible that exposure to smoking from peers leads to smoking through socialisation effects. A recent systematic review of 40 studies of adolescent substance use found support for peer selection, with relatively few studies reporting socialisation effects(Henneberger et al., 2021).

The second structural-level explanation put forward by this paper is based on the work of Factor and colleagues, and is a group-based explanation for social inequalities in adolescent smoking(2011; 2013). The authors observed that, within societies, non-dominant minority groups exhibit higher rates of risky health behaviours, compared to the dominant majority group. They argue that this pattern emerges as a result of experiences of discrimination, which makes non-dominant groups feel alienated or rejected from the larger society. By engaging in risky behaviours, such as smoking, the non-dominant groups ‘express their willingness and ability to defy… the dominant group’ (p.1293). Their theory builds on Scott’s (1990) theory of ‘everyday resistance,’ which seeks to understand the ways in which marginalised groups defy dominant groups and ‘the system,’ with small acts of resistance, in the context of peasant societies, some of which may improve their standard of living, but many of which provide symbolic empowerment and take back agency. Johnson and Hoffmann (2000)compared smoking initiation between members of minority groups attending ‘minority’ schools (where there is no opposing dominant majority) and members attending majority schools (where there is an opposing dominant majority). Although minority schools have been linked with low educational achievement and high risk of dropout, their results indicated that attendance

results in a reduced risk of smoking among minority adolescents, supporting our argument for smoking as a ‘resistance’ behaviour. Lister, also using Scott’s theory, has proposed a similar characterisation of actions among non-dominant groups, particularly people in poverty, as ‘getting back at’ behaviours.

As well as ‘getting back at,’ smoking can be used to differentiate minority groups from the dominant majority group by asserting an oppositional social identity. In the same way that adopting smoking was initially seen as a form of distinction between those who had the resources to adopt it, and those who did not(Elias, 1939).Factor et al. (2011), in the context of racial differences in risky behaviours in the US, observed that non-dominant groups feel a pressure not to embrace the attitudes and behaviours identified with the dominant group, what they term ‘acting white,’ even though they understand that these behaviours may have harmful consequences for their health. Similar processes of group identity have been central to the educational resistance literature, most famously in the work of Willis (1977) on the activities of the ‘lads’ in a British ‘working-class’ boys school. The ‘lads’ created their own oppositional subculture, which exerted maximum distinction from institutional conformity. This included their own take on the school uniform, getting out of doing homework and having ‘a crafty smoke’ during the school day (p.23). Their ostracism of those who strove for academic success had the effect of reproducing their family’s educational disadvantage. In this way, there is a ‘complexity and even irrationality within resistance’(Johansson & Vinthagen, 2016). This argument refers to the recognized ‘dark side’ of social capital, where communities can reject certain behaviours that may be deemed as aligned with the dominant majority group(Schreuders et al., 2017).

It could be argued that the central theoretical problem of sociology since Durkheim (1895) has been the identification of the processes through which social structures shape individual behaviour, and the role of individual agency in this. This study provides a concrete example of one such process that can be tested empirically. This paper first quantifies the social gradient in smoking in a representative sample of young people from Ireland. The paper then uses path analysis to empirically examine the contribution of an individual-level theory and structural-level theory for the social patterning of smoking. Crucially, our approach tests whether the same pathways operate differently depending on socio-economic subgroup.

Data and methods

Data

(see Data section in the Introduction)

Levels of missing data across variables was low, on average, but reached 3.7% on some variables (see table one in appendices). Survey attrition was, in part, due to the lack of retention of more disadvantaged participants, meaning there were systematic differences between missing and complete data according to socio-demographic variables observed in our dataset (Donders et al., 2006). This had the potential to bias our analysis, and so missing values were imputed for analysis using multiple imputation by chained equations (MICE) to produce ten imputed datasets, using the Rubin method (Rubin, 1987) as implemented in STATA 15. All of the variables in table one (see appendix) were included in the imputation process, with the exception of the smoking status (dependent variable) and family SEP (independent variable). The data were also reweighted for analyses to take account of the original sample error and subsequent attrition using a range of factors and a minimum distance algorithm. The final imputed and re-weighted sample (n=6,039) is representative of young people who were residing in Ireland at age nine in 2006 and who continued to live in Ireland at age 17/18 in 2016.

Measures

Dependent variable – smoking status at age 17/18.

Smoking status was self-reported by the young person at wave three, when they were aged 17 or 18yrs (henceforth 17/18): ‘have you ever smoked a cigarette?’ (yes/no) and subsequently ‘which of the following best describes you?’ (only ever tried smoking once or twice/used to smoke but not now/smoke occasionally/smoke daily/do not smoke). The answer categories were recoded into ‘smokers’ and ‘non-smokers,’ with respondents who answered ‘no’ to the first question and ‘only ever tried smoking once or twice,’ ‘used to

smoke but not now' and 'do not smoke' being classified as non-smokers. The proportion of occasional smokers was too low to consider separately for the proposed sub-group analysis (discussed further in the limitation section of this paper). Due to the sensitive nature of the questions (smoking is illegal under the age of 16 in Ireland and parents may not be aware of the participant's smoking) the questions were administered on a supplemental self-complete questionnaire, and the young person was assured of confidentiality. The young person, at wave three, also reported what age they were when they initiated smoking and the answers ranged from aged 12 to 18yrs. This variable was used in a supplemental analysis.

Treatment variable - family SEP

SEP was assigned to the participant and their household based on parental occupation at wave one (age 9). For two-parent families in which both partners were economically active outside the home, the family's SEP was assigned using a 'dominance' procedure with family SEP equal to that of the higher of the two parents. A fivefold classification of SEP (professionals and higher management/lower management or technical worker/non-manual worker/skilled manual/semi or un-skilled manual) based on the Standard Occupational Classification of the Central Statistics Office (CSO, 2023) was applied.

Mediating variables

Psychological

Self-efficacy was measured at wave three (age 17/18) by asking the young person about their belief in their ability to succeed in specific situations and to accomplish tasks. The scale contained six items relating to general self-efficacy (e.g. 'If I cannot do a job the first time I keep trying until I can') and social self-efficacy (e.g. 'I find it easy to make

new friends') with four answer categories ranging from 'strongly agree' to 'strongly disagree.' The scores on the items relating to general and social efficacy were reverse coded where necessary and summed to produce an overall self-efficacy score, which ranged from zero to twenty-four. Higher scores were indicative of greater self-efficacy. Self-efficacy has been found to be strongly predictive of health behaviour initiation and cessation (Lawrance & Rubinson, 1986; Van De Ven et al., 2007). The scale used in the present study was adapted from the Sherer et al. (1982) measure by researchers on the ESRC 16-19 initiative research programme. Internal reliability for this scale at age 17/18 of Growing Up in Ireland was α= .72 (Murphy et al., 2019)

Self-esteem was measured using the Rosenberg Self-Esteem scale (Rosenberg, 1965). At wave three, the young person was asked to rate six items (e.g. 'on the whole, I am satisfied with myself' and 'I certainly feel useless at times') on a four-point scale ranging from 'strongly disagree' to 'strongly agree.' The scores on six items were combined, and reverse coded where necessary, to get an overall self-esteem score, which ranged from zero to eighteen. Higher scores were indicative of higher levels of self-esteem. Previous research has highlighted the role of low self-esteem on the initiation of risky health behaviours(Bozzini et al., 2020). The scale is the most widely-used and well-validated measure of self-esteem(Robins et al., 2001).

Smoking exposure

At wave two (age 13), the primary caregiver (PCG; the mother in 98% of the sample) reported on their own smoking status: 'do you currently smoke daily, occasionally or not at all?' The three categories (daily/occasionally/not at all) were recoded into two categories 'parental smoking' and 'no parental smoking' where both daily and occasional smokers were considered smoking parents. The PCG also reported on the number of

smokers in the young person's primary residence: 'including yourself, how many members of the household smoke?' This question allowed us to distinguish between households where only the PCG smoked and households with additional smokers.

At wave three (age 17/18), the young person was asked: 'How many of your regular friends do or have ever smoked cigarettes?' There were four possible response categories (none/a few/some/most/all). The four response categories were recoded into two categories which indicated (1) 'none/few friends smoke' or (2) 'most/all friends smoke.' This question was asked as part of the supplemental self-complete questionnaire where administration was designed with sensitive questions in mind.

Social resistance

The Everyday Discrimination Scale (EDS) was administered at wave three in the supplemental self-complete sensitive questionnaire to the young person directly. The EDS was constructed using five statements about how often participants felt they had experienced various forms of interpersonal mistreatment (e.g. 'being threatened or harassed,' 'receiving less courtesies or respect than others'). The young person indicated the frequency of these acts on a six-point scale from 'never' to 'almost every day.' A total discrimination score was generated from the sum of all five items. Higher scores are indicative of more frequent discrimination. The scores range from zero to five. This 5-item scale was adapted from the original 9-item version of the EDS(Williams et al., 1997). The scale was found to have good internal reliability at age 17/18 of Growing Up in Ireland with $\alpha = .74$(Murphy et al., 2019).

The extent to which the young person had developed oppositional values was measured using a scale from the ESRC 16-19 initiative research programme to measure relationships with authority. The scale was administered at wave three to the young person. The scale contained eight-items:

1. It can be okay to do something which is against the law if it is to help a friend.
2. People in authority, like teachers, always pick on me.
3. Most of the rules in places like schools are stupid and petty.
4. School has been a waste of time for me.
5. Defying people in authority is alright if you can get away with it.
6. The police are often unnecessarily brutal to people.
7. If I saw someone make a break in I would tell the police about it.
8. Most police officers are honest.

The young person indicated their level of agreement with the statements on a 4-point scale (strongly agree/agree/disagree/strongly disagree). Scores on the eight items were reverse coded where necessary and summed to produce an overall score ranging from zero to thirty-two. Higher scores are indicative of more opposition to authority. For some descriptive statistics it was necessary to split the responses into tertiles: 'low,' 'medium' and 'high' levels of oppositional values. The Cronbach alpha value for the scale at age 17/18 of Growing Up in Ireland was $\alpha = 0.72$(Murphy et al., 2019).

Control variables

At wave one, basic descriptive information about the young person was captured including sex (male/female) and the young person's personality. It was not possible to split the sample by gender, in order to observe unique pathways for males and females, due to the small number of smokers at wave three. Since previous research suggests that

smoking is significantly associated with certain personality traits(Vollrath et al., 1999), the Ten Item Personality Inventory (TIPI) was used to measure aspects of the young person's personality, at wave three, as reported by the young person. TIPI consists of five subscales: agreeableness, extraversion, conscientiousness, emotional stability, and openness to experience (Gosling et al., 2003). Classifying individual differences in personality into these five broad empirically derived domains is one of the most widely used and extensively researched models of personality. Each personality dimension was rated on a seven-point scale, with answer categories ranging from disagree strongly to agree strongly, where strongly agree indicates a stronger presence of the respective personality trait. Gosling et al. (2003)noted the scale has good test-retest reliability (r= .72), and report convergent correlations with the personality scale: the 'Big-Five Inventory.' Furthermore it was not hypothesized that gender or personality mediated the relationship between SEP and smoking, so they were included as a control as opposed to mediating variables in the analysis.

Analysis Strategy

Descriptive statistics were computed to understand the sample characteristics, and patterns according to SEP and smoking status. Cross-tabulation chi-squared (χ^2) tests, with 95% CIs, were performed to examine the relationship between SEP and smoking, as well as SEP and gender (control variable), parental, friend's and household smoking (mediating variables). The association between SEP and self-esteem, self-efficacy, everyday discrimination, and levels of opposition to authority (mediating variables, continuous) was assessed through one-way anova analyses. Levene's test for equality of variance was used to asses that the anova were valid. The associations between smoking status and continuous control (personality) and mediating variables (self-esteem, self-

efficacy, everyday discrimination, levels of opposition to authority) were assessed by performing independent sample t-tests. The association between smoking and the remaining mediating variables (parental, friend's and household smoking, binary) were assessed by performing cross-tabulation chi-squared (χ^2) tests, with 95% CIs. Results were visually displayed and interpreted using bar chats and line graphs.

This study employed generalised structural equation modelling (GSEM) to test the hypothesised causal pathways by which family SEP (age 9) would contribute to the probability of smoking (age 17/18). GSEM relaxes the constraints of structural equation models (SEM) by allowing for binary modelling of dependent variables. Using GSEM, we fit our models of interest separately allowing us to compare their efficacy, before fitting them all together. All variables included in the models were observed variables (none were latent). There are some disadvantages to using GSEM: (1) it does not provide standardised coefficients, therefore only the significance/non-significance of coefficients can be used to *compare* the strength of potential pathways, although coefficients can still be interpreted in their own right. (2) The default estimation method in most SEM programs is maximum likelihood; an underlying assumption of this method is multivariate normality of the observed variables. Our data clearly violates this assumption, as many of our variables are binary, and so assume a Bernoulli distribution. Therefore, we examined model fit using summary statistic data. All analyses were carried out on imputed data, with appropriate study weights.

Path analysis allows us to examine how the effect of SEP on smoking probability is mediated, by estimating the relationship between SEP and our mediating variables, as well as, the effect of the mediators on smoking probability. If both equations reveal

significant effects we have evidence of a statistically significant mediating pathway. Path analysis cannot demonstrate causality and significant results should be interpreted as correlational; however, it does allow us to determine whether the hypothesised causal pathway is plausible or not. Moreover, path analysis, using longitudinal data, allows us to test causal ordering among sets of variables. We examined the pathways associated with each hypothesized model, which can be summarised as follows:

Model one (M1) – Baseline + controls: childhood SEP + control variables (gender and personality)

Model two (M2) – Psychological model: M1 + self-efficacy + self-esteem

Model three (M3) – Exposure model: M1 + parental smoking + household smoking + peer smoking.

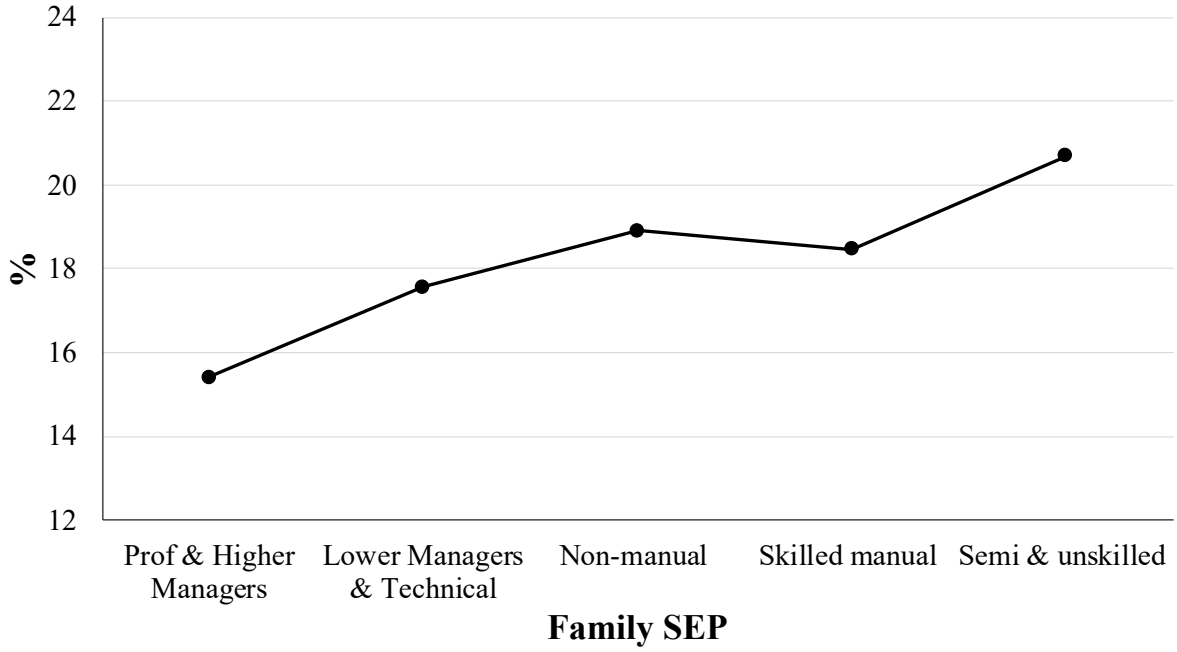
Model four (M4) – Social-resistance model: M1 + experiences of everyday discrimination + levels of opposition to authority.

Model five (M5) - Fully adjusted model.

Results

As shown in figure six, there is a SEP gradient in adolescent smoking at 17/18, where belonging to a more disadvantaged SEP increases the likelihood of being a smoker. This relationship was only marginally significant (p=.0587).

Figure 6. Proportion of smokers by family SEP and gender.



To descriptively assess whether our mediating processes are adversely distributed by SEP, figures 7.1 and 7.2 provide the means and percentages for each mediating variable by SEP category. Self-esteem followed a clear SEP gradient, where belonging to more disadvantaged SEP categories was significantly (p<.001) associated with decreasing levels of self-esteem, on average. Reported levels of self-efficacy were not patterned according to SEP, and group differences were not significant. There is a clear SEP gradient in the levels of exposure to smoking from parents (p<.001) and other household smokers (p<.001), where young people from more disadvantaged SEP categories were significantly more likely to be exposed to more smoking in the home environment. In

terms of exposure to peer smoking, there is no clear SEP gradient, and group differences were not significant. Mean levels of everyday discrimination did not follow any clear SEP patterning, and differences between groups were not significant. However, mean levels of opposition to authority increased, in a stepwise fashion, as SEP decreased, and this relationship was highly significant ($p<.001$).

Figure 7.1. Mean levels of (continuous) mediating variables by family SEP.

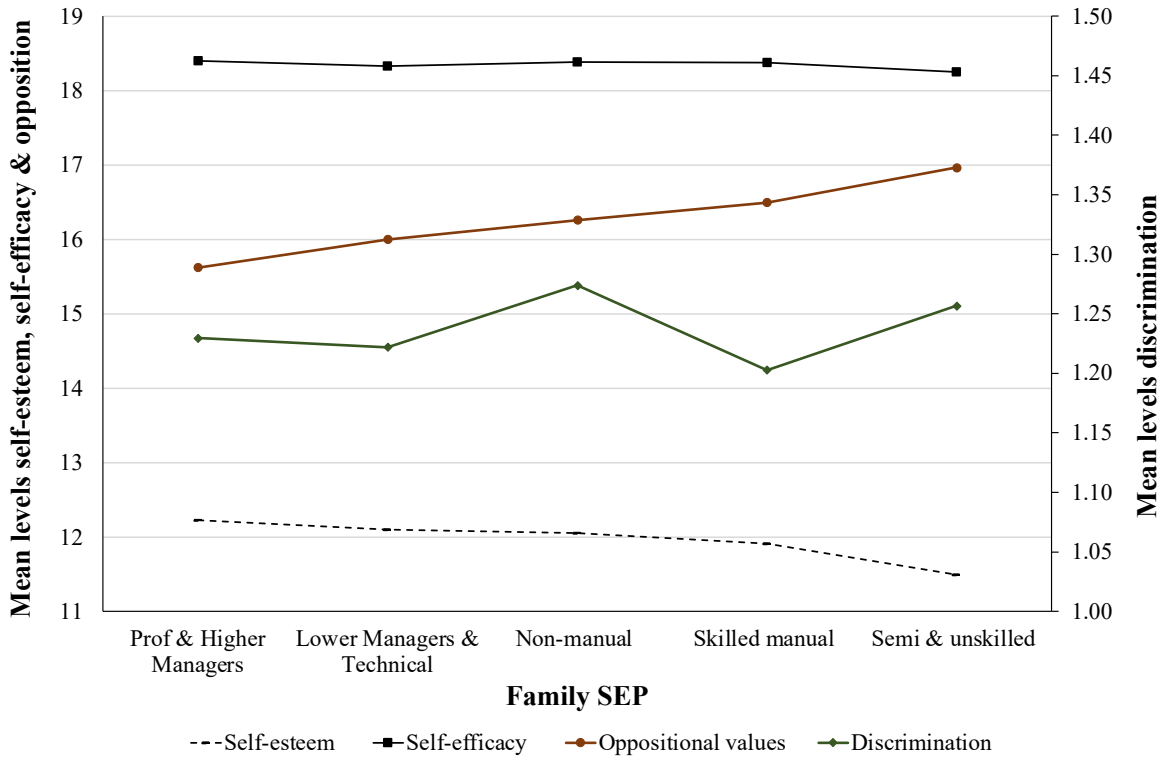
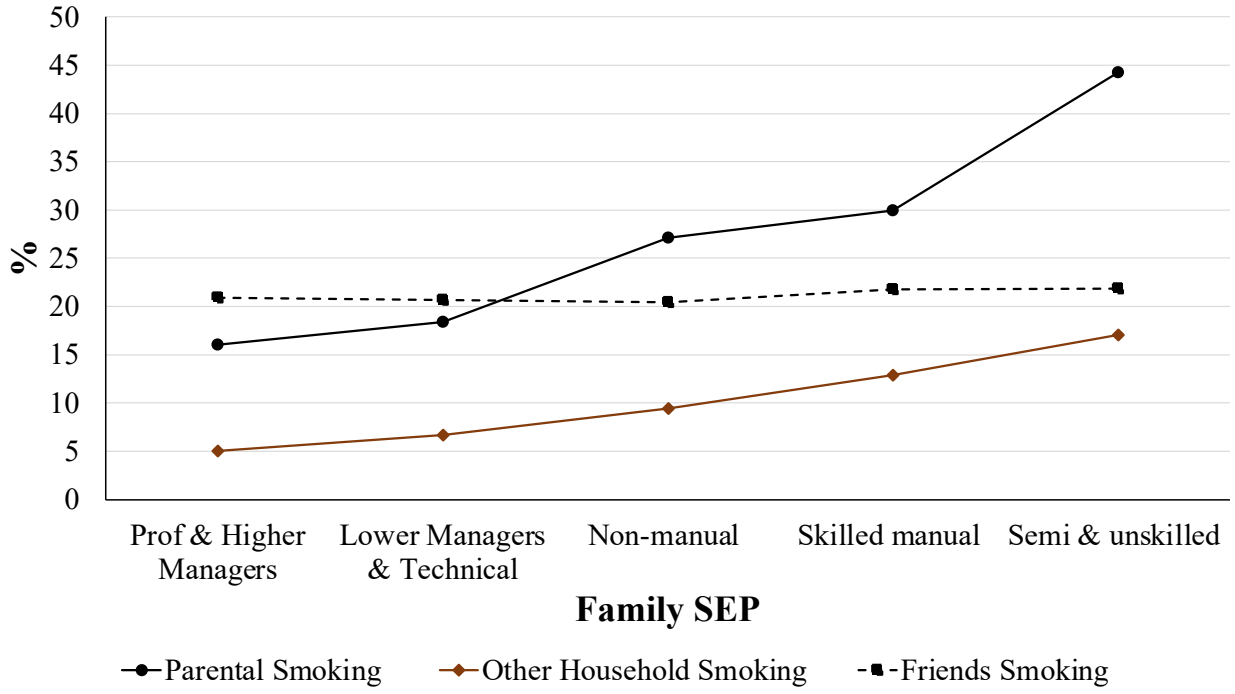


Figure 7.2. Proportion of (categorical) mediating variables by family SEP.



Do adolescents who smoke have more adverse patterns of our chosen mediating variables compared to those who do not? Figures 8.1 and 8.2 provide insight into this by providing means and percentages for the same mediating variables by adolescent smoking status. On average, young people who smoke have significantly higher exposure to smoking from their parents ($p<.001$) and other household members ($p<.001$), compared to those who do not smoke. Young people who smoke have a significantly higher proportion of smoking friends, compared to those who do not smoke ($p<.001$). Young people who smoke, on average, exhibit higher levels of opposition to authority and also tend to have more experiences of everyday discrimination, compared to those who do not smoke ($p<.001$). Reported levels of self-esteem and self-efficacy were higher amongst for those who do not smoke, compared to those that do smoke ($p<.001$).

Figure 8.1. Mean levels of (continuous) mediating variables by smoking status.

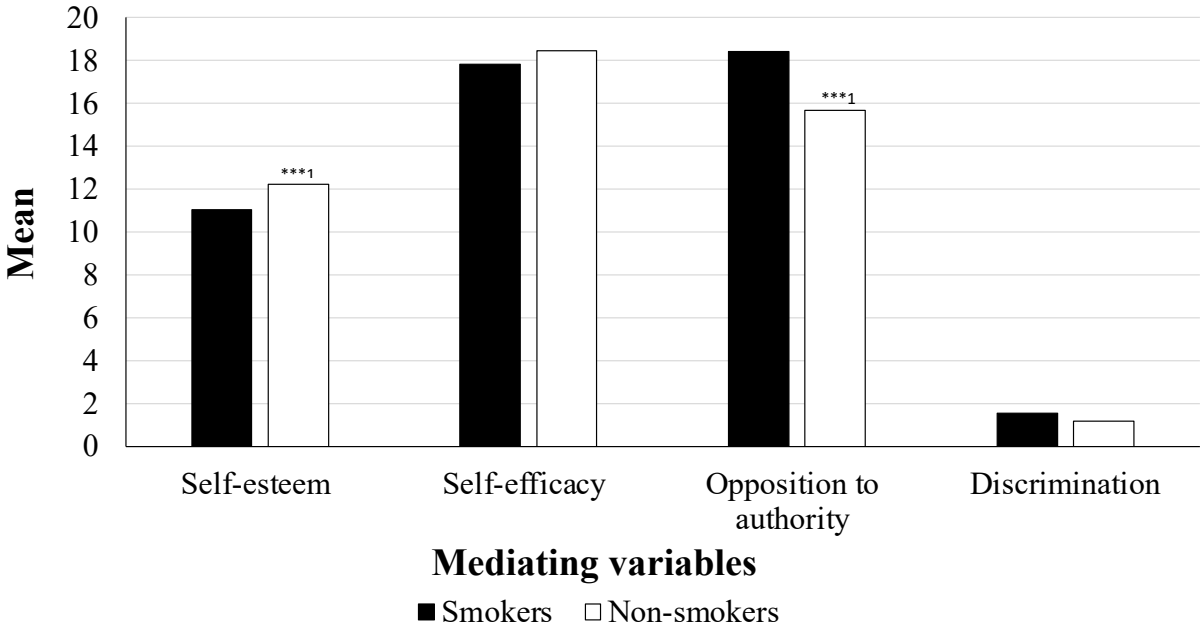
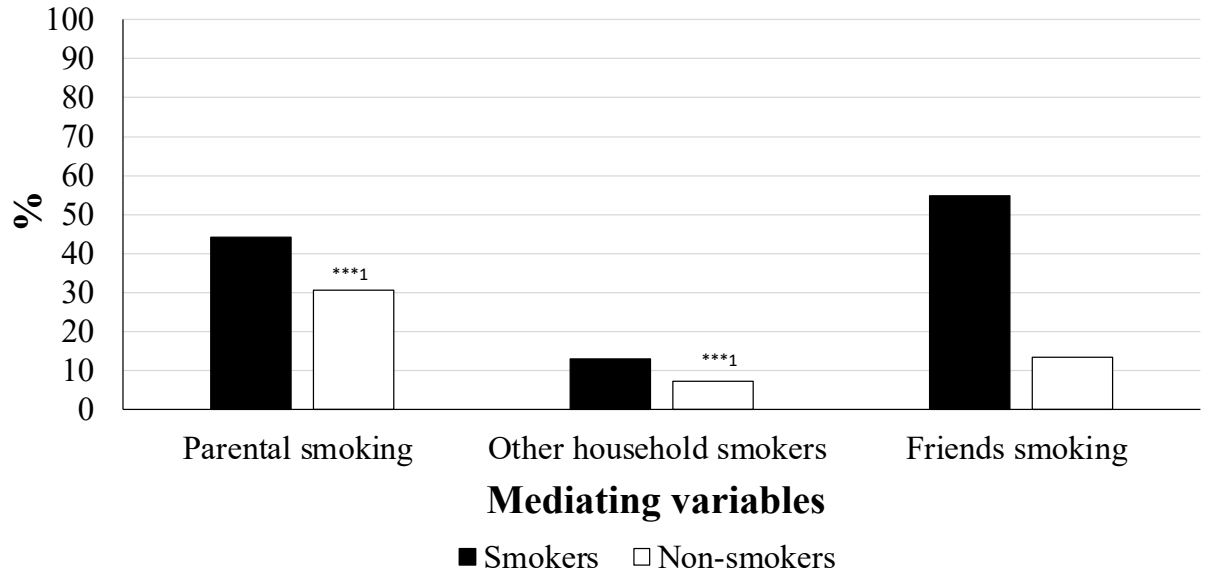


Figure 8.2. Proportion of (categorical) mediating variables by smoking status.



Do socio-economically disadvantaged adolescents, with high levels of oppositional values, have more smoking friends? Table five confirms that the proportion of friends that smoke increases as levels of oppositional values increase. Of those with high-levels of oppositional values young people from the non-manual households (34.95%) and young

[1] *p<.05, **p<.01, ***p<.001

people from professionals and higher managerial households (34.26%) report the highest proportion of smoking friends, compared to the other SEP household categories. These results illustrate that the combination of high oppositional values and an advantaged SEP seems to increase the likelihood of having friends' who smoke.

Table 5. Proportion smoking friends with oppositional values by SEP.

	Low	Medium	High
Prof & Higher Managers	13.47%	19.82%	34.26%
Lower Managers & Technical	14.27%	20.34%	30.66%
Non-manual	11.06%	15.71%	34.95%
Skilled manual	13.80%	19.70%	31.36%
Semi & unskilled	11.81%	18.23%	31.65%

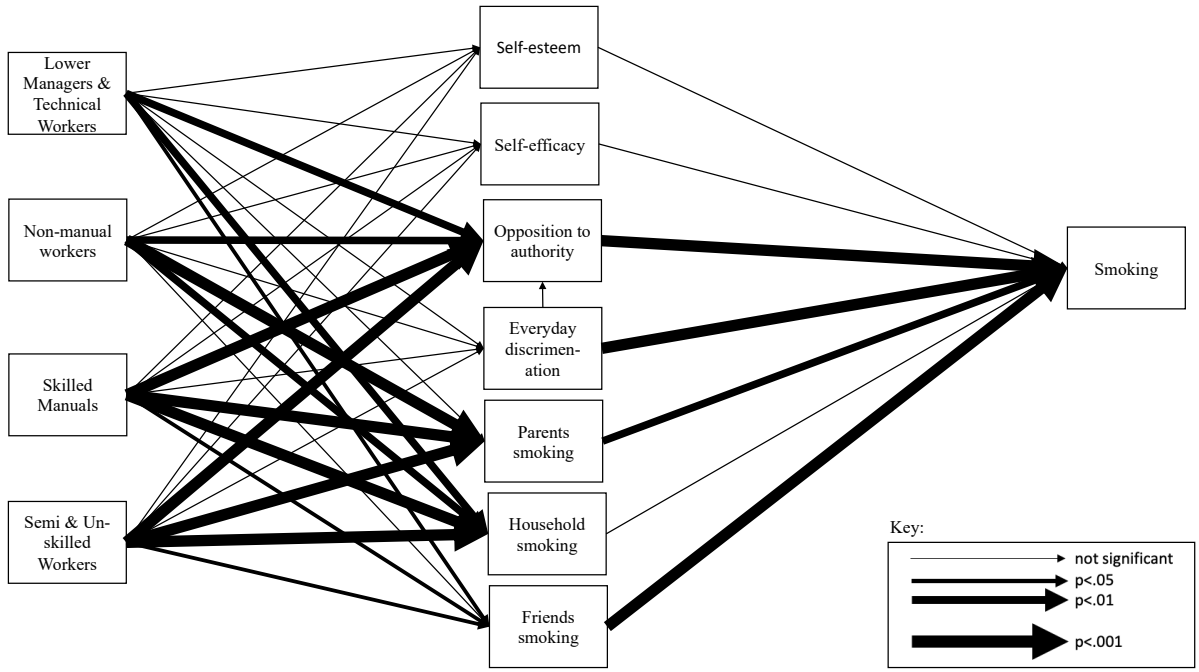
A total of 6,039 subjects were eligible for multivariate analysis. Using GSEM, we tested the ability of our three theoretical models to predict smoking, and the results are displayed graphically in figure nine (fully-adjusted model) with full tabular results available in the appendix (table four). Professionals and higher managerial households are the reference group for family SEP, our independent variable. This category was chosen as it represents the most socio-economically advantaged families with the lowest proportion of children smoking. Line thickness indicates level of significance for each of the pathways, with thicker lines indicative of more significant relationships i.e. lower p-values. Missing from the figure is the direct pathway from family SEP to smoking, which was removed to simplify the illustration, but is included as a pathway in the analyses. Firstly, we tested the variables associated with each model in isolation.

The direct effect of self-esteem on the probability of smoking was not significant. The direct effect of self-efficacy on the probability of smoking was also not significant. In short, there was no significant association between the variables that represent the psychological model and family SEP. However, there was a significant ($p<.01$) relationship between self-esteem and smoking status, where low levels of self-esteem meant the young person was more likely to be a smoker. The combined results demonstrate that there is no significant indirect pathway between SEP and the probability of smoking via our chosen psychological mediating variables, although lower self-esteem is associated with a higher likelihood of smoking at age 17/18.

Next, we tested the variables associated with the smoking exposure model. The results showed statistically significant direct effects of more disadvantaged SEP on exposure to parental smoking (non-manual, $p<.001$; skilled manual, $p<.001$;semi & unskilled, $p<.001$) and other household smokers (non-manual, $p<.01$; skilled manual, $p<.001$;semi & unskilled, $p<.001$). Our results also showed significant direct effects of exposure to parental smoking ($p<.001$), and exposure to other household smokers ($p<.01$), on the probability of smoking. This acts as evidence for statistically significant mediating pathways between SEP and the probability of smoking via exposure to parental smoking and other household smokers. There was no significant relationship between SEP and being exposed to friends' smoking. However, the direct effect on exposure to friends smoking and probability of smoking was statistically significant ($p<.001$). Taken in unison, this demonstrates that being exposed to friend's smoking does not mediate the relationship between childhood SEP and smoking status at age 17/18, despite smoking friends' being strongly associated with a higher likelihood of smoking during this period.

For the social-resistance model, there were significant direct effects of disadvantaged SEP (lower managers & technical workers, p<.01;non-manual, p<.01; skilled manual, p<.001;semi & unskilled, p<.001) on opposition to authority. There were also significant direct effects of opposition to authority (p<.001), and exposure to everyday discrimination (p<.001), on the probability of smoking. These results suggest that there are significant mediating pathways between SEP and the probability of smoking via levels of opposition to authority, but not via the experiences of discrimination. Furthermore, the direct effect of experiences of discrimination on opposition to authority was also statistically significant (p<.001). It seems that experiencing discrimination is not a causal pathway through which (1) childhood SEP predicts smoking or (2) childhood SEP results in increased experience of everyday discrimination which leads to higher levels of opposition to authority, which in turn leads to a higher probability of smoking, as put forward by this paper. However, experiencing discrimination does result in increased oppositional values, as argued by social-resistance framework as argued byFactor et al. (2011).

Figure 9. Fully adjusted GSEM predicting smoking status at age 17/18.



We used the Akaike information criterion (AIC) (Akaike, 1974)and the Bayesian information criterion (BIC) (Stone, 1979) to assess which of the three models best fits the observed data. According to these criterion, the smoking exposure model (model 2; AIC=24954.26, BIC=25279.65) is the superior model in terms of fit, followed by the social-resistance model (model 3; AIC=49805.89, BIC=50060.15) with the psychological model (model 1; AIC=65535.05, BIC=65782.67) explaining the least variation in smoking across our sample.

[2] Controls: sex and personality, N=6039

Discussion

Although overall prevalence of smoking has declined, social inequalities in smoking continue to increase in adult (Lahelma et al., 2016; Pampel, 2009; WHO, 2022) and youth populations(Rasmussen et al., 2009). This paper confirmed the inverse gradient in smoking, found in previous research, where disadvantaged SEP is associated with a higher likelihood of smoking, in the Irish context, using a nationally representative sample of young people. In the following sections, we have presented the results for our three models, namely: 'the psychological model,' 'the smoking exposure model' and the 'social-resistance model.' For each model we theorised how family disadvantage in childhood might lead to smoking in late adolescence, and there is a clear differentiation between individual-level and structural-level explanations for inequalities in smoking.

The psychological model

The psychological model represented an established individual-level explanation for smoking within the literature. The results of our preliminary bivariate analysis confirmed that self-esteem and self-efficacy are important predictors of adolescent smoking, as shown elsewhere(Khosravi et al., 2016; Lawrance & Rubinson, 1986). However, contrary to the underlying argument of the 'psychological model,' the results of our bivariate analysis did not show a significant relationship between these psychological traits and family SEP. Furthermore, our path analysis demonstrated that neither self-esteem nor self-efficacy were mediating pathways between childhood disadvantage and smoking at age 17/18. This suggests that variation in individual psychological traits can aid our understanding of smoking initiation in adolescence, in general, but crucially does not provide insight into how inequalities in smoking emerge during this period.

The smoking exposure model

Using the same statistical techniques, we found parental smoking to be a statistically significant mediating pathway between childhood disadvantage and smoking at age 17/18, when controlling for all other pathways in the fully adjusted model. Our results revealed that, for young people from disadvantaged families, there was increased exposure to parental smoking, and this exposure increased the likelihood of the young person themselves smoking. Research has previously demonstrated how parental smoking influences the onset of smoking in off-spring(Bandura & Walters, 1977; Wellman et al., 2016). We demonstrated how this socialisation process, whereby behaviours are observed and learned in the home environment, explains differentials in smoking across SEP groups. Alves et al. (2016)also previously found that parental smoking contributes to the transmission of SEP inequalities in adolescent smoking in a sample of students across six European cities.

Being exposed to smoking friends was a strongly significant positive predictor of smoking in adolescence, a relationship that is well documented(Simons-Morton & Farhat, 2010; Wellman et al., 2016). Less is known about whether this relationship differs according to SEP. Exposure to friends' smoking did not differ significantly by SEP, which suggests that disadvantaged adolescents, although more likely to be smokers themselves, are not more likely to be exposed to more smoking friends than adolescents from more advantaged backgrounds. Harris (2000) argues, in her work, that young people inherit a 'shared culture' from their parents that demands the same behaviours, such as smoking. However, our results contradict this idea that young people from disadvantaged backgrounds are more vulnerable to being socialised into smoking via their peer networks. Previous investigation (Moor et al., 2015) on the role of peers in explaining

socioeconomic inequalities in adolescent smoking found that “peer effects” are equally important among advantaged and disadvantaged young people, and therefore do not contribute to understanding inequalities. It seems that the effects of the peer group and youth culture cut across socioeconomic strata, as theorised by the equalisation hypothesis(West, 1997). In this paper we consider the effect of peer smoking as opposed to quality of peer relationships considered by Moor et al. (2015) for the “peer effect,” which may be considered a more direct measure for peer smoking influence. Nonetheless, we find the same results; peer factors did not mediate the relationship between SEP and smoking in adolescence.

The social resistance model

The results of our path analysis showed that varying levels of oppositional values is a statistically significant mediating pathway between disadvantaged childhood SEP and probability of smoking at age 17/18, even when controlling for all other pathways in the fully adjusted model. This suggests that the ‘social-resistance framework’ is a plausible structural explanation for inequalities in smoking in adolescence(Factor et al., 2011). It has been theorised that smoking cessation strategies have an adverse effect in that they strengthen further the social meaning attached to smoking as a physical manifestation of challenging adult authority (Schreuders et al., 2017). Our results seem to support this hypothesis.

In order to contribute to the ongoing debate between the role of ‘peer selection’ and ‘peer socialization’ processes in the development of substance use in adolescence(Henneberger et al., 2021), we considered the relationship between having smoking friends and oppositional values. Our results showed that young people with high-levels of

oppositional values had the highest proportion of smoking friends, compared to those that had low or medium-levels of oppositional values. Moreover, we found that 'oppositional' young people whose parents were 'non-manuals' or 'professionals' had the highest proportion of smoking friends. Surprisingly, these young people are some of the least socio-economically deprived and are also the least likely to be smokers themselves or have smoking parents. We reason that 'peer selection' and 'social-resistance' processes work in tandem for these (more advantaged) young people, where they seek out smoking friends as a way to assert an oppositional identity to their non-smoking parents. In this way the social resistance process remains the same, however, who they are distinguishing themselves from (Bourdieu, 2018) changes.

Experiencing discrimination was found to be an important predictor of smoking. Moreover, the process, put forward by Factor and colleagues (2011; 2013) whereby exposure to discrimination prompts 'resistance,' was supported here. However, experiences of discrimination were not disproportionately experienced by adolescents from a disadvantaged SEP. Therefore, experiencing discrimination did not prove to mediate the relationship between disadvantaged SEP and smoking initiation. It may be that our measure of discrimination cannot measure the more subtle and nuanced ways in which disadvantaged adolescents experience 'status anxiety' (Layte & Whelan, 2014) or feel their own cultural capital is under-valued within society.

Study limitations

There are limitations to our approach. Our data is concerned with risky (and often illegal) behaviours which may result in potential response bias from young people. We discussed the attempts used to mitigate this risk in the methods section of this paper, which include

supplemental self-complete questionnaires and ensuring the young person of confidentiality.

In terms of measuring the young person's SEP, more recent literature (Moor et al., 2015) advocates for the use of measures relevant to the age group, e.g. young person's report of their expected education-level(Doku et al., 2010; Gagné et al., 2018; LK et al., 2006). For a number of reasons we did not use this measurement: (1) the advantage of longitudinal panel data meant that we could assess the relationship between childhood SEP and later outcomes. (2) GUI consists of a general measure of expected education-level, and it has been shown that different dimensions of school-based status relate to adolescent smoking in opposing directions, meaning that one measure based on several dimensions might show inconsistent relationships(Sweeting & Hunt, 2015). (3) We argue that our measure captures the multidimensionality of SEP, incorporating economic, social, occupational and cultural aspects. Whereas education based measures reflects academic attainment and income measures do not incorporate the more nuanced elements of social class(Erikson & Goldthorpe, 1992; Goldthorpe et al., 1987).

Due to the use of smoking as a 'yes' or 'no' binary variable, it was not possible to carry out path analysis using Structured Equation Modelling (SEM). This study employed generalised structural equation modelling (GSEM) in its place, which has some drawbacks as discussed in the methods section of this paper. Another important limitation related to the use of a simple binary variable for smoking was the lack of specific analysis of 'occasional smokers,' which have been shown to display a social gradient(Alves et al., 2023). The proportion of 'professionals' that were occasional smokers was less than 5% i.e. too small for further analysis.

We put forward that smoking, as a consumption practice, is an outward material representation of inner feelings of opposition to authority used as a symbolic device to demonstrate distinction. However, we so not wish to imply that this is a deterministic process; a much higher proportion of young people with oppositional values are likely to smoke, compared to young people with low-levels of oppositional values, yet, the majority of young people do not smoke at all, which implies that the majority of those with oppositional values still do not smoke.

Conclusion

This paper is an empirical contribution to one of the central problems of sociology: what is the relationship between social structures and individual agency? This paper provided empirical evidence that the social context, within which the young person is embedded, has a more important influence on smoking risk than the young person's individual-level psychological or personality traits. Individualistic theories, based on human agency, although important for explaining smoking in adolescence, did not explain patterns of smoking behaviour at the aggregate class-level. Instead, we found that young people born into disadvantaged households are more likely to have smoking parents, and this exposure makes them more likely to smoke. This would suggest that they are structurally pre-disposed to smoke. We also found that young people from disadvantaged households have higher levels of oppositional values, compared to their peers from more advantaged households, which is in-turn positively associated with smoking initiation. This suggests that smoking risk is determined by one's positioning in the class-structure. However, we argue that individuals possess perceptual boundaries of what is possible and choose symbolic acts of resistance within these boundaries, where outright resistance is not

feasible(Scott, 1990). In this way, an economic and social environment produces a socially meaningful behaviour which is inherently concerned with taking back agency.

4 Can Social Exclusion Explain Inequalities in Health Behaviours? Insights from a Longitudinal Study of Irish Young People.

Introduction

Non-communicable diseases (NCDS; e.g. heart disease, lung disease, cancer, diabetes, overweight and obesity) are the leading cause of death globally. The risk of NCDs is inversely related to social and economic position (SEP) and, for this reason, has come to be known as the 'social gradient in health' (Marmot et al., 1978). The World Health Organization (WHO) estimates that about a third of the burden of disease in high-income countries is directly attributable to four key health behaviours: smoking, excessive consumption of alcohol, poor diet, and low levels of physical activity (2021). The social patterning of NCDs can be explained, in part, by differentials in these 'key' health behaviours by SEP. They have been shown to cluster according to SEP (Pampel et al., 2010), and increase the risk of mortality from NCDs (Petrovic et al., 2018).

Despite most deaths (attributable to NCDs) occurring in adulthood, exposure to risk factors for the development of health behaviours begins earlier in life. This paper focuses on the period of childhood and adolescence, the latter is known as a 'critical period' for the formation of health behaviours (Kuh et al., 2003). While the importance of investment in child infancy is broadly accepted, the comparable importance of this age is sometimes neglected. It is a time when major biological, psychological, and social changes occur, laying the foundations for adult life (Viner et al., 2015). Social transitions from being a dependent child towards stronger peer affiliation, the development of intimate partner relationships, and the transition from primary through second level to further education and employment are accompanied by new health, social and personal behaviours (Viner et al., 2015). Adolescence is a unique period of discovery and experimentation, during which

health practices emerge or are discarded. Cross-national data from the Health Behaviour of School Age Children (HBSC) study has been used to demonstrate large cross-national consistencies in ‘problem-prone’ behaviour and ‘experimentation’ in adolescence (Ahmad et al., 2023; de Looze et al., 2015).

As well as being a period of initiation it is also a period where harmful health behaviours (HHBs) often cluster together (Whitaker et al., 2021). This may be due to some behaviours increasing the risk of others through disinhibition or ‘gateway’ mechanisms or because of the influence of common psychological or social risk factors. Moreover, health behaviours established during childhood and adolescence tend to track into adulthood (Craigie et al., 2011), making health-related behaviours in adolescence an important predictor of health inequalities later in the life course (Dos Santos et al., 2020). Patterns of health-compromising behaviours are said to be 'crystallized' after a final stage of movement just prior to age 19 (van Nieuwenhuijzen et al., 2009). Despite this, fewer studies have examined the clustering of health behaviours among young adults, compared to studies on adult populations (Meader et al., 2016).

Evidence is accumulating regarding behaviours which tend to cluster together. The clustering of dietary patterns and physical inactivity is well-established (Martin et al., 2022) and there is evidence for the clustering of substance use, alcohol consumption and use of tobacco (Noble et al., 2015). However, with new concerns emerging about the harmful consequences of screen-time for young people, there is a lack of evidence on the overuse of internet-based technologies. Research in this area has, to date, focused on the clustering of screen-time, sleep quality, physical activity and dietary patterns only (Magee et al., 2013; Nuutinen et al., 2017) and has not integrated alcohol consumption and use of tobacco.

The evidence base is mixed and points towards covariance in sets of behaviours (e.g. substance use and poor eating and exercising habits) but also distinct clusters of behaviours, where individuals with poor eating and exercising habits are not more likely to be substance users (Dos Santos et al., 2020; Heikkala et al., 2014; Jonsson et al., 2023; Lazzeri et al., 2018; Noble et al., 2015; Nolan & Smyth, 2020; Skalamera & Hummer, 2016; van Nieuwenhuijzen et al., 2009; Whitaker et al., 2021). Depending on the patterns of behaviours that emerge, young people could be viewed as generalists (i.e. seeking out any harmful behaviour) or specialists (i.e. seeking out specific health risk behaviours). The characteristics and motivations of 'specialists' remains unclear, however, the characteristics of 'generalists' have been broadly established, and include those from a disadvantaged SEP (Dos Santos et al., 2020; Heikkala et al., 2014; Jonsson et al., 2023; Lazzeri et al., 2018; Noble et al., 2015; Skalamera & Hummer, 2016; van Nieuwenhuijzen et al., 2009).

This paper will put forward, and test empirically, potential risk factors for engagement in the clusters of HHBs that we find in our data, assuming there are one or more corresponding underlying risk factors for members of the same cluster. Our focus on risk factors is a novel approach, as previous research has focused on highlighting the consequences of the clustering of HHBs for adolescent health (Ahmad et al., 2023; Akasaki et al., 2019; Boone-Heinonen et al., 2008; Conry et al., 2011; Magee et al., 2013; Nuutinen et al., 2017; Walsh et al., 2020).

Background

Having established the social structuring of health in the 1970s, epidemiological research turned to identifying risk factors more proximate to the cause of disease (Black et al.,

1988; Blane et al., 2013; Krieger, 2011; Marmot & Wilkinson, 2005; Solar & Irwin, 2007), which led to important advances in understanding how SEP shapes health and mortality, as well as, the identification of potential modifiable mechanisms for policy intervention. The question remains as to how SEP shapes the proximate drivers of health e.g. health behaviours. Link and Phelan (1995) argue that social conditions are not proxies for more proximate causes but are themselves 'fundamental causes' of ill-health.

Fundamental Causes Theory (FCT) posits that avoiding disease and death, requires an agentic response to public health advice on how to protect against disease, or how to gain a health advantage. However, one's ability to benefit from public health knowledge relies on access to resources, such as money, knowledge, prestige, power, and beneficial social connections (Link & Phelan, 1995; Link & Phelan, 2002; Phelan et al., 2010). Access to these resources shapes individual health behaviours by influencing whether people know about, have access to, can afford to, and are supported in their efforts to engage in health enhancing behaviours. In this way, resources influence the 'causes of the causes' or proximate causes of disease (i.e. health behaviours) as opposed to the disease itself. FCT has been very influential in shaping research on how and why inequalities in health have persisted over time, even as population health improves and the proximal causes of disease evolve (Kadushin, 1964; McCartney et al., 2021). Despite this, empirical studies concerning FCT have only focused on the implications of the lack of resources (or SEP) on health and mortality directly. In this paper, we investigate the relationship between resource deprivation (as representee of a fundamental cause), social exclusion and health behaviours.

Fundamental Cause Theory and Social Exclusion

The term ‘social exclusion’ has its origins in French political thinking; René Lenoir (Secretary of State for Social Action in the 1970s) categorised a tenth of the French population as ‘excluded’ (e.g. those with illness or disability, single parents). The concept was used to legitimise policy reform around social provisions (social insurance) to enable ‘les exclus’ to integrate further (Béland & Hansen, 2000; Rawal, 2008) due to concern that groups pushed to the margins of society threaten the social order, with consequences of detachment from the labour market, delinquency and criminality (Layte et al., 2010; Smith & Bohm, 2008).

Social exclusion has been frequently redefined and more groups included (e.g. school leavers, unemployed) (Aasland & Fløtten, 2001). A number of different definitions of social exclusion have been offered (Levitas, 2006) but most emphasize that a persistent lack of income leads to exclusion from economic, social and often political life (Aasland & Fløtten, 2001), with a focus on relativity i.e. deprivation relative to a community-level norms as opposed to an absolute lack of resources relative to needs (Townsend, 1979). The ‘social exclusion framework’ maintains that exclusion arises from low levels of resources and the subsequent fall in the consumption of the goods and services necessary for individuals, families and groups to obtain a healthy lifestyle similar to their fellow citizens. This includes consumption of goods and services pertaining to diet and exercise. The absence of resources may help to explain why some groups are less able to adopt health protecting behaviours, but it is less clear how it helps us understand why some groups are more likely to adopt health damaging behaviours (such as smoking and alcohol misuse). The following section sets out the theory of ‘resistance’ behaviours which provides a theoretical explanation for how social exclusion could lead to the adoption of health damaging behaviours.

Social Exclusion and Social Resistance

Factor et.al (2011; 2013) have observed that, within societies, marginalised (or excluded) groups engage in more HHBs relative to the majority population group. Building on Scott's (1990) theory of 'everyday resistance,' which seeks to understand the ways in which marginalised groups defy dominant groups with small acts of resistance in the context of peasant societies, Factor and colleagues propose that, in response to feelings of alienation and rejection from larger society, marginalised groups engage in risky behaviours to gain a sense of symbolic empowerment. This suggests that social exclusion influences behaviours related to health in the same way it influences behaviours that represent an absence of commitment to the country's laws. Lister (2015), also using Scott's theory, has characterised these behaviours among people in poverty as 'getting (back) at' behaviours that elicit a sense of social justice.

Considering Bourdieu's (1989, 2018), Bartley's (2016) and Cockerham's (2005) ideas about the influence of status groups on health lifestyles, it is clear that groups differentiate themselves by the development of patterns and tastes and consumption. We propose that engaging in HHBs works to differentiate socially excluded individuals from the majority (included) group by creating an oppositional social identity, which is visible and replicable if others wish to show their allegiance to their marginalised peers. Young people, at an early age, observe that clothing worn, objects owned, and activities pursued all have social value in shaping their identity to others (and themselves), and thus help to define which groups they belong to.

In this way, motivation for engagement in HHBs could be fuelled by a desire for group membership, where such an affiliation may also offer an opportunity to express resistance.

The desire for group membership, over and above the desire to promote one's health is referred to as a 'countervailing mechanism,' within the literature on FCT. This is where other competing goals (e.g. manliness, beauty) have been shown to be more powerful motivators than health, and are pursued to the detriment of health (Lutfey & Freese, 2005; Phelan et al., 2010). There is a similar 'complexity and even irrationality' discussed within the educational resistance literature (Johansson & Vinthagen, 2016), most famously in the work of Willis (1977) where their oppositional subculture had the effect of reproducing their family's educational disadvantage.

This paper focuses on HHBs during the period from 13-18 years. This allows us to examine multiple behaviours, some of which only manifest in adolescence. Using latent class analysis, we identify groups that have distinct patterning of our chosen health behaviours (diet, physical activity, screen-time, smoking, underage-drinking). Using multi-nominal logistic regression, we examine whether level of resources (income), in and of itself, is a risk factor for membership of latent class groups characterised by HHBs. Our measure of income poverty covers three waves of the study spanning a period of 9-18 years. Longitudinal analysis has led the way in highlighting poverty dynamics, and this paper offers an opportunity to assess the consequences of periodic as well as sustained exposure to poverty (Fischer, 2011; Rawal, 2008; Redmond et al., 2022). Using the same method we then test whether the formation of, and level of, oppositional values is a risk factor for latent class group membership. In doing so, we aim to extend the body of empirical evidence supporting FCT to include health behaviours, whilst also testing our proposed theory that a 'fundamental' lack of resources and the social detachment that accompanies the experience of persistent poverty (social exclusion) can lead to the engagement in HHBs as 'resistance behaviours.'

Material & Methods

(see Data section in the Introduction)

Levels of missing data across variables were low, on average, but reached 10.4% on one variable (see table one in the supplementary material). Non-response is a feature of all surveys, and by using tracing procedures it is possible to establish if inter-wave attrition is systematically associated with family or other characteristics measured in our survey (e.g. household social class), as is the case in GUI(Thornton et al., 2010). This has the potential to bias our analysis and for this reason, missing values on the control and predictor variables were imputed for analysis using multiple imputation by chained equations (MICE) to produce ten imputed datasets, using the Rubin method (Rubin, 1987) as implemented in STATA 18. The data were also reweighted for analyses to take account of the original sample error and subsequent attrition using a range of factors and a minimum distance algorithm. The final sample is representative of young people who were residing in Ireland at nine years of age in 2007/2008 and who continued to live in Ireland at 20 years of age in 2018/2019(Thornton et al., 2010).

Measures: Dependent Variable

Our outcome variable for this analysis is placement into the groups produced by our latent-class analysis (LCA). The LCA is based on five harmful health behaviours (HHBs; physical activity, screentime, diet, smoking, underage-drinking). Young people with shared patterns of engagement in the selected HHBs were identified, and assigned to their corresponding group.

Physical Activity (PA)

PA levels were calculated using the Godin-Shephard Leisure Time Exercise Questionnaire (GSLTEQ) at wave three (age 17). The young person reported the number of days out of the previous 14 that they had engaged in 'hard' or 'light' exercise for at least 20 min. Hard exercise was defined as 'enough to make him/her breathe heavily and make his/her heart faster'. Light exercise was defined as 'not hard enough to make him/her breathe heavily and make his/her heart beat fast.' The total activity score is computed by weighting the frequency of strenuous and light exercise per week by the values established by the developers. For descriptive and latent-class analyses it was necessary to categorise the activity score into tertiles: 'low' 'moderate' and 'high.' The GSLTEQ has previously demonstrated concurrent validity with measures of maximum oxygen intake and muscular endurance (Godin et al., 1986) and construct validity (Godin & Shephard, 1985; Zelener & Schneider, 2016) and test-retest reliability (Sallis et al., 1993; Zelener & Schneider, 2016).

Screentime

At wave three (age 17) the young person was asked 'on a normal weekday during term-time, about how much time do you spend…' on each of the following: watching TV, interacting online (social media/browsing), computer gaming. These variables were combined to ascertain total minutes of screentime on a normal weekday. For descriptive and latent class analyses it was necessary to categorise total screentime into tertiles: 'low' 'moderate' and 'high.'

Diet

Dietary quality is proxied using the Amhurst Sallis Food Frequency Questionnaire, by asking the young person how often they had eaten thirteen different food types in the previous 24hrs (never/once/more than once), as part of the main questionnaire at wave three (age 17). Fresh fruit, cooked or raw vegetable and cheese, yogurt and other dairy

products were defined as ‘heathy’. Processed meats (e.g. sausages hamburgers), fried foods (e.g. chips/crisps) and sweet deserts (e.g. biscuits, donuts, cake) were defined as ‘unhealthy.’ ‘healthy lifestyle’ foods were scored positively (once = 1; more than once = 2) and ‘unhealthy lifestyle’ foods negatively. Summing the food types yields a scale which varies between -12 and 12+. For descriptive and latent class analyses it was necessary to categorise the dietary score into tertiles: ‘low’ ‘moderate’ and ‘high’ quality diet.

Smoking

Smoking status was self-reported by the young person at wave three (age 17): ‘have you ever smoked a cigarette?’ (yes/no) and subsequently ‘which of the following best describes you?’ (only ever tried smoking once or twice/used to smoke but not now/smoke occasionally/smoke daily/do not smoke). The answer categories were recoded into ‘daily smokers’ ‘occasional smokers’ and ‘non-smokers,’ with respondents who answered ‘no’ to the first question and ‘only ever tried smoking once or twice,’ ‘used to smoke but not now’ and ‘do not smoke’ being classified as non-smokers. Due to the sensitive nature of the questions (smoking is illegal under the age of 16 in Ireland and parents may not be aware of the participant’s smoking) the questions were administered on a supplemental self-complete questionnaire, and the young person was assured of confidentiality.

Underage Drinking

The young person reported, retrospectively, on the age ‘when had [their] first full drink of alcohol – more than a few sips’ at wave three (age 17). Due to the sensitive nature of the questions (consumption of alcohol is illegal under the age of 18 in Ireland and parents may not be aware of the participant’s drinking) the questions were administered on a supplemental self-complete questionnaire, and the young person was assured of

confidentiality. The responses were used to ascertain if the young person drank and how young they were when they started, and the responses were categorised to demonstrate the differences (15 or younger/16/17/18/don't drink).

Predictor Variables: Persistent Poverty

The primary care giver (PCG) reported, prospectively, on income sources from all household members at wave one (age 9), wave two (age 13) and wave three (age 17/18). In order to make meaningful comparisons between households on their income, household size and structure was taken into account by assigning a weight to each household member to create an 'equivalised' total disposable income (after tax and welfare transfers). The first adult is assigned a weight of 1 with a weight of 0.66 allocated to all subsequent adults and 0.33 to each child (14 years or less). The weightings used for the Irish official income poverty line(Roantree et al., 2022). Equivalised income was ranked and transformed into income quintiles. Young people whose families appeared in the bottom two equivalised income quintiles were considered to have experienced income poverty, and the number of times they appeared in the bottom two income quintiles, over the period of the three waves of data collection, was used to assess the persistence of their experience (e.g. 0 – no experience; 3 – experienced poverty at all three waves). Income quintiles were used to capture relative, as opposed to absolute levels of deprivation. A measure of relative deprivation measure was deemed more appropriate for capturing the processes associated with fundamental cause theory (FCT) and social exclusion.

It is well established that cross-sectional analyses do not give a representative picture of the contrasting consequences for those who experience a spell of poverty compared to those who are persistently poor(Li & Chzhen, 2023; Whelan et al., 2002).

Oppositional Values

Opposition to authority was measured using a scale from the ESRC 16-19 initiative research programme. The scale was administered at wave three (age 17/18) to the young person. It contained eight-items (e.g. 'it can be okay to do something which is against the law if it is to help a friend,' 'people in authority like teachers always pick on me'). The young person indicated their level of agreement with the statements on a 4-point scale (strongly agree/agree/disagree/strongly disagree). Higher scores are indicative of more oppositional values. For descriptive analyses it was necessary to categorise level of oppositional values into two groups: 'low' and 'high.' The Cronbach alpha value for the scale at age 17/18 was 0.72(Murphy et al., 2019).

Control Variables

Whether or not the young person had a chronic illness (physical or mental health problem, illness or disability) and gender (male/female) were included, from wave one (age 9), as control variables in the analysis. Since previous research suggests that health-related behaviour is significantly associated with certain personality traits(Vollrath et al., 1999), the Ten Item Personality Inventory (TIPI) was included as a control variable to measure aspects of the young person's personality, as reported by the young person at wave three (age 17/18)(Gosling et al., 2003). The 'Big-Five' framework is one of the most widely used and extensively researched models of personality; classifying individual differences in personality into five broad empirically derived domains: 'Openness to Experience,' 'Conscientiousness,' 'Extraversion,' 'Agreeableness,' and 'Neuroticism.' Each personality dimension was rated on a seven-point scale, with answer categories ranging from disagree strongly to agree strongly, where strongly agree indicates a stronger presence of the respective personality trait. For descriptive analyses it was necessary to categorise each

answer category into two groups: ‘low’ and ‘high.’ Gosling et al. (2003)noted the scale has good test-retest reliability (r= .72), and report convergent correlations with the personality scale: the ‘Big-Five Inventory.’

Analysis Strategy

Our principle aim was to assess whether the young person's participation in HHBs was patterned, using Latent Class Analysis (LCA) and if so, to examine whether young people with similar patterns of behaviours have distinctive risk profiles, using multi-nominal logistic regression.

LCA is a statistical technique which relates observed variables to unobserved or 'latent' categorical variables. The technique allows the analyst to vary the number of 'latent classes' to find the best statistical 'fit' with the observed variables and then provides a probability that each young person will be a member of each latent class. LCA is a data-driven approach, identifying the optimal subdivision using the data themselves rather than a pre-existing theory.

The optimal number of latent classes was established using an overall assessment of measures of fit, with additional consideration of the value of the solution in terms of providing insight into the empirical problem (based on cluster interpretability and parsimony of the model). Entropy, Akaike's Information Criterion (AIC), Bayesian Information Criteria (BIC), sample size adjusted BIC (aBIC*) are shown in table two in the supplementary material. Entropy with values approaching one indicate clear delineation of classes(Celeux & Soromenho, 1996). Results for our entropy values do not indicate a good separation of classes, with no values reaching the standard i.e. >.80, which represents a good classification of participants into classes. In this case, it is recommended to consider all fit statistics to enumerate the classes, as well as, model interpretability and utility(Nylund et al., 2007). Lower values of the AIC, BIC and aBIC suggest a better fitting model. In general, if more than one models is "true," AIC fails to choose the smaller model,

while BIC chooses the more close fitting model (Chakrabarti & Ghosh, 2011). This is because AIC does not depend directly on sample size. Moreover, AIC presents the danger that it might overfit, whereas BIC presents the danger that it might underfit, simply in virtue of how they penalize free parameters(Nylund et al., 2007). We compared models for two to six classes. AIC values indicated a six-class solution, BIC values indicated a three-class solution and aBIC values indicated a four-class solution (see table two in the supplementary material). The results reflect our previous note that AIC tends to overfit and BIC tends to underfit. aBIC was favoured due to its consideration of sample size, and particularly a large sample, suggesting a four-class. On further inspection, the five-class (proposed by BIC) was disqualified as the smallest latent group had less than 6% of the total participants. In short, a four-class solution was found to fit the data best.

To facilitate the LCA analysis, the continuous variables screentime, diet and physical activity were categorised into 'low' 'moderate' and 'high' levels of engagement. LCA analysis was performed using the doLCA STATA Plug-in for STATA 18(Lanza et al., 2018). Class membership for each study participant was identified as the class with the highest probability for each participant. Once class membership was established, we examined the profile of each latent class group on the variables from which it is derived (physical activity, screentime, diet quality, underage drinking and smoking), as well as, additional demographic variables and variables of interest. For interpretability, we reverse coded the variable for physical activity and diet (in its continuous form), so that higher scores on all variables relating to HHBs would run in the same direction i.e. indicate more harmful engagement. The continuous variables (physical activity, dietary and screentime) were standardised to calculate z-scores for these variables. The purpose of standardizing was to provide a common scale for comparison across variables. In addition to this, z-scores

measure how many standard deviations below or above the population mean a raw score is, which gives a clearer picture of how far from the average score a data point is. The z-scores run from minus one to plus one where values closer to plus one represent unhealthy behaviours. We then plotted the mean level of engagement with each health behaviour variable by latent class group, to display the patterns of behaviour graphically (using the reverse coded and standardised variables).

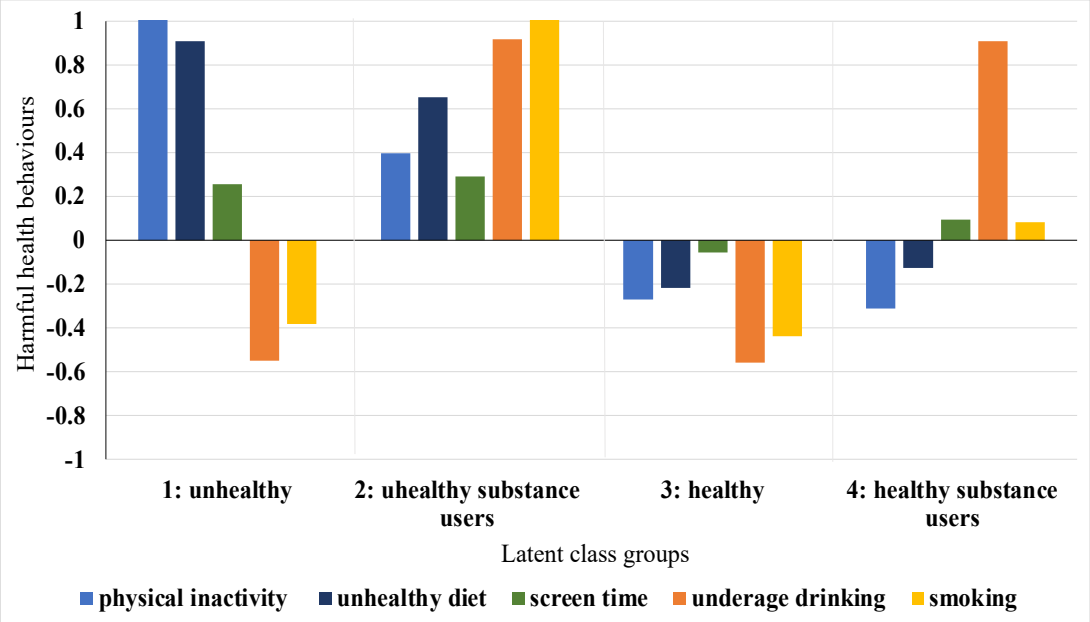
We then assessed the relationship between the controls, predictors and LCGs descriptively before carrying out multivariate analyses. Multinomial logistic regression was used to evaluate predictors of latent class group membership, adjusting for child's health status, gender and personality (controls). We looked at the predictor variables representing social exclusion (periodic and persistent poverty) and social resistance (oppositional values) separately and then together. The likelihood of cluster membership were reported as relative risk ratios (RRR) with 95% confidence interval (CI).

Results

Latent- class Analysis

Figure one (below) presents the standardised group means for each health behaviour across the four latent class groups (LCGs), with higher scores indicating worse health behaviour. The four LCGs are clearly distinguishable across the five dimensions of health behaviours. The first and second classes are characterised by physical inactivity, low quality diet and above average screentime ('lifestyle behaviours') but differ in terms of alcohol and tobacco use ('substance use'). The first class has low substance use and is labelled the 'unhealthy lifestyle' group whereas the second class reported the highest proportion of underage-drinking and smoking across the four LCGs, and is thus labelled 'unhealthy lifestyle and substance users.' The third class reported a moderate-high quality diet, low levels of screentime and moderate-high levels of physical activity, the majority did not drink alcohol until legal age and there were no smokers. This group is thus labelled the 'healthy' group. The fourth class, whilst also characterised by a healthy lifestyle (moderate-high quality diet, physically active and low-moderate screentime), reported high levels of underage-drinking and some occasional smoking. For this reason the group is labelled 'healthy lifestyle but substance users.'

Figure 10. Mean level of harmful health behaviours (standardised) by latent class groups.



The patterns that emerge suggest that the domains (health behaviours) are not independent. There are clear distinctions between engagement in healthy and unhealthy behaviours, in terms of diet, physical activity and screentime, and there is a further distinction between substance users and non-substance users. This would suggest that there is an underlying process which increases the risk of engagement in the triad of poor health behaviours and/or substance use, rather than the risk of exhibiting each behaviour independently.

Further sensitivity analyses by sex shows that the overall pattern in figure one does not vary significantly between male and female participants.

Descriptive Statistics

Table one provides the frequency and proportion of our variables of interest overall, and by each latent class group (LCG), with study weights applied. Overall, the sample contained a marginally larger proportion of male (50.7%) participants and the majority of the sample

did not have a chronic condition (93.9%). In terms of health behaviours, the sample contained a larger proportion of non-smokers (79.5%), compared to occasional smokers (12.3%) and daily smokers (8.2%). Many young people had their first drink under the age of 15 (40.1%) or by 16yrs (29.8%). Young people were evenly distributed across the physical activity categories (low, moderate and high) with a marginal majority in the lowest category (35.0%), and screentime was low (41.3%) on average, with no distinction between types of usage (e.g. educational, recreational). More young people had a low (44.9%) or moderate (35.55%) quality diet, compared to a high quality diet (19.5%).

Table one also provided information on the distribution of the predictor variables, enabling us to compare values across LCGs. This provides an initial descriptive assessment of the relationship between the LCGs and the predictor variables, which will be supplemented in the following section with multivariate analyses. LCG one ('unhealthy lifestyle') and LCG two ('unhealthy lifestyle and substance users') had the largest proportion of young people who had experienced three waves of income poverty/persistent poverty (25.9% and 23.8% respectively). Young people who experienced two waves of income poverty were also more likely to be grouped into these same LCGs (unhealthy: 22.9%, unhealthy lifestyle and substance users: 24.0%). LCGs that were characterised by healthy lifestyle behaviours, in terms of screentime, diet and exercise, consisted of young people whose families had never experienced income poverty (healthy: 40.8%, healthy lifestyle and substance users: 43.6%) or experienced for one wave (healthy: 23.9%, healthy lifestyle and substance users: 22.4%). The relationship between persistent poverty and LCGs is shown graphically in Fig. 2 (below). As shown in Fig. 3 (below), young people in the LCGs characterised by substance use reported higher than average levels of oppositional values (healthy lifestyle

and substance users: 0.27, unhealthy lifestyle and substance users: 0.86) compared to the LCGs labelled healthy (-0.21) and unhealthy(-0.04).

Table 6. The distribution of variables in the sample overall and by LCGs.

HHBs	Overall		Class 1:'unhealthy lifestyle'		Class 2: 'unhealthy lifestyle and substance users'		Class 3: 'healthy lifestyle'		Class 4: 'healthy lifestyle and substance users'	
	N	%	N	%	N	%	N	%	N	%
Smoking status										
Non-smoker	4845	79.5	934	97.1	0.0	0.0	2808	100.0	1222	71.2
Occasional smoker	749	12.3	25	2.6	194	32.1	0	0.0	492	28.7
Daily smoker	497	8.2	2	0.2	411	67.9	0	0.0	2	0.2
Underage Drinking										
Age first alcoholic drink										
Under 15	2442	40.1	244	25.4	551	91.0	0	0.0	1523	88.8
age 16	1815	29.8	208	21.6	39	6.5	1476	52.6	172	10.1
age 17/18	1204	19.8	216	22.5	15	2.5	1001	35.6	20	1.2
no alcohol yet	630	10.3	294	30.6	0	0.0	332	11.8	0	0.0
Diet Quality										
Low	2728	44.8	879	91.4	419	69.3	770	27.4	550	32.1
Moderate	2172	35.7	82	8.6	148	24.4	1270	45.2	730	42.6
High	1191	19.6	0	0.1	38	6.3	768	27.3	436	25.4
Physical activity										
Low	2129	35.0	879	91.4	302	49.9	544	19.4	301	17.5
Moderate	1985	32.6	81.841	8.5	204.78	33.9	1,086.49	38.7	645.9629	37.6
High	1976	32.5	0.79	0.1	98.56	16.3	1177.61	41.9	769.07	44.8
Screentime (minutes)										
Low	2507	41.2	346	36.0	214	35.4	1244	44.3	722	42.1
Moderate	1963	32.2	278	28.9	167	27.6	998	35.5	536	31.2
High	1621	26.6	338	35.2	224	37.0	567	20.2	458	26.7
Controls	N	%	N	%	N	%	N	%	N	%
Gender										
Male	3085	50.7	596	62.0	337	55.7	1305	46.5	743	43.3
Female	3006	49.4	366	38.0	268	44.4	1503	53.5	973	56.7
Personality										
Extraversion										

Low	3759	61.7	695	72.3	346	57.3	1799	64.1	917	53.5
High	2332	38.3	267	27.8	259	42.7	1009	35.9	799	46.6
Agreeableness										
Low	3373	55.4	506	52.6	341	56.4	1472	52.4	1049	61.2
High	2718	44.6	456	47.4	264	43.6	1336	47.6	667	38.8
Conscientiousness										
Low	3806	62.5	574	59.7	463	76.5	1641	58.5	1106	64.4
High	2285	37.5	388	40.3	142	23.5	1167	41.6	610	35.6
Emotional stability										
Low	3694	60.6	666	69.2	409	67.7	1612	57.4	983	57.3
High	2397	39.4	296	30.8	196	32.3	1196	42.6	733	42.7
Openness to experience										
Low	3262	53.6	578	60.1	308	50.9	1570	55.9	807	47.0
High	2829	46.5	384	39.9	297	49.1	1238	44.1	909	53.0
Chronic illness										
No	5666	93.9	885	92.3	567	95.0	2613	94.0	1602	94.2
Yes	370	6.1	73	7.7	30	5.0	167	6.0	99	5.8
Predictors	N	%	N	%	N	%	N	%	N	%
Persistent poverty										
No waves	2346	38.5	289	30.1	185	30.5	1146	40.8	748	43.6
One wave	1386	22.8	204	21.2	131	21.7	671	23.9	385	22.4
Two waves	1247	20.5	220	22.9	145	24.0	538	19.2	335	19.5
Three waves	1112	18.3	249	25.9	144	23.8	452	16.1	248	14.5
Oppositional values										
Low	3118	51.2	530	55.1	139	23.0	1761	62.7	740	43.2
High	2973	48.8	432	44.9	466	77.0	1047	37.3	976	56.9

Figure 11. No. waves experienced income poverty by latent class groups.

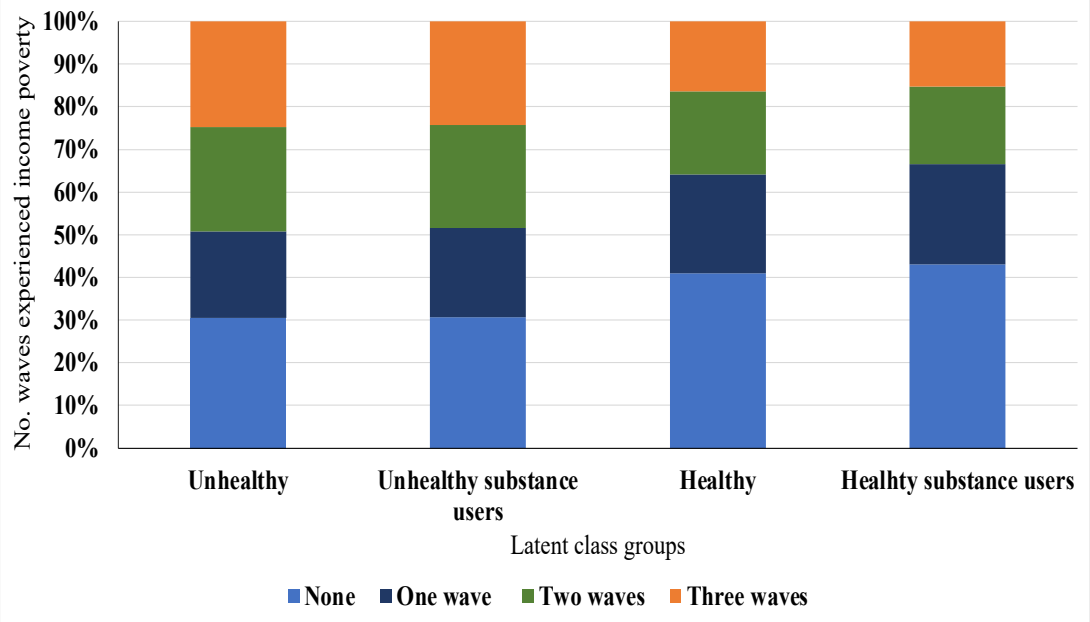


Figure 12 Mean level of oppositional values by latent class groups.

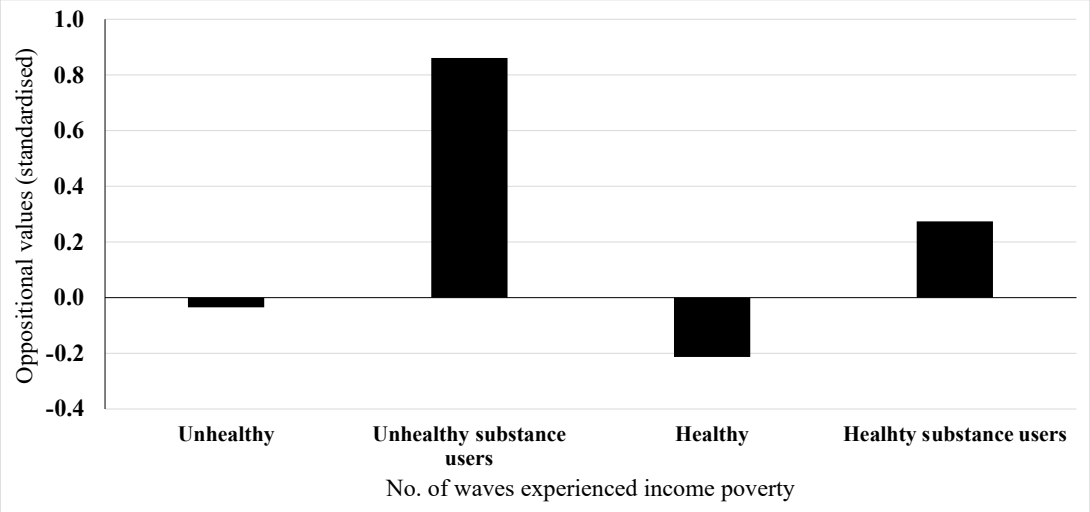
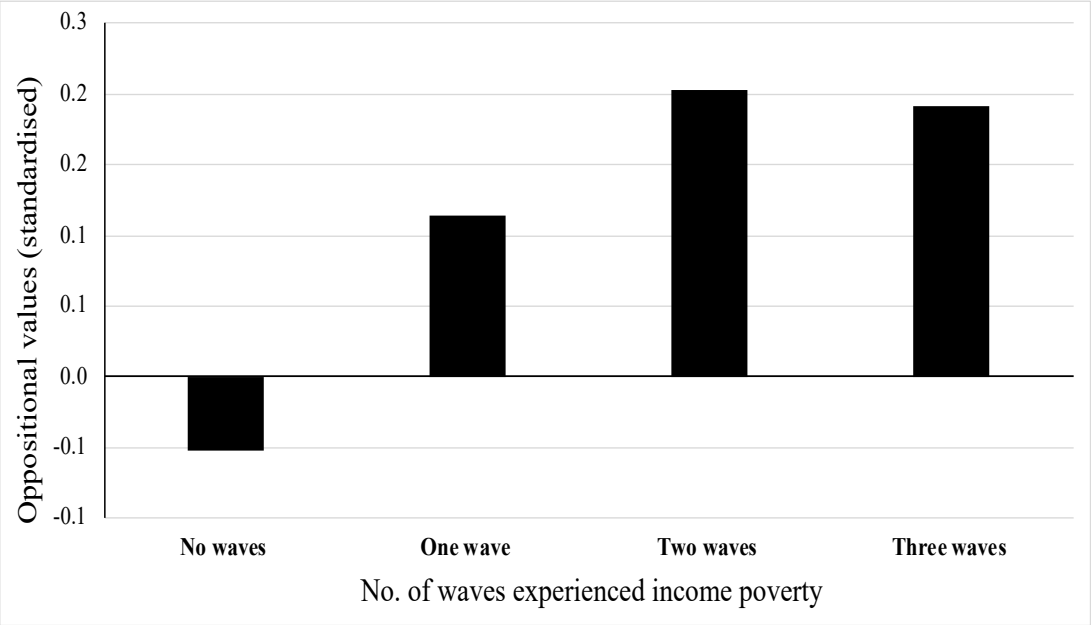


Figure 13. Mean level of oppositional values (standardised) by duration of income poverty.



Multivariate Analysis

Our analyses have shown that young people can be differentiated by their patterns of engagement in HHBs. In addition to this, our initial bivariate analysis, shown in table one, figure three and figure four, indicates varying distributions of our chosen risk factors (persistent poverty and social resistance) across the LCGs. Moreover, there is a clear gradient in oppositional values by duration of the experience of poverty, displayed in figure four below.

Multi-nominal logistic regressions were used to further examine associations between latent-class group membership and our predictor variables. LCG three (or the 'healthy lifestyle' group) was used as the reference category, so that we could distinguish between the healthiest patterns of behaviours and all other groups. The results are weighted, and data is imputed. , and figure five, two shows the relative risk ratios (RRR) for our four

models: baseline model, social exclusion model, social resistance model and fully adjusted model (with interaction), all of which include our control variables. By adjusting for our control variables (gender, health status and personality), it was possible to account for the influence of our risk factors on cluster membership.

The results of model two show that reporting income poverty (two or three waves), compared to never reporting income poverty, significantly increases the likelihood of membership in the 'unhealthy lifestyle' (two waves: 71%, three waves: 100%) and 'unhealthy lifestyle and substance users' (two waves: 94%, three waves: 138%) LCGs, compared to the 'healthy lifestyle' (reference) LCG. These relationships retain their significance in the fully adjusted model (or model four), however, the magnitude of the effect of persistent poverty is reduced with the addition of the oppositional values variable for the 'healthy lifestyle' LCG (see figure two). In contrast, experiencing income poverty (over one, two or three waves), compared to never experiencing income poverty, significantly reduces the likelihood of being a member of the 'healthy lifestyle but substance users' compared to the 'healthy lifestyle' LCG. However this relationship is not significant. Overall, the results suggest that poverty is uniquely associated with patterns of HHBs defined by unhealthy lifestyle (exercise, diet, screentime) as opposed to HHBs related to substance abuse.

The results of model three show that an increase in oppositional values significantly increases the risk of being a member of the 'unhealthy lifestyle' (by 23%), 'healthy lifestyle but substance users' (by 65%) and 'unhealthy lifestyle and substance users' (by 190%), compared to 'healthy lifestyle' LCG. This suggests that high oppositional values increases the risk of membership of all latent class groups characterised by unhealthy behaviours, but

particularly those characterised by substance use. These associations retain their significance in the fully adjusted model, as displayed in table two and figure five. This suggests that substance users are particularly likely to be characterised as having oppositional values.

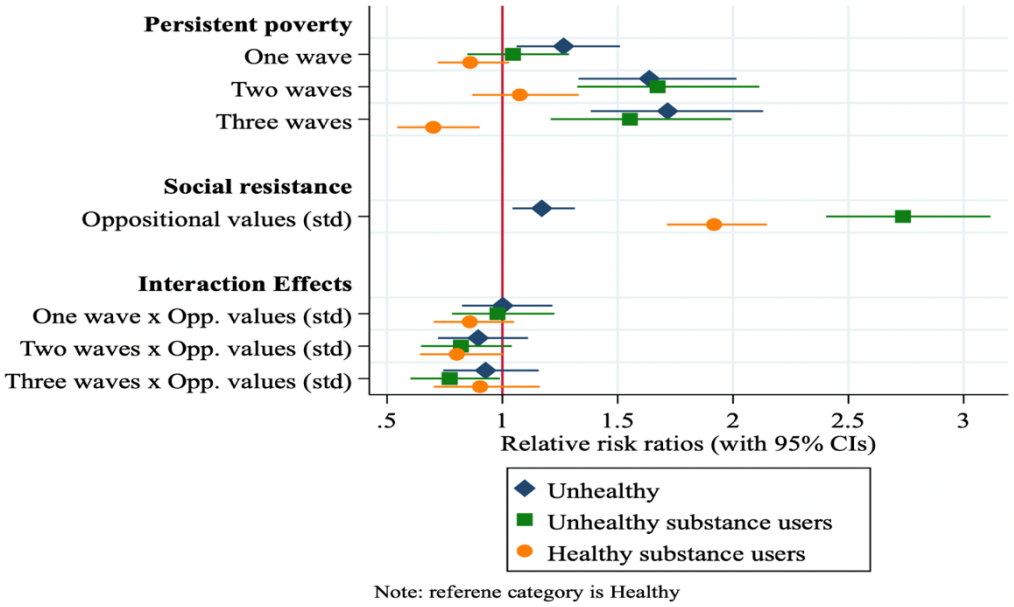
The interaction effect (persistent poverty and oppositional values) was not significant, with the exception of the comparison between the healthiest and unhealthiest LCGs. Experiencing persistent poverty (three waves) and higher oppositional values significantly decreased the risk (by 33%) of being in the 'unhealthy lifestyle and substance users,' compared to the 'healthy lifestyle' LCG.

Table 2. Results from multi-nominal logistic regression predicting latent class membership.

	Model 1[1]			Model 2[1]			Model 3[1]			Model 4[1]		
	Baseline			Social Exclusion			Social resistance			Interaction social exclusion and social resistance		
	RRR	*S.E*	*pvalue*	*RRR*	*S.E*	*pvalue*	*RRR*	*S.E*	*pvalue*	*RRR*	*S.E*	*pvalue*
Unhealthy												
Persistent poverty												
One wave (vs. no waves)				1.19	0.12	0.10				1.16	0.12	0.16
Two waves (vs. no waves)				1.71	0.17	**0.00**				1.63	0.17	**0.00**
Three waves (vs. no waves)				2.00	0.21	**0.00**				1.91	0.20	**0.00**
Oppositional values							1.23	0.05	**0.00**	1.18	0.09	**0.02**
Persistent poverty # oppositional values												
One wave (vs. no waves)										0.97	0.11	0.80
Two waves (vs. no waves)										1.08	0.12	0.48
Three waves (vs. no waves)										0.97	0.10	0.79
Constant	0.28	0.02	**0.00**	0.21	0.02	**0.00**	0.28	0.02	**0.00**	0.22	0.02	**0.00**
Healthy (reference category)												
Unhealthy drinkers and smokers												
Persistent poverty												
One wave (vs. no waves)				1.30	0.16	**0.03**				1.14	0.17	0.38
Two waves (vs. no waves)				1.94	0.23	**0.00**				1.49	0.23	**0.02**

Three waves (vs. no waves)				2.38	0.29	**0.00**				2.34	0.33	**0.00**
Oppositional values							2.90	0.14	**0.00**	3.17	0.27	**0.00**
Persistent poverty # oppositional values												
One wave (vs. no waves)										0.95	0.13	0.72
Two waves (vs. no waves)										1.02	0.14	0.91
Three waves (vs. no waves)										0.67	0.08	**0.00**
Constant	0.22	0.01	**0.00**	0.15	0.01	**0.00**	0.14	0.01	**0.00**	0.10	0.01	**0.00**
Healthy drinker (& smokers)												
Persistent poverty												
One wave (vs. no waves)				0.98	0.08	0.78				0.90	0.08	0.20
Two waves (vs. no waves)				0.93	0.08	0.44				0.86	0.08	0.09
Three waves (vs. no waves)				0.95	0.09	0.57				0.81	0.08	**0.03**
Oppositinal values							1.65	0.06	**0.00**	1.71	0.10	**0.00**
Persistent poverty # oppositional values												
One wave (vs. no waves)										1.02	0.09	0.82
Two waves (vs. no waves)										0.86	0.08	0.13
Three waves (vs. no waves)										0.99	0.09	0.88
Constant	0.68	0.03	**0.00**	0.70	0.04	**0.00**	0.63	0.03	**0.00**	0.69	0.04	**0.00**
N	6,091			6,091			6,091			6,091		

Figure 14. Coefplot displaying the results from a multi-nominal logistic regression for the fully-adjusted model with interaction (or 'model four').



Discussion

Social gradients in harmful health behaviours (HHBs) are a major contributor to health inequalities and adolescence is regarded as a 'critical period' for the emergence of these behaviours. We use a nationally representative longitudinal sample of young people aged 9-18yrs from Ireland to examine whether there are unobserved (or 'latent') groups of HHBs, and whether the members of these latent groups share common risk factors that predict group membership.

The results of our LCA support previous research that shows that HHBs cluster together. What we have termed 'unhealthy lifestyle' behaviours (poor diet, low physical activity and high screentime) tend not to occur individually, but instead occur together. However, these lifestyle behaviours appear to be relatively independent of tobacco smoking and underage drinking, or what we have termed 'substance use.' The four groups that emerged were labelled accordingly: 'healthy,' 'healthy lifestyle but substance users,' 'unhealthy,' and 'unhealthy lifestyle and substance users'. To our knowledge, this is the first paper to consider a combination of these behaviours including screentime, a new and emerging health-risk. Four distinct groups would suggest that there are also distinct underlying processes, which increase the risk of engagement in either behaviours associated with an unhealthy lifestyle or substance use, as opposed to independent risk factors for each behaviour individually.

Fundamental Cause Theory

Using multi-nominal logistic regression, we explored whether experiencing resource deprivation, through periodic or persistent poverty, predicted membership of these four latent class groups. Controlling for child's gender, health status and personality, we found

that experiencing persistent (income) poverty, over the ten-year period of data collection, was highly predictive of membership in the 'unhealthy lifestyle' and 'unhealthy substance user' LCGs.

Our findings support existing evidence that young people from disadvantaged social and economic backgrounds are more likely to have poor diets(Gebremariam et al., 2017; Layte et al., 2023), tend to be less physically active (Inchley et al., 2016; McEvoy et al., 2022) and have higher levels of screentime (Brannigan et al., 2022) compared to their more advantaged peers. Our results support the argument put forward by Fundamental Cause Theory, as outlined in the introduction of this paper: that a lack of resources (in this case money) shapes health inequalities by influencing individual health behaviours. We further hypothesized, using the social exclusion framework, that persistent poverty leads to a fall in the consumption of the goods and services necessary for individuals, families and groups to obtain a lifestyle similar to their fellow citizens. Although this mechanism is not tested directly, our results suggest support for this hypothesis.

The strong association between persistent poverty and belonging to the 'unhealthy lifestyle' LCG remained after adjustment for oppositional values in the fully adjusted model. The strength of the association between persistent poverty and belonging to the 'unhealthy but substance user' LCG weakened. This indicates that whilst experience of persistent poverty increases the probability of poor-quality diet, low levels of physical activity and high levels of screentime, it is the emergence of oppositional values which increases the probability of substance use.

Using the same methods, we examined the relationship between LCG membership and oppositional values. The results of our fully adjusted model revealed that reporting oppositional values significantly increases the risk of being a member of all latent class groups characterised by unhealthy behaviours, but particularly those characterised by substance use ('healthy lifestyle but substance users' and 'unhealthy lifestyle and substance users'). The relationship between oppositional values and substance use behaviours might be explained by the illegal nature of these activities, making them more alluring for young people wishing to demonstrate a 'resistance' behaviour. Here we demonstrates the motivations of 'specialists' (seek out specific health risk behaviours) as opposed to generalists (seeking out any harmful behaviour), which is a unique contribution to research on multiple health behaviours, as outlined in the introduction section of this paper.

Our findings provide support for our hypothesis that the development of oppositional values fuels engagement in HHBs, possibly via our proposed mechanisms: 'getting back at' and processes of group identity. However, it was not possible to test this here. Nonetheless, it seems that resistance processes are particularly helpful in understanding why young people engage in harmful behaviours, which have serious long-term repercussions for their health. The findings also support the theory of 'countervailing mechanisms' (Lutfey & Freese, 2005; Phelan et al., 2010), which argues that other, often socially-fuelled, desires can be more powerful motivators for behaviour than health. Whilst the adoption of harmful health behaviours by individuals from disadvantaged SEP has been defined as 'irrational' by some analysts(Johansson & Vinthagen, 2016), they should instead be seen as meaningful social action which expresses social identity.

We theorised, using Factor & colleagues social resistance framework(2011), that oppositional values were prompted by feelings of alienation and rejection that accompany being part of a marginalised (or excluded) group, within society. However, the results of our multivariate analysis showed that reporting oppositional values was predictive of substance use for members of the 'healthy lifestyle but substance users' LCG as well as the 'unhealthy lifestyle and substance users' LCG. The former consists of young people whose families are the least resource (income) deprived in our sample. This would suggest that oppositional values predict substance use behaviours independent of SEP. However, the risk of being an 'unhealthy substance user' is more than double the risk of being a 'healthy substance user,' for young people with oppositional values. This may be due to the fact that oppositional values increase incrementally as the number of years experiencing income poverty increase, making young people who experience persistent poverty more likely to develop oppositional values, and therefore more likely to engage in these substance use behaviours.

Limitations

Latent Class Analysis (LCA) is a data-driven approach, identifying the optimal subdivision using the data themselves rather than a pre-existing theory. The optimal number of latent classes reflects, in large part, an overall assessment of measures of fit including entropy values, results for our entropy values do not indicate a good separation of classes. As shown elsewhere, our identification of classes was then reliant on interpreting other fit statistics i.e. AIC, BIC and aBIC, as well as, model interpretability and utility (Nylund et al., 2007).

Another common approach to data classification, particularly, in deviance and substance use research is Guttman scaling (Andrich, 1985). This approach necessitates one-dimensionality and cumulativeness of selected variables. Our chosen health behaviours did not possess these Guttman scale properties in that they did not scale in terms of severity, rather lifestyle type behaviours appeared to be distinct to substance use in that being a substance user did not necessitate being physically inactive or having a poor diet or vice versa. Therefore, this approach was not used. As proposed in the introduction of this paper, adolescence is a time when harmful health behaviours tend to emerge and often cluster together. We set out to establish if this is due to a disinhibition or 'gateway' mechanism or because of the influence of common psychological or social risk factors. It seems that lifestyle type behaviours may have a gateway mechanism in that poor diet, physical inactivity and screentime seem to co-exist but out data suggests that this influence does not extend to substance use behaviours. It seems there are distinct social and psychological mechanisms prompting these different sets of behaviours respectively.

One of the important strengths of this study lies in the use of the GUI cohort data because of its sample size, representativeness, and longitudinal nature. Prospective collection of longitudinal data allows us to examine how the experience of persistent, as well as periodic income poverty influences health behaviours. It also enables us to consider underage drinking, as opposed to drinking behaviour at legal age (17/18yrs), as a harmful health behaviour. This is particularly useful for this paper as measuring the consumption of alcohol at age 17/18 would not necessarily be considered a harmful or norm-violating behaviour. However, the frequency in which alcohol is consumed is not measured by this variable. The age at which the young person 'had [their] first full drink of alcohol – more than a few sips,' does not indicate how much they drank, if they continued to drink and

how frequently, and in what environment their drinking took place. Our study results suggest that underage drinking appears to be the only harmful health behaviour that is not adversely patterned by family poverty. It might be that a more accurate measure of alcohol misuse would show a positive correlation with family poverty, especially considering the finding that “employed” and “more educated” parents of are more likely to permit early adolescent drinking(Maggs and Staff, 2018). A further strength of this data is the availability of both lifestyle and substance related health behaviours, whereas previous studies have had to consider these separately. However, it was not possible to give consideration to other relevant risky health behaviours such as risky sexual activity as these measures are only accessible through accessing the GUI restricted data files.

Latent Class Analysis (LCA) is a data-driven approach, identifying the optimal subdivision using the data themselves rather than a pre-existing theory. The optimal number of latent classes reflects, in large part, an overall assessment of measures of fit. Another common approach to data classification, particularly in deviance and substance use research, is Guttman scaling(Andrich, 1985). This approach necessitates one-dimensionality and cumulativeness of selected variables. Our chosen health behaviours did not possess these Guttman scale properties in that they did not scale in terms of severity, rather lifestyle type behaviours appeared to be distinct to substance use in that being a substance user did not necessitate being physically inactive or having a poor diet or vice versa. Therefore, this approach was not used. As proposed in the introduction of this paper, adolescence is a time when harmful health behaviours tend to emerge and often cluster together. We set out to establish if this is due to a disinhibition or ‘gateway’ mechanism or because of the influence of common psychological or social risk factors. It seems that lifestyle type behaviours may have a gateway mechanism in that poor diet,

physical inactivity and screentime seem to co-exist but our data suggests that path from this gateway does not extend to substance use behaviours. Rather, it seems there are distinct social and psychological mechanisms prompting these different sets of behaviours respectively.

Policy Intervention

Knowing the patterning of health behaviours can contribute to the development of public health policy and interventions that take a multi-faceted approach. There is already a recognition that unhealthy behaviours often co-occur and are influenced by the same factors and our results confirm this. We suggest interventions that will target multiple behaviours, as opposed to individual health behaviours, and reach those who socio-economically disadvantaged.

There is a long-standing literature that argues that health behaviours must be understood in their social and economic context, and viewed as a response to conditions imposed by social structures. This paper further emphasises the need to address the 'fundamental causes' of health problems if we are to address inequalities related to health. Specifically, we show the impact of persistent, compared to periodic, poverty on the engagement with a host of HHBs. Reducing income inequality should help to re-shape the distribution of HHBs by SEP(Link & Phelan, 2002; McCartney et al., 2021). This first recommendation falls outside the explicit domain of public health policy, but according to FCT is closely tied to it. Policy intervention that focuses on the more proximate causes of disease can have an important impact on health outcomes, but has been shown to have a transitory impact on health inequalities and may increase them (as advantaged groups may ultimately benefit more). In the process of elucidating the mechanisms connecting social conditions to health, it is

important not to lose sight the importance of the social condition whose effect on health we originally sought to explain.

Our conclusion that marginalised groups are resistant to behaviours that are in line with public health guidelines (and that of the dominant majority group) informs our second recommendation. It is important to acknowledge that HHBs, particularly those related to substance use, may be fuelled by oppositional values, especially among those experiencing poverty and social exclusion. Recent research, on unsuccessful school tobacco policies, proposes that increased restrictions on smoking and reduced adult consumption of tobacco can actually increase the incentive to smoke among adolescents as it enhances the contrast with adult behaviour and demonstrates personal autonomy(Schreuders et al., 2017). We suggest that future research consider the effectiveness of peer-led interventions, which have shown to be effective in addressing socio-emotional outcomes elsewhere(O'Keeffe et al., 2021).

5 Does Family Economic Strain increase the likelihood of Child Smoking? An extension of the Family Stress Model using longitudinal population-based data and the Great Recession in Ireland.

Introduction

Non-communicable diseases (NCDs) such as heart and lung disease, cancer and diabetes represent the leading cause of death globally (WHO, 2022). Estimates suggest that, in high-income countries, approximately one third of NCDs can be attributed to four health behaviours: smoking, excessive consumption of alcohol, poor diet and low levels of physical activity (Pampel et al., 2010; Petrovic et al., 2018). Of these behaviours, smoking is the leading cause of preventable death globally, and is the single largest avoidable health risk (WHO, 2022). The burden of NCDs disproportionately impacts on socio-economically disadvantaged groups resulting in significant health inequalities (Krieger, 2011; Marmot et al., 1978; Stringhini et al., 2017; Tanaka et al., 2019). Differentials in health behaviours, smoking in particular, by social and economic position (henceforth SEP) clearly plays a significant role in the formation of these health inequalities. This begs the question as to why smoking varies by SEP? Marmot (2018) has characterised this as the question of the 'causes of the causes' of health inequalities.

Tobacco smoking presents a particularly difficult challenge to researchers trying to understand the social and economic structuring of health behaviours. In the first instance, public health messaging, internationally, means that few are unaware of the negative health consequences associated, and so differentials in knowledge and information by SEP are likely to play a small role in explaining inequalities (Layte & Whelan, 2009; Mazanov & Byrne, 2007). Secondly, tobacco smoking is likely to be correlated, at the individual level, with a number of unobserved characteristics which vary by SEP, which

presents the additional challenge of how to separate correlation from causation in analyses of disadvantaged subpopulations. Lastly, evidence suggests that when smokers begin smoking, in adolescence, the social gradient is already present cross nationally, and this is coupled with lower rates of cessation among those from a disadvantaged SEP as they transition into adulthood (Ahmad et al., 2023; de Looze et al., 2015; Viner et al., 2015).

In response to these three challenges, in this paper, we study the social determinants of smoking initiation in adolescence between the ages of 9 and 17/8 using data from a nationally representative longitudinal cohort study from Ireland. Our data were collected before, during and in the aftermath of the 'great recession' in Ireland (2009 to 2014) and this allows us to test a specific theory about the effects of SEP on smoking initiation in adolescence using quasi-experimental methods.

Background

Social Determinants of Smoking

Since the publication of the landmark Black Report (1980), theories as to why health behaviours vary across SEP have tended to be grouped into three main types: materialist, behavioural and cultural and psycho-social (Bartley, 2016). Materialist theories, in their simplest form, focus on the role of variation in resource availability, which then structures the incentives and opportunities available to individuals and groups. 'Direct' behavioural theories often invoke psychological characteristics as the proximate cause of behaviours. For example, low or decreasing adolescent self-efficacy has been shown to predict smoking initiation (Lawrance & Rubinson, 1986; Van De Ven et al., 2007) and there is a well-established literature on the role of low self-esteem in adolescent smoking initiation (Khosravi et al., 2016). There is rarely consideration as to why there may be variations in

these psychological traits by SEP instead the prevailing assumption is that these individual differences are rooted in biology, which selects individuals into smoking. Cultural theories often draw upon the work of Bourdieu (1984). Cockerham (2005, 2013) and Bartley (2016) employ the work of Bourdieu (1989) to unpack the symbolic dimensions of risky health behaviours, especially smoking. They argue that smoking is a strategy adopted by individuals to display group membership, and distance themselves from other non-smoking groups. In this way, smoking acts as a consumption practice for identity and status.

Lastly, psycho-social explanations are concerned with the way social inequality can make people feel, and how these feelings may alter body chemistry (e.g. stress). Early theories drawing on psychosocial explanations implicated the hypothalamic-pituitary-adrenocortical (HPA) system in response to the experience of inequalities in social status as a contributor to inequalities in disease and mortality (Brunner, 1997; Dickerson & Kemeny, 2004). This mechanism has also been applied to health behaviour research. There is a well-established relationship between the experience of poverty and smoking risk (Ciapponi et al., 2014) and this relationship has been explained through the use of tobacco as a way of regulating mood, managing stress and coping with the mental strain of living with deprivation (Graham, 1987). However, self-medication hypotheses are inherently contradictive considering that that nicotine is a stimulant, not a sedative or anxiolytic and simply does not supply the calming effect claimed for it, unless the person is already experiencing withdrawal (Bobak et al., 2000). Nonetheless, even without the pharmacological effects claimed for it, smoking may still provide a coping mechanism for deprived individuals.

Family Stress Model and Smoking

One process through which the impact of 'materialist' and 'psychosocial' circumstances may impact on smoking behaviours, in adolescence at least, is offered by what has come to be known as the Family Stress Model (FSM). The FSM postulates that economic hardship and deprivation increase parent psychological distress with consequences for their risk of anxiety and depression. The experience of psychological distress and poor mental health also increase the risk of marital conflict which together may lead to diminished parenting quality, which in-turn increase the risk of internalizing and externalizing symptoms in children with whom they live (Conger et al., 2000). Social and emotional problems in young people have consistently been related to a range of poor health behaviours, including an increased risk of smoking (Kekäläinen et al., 2023; Papachristou & Flouri, 2020; Šablatúrová et al., 2021; Thomas et al., 2020; Voisin et al., 2016).

Putting these two literatures together, it may be that smoking, and other harmful health behaviours, are adopted by adolescents as coping mechanism, to help mitigate the stress that is generated via the worsening of their parental relationship as a function of parent experience of economic strain and deprivation (Boardman & Alexander, 2011; Umberson et al., 2008). We argue that SEP differentials in the initiation of smoking may emerge because of differential exposure to economic strain and thus inequalities in the emergence of smoking as a coping mechanism related to child and adolescent socio-emotional adjustment. Our hypothesis is based on previous research, which has shown that low levels of adolescent 'mastery,' referring to the young person's ability to effectively navigate and manage social and emotional challenges, results in the adoption of harmful

health behaviours as a coping mechanism for family stress (Kwon & Wickrama, 2014; McCubbin et al., 1985; Pearlin & Radabaugh, 1976; Ryan et al., 2010; Wen & Hui, 2012). The FSM has garnered support from numerous studies, predominantly form the US (Masarik & Conger, 2017). Existing evaluations are limited in that they are cross-sectional in design and reliant on non-population based samples, with few exceptions (Chen et al., 2023; Gard et al., 2020; Layte, 2022; Simons et al., 2016; Solantaus et al., 2004), making it challenging to disentangle the causal effect of economic pressure, on socio-emotional difficulties, from pre-existing risk factors. Identifying any causal relationship between childhood economic disadvantage and smoking status is challenging, as family hardship is likely to be correlated with other characteristics, such as parental education and occupational status, which also influence smoking behaviour independently of the experience of economic strain and psychological distress. In response to this, researchers utilise economic crises, in Korea (Kwon et al., 2003), Turkey (Aytaç & Rankin, 2009), the US (Solantaus et al., 2004) and Ireland (Layte, 2022), to identify the impact of financial strain on parenting and child socio-emotional problems. Using exposure to recession as an exogenous effect to measure the effect of family economic strain on smoking risk, net of other factors.

The ‘Great Recession’ in Ireland is usually defined as starting with the collapse of Lehman Brothers’ Bank on the 15 September 2008 and the Irish Government’s subsequent blanket guarantee of all deposits, debt, and securities in the Irish pillar banks during the night of the 29th and 30th September. Over the following four years, there were deep cuts in public services, social protection, and salaries as the government struggled to balance falling tax revenue with increasing demand for social protection. During the recession, average household income fell by over 17.0 per cent and

unemployment tripled from 6.0 per cent to 18.0 per cent, leading to the proportion of households experiencing the Government's official definition of lifestyle deprivation to increase from 11.8 per cent in 2007/2008 to over 30.0 per cent by 2013 (CSO, 2018). Most families, regardless of prior socio-economic standing, experienced financial strain and uncertainty during this period. In this way, the Great Recession presents a unique opportunity to assess the causal relationship between parental economic strain and child and adolescent smoking.

Previous research on the effect of the recession on health behaviours has shown that economic strain can evoke a marked increase in health-risk behaviours, across all strata in society; population prevalence of smoking, drinking and overweight increased across geographic regions in Italy (Castellari et al., 2021; Mattei et al., 2017) in Germany (Kaiser et al., 2018), and in Spain (Bassols & Castelló, 2016) as a result of the economic downturn. However, in Iceland, the reverse was true, national prevalence of smoking declined following the economic crisis (McClure et al., 2012).

Although the onset of the Great Recession is said to be an exogenous 'shock' to all families, and so independent of family characteristics, in reality, some families are more vulnerable to the effects of recession (e.g. job loss, behind on bills) than others. In order to isolate the effect of the recession on smoking, it is important to adjust for this non-random probability of experiencing the effects of recession. Using a nationally representative longitudinal sample of Irish families, we identify families with a similar propensity to experience recession events, based on their socio-demographic characteristics prior to recession in 2009. By then matching families who had a similar propensity to experience recession, but who varied in terms of their actual experience of

recession, we can test whether the economic strain as a result of the recession increased the probability of smoking initiation, and whether the processes associated with the FSM mediated this relationship.

Methods

Data

(see section on Data, introduction)

Although the study was not planned as such, baseline data collection occurred as the economic boom of the previous 12 years was coming to an end and just prior to the onset of the Great Recession in the first quarter of 2009. The second wave of data collection occurred at the trough of the same recession and wave three occurred as the economy was recovering strongly. In this paper, we exploit the fortuitous timing of data collection to carry out a quasi-experiment.

Treatment of Missing values

Levels of missing data across variables were low, on average, but reached 6.8 per cent on one variable (see table 8, appendix C). Non-response is a feature of all surveys, and by using tracing procedures it is possible to establish if inter-wave attrition is systematically associated with family or other characteristics measured in our survey (e.g. household social class), as is the case in GUI (Thornton et al., 2010). This missing data has the potential to bias analysis, and for this reason, missing values on the control and predictor variables are imputed for analysis using multiple imputation by chained equations (MICE) to produce ten imputed datasets, using the Rubin method (Rubin, 1988) as implemented in STATA 16. The data are also reweighted for analyses to take account of the original sample error and subsequent attrition using a range of factors and a minimum distance algorithm. The final sample is representative of young people who were residing in Ireland at nine years of age in 2006 and who continued to live in Ireland at 20 years of age in 2021 (Thornton et al., 2010).

Measures

Dependent Variable

Smoking status was self-reported by the young person at T3 (age 17). Participants were asked: 'have you ever smoked a cigarette?' (yes/no) and subsequently 'which of the following best describes you?' (only ever tried smoking once or twice/used to smoke but not now/smoke occasionally/smoke daily/do not smoke). The answer categories were recoded into 'smokers' and 'non-smokers,' with respondents who answered 'no' to the first question and 'only ever tried smoking once or twice,' 'used to smoke but not now' and 'do not smoke' being classified as non-smokers. Due to the sensitive nature of the questions (parents may not be aware of the participant's smoking) the questions were administered on a supplemental self-complete questionnaire, and the young person was assured of confidentiality. Information on vaping was not available until T4 (age 20), where there was more survey attrition, and so was not included in this analysis.

Exposure Variable

At T2 (age 13; 2011/2012), the PCG was asked whether the recession had affected their family since the first interview (age 9; 2007/2008) to gauge the impact of the recession on households. This was a routed question with four response categories ranging from a 'very significant effect' to 'no effect at all' on your family. Those who indicated that the recession was having an impact on their family were routed to a 9-item multi-response question to ascertain the number of ways the recession had affected the family:

1. You were made redundant/lost your job.
2. Your spouse/partner was made redundant/lost their job.
3. Your or your spouse/partner's working hours were reduced.
4. Your or your spouse/partner's wages were reduced.

5. Your or your spouse/partner's social welfare benefits were reduced.
6. Your family cannot afford luxuries (holidays, meals out, etc.).
7. Your family cannot afford/had to cut back on basics (food, clothes).
8. You are behind with rent/mortgage payments.
9. You are behind with utility bills (e.g. electricity, gas bills, etc.).

The first two response categories (redundancy relating to both parents) were combined. The categories were then renamed: redundancy, hours, wages, social welfare, luxuries, basics, bills, mortgage. Our hypothesis is that exposure to economic strain via one or more of these recession events triggers our primary mediating process (psychological distress) which then leads to smoking initiation.

Mediating Variables

Parental Depression

At T1 and T2, the primary caregiver (PCG) completed the Centre for Epidemiological Studies Depression Scale (8-item) (CESD-8) is a short self-report screening instrument for depression for general populations. Responses are indicated on a four-point rating scale: 'rarely or none of the time (less than 1 day)', 'some or a little of the time (1–2 days)', 'occasionally or a moderate amount of the time (3–4days)', and 'most or all of the time (5–7 days)' with a reference period of the previous 7 days. A composite score is calculated by summing responses across items (range: 0–24). CESD-8 has good internal consistency, reliability (α=0.86) and correlates highly (0.93) with the longer 20-item version of the CESD scale. The concurrent validity of the scale has been established through its association with other measures of depression such as the Beck Depression Inventory (Melchior et al., 1993).

Child Social-Emotional Difficulties

Child's social-emotional difficulties are measured using the Strengths and Difficulties Questionnaire (SDQ), a 25-item screening questionnaire designed to assess emotional health and problem behaviours (Goodman, 1997). The instrument produces scores for each of the five sub-scales: emotional symptoms, conduct problems, hyperactivity/inattention, peer problems, and prosocial behaviour. A total difficulties score is obtained by summing scores across the four deficit-focused scales (i.e. all except the prosocial behaviour scale). Sub-scale scores range from 0-10 and the total difficulties score ranges from 0-40. Higher scores are indicative of more problems. SDQ has been shown to correlate highly with longer-deficit focused instruments such as the Rutter scales and the Child Behaviour Checklist; to differentiate well between clinical- and community-based samples(Goodman & Scott, 1999); and to be sensitive to changes in behaviour following intervention(Mathai et al., 2003).

Control Variables

Gender (male/female) included as a control variable in this analysis. Baseline (or T1) levels of the mediating variables (i.e. parental depression and child socio-emotional difficulties) were also included as control variables. This was to ensure we examine the influence of family processes that are a result of the increased economic strain that accompanied the Great Recession by holding constant any pre-existing family conditions. Following the same logic, child smoking status pre-recession was also included as a control variable. Measures of self-efficacy (Sherer et al., 1982) and self-esteem (Rosenberg, 1965) were included to control for the individual determinants of smoking behaviour.

Analysis strategy

The objectives of this paper are two-fold: our first objective is to assess whether exposure to economic recession influences the probability of the young person smoking. Our second objective is to identify whether any association that does exist, is mediated by the mechanisms put forward by the FSM.

Table 9 displays the distribution of our variables of interest in our sample. To establish whether our hypothesised associations exist, we first descriptively examine the distribution of smokers vs. non-smokers (our dependent variable) and variation in parental depression and child social emotional adjustment (mediating variables) according to the exposure (number of recession events). This is to demonstrate the adverse relationship between experiencing recession and likelihood of becoming a smoker, increased reporting of poor parental mental health and child social-emotional problems.

Although the onset of recession could be said to be an exogenous 'shock' to all families, and so independent of the observed and unobserved characteristics of each family, in reality some families are more vulnerable to the effects of recession than others. We identified baseline characteristics that may influence the likelihood of experiencing more recession events, drawing on existing theory and empirical research. Table 7 demonstrates the patterning of recession events according to these baseline characteristics.

Ideally, we would evaluate whether a family's experience of recession had an effect on the young person smoking by closely approximating the conditions for valid causal (or counterfactual) inference, i.e. a randomised control trial (RCT). Here families would be randomly allocated to be exposed or not to 'recession' (the treatment in this experiment) and then followed over time to measure the subsequent impact on smoking. For practical

and ethical reasons, this is clearly not possible. Observational research differs from this ideally designed experiment in that we observe average effects when the cause is present and average effects when it is not. Therefore, we cannot infer causality as the groups being compared are not 'essentially' similar at the beginning of the experiment, a problem called sample selectivity or selection bias (Cochran, 1957; Imbens & Rubin, 2010). We use propensity score matching (PSM) techniques to reduce such selection bias into the treatment group (group that experience recession). We assign "propensity scores" to all families, which represent the conditional probability that a person will be in a treatment group based on their specific characteristics pre-treatment. $P(D = 1 \mid X) = P(X)$, i.e. the probability for an individual to participate in a treatment given his observed covariates X. Propensity scores control for much of the selection effect by matching families who have a similar propensity to experience recession, but who vary in terms of their actual experience of recession. Approximating an RCT in this way, as opposed to traditional covariate adjustment, produces a more precise and reliable estimate (Luellen et al., 2005).

A poisson regression model is used to compute the probability that a respondent will experience recession events (i.e. a propensity score), as the outcome is a count variable and therefore demonstrates a poisson distribution, as opposed to a normal distribution. Meaning, it possesses a low mean, a positive skew and the possibility of zero as an outcome, all of which can be addressed by adopting the poisson estimator. One alternative practice is to dichotomize data of this nature into an occurred or did not occur event. This approach does not use all the available data and results in loss of information, reduced statistical power, and misleading analytic results and inferences (Caliendo & Kopeinig, 2008; Hayat & Higgins, 2014; Owen & Froman, 2005).

Finally, observations are matched based on similar propensity scores, by stratifying the continuous propensity scores into 20 strata. Using these strata, the effects of the recession can be compared using a fixed- effects or 'within' estimator. Using this method, we control for all differences between strata and confine analysis to the impact of recession events on our desired outcome within each strata. To determine the extent of reduction in selection bias, we first tested the balancing property of propensity score strata using each of the pre-treatment baseline characteristic variables (as in table 7) separately to predict experience of recession events, comparing the results for a poisson regression with the results for fixed-effects approach, the latter confining analysis within our propensity score subgroups. The results are displayed in table 8.

Using the matched groups, we were able to compare families with a similar likelihood of being exposed to the effects of the recession, but who nonetheless differ in their actual experience of recession. We used three fixed-effects, or 'within-estimator,' logistic regression models and the 'difference method' (VanderWeele, 2016), to measure the mediating effect of variables associated with the FSM in the association between recession and smoking risk (Baron & Kenny, 1986). Model one acts as the baseline model where the sole predictor is the number of recession events experienced between baseline and follow-up, adjusting for the non-random probability of selection into experiencing recession. In model two, we control for characteristics of the young person at baseline that may influence the decision to smoke and baseline measures of variables derived from the FSM theory. Model three adjusts for the measures derived from the FSM theory at follow-up to assess the extent to which these mediate the association between number of recession events experienced and adolescent smoking. If this is the case, we expect that our measures of parental mental health and the socio-emotional behaviours of the young

person will account for the association between recession and smoking initiation (Baron & Kenny, 1986).

Results

Table seven provides the proportion of recession events experienced by selected background characteristics that are theorised to increase a family's vulnerability to recession. Recession events were non-randomly distributed across selected background characteristics. On average, disadvantaged households were more likely to be exposed to the recession events: manual-class households, single-parent households, younger parents, local authority homeowners and parents with less education were all more likely to experience reduced working hours, reduced social-welfare payments, cutting back on basics and luxuries, and being behind on mortgage and utility payments, compared to their more advantaged counterparts. However, contrary to our expectations, experiencing a wage or salary reduction was more common among households with baseline characteristics that indicate socio-economic advantage (non-manual and professional class households, parents with a third-level degree, older parents, household owners and non-migrant parents). Only a small proportion of the overall sample experienced job loss, and it was most common among non-manual and professional classes, those with a leaving certificate but no third-level education, private renters and single-parent and younger parent household.

Table 7. Proportion experiencing specific recession events between 2007/2008 and 2012 by household, parent, and child characteristics.

		Experienced specific recession event							
		Wages (n=4,272) %	SW (n=2,827) %	Luxuries (n=2,808) %	Basics (n=1,517) %	Hours (n=1,342) %	Redundancy (n=456) %	Mortgage (n=440) %	Bills (n=517) %
Household Class	Professionals	73.0	40.3	38.8	20.2	16.5	7.5	5.4	3.4
	Managerial & Technical	72.3	43.4	42.9	22.3	19.1	8.4	6.1	6.2
	Non-manual	67.6	48.9	51.3	24.9	26.2	10.2	7.0	9.6
	Skilled-manual	61.8	55.8	52.3	33.5	34.6	4.9	11.7	12.3
	Semi & Unskilled	40.3	68.3	52.9	37.7	18.5	7.5	13.7	18.4
Maternal Education	Third level	71.8	42.2	40.9	19.2	15.0	7.3	4.7	5.2
	Post-secondary	70.5	49.0	47.0	23.1	24.6	9.2	8.8	10.2
	Leaving certificate	64.8	50.8	47.6	27.6	23.8	23.8	7.9	9.5
	Junior certificate	50.4	60.0	52.9	36.0	24.9	6.2	12.4	14.4
PCG age	45+	61.1	47.3	43.9	23.0	20.2	7.3	5.8	7.1
	40-44	69.5	49.0	44.5	24.6	20.8	6.1	6.4	6.9
	35-39	64.7	53.7	49.3	29.5	24.7	8.4	10.4	11.6
	<35	49.2	57.6	55.7	35.9	25.5	10.7	13.6	17.6
Migrant status	Not migrant	63.4	51.3	47.3	27.4	22.6	7.5	8.8	9.5

	Migrant	59.4	53.8	51.3	30.3	23.6	10.1	9.0	14.7
Household type	Couple 1-2 children	67.9	46.3	43.7	24.4	26.4	8.9	7.8	7.2
	Couple 3+ children	67.9	50.1	49.5	27.1	23.3	5.9	8.2	9.4
	Single 1-2 children	45.1	59.4	48.8	34.7	14.4	13.2	9.1	16.6
	Single 3+ children	28.0	77.8	56.3	39.8	13.1	7.9	18.0	22.3
Housing tenure	Owner	68.4	47.2	45.7	24.6	23.1	7.4	7.6	7.7
	Private Renter	48.6	60.5	54.4	32.2	26.2	12.0	9.5	18.2
	Local authority	36.3	73.7	73.7	73.7	18.5	8.2	15.5	21.7

Table eight illustrates the balancing property of the propensity score strata. Here, number of recession events is regressed on variables theorised to be predictive of recession. The unadjusted results are derived from a poisson regression without stratification, which are compared to the results derived from a fixed-effects approach using an estimator based on the effects within propensity score strata. The balancing property of the propensity score is largely satisfied as the majority of the effects of pre-treatment variables on recession effects is non-significant within strata. Only the coefficient for the local authority tenure (vs. homeowner) and parental achieving post-secondary education (vs. third-level) at baseline remains significant, although the coefficients are reduced.

Table 9. Regression coefficients and t-statistics for predictors of treatment with and without matching by propensity score strata.

Domain	Variable	Unadjusted		Within PSM strata	
		Coef.	t-stat	Coef.	t-stat
Household class	Professionals (ref.)				
	Managerial & Technical	0.18	**0.08***	0.08	0.07
	Non-manual	0.40	**0.09*****	0.10	0.08
	Skilled-manual	0.64	**0.10*****	0.19	**0.10***
	Semi & Unskilled	0.52	**0.11*****	0.03	0.10
Maternal Education	Junior certificate	0.53	**0.08*****	0.01	0.08
	Leaving certificate	0.36	**0.07*****	0.01	0.06
	Post-secondary	0.39	**0.08*****	0.12	**0.06***
	Third level (ref.)				
PCG age	<35	0.41	**0.10*****	-0.05	0.08
	35-39	0.30	**0.08*****	-0.10	0.07
	40-44	0.07	0.08	-0.08	0.06
	45+ (ref.)				
Migrant status	Migrant (ref.)				
	Not migrant	0.13	0.09	0.05	0.06
Household type	Single 1-2 children	0.07	0.11	0.02	0.08
	Single 3+ children	0.31	0.16	0.16	0.12
	Couple 1-2 children (ref.)				
	Couple 3+ children	0.08	0.06	0.03	0.04
Housing tenure	Owner (ref.)				
	Local Authority	0.45	**0.11*****	0.24	**0.08****
	Private renter	0.23	**0.13***	0.03	0.09

*p<.05, *p<.01. p<.001

Descriptive Analyses

Table ten shows that there are more non-smokers (80.0 per cent) than smokers (20.0 per cent) at T3, slightly more males (51.0 per cent) than females (49.0 per cent) in the sample, and only a small proportion of children smoking pre-recession (4.0 per cent). On average,

families have experienced 2.46 of the nine recession events. We note that the child social-emotional difficulty scores at T1 (7.90) is higher than at T2 (6.98), indicating an overall improvement in social-emotional behaviours as participants grew older. Parental depression scores increase between T1 (2.19) and T2 (2.56) indicating higher parental symptoms of depression, on average, at T2 compared to T1. The average self-esteem score is 11.97 and the average self-efficacy score is 18.35 in our sample, with scale ranges displayed in table 10.

Table 10. Descriptive statistics for our variables of interest.

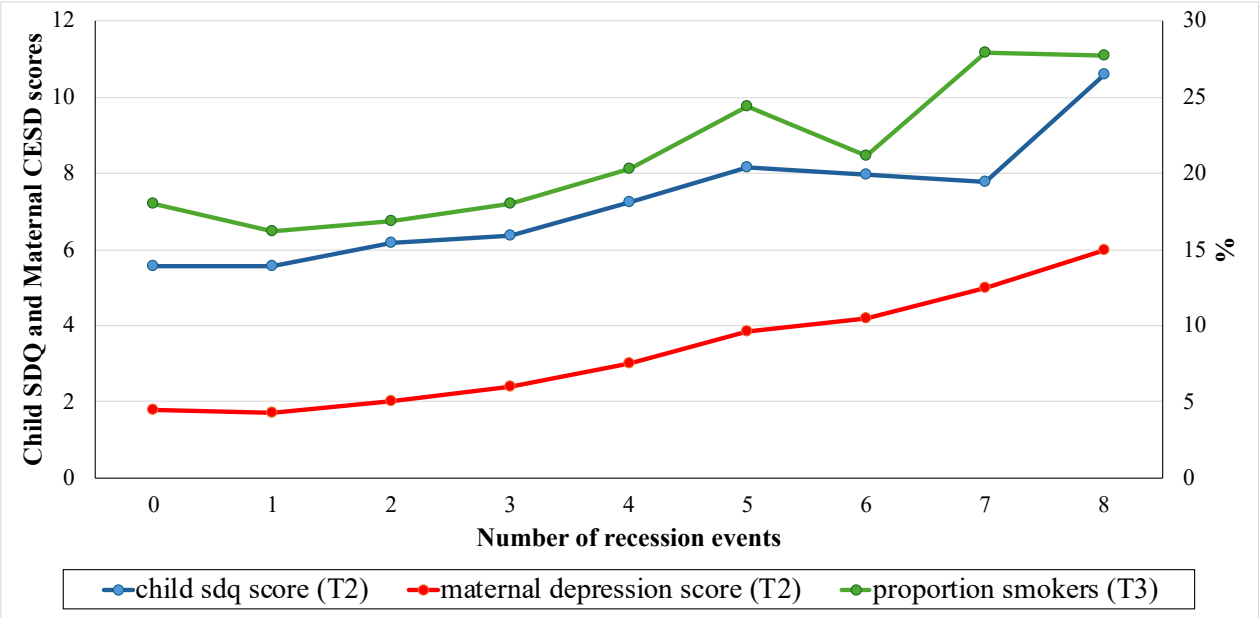
	N	Mean	SD	Minimum	Maximum
Outcome					
Child Smoking Status (T3)	6,089	0.20	0.40	0	1
Exposure					
Family's Number of Recession Events (T2)	6,089	2.46	1.62	0	9
Mediating Variables					
Child Social-Emotional Difficulties (T2)	6,089	6.98	5.23	0	35
Parental Depression (T2)	6,089	2.56	3.59	0	24
Control Variables					
Child Sex: Male	6,089	0.51	0.50	0	1
Child pre-recession smoking: Yes (T1)	6,089	0.04	0.20	0	1
Child Self-efficacy (T2)	6,063	18.35	2.79	7	24
Child Self-esteem (T2)	6,056	11.97	3.55	0	18
Child Social-Emotional Difficulties (T1)	6,089	7.90	5.27	0	33
Parental Depression (T1)	6,089	2.19	3.47	0	24

On average, young people who were smokers' post-recession had experienced marginally more recession events (smokers: 2.51, non-smoker: 2.31) compared to those who were non-smokers post-recession. Reported parental depression scores are higher for parents of smokers compared to non-smokers at T1 (smokers: 2.36, non-smoker: 1.91) and T2 (smokers: 2.96, non-smoker: 2.21). Reported child social emotional adjustment scores are

also higher for smokers compared to non-smokers at T1 (smokers: 7.94, non-smoker: 7.06) and T2 (smokers: 7.45, non-smoker: 6.12).

Figure 15 illustrates the relationship between recession events and smoking and the FSM mediating variables. The horizontal axis represents the number of recession events the family experienced, and the vertical axis (left) indicates mean score on the CES-D (representing parental depression) and child SDQ (representing child social-emotional difficulties) scales. Higher scores indicate worse outcomes on both variables. The vertical axis (right) indicates proportion of smokers measured as a percentage. FSM theory argues that increasing economic strain, in this case represented by number of recession events, will be accompanied by increased symptoms of parental depression and child socio-emotional difficulties. Figure 15 shows that the relationship is positive in our data. The figure also suggests that as the number of recession events increases, so too does the likelihood of the young person becoming a future smoker.

Figure 15. Child socio-emotional difficulty score, parental depression score and proportion smoking at age 17 (T3) by number of recession events family experienced.



Multivariate Analysis

Table 11 provides the expected probability, t-statistics, and levels of significance for the probability of smoking at age 17/18 (or T3) within the twenty strata of the propensity scores. Three models are estimated with groups of variables entered in stepwise fashion.

Table 11. Fixed effect estimates of probability of smoking at T3.

		Model 1		Model 2		Model 3	
		Coef.	t-stat	Coef.	t-stat	Coef.	t-stat
Recession	Number of recession events	0.07	**3.41****	0.06	**2.75***	0.05	**2.05***
Child characteristics	Female			-0.10	-1.45	-0.09	-1.29
	Male (ref.)						
	Pre-recession smoking						
	Yes (vs. No)			2.26	**14.00*****	2.21	**13.67*****
	Self-efficacy			-0.01	-0.84	-0.01	-0.67
	Self-esteem			-0.09	**-7.35*****	-0.09	**-7.10*****
FSM T1	Parental Depression			0.02	**2.00***	0.01	0.72
	Child SDQ			0.01	**1.68***	-0.01	-0.85
FSM T2	Parental Depression					0.03	**2.71*****
	Child SDQ					0.03	**3.25*****
	Observations overall	6,082		6,082		6,082	
	Minimum per group	144		144		144	
	Maximum per group	663		663		663	

*p<.05, *p<.01. p<.001

Model one provides an estimate of the effect of recession on the young person's smoking status. There is statistically significant positive association at p<.001 between experiencing a facet of the recession in 2011/2012 (T2) and becoming a smoker in 2018/2019 (T3). Model two adjusts for our selected control variables, which include variables representing the individual determinants of smoking literature (i.e. self-efficacy

and self-esteem) at T2 (2011/2012) and variables representing FSM theory (i.e. parental depression and child social emotional difficulties) measured at pre-recession baseline (T1;2007/2008). In this model, the association between experiencing a recession event and smoking remains positive and statistically significant at p<.05. The estimate is reduced by 15.9% compared to model one (see table 12, below). The results suggest that self-esteem mediates the relationship between experience of recession and smoking. Decreasing self-esteem positively predicts later smoking at age 17/18.

Model three provides an opportunity to assess the explanatory value of the FSM theory in mediating the effect of recession events on smoking risk. With the addition of variables implicated in the FSM theory at T2 (2011/2012), the estimate is reduced by 36.5% from model one (see table 12) but remains significant at p<.05. There is a significant positive association (p<.001) between the variables representing the FSM as measured during the recession (T2, 2011/2012) and smoking at age 17/18 or post-recession. The relationship between smoking and the baseline measures of the same mediators are rendered insignificant with the addition of these variables at T2 (parental CES-D score at T1, p=0.366; child SDQ at T1, p=0.759). Our results suggest that poor parental mental health and poor child social-emotional difficulties mediate, in part, the relationship between the experience of recession in childhood and smoking at age 17/18, as proposed by the FSM.

Table 12. Average proportionate reduction in the "recession events" estimate for probability of smoking at age 17 with the addition of our control variables (model 2) and our mediating variables (model 3).

	Model 1 Unadjusted	Model 2 Controls	Model 3 FSM at follow-up
Number of recession events	0.0%	-15.9%	-36.5%

Discussion

This aim of this paper was to examine the relationship between family economic circumstances and subsequent smoking in a nationally representative cohort study of young people. Smoking is the leading cause of preventable death globally making it the single largest avoidable health risk (WHO, 2022). This relationship has been the subject of sociological, public health and multi-disciplinary research (Casetta et al., 2017) for some time, with a common goal to address inequalities in health. Ethical and practical difficulties prevent the application of experimental methodology in this research area, which presents a key challenge to the achievement of this goal, as it is not possible to disentangle the influence of economic strain on the young person's smoking behaviour from other common features of poverty such as parental education and occupational status, that also influence smoking behaviour. The Great Recession in Ireland, and its sprawling repercussions for families, presents an opportunity to adopt a quasi-experimental methodology to the examination of family economic circumstances and smoking.

After adjustment for relevant control variables and using PSM techniques to reduce selection bias, our results show that increased economic strain, as a result of the family experiencing one or more recession events such as redundancy, job loss, falling into arrears etc., results in a significantly increased likelihood of child being a smoker post-recession. The use of this quasi-experimental approach enables us to contribute to the growing body of literature debating social vs. individual determinants of health (Marmot & Wilkinson, 2005) by showing how the 'force of circumstance' (Bartley, 2016) in the form of the exogenous shock of an economic downturn, fuels engagement in smoking.

Our results suggest that there is a relationship between economic strain and smoking independent of other correlates of the experience of poverty.

Continuing to leverage the experience of the Great Recession in Ireland, we tested the pathways that mediate the relationship between family economic strain, in the form of one or more recession events, and smoking. Considering the large body of evidence for the role of the Family Stress Model (FSM) in explaining the relationship between childhood disadvantage and development (Conger & Elder, 2020; Masarik & Conger, 2017), we hypothesised that the FSM would continue to act as a key mediating process when a family is faced with temporary economic strain as a result of the economic downturn. To the best of our knowledge, we are the first to extend the consequences of the FSM to the specific facet of adolescent problem behaviour that is risky health behaviours, in this case smoking. Moreover, this study is one of few emerging studies testing the FSM exploiting the temporal ordering of longitudinal data (Chen et al., 2023; Layte, 2022; Simons et al., 2016; Solantaus et al., 2004).

The results of our models provide some evidence for the FSM as a mediating process between temporary family economic strain as a result of the recession and smoking. The average proportionate reduction in the "recession events" estimate for probability of smoking at age 17 is 36.5% with the addition of our mediating and control variables. It seems that the psychological consequences of economic strain play a part in increasing the likelihood of subsequent child smoking. This suggests that the mechanistic processes that lead young people from disadvantaged families to problem behaviour, more generally, are the same as for advantaged families who experience temporary economic strain as a result of economic downturn. Our findings from this mediation analysis

provide further evidence that family conditions and context influence individual risk of initiating smoking.

Our findings contribute to the body of evidence on the impact of economic recessions on health and health behaviours (Glonti et al., 2015). It could be argued that the impact on health as a result of recessions is temporary, in that once the recession abates then household incomes will recover the family context will returns to its prior state. Regardless, it's plausible that the initiation of smoking has lasting effects. Firstly, smoking has an addictive quality making cessation more difficult and secondly, health behaviours established during adolescence tend to track into adulthood (Craigie et al., 2011; Dos Santos et al., 2020) with evidence for patterns of behaviours being firmly established prior to age 19 (van Nieuwenhuijzen et al., 2009). As our study looks at smoking at age 17/18, the young person's health trajectory may be permanently altered.

Study Limitations

This longitudinal design, which involves interviewing the same participants on several occasions, is one of the key assets of this dataset. Longitudinal data tracks the development of the young person, meaning we can observe trajectories in our variables of interest over time. We can examine the effects of earlier exposures to risk factors for later development of HHBs, as contended by the life-course approach (Kuh et al., 2003).

Due to a combination of a detailed understanding of what makes some families more vulnerable to the recession than others and the informational richness of the GUI data, it was possible to apply PSM (Caliendo & Kopeinig, 2008; Wang, 2021). Following the advice of Caliendo and Kopeinig (2008), theory guides our variable selection to avoid over-parameterized models that include extraneous variables in the propensity score

specification. Matching is an increasingly popular method for preprocessing data to improve causal inferences in observational data, but we are careful not claim causal inference or to approximate exactly an experimental design. The propensity score analysis reduces the selection bias on observed characteristics but it is possible that other, unobserved factors that were not strongly correlated with the observed characteristics of the family continue to bias the analysis. If so, this could mean that our results overestimate the size of the effect of recession. Moreover, this study may overestimate the impact of recession as recession limits the ability of marital partners to separate, which may lead to an intensification of conflict between partners, a key part of the FSM process. It's also possible that the magnitude of the effects that we see in this paper would be larger outside of the context of a recession, particularly one as profound as the Great Recession. There is evidence that the psychological impact of unemployment is lower when the proportion of people in your reference group who are unemployed is higher (Clark, 2003), which is theorised to be as a result of less social comparisons or 'status anxiety' (Layte & Whelan, 2014). If true, some of the effects we observe will be lower than those experienced outside of a recession.

Our outcome variable is a count of the number of recession events the family experienced. As with all count variables there is the potential issue of "the consistency assumption:" different variants of the exposure have different effects on the outcome. To acknowledge this assumption, we further clarify the nature of our exposure variable i.e. recession events (Rehkopf et al., 2016). We carried out additional sensitivity analyses replicating our current models (table 11) with "number of recession events" but each recession event is fitted separately to predict smoking (see table 9, appendix C). Here it is possible to see the unique influence of each type of recession event on our outcome, smoking. The variables are modelled separately as binary variables (e.g. 0 no redundancy,

1 redundancy) as there is likely strong linear associations among these sets of predictors. There is a positive relationship between each recession event and smoking. The relationship is highly significant at $p<.001$ for reduced hours, mortgage arrears and bill arrears and significant at $p<.01$ for cutting back on basics and luxuries and $p<.05$ for a parent becoming redundant. This preliminary investigation suggests that each variant of the exposure does not have different effects on the outcome. However, this is with the exception of 'reduced wages,' which appears to have a significant negative effect on smoking ($p<.001$), indicating that a family experiencing this recession event reduces the likelihood of child smoking. In our initial descriptive analysis, we observed that families who experienced 'reduced wages' tended to have demographic characteristics that indicated social and economic advantage (non-manual and professional class households, parents with a third-level degree, older parents, household owners and non-migrants), which suggests this might be a compositional effect. For example, families who experienced this had other advantages (e.g. savings) that protected them from the otherwise negative consequences of reduced wages. To investigate this possible compositional effect further, we ran a series of three models (see table 10, appendix C). Model three considers the interaction of household social class and reduced wages. It shows that experiencing reduced wages (vs. not experiencing reduced wages) has a negative effect on the probability of the study child smoking. However, the interaction term shows that the children of professional and non-manual households who experience reduced wages are more likely to smoke than others from professional and non-manual households who did not experience a reduction in wages. This finding supports the combination of all of the indicators of recession in the same composite index.

However, according to the interaction term, the children of professional and non-manual households who experience reduced wages are not more likely to smoke than their more disadvantaged counterparts. This further sub-analysis aided in demonstrating that it is likely not a compositional effect but may be due to a unique mechanism that is triggered when a family income is reduced that results in less smoking in their offspring.

Another limitation was the use of a simple binary variable for smoking: smokers vs. non-smokers, and the resulting lack of specific analysis of 'occasional smokers,' which have been shown to display a social gradient (Alves et al., 2023). This was due to the small number of occasional smokers from an advantaged SEP.

Discussion & Conclusion

Overview

Paper one: The Role of Family, School and Neighbourhood in Explaining Inequalities in Physical Activity Trajectories between age 9 and 18.

PA is the first of the common modifiable risk factors for the emergence of NCDs that we consider in this thesis. This paper quantifies the role of aspects of family, school and neighbourhood in accounting for the social gradient in PA. Having established that BMI, health-status and financial resources have a bearing on varying levels of participation in PA by SEP, paper one brings to light other more structural influences. Both the behaviours of parents (own PA levels) and the specific parenting practices that they adopt ('concerted cultivation' or 'natural growth') partially explain the social patterning of PA. Considered separately, parents enrolling their children in organised sport was the most explanatory family-level mechanism. Participation in organised sport has already been shown to account for both the age decline and gender differences in levels of PA, our findings indicate that it also accounts, in part, for social inequalities in PA. Our chosen aspects of the school environment (sports facilities and commute) did not explain the social patterning of PA, but it is possible that there are other important but unmeasured school-level processes that were not captured in this study. The role of our chosen aspects of the neighbourhood environment (safety and crime) was also modest.

Paper two: Bringing the Group Back in: Social Class and Resistance in Adolescent Smoking.

Smoking is the next common modifiable risk factor under the examination. Although overall prevalence of smoking has declined, social inequalities in smoking continue to increase. In this paper we present 'the psychological model,' 'the smoking exposure

model' and the 'social-resistance model,' where the first model represents individual-level explanations based on established theories in the field of public health, and the second and third model represent the structural-level explanations put forward by this paper. Our results indicate that 'the psychological model' does not provide insight into how inequalities in smoking emerge. In terms of 'the smoking exposure' model, parental smoking is a mediating pathway between childhood disadvantage and smoking i.e. young people from disadvantaged families are more exposed to parental smoking and this increases their likelihood of smoking. Being exposed to smoking friends was a strongly significant positive predictor of smoking, a relationship that is well documented. However, this exposure does not aid in explaining smoking inequalities in young people. A finding that has been attributed elsewhere to equalisation hypothesis(West, 1997), which suggests that youth culture cuts across socioeconomic strata. For 'the social resistance model,' varying levels of oppositional values is a mediating pathway between childhood SEP and smoking, which suggests that smoking as a symbolic device for the 'social-resistance framework' is a plausible structural explanation. In sum, we demonstrate how the social context, within which the young person is embedded, has a more important influence on inequalities in smoking risk than the young person's individual-level psychological or personality traits.

Paper three: Social Exclusion and Health Behaviours: Insights from a Longitudinal Study of Irish Young People.

Paper three examines patterns of engagement with HHBs. The results of our LCA confirm that 'unhealthy lifestyle' behaviours (poor diet, low physical activity and high screentime) tend to occur together, while substance use behaviours (smoking and underage drinking) appear to be independent of lifestyle behaviours, and cluster together. The four clusters

that emerged were labelled accordingly: 'healthy,' 'healthy lifestyle but substance users,' 'unhealthy,' and 'unhealthy lifestyle and substance users'. Using multi-nominal logistic regression, we tested whether the members of these groups share common risk factors that predict group membership. In sum, we found that experiencing periodic and persistent (income) poverty was highly predictive of membership in the 'unhealthy lifestyle' LCG, and to a lesser extent predictive of membership of the 'unhealthy substance user' LCG, supporting existing evidence from previous poverty research. Using the same methods, we found that reporting oppositional values significantly increases the risk of being a member of 'healthy lifestyle but substance users' and 'unhealthy lifestyle and substance users' LCG i.e. those characterised by substance use. In summary, our results provided support for our hypotheses that persistent poverty leads to a fall in the consumption of the goods and services necessary for individuals, families and groups to obtain a lifestyle similar to their fellow citizens, and that resistance processes are particularly helpful in understanding why young people engage in HHBs. It seems that adolescents who develop oppositional values, regardless of SEP, are more likely to engage with substance use behaviours. However, as predicted by the social Resistance framework, oppositional values are higher amongst those who experience social exclusion in the form of income poverty.

Paper four: Does Family Economic Strain increase the likelihood of Child Smoking? A Longitudinal Assessment Using the Great Recession in Ireland.

This aim of the final paper of this thesis was to examine the relationship between family economic circumstances and smoking. The key challenge was to disentangle the influence of economic strain on the young person's smoking behaviour from other common features of poverty such as parental education-level and occupational status. To overcome this challenge, we adopted a quasi-experimental design to consider the relationship

between increased economic strain, only as a as a result of the Great Recession, and the likelihood of being an adolescent smoker. The experience of recession increased the likelihood of the young person being a smoker, suggesting that there is a relationship between economic strain and smoking independent of other correlates of the experience of poverty. Continuing to leverage the Great Recession in Ireland as a quasi-experiment, we tested the pathways that mediate this relationship. The results of our models provide evidence for the FSM as a key mediating process between family economic strain and smoking. It seems that the psychological consequences of economic strain, in the form of deteriorating parental mental health and child social-emotional mal-adjustment, play a part in increasing the likelihood of youth smoking. Our findings provide further evidence that family conditions and context influences individuals initiating smoking.

Research Contribution

Our research aims to fill a specific gap in the literature by gaining insight into the 'causes of causes' of health inequalities i.e. why health behaviours vary across groups in a structured manner. Previous research has stopped short by highlighting the important role that *behavioural* factors play in the formation of disease, and disparities in health. Here we have both theorised and tested empirically the specific mechanistic processes that link SEP and risk of specific HHBs.

Our data confirms adverse gradients in physical activity, screentime, diet, smoking and underage drinking by SEP in the Irish context, to add to evidence from other national contexts that also show a strong associations for these potentially harmful health behaviours(J Inchley et al., 2020). We also confirm a sharp decline in PA and the uptake of smoking during adolescence, in this context, as hypothesised by Viner et al. (2015) 'critical period' research, and shown elsewhere.

GUI data is renowned for its multifaceted approach to data collection, collecting variables related to the individual and their social environment. Due to this richness of the data, we were able to compare individual-level and structural-level influences on the young person's behaviour. This allows us to contribute to the growing literature on the social determinants of health (Marmot & Bell, 2019) but also speak to the fundamental sociological debate on the role of structure vs. agency(Durkheim, 1895). The timing of the GUI study, where young people experienced the Great Recession during the period of transition from childhood to adolescence, enables us to make a novel contribution to the growing body of literature debating individual vs. structural determinants of health in the form of a quasi-experiment(Marmot & Wilkinson, 2005). We demonstrate that an exogenous shock in the form of an economic downturn can fuel the individual behaviour that is smoking.

This thesis answers the call for research on health inequalities that focuses on disadvantaged sup-groups in the population(Marmot & Bell, 2019). Historically as population health has improved, health inequalities persist(Hiscock et al., 2012; Lawlor et al., 2003). Regarding HHBs, the focus of this thesis, their prevalence is in large decreasing (Abuse & Administration, 2014; Bauman et al., 2012; Gowing et al., 2015; OECD, 2021) but reductions have been larger among more socio-economically advantaged groups, which has led to widening socioeconomic disparities.

Overall, our research provides empirical evidence for established theories from the disciplines of sociology, psychology, and social epidemiology.

Conclusion

In summation, we argue that inequalities in health behaviours are best explained from a structural theoretical perspective. We put forward that the young person's social and cultural environment is an important factor in understanding how social gradients in physical inactivity, poor diet, excessive screentime, underage drinking and smoking emerge, at a critical juncture in the life-course. We used advanced quantitative methodology on a large longitudinal dataset to test this perspective. Our results highlight the key role of the family context and social exclusion in explaining gradients in HHBs. Our findings highlight the broader structural processes that shape individual health outcomes.

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Appendices

Appendix A – Chapter 3

Table 1. Proportion of missing values.

Dependent variable	% missing
Smoking status at 17	1.09%
Treatment variable	
Family SEP	0.07%
Control variables	
Gender	0.00%
Extraversion	0.13%
Agreeableness	0.07%
Conscientiousness	0.09%
Emotional stability	0.09%
Openness to experience	0.19%
Mediating variables	
Psychological model	
Self-esteem	1.43%
Self-efficacy	1.35%
Exposure model	
Parental smoking	2.43%
Other household members smoking	2.43%
Friends smoking	0.00%
Social resistance model	
Everyday discrimination	1.24%
Opposition to authority	3.68%

Table 2. Descriptive statistics for mediating variables by family social class.

		Five Household Social Class Categories										
		Professionals & Higher Managers (N=1172)		Lower Manager & Technical (N=3317)		Non-manual (N=1686)		Skilled Manual (N=1196)		Semi & Unskilled (N=1191)		
		Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	*p value*
	Exogenous Variables											
Psychological Model	Self-esteem	12.22	3.58	12.10	3.48	12.05	3.48	11.91	3.54	11.49	3.69	<.001
	Self-efficacy	18.40	2.74	18.33	2.75	18.38	2.88	18.38	2.81	18.25	2.81	0.80
Social Resistance Model	Opposition to authority	15.62	3.41	16.00	3.56	16.26	3.60	16.49	3.59	16.97	3.74	<.001
	Everyday discrimination	1.23	0.78	1.22	0.83	1.27	0.89	1.20	0.89	1.26	0.93	0.33
		N	%	N	%	N	%	N	%	N	%	
Smoking Exposure Model	Parent's smoking	172	16.06	549	18.42	386	27.09	300	29.94	429	44.27	<.001
	Other household smokers	54	5.05	200	6.72	134	9.42	129	12.91	165	17.1	<.001
	Friends smoking	195	20.9	516	20.66	236	20.43	176	21.81	162	21.83	0.91

Table 3. Descriptive statistics for mediating variables for smokers and non-smokers.

		Smoking status at 17				
		Smokers (N=1118)		Non-smokers (N=5098)		
		Mean	(SD)	Mean	(SD)	*p value*
	Exogenous Variables					
Psychological Model	Self-esteem	11.04	0.11	12.23	0.05	<.001
	Self-efficacy	17.81	0.08	18.46	0.04	<.001
Social Resistance Model	Opposition to authority	18.42	0.11	15.67	0.05	<.001
	Everyday discrimination	1.55	0.03	1.16	0.01	<.001
		N	%	N	%	*p value*
Smoking Exposure Model	Parent's smoking	352	32.77	1067	21.7	<.001
	Other household smokers	140	13.06	358	7.29	<.001
	Friends smoking	1091	13.46	3825	54.72	<.0.01

Table 4. Path coefficients of exogenous and endogenous variables estimated using logistic regression as part of generalised structural equation modelling.

		Controls Model (N=6039)		Psychological Model (N=6039)		Exposure model (N=6039)		Resistance Model (N=6039)		Fully Adjusted Model (N=6039)	
		coef .	S.E	coef .	S.E	coef .	S.E	coef .	S.E	coef .	S.E
Predicting smoking status	female	-0.13	0.09	0.02	0.10	-0.09	0.10	-0.34**	0.10	-0.25*	0.11
	Lower Manager & Technical (vs. professional)	0.09	0.16	0.09	0.16	0.06	0.16	0.00	0.16	-0.01	0.17
	Non-manuals (vs. professionals)	0.23	0.17	0.21	0.16	0.17	0.19	0.05	0.18	0.04	0.19
	Skilled manuals (vs. professionals)	0.17	0.18	0.18	0.18	0.06	0.19	-0.00	0.19	-0.05	0.20
	Semi & unskilled (vs. professionals)	0.23	0.18	0.20	0.18	0.10	0.20	-0.07	0.19	-0.11	0.21
	Extraversion	0.075699**	0.02	0.09**	0.03	0.05	0.03	0.07*	0.03	0.05	0.03
	Agreeableness	-0.11***	0.02	-0.11***	0.03	-0.09**	0.03	-0.07*	0.03	-0.07*	0.03
	Conscientiousness	-0.13***	0.02	-0.12***	0.24	-0.11***	0.03	-0.10***	0.02	-0.09**	0.03
	Emotional stability	0.01	0.02	0.02	0.03	0.01	0.03	0.01	0.03	0.02	0.03
	Openness to experience	0.03	0.03	0.03	0.03	0.02	0.03	0.03	0.03	0.02	0.03
	Self-esteem			-0.07***	0.02					-0.02	0.02
	Self-efficacy			-0.02	0.02					0.01	0.02
	Parental smokers					0.45***	0.12			0.39**	0.12
	Household smokers					0.14*	0.07			0.12	0.07
	Friend smokers					1.96***	0.10			1.75***	0.11

	Opposition to authority							0.18***	0.02	0.14***	0.02
	Experience everyday discrimination							0.23***	0.06	0.18*	0.07
	Constant	-0.93	0.24	0.10	0.36	-1.74	0.27	-4.28	0.36	-4.40	0.56
Predicting levels of self-esteem	female			1.95***	0.12					1.96***	0.12
	Lower Manager & Technical (vs. professional)			0.00	0.17					0.01	0.17
	Non-manuals (vs. professionals)			-0.17	0.20					-0.18	0.20
	Skilled manuals (vs. professionals)			-0.05	0.21					-0.04	0.21
	Semi & unskilled (vs. professionals)			-0.41	0.22					-0.37	0.22
	Extraversion			0.07*	0.03					0.07	0.03
	Agreeableness			0.04	0.03					0.04*	0.03
	Conscientiousness			0.10**	0.03					0.10**	0.03
	Emotional stability			0.15***	0.03					0.15***	0.03
	Openness to experience			-0.04	0.03					-0.04	0.04
	Constant			9.76	0.30					9.74	0.29
Predicting levels of self-efficacy	female			0.58***	0.10					0.58***	0.10
	Lower Manager & Technical (vs. professional)			0.14	0.15					0.14	0.15
	Non-manuals (vs. professionals)			0.04	0.17					0.04	0.17

	Skilled manuals (vs. professionals)	0.28	0.18			0.28	0.18
	Semi & unskilled (vs. professionals)	0.11	0.18			0.11	0.18
	Extraversion	0.14*	0.03			0.14***	0.03
	Agreeableness	0.02	0.03			0.02	0.03
	Conscientiousness	0.06**	0.03			0.06*	0.03
	Emotional stability	0.09***	0.03			0.09***	0.03
	Openness to experience	0.02	0.03			0.02	0.03
	Constant	16.57	0.24			16.57	0.24
Predicting exposure to parents smoking	female			-0.12	0.08	-0.12	0.08
	Lower Manager & Technical (vs. professional)			0.16	0.13	0.16	0.13
	Non-manuals (vs. professionals)			0.73***	0.14	0.73***	0.14
	Skilled manuals (vs. professionals)			0.93***	0.14	0.93***	0.14
	Semi & unskilled (vs. professionals)			1.24***	0.15	1.24***	0.15
	Extraversion			0.03	0.02	0.03	0.02
	Agreeableness			-0.03	0.02	-0.03	0.02
	Conscientiousness			-0.06**	0.02	-0.06**	0.02
	Emotional stability			-0.02	0.02	-0.02	0.02
	Openness to experience			-0.02	0.04	0.02	0.02

	Constant	-0.79	0.19	-0.79	0.19
Predicting exposure to smokers in the household (excluding parents)	female	-0.13	0.12	-0.13	0.12
	Lower Manager & Technical (vs. professional)	0.21	0.25	0.21**	0.25
	Non-manuals (vs. professionals)	0.71**	0.27	0.71**	0.27
	Skilled manuals (vs. professionals)	0.97***	0.26	0.97***	0.26
	Semi & unskilled (vs. professionals)	1.27***	0.26	1.27***	0.26
	Extraversion	0.09*	0.03	0.09	0.03
	Agreeableness	-0.04	0.03	-0.04***	0.03
	Conscientiousness	-0.06*	0.03	-0.06***	0.03
	Emotional stability	-0.00	0.04	0.00	0.03
	Openness to experience	-0.02	0.04	-0.02	0.04
	Constant	-2.45	0.30	-2.45	0.30
Predicting exposure to smoking friends	female	-0.02	0.09	-0.02	0.09
	Lower Manager & Technical (vs. professional)	-0.01	0.14	-0.01	0.14
	Non-manuals (vs. professionals)	-0.29	0.15	-0.03	0.15
	Skilled manuals (vs. professionals)	-0.06	0.16	0.06	0.16

	Semi & unskilled (vs. professionals)	-0.03	0.16			0.09	0.17
	Extraversion	0.09***	0.02			0.09***	0.02
	Agreeableness	-0.06*	0.02			-0.06**	0.02
	Conscientiousness	-0.06	0.02			-0.06**	0.02
	Emotional stability	-0.02	0.02			-0.02	0.02
	Openness to experience	0.02	0.03			0.03	0.03
	Constant	-1.01	0.21			-1.01	0.21
Predicting levels of opposition to authority	female			0.67***	0.08	0.66***	0.13
	Lower Manager & Technical (vs. professional)			0.57**	0.19	0.53**	0.18
	Non-manuals (vs. professionals)			0.75**	0.22	0.72**	0.22
	Skilled manuals (vs. professionals)			1.07***	0.23	1.02***	0.23
	Semi & unskilled (vs. professionals)			1.58***	0.24	1.55***	0.23
	Extraversion			-0.00	0.03	0.00	0.03
	Agreeableness			-0.14***	0.04	-0.14***	0.03
	Conscientiousness			-0.14***	0.03	-0.14***	0.03
	Emotional stability			0.01	0.03	0.00	0.03
	Openness to experience			0.02	0.04	0.01	0.04
	Everyday discrimination			1.44***	0.08	1.43***	0.08
	Constant			14.66	0.35	14.72	0.34

Predicting experiencing everyday acts of discrimination	female	0.12***	0.03	0.12***	0.03
	Lower Manager & Technical (vs. professional)	-0.05	0.05	-0.05	0.05
	Non-manuals (vs. professionals)	0.04	0.06	0.04	0.06
	Skilled manuals (vs. professionals)	-0.07	0.06	-0.06	0.06
	Semi & unskilled (vs. professionals)	-0.00	0.06	-0.00	0.06
	Extraversion	0.03*	0.01	0.27**	0.01
	Agreeableness	-0.04***	0.01	-0.04***	0.01
	Conscientiousness	-0.03***	0.01	-0.03***	0.01
	Emotional stability	-0.02	0.01	-0.02	0.01
	Openness to experience	0.01	0.01	0.01	0.01
	Constant	1.46	0.07	1.46	0.07

***p<.05, ***p<.01, ***p<.001**

Appendix B – Chapter 4

Table 5. Level of missing

Health Behaviours	% missing
Smoking status	1.1%
Age first alcoholic drink	1.6%
Diet Quality	0.1%
Physical activity	0.1%
Screen time (minutes)	1.2%
Controls	
Gender	0.0%
Personality	
Extraversion	0.2%
Agreeableness	0.2%
Conscientiousness	0.2%
Emotional stability	0.2%
Openness to experience	0.2%
Chronic illness	0.9%
Predictors	
Persistent poverty	10.4%
Oppositional values	3.7%

Table 6. Fit statistics

Number of classes	AIC	BIC	aBIC*
2	445.77	573.34	512.97
3	265.79	460.51	368.36
4	217.66	479.52	355.59
5	187.86	516.87	361.16
6	184.59	580.75	393.26

Table 7. Results from multi-nominal logistic regression predicting latent class membership.

	Model 1[1]			Model 2[1]			Model 3[1]			Model 4[1]		
	Baseline			Social Exclusion			Social resistance			Interaction social exclusion and social resistance		
	RRR	*S.E*	*pvalue*	*RRR*	*S.E*	*pvalue*	*RRR*	*S.E*	*pvalue*	*RRR*	*S.E*	*pvalue*
Unhealthy												
Persistent poverty												
One wave (vs. no waves)				1.19	0.12	0.10				1.16	0.12	0.16
Two waves (vs. no waves)				1.71	0.17	**0.00**				1.63	0.17	**0.00**
Three waves (vs. no waves)				2.00	0.21	**0.00**				1.91	0.20	**0.00**
Oppositional values							1.23	0.05	**0.00**	1.18	0.09	**0.02**
Persistent poverty # oppositional values												
One wave (vs. no waves)										0.97	0.11	0.80
Two waves (vs. no waves)										1.08	0.12	0.48
Three waves (vs. no waves)										0.97	0.10	0.79
Constant	0.28	0.02	**0.00**	0.21	0.02	**0.00**	0.28	0.02	**0.00**	0.22	0.02	**0.00**
Healthy (reference category)												
Unhealthy drinkers and smokers												
Persistent poverty												
One wave (vs. no waves)				1.30	0.16	**0.03**				1.14	0.17	0.38
Two waves (vs. no waves)				1.94	0.23	**0.00**				1.49	0.23	**0.02**
Three waves (vs. no waves)				2.38	0.29	**0.00**				2.34	0.33	**0.00**
Oppositinal values							2.90	0.14	**0.00**	3.17	0.27	**0.00**

Persistent poverty # oppositional values												
One wave (vs. no waves)										0.95	0.13	0.72
Two waves (vs. no waves)										1.02	0.14	0.91
Three waves (vs. no waves)										0.67	0.08	**0.00**
Constant	0.22	0.01	**0.00**	0.15	0.01	**0.00**	0.14	0.01	**0.00**	0.10	0.01	**0.00**
Healthy drinker (& smokers)												
Persistent poverty												
One wave (vs. no waves)				0.98	0.08	0.78				0.90	0.08	0.20
Two waves (vs. no waves)				0.93	0.08	0.44				0.86	0.08	0.09
Three waves (vs. no waves)				0.95	0.09	0.57				0.81	0.08	**0.03**
Oppositinal values							1.65	0.06	**0.00**	1.71	0.10	**0.00**
Persistent poverty # oppositional values												
One wave (vs. no waves)										1.02	0.09	0.82
Two waves (vs. no waves)										0.86	0.08	0.13
Three waves (vs. no waves)										0.99	0.09	0.88
Constant	0.68	0.03	**0.00**	0.70	0.04	**0.00**	0.63	0.03	**0.00**	0.69	0.04	**0.00**
N	6,091			6,091			6,091			6,091		

[1] this model includes the control variables chronic illness, gender, personality but the coefficients are not displayed.

Appendix C – Chapter 5

Table 8. Level of Missing.

	T	% missing
PSM Variables		
Household Class	1	0.0 %
Maternal Education	1	0.0 %
PCG Age	1	0.0 %
Migrant Status	1	0.1 %
Household Type	1	0.0 %
Housing Tenure	1	0.1 %
PCG Self-Rated Health	1	0.0 %
ACE Score	1	0.0 %
Dependent Variable		
Smoking Status at 17	3	1.09 %
Exposure Variable		
Number of Recession Events	2	2.85 %
Change in Level of Economic Strain	1&2	2.96 %
Control Variables		
Gender	1	0.00 %
Chronic Illness	1	0.00 %
Parental CES-D	1	6.87 %
Child Social-Emotional Difficulties	1	0.50 %
Mediating Variables		
Parental CES-D	2	3.57 %
Child Social-Emotional Difficulties	2	2.86 %

Table 9. Separate logistic regressions predicting probability of smoking at T3 for each recession event.

		Coef.	t-stat
Recession event	Redundancy	0.22	1.86
	Reduced hours	0.22	**2.81****
	Reduced wages	-0.24	**-3.31*****
	Reduced social transfers	0.07	1.10
	Cut back luxuries	0.16	**2.32***
	Cut back basics	0.16	**2.05***
	Mortgage arrears	0.50	**4.34*****
	Bills arrears	0.57	**5.34*****
	Other	0.16	1.16

*p<.05, **p<.01, ***p<.001

Table 10. Logistic regression estimates of probability of smoking at T3.

		Model 1		Model 2		Model 3	
		Coef.	t-stat	Coef.	t-stat	Coef.	t-stat
Reduced wages	No (ref.)						
	Yes	-0.30	**-4.27*** **	-0.27	**-3.86*** **	-0.59	**-3.16** **
Household social class	Professionals			-0.31	**-2.36***	-0.53	**-2.55** **
	Managerial & Technical			-0.14	-1.36	-0.34	**-2.20***
	Non-manual			-0.05	-0.45	-0.13	-0.73
	Skilled-manual			-0.09	-0.72	-0.30	-1.57
	Semi & Unskilled (ref.)						
Household social class# Reduced wages	Professionals#Yes					0.44	1.61
	Managerial & Technical#Yes					0.40	1.82
	Non-manual#Yes					0.23	0.93
	Skilled-manual#Yes					0.43	1.63
	Constant	-1.30	-23.17***	-1.19	-12.41***	-1.06	-9.05***

*p<.05, **p<.01, ***p<.001