

**Bangor University**

## **DOCTOR OF PHILOSOPHY**

### **Movin' On Up? The economics of Adverse Childhood Experiences (ACEs) in terms of health care costs and social mobility**

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*Award date:*  
2023

*Awarding institution:*  
Bangor University

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Movin' on up? The economics of Adverse Childhood Experiences

(ACEs) in terms of health care costs and social mobility

Huw Lloyd-Williams

**Thesis submitted to the School of Healthcare Sciences,  
Bangor University, in fulfilment of the requirements for the  
degree of Doctor of Philosophy**

## Declaration and Consent

'Yr wyf drwy hyn yn datgan mai canlyniad fy ymchwil fy hun yw'r thesis hwn, ac eithrio lle nodir yn wahanol. Caiff ffynonellau eraill eu cydnabod gan droednodiadau yn rhoi cyfeiriadau eglur. Nid yw sylwedd y gwaith hwn wedi cael ei dderbyn o'r blaen ar gyfer unrhyw radd, ac nid yw'n cael ei gyflwyno ar yr un pryd mewn ymgeisiaeth am unrhyw radd oni bai ei fod, fel y cytunwyd gan y Brifysgol, am gymwysterau deuol cymeradwy.'

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## Acknowledgements

I'd like to thank my supervisors Professor Rhiannon Tudor Edwards at the Centre for Health Economics and Medicines Evaluation (CHEME) at Bangor University and Professor Mark Bellis at Public Health Wales for all their help, support, and supervision throughout the process of this PhD. They have provided invaluable expertise and knowledge which has enabled me to develop both academically and professionally. Without their continued guidance and feedback this thesis would not have been possible. Dr Rebecca Hill at Public Health Wales for stepping in as my second supervisor towards the end of my PhD. Also, Dr Llinos Haf Spencer who acted as a second reviewer in my systematic review chapter and helped with submitting a paper from the thesis for publication. Dr Nathan Bray, Dr Catrin Plumpton and Dr Victory Ezeofor for reading drafts of sections of the thesis and providing invaluable advice on the technical aspects of the thesis. Andrew Bennet, Professor Gill Windle, Yasmin Noorani and Dr Siobhan Adams who provided advice and Karen Hughes for help with the data. I would especially like to thank Professor Shanti Chakravarty for his continued support over the years and for helping me get over the line with this PhD. Finally, I'd like to thank my mum Iona, my partner Iola and my son Gruffudd for whom this thesis is dedicated along with my late father Gareth.

## Presentations

The systematic review in this thesis was delivered as an oral presentation at the European Health Economics Association (EuHEA) in Maastricht in 2018 and also at the Welsh Health Economics Group's (WHEG) annual meeting in 2018. An abstract of the whole thesis was also presented at the International Health Economics Association (iHEA) in 2021. Chapter 2 has been adapted into a paper and submitted to the 'Journal of Child Psychology and Psychiatry'.

## Abstract

Adverse childhood experiences (ACEs) is a term developed to define one of ten possible experiences that affect children as they are growing up that can lead to problems later in life.

In the early stages of the PhD relatively little was known about how much ACEs cost society and how ACEs affect lifetime socioeconomic status and social mobility. Research published in 2020, however, estimated that ACEs cost the economy of England and Wales £48.2 billion.

This thesis is in two parts. The first part deals with testing a methodology for attributing lifetime health costs to the presence or absence of ACEs across five main diseases in terms of expenditure (Chapters 2-3). This thesis proposes a novel approach to achieve this aim which can be broadly defined as performing an extrapolation of the data that *is* available or a pro-rata approach to estimate the missing data. The second part (Chapters 4-5) of the thesis addresses the issue of whether ACEs are associated with a degree of lifetime social mobility defined by wealth in adulthood compared with wealth in childhood. Both parts of the thesis employ systematic informed reviews of current evidence (Chapters 2 and 5). In part 1, looking at attributable health costs, a population attributable fraction (PAF) methodology is used (Chapter 3). In part 2, social mobility is explored using part of the ACEs dataset (which is a large survey dataset conducted in Wales, England, Blackburn with Darwen and southern England between 2012 and 2015 (N=13,130) of the general public asking them to reflect on their ACEs). The areas used in the analytical chapter were a subset of this broader dataset and was confined to Wales and Southern England (N=7,429) as only these areas had the variable 'wealth in childhood/adulthood' used to calculate social mobility. The two parts of the thesis are connected. It is posited that an increase in investment to tackle ACEs, the amount of which is identified in the costing section, leads to an increase in social mobility and hence a commensurate increase in tax revenues for the government that could, if the

government were so inclined, be invested back into society to deal with the outcomes associated with ACEs.

This thesis provides two novel findings. First, that it was possible to find lifetime attributable costs for mental health, cancer and circulatory disease but not the other two areas of musculoskeletal and genitourinary disease. This was because of a lack of information on odds ratios for ACE counts for musculoskeletal disease and a lack of data on odds ratios *and* costs for genitourinary disease. Secondly, with respect to social mobility, this thesis found a counter intuitive, but statistically significant outcome where increasing ACEs were associated with an increased likelihood of upward social mobility. This may be because access to health and social care increases as ACE counts increased. Another explanation may be that people become upwardly mobile *despite* ACEs by having access to a trusted adult and developing resilience. One way of preventing ACEs from being passed down the generations is through social mobility. The question will be asked if there are any enablers in terms of the promotion of resilience that aid social mobility in children who have experience of ACEs. This relationship is tested with the data in Chapter 5 and it is found that having a trusted adult is linked with upward social mobility given that the respondents have ACEs. That is children become upwardly mobile *despite* adversity given the help of a trusted adult.

The second finding also gave rise to a research dilemma. I could not reject the counterintuitive result because it was significant and could not rejoice over the intuitive result as it was not significant. Suffice to say that the knowledge base of this field of study has been expanded by this thesis.

In the final chapter, policy recommendations are offered based on the results of this thesis, around approaches to dealing with ACEs especially considering improving social mobility.

Developing access to a 'trusted adult' is seen as key in providing children with an element of resilience against the harmful effect of ACEs.

This thesis argues that if the role of trusted adults can be maintained and developed then this can protect children against the possibility of downward social mobility and even lead to upward social mobility. That is, children can 'move on up' despite having several ACEs, if given the right support with economic benefits both to themselves and the economy as a whole.

## Chapter 1 – Introductory chapter

### 1.1 Chapter summary

Adverse childhood experiences have a huge cost to children, families, and wider society.

They lead to poor health and social problems through the life course. Chapter 1 of this thesis presents the rationale, research questions and approaches used in subsequent chapters. The basic premise is that ACEs have a substantial cost, both directly in terms of the harm caused to children, and indirectly to society as a whole. It has been estimated that in 2020 ACEs cost the economy of England and Wales £42.8 billion (Hughes et al 2020).

Further, nearly £17 billion per year is spent in England and Wales by the state on the cost of late intervention. This works out at around £287 per person.

- The largest individual costs are:
  - £5.3 billion spent on Looked After Children
  - £5.2 billion associated with cases of domestic violence
  - £2.7 billion spent on benefits for young people who are not in education, employment, or training (NEET).
- The cost of late intervention is spread across different areas of the public sector, with the largest shares borne by:
  - local authorities (£6.4 billion)
  - the NHS (£3.7 billion)
  - DWP (£2.7 billion).

Source: EIF (2016)



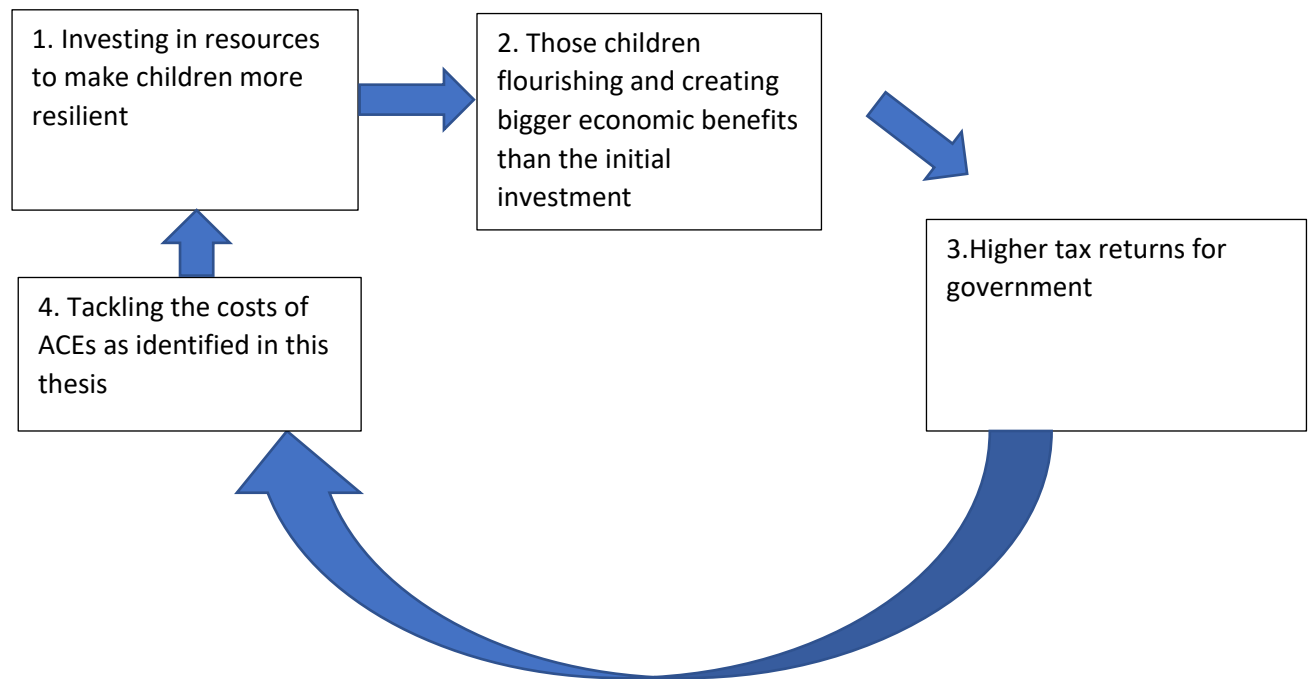
These are huge and preventable figures. The reduction of ACEs could therefore save the public purse substantial amounts let alone avoiding the human cost in terms of harm to children. This chapter explains exactly what is meant by ACEs delineating the ways in which they can impact on lifelong health outcomes both directly and indirectly. It also describes the prevalence of ACEs in the UK so that it can be established how much of a problem they pose. Also described in this chapter is the overarching aim of this thesis and the methods that will be used to tackle these issues.

## **1.2 Rationale for the thesis**

In the early stages of writing this thesis little was known about the true costs of ACEs to society. ACEs, as can be seen later in this chapter, contribute to a plethora of diseases in the later life of individuals affected by them. The rationale for the first part of this thesis was to find out if it is possible, with the data available, to put a societal lifetime cost on those diseases that ACEs contribute towards. Further, a decade of austerity has led to socio-economic inequality becoming sharpened in our society. Having a difficult start in life leads to adverse *economic* outcomes as well as adverse health outcomes. Most recently the link between disadvantage and health has been sharpened even further due to the Covid-19 pandemic. Those from a disadvantaged background are twice as likely to die because of the pandemic (Nuffield Trust 2020). Set against this is the idea that ACEs are prevalent, especially in disadvantaged communities, and they may have a bearing on whether individuals can escape this poverty by means of social mobility. The hypothesis to be tested is 'do ACEs lead to upward or downward social mobility?' If so, are there mitigating factors that

enable this transition? Such a factor could be having a trusted adult, and this will be explored in the analysis chapter of this thesis (Chapter 5). The rationale behind this part of the thesis is that as people become more upwardly mobile and have a better standard of living this will generate more tax revenues that government then has the option to channel into combating the ill effects of ACEs in society today. This proposition can be seen as the conceptual underpinning of this thesis. In effect this thesis explores the idea that combating ACEs and their effect is not a zero-sum game. That is, taking resources away from current spending priorities and putting them towards dealing with ACEs need not mean that the amount of ACEs is reduced by a commensurate amount. Indeed, it can be argued that channelling resources towards making children more resilient may lead to those same children flourishing and creating bigger economic benefits to themselves and society than the initial outlay in terms of investment. A positive sum game therefore where, given the right investment in the right area, children that suffer ACEs can become upwardly mobile and experience a higher standard of living thus increasing the tax revenues of government and their ability to invest further in the ways of addressing ACEs as a social ill. This thesis explores, firstly, how much of an outlay should be expected by government in terms of the costs of ACEs, or more specifically the diseases that are caused, albeit partially, by ACEs. Secondly it explores whether or not investing in resources to increase resilience among children that suffer ACEs is worthwhile by positing a hypothesis that ACEs lead to upward social mobility when we include the confounding effects of resilience.

**Figure 1.1: Virtuous cycle of investment in resilience tackling the costs of ACEs**



### **1.3 Aims of the thesis**

The aims of this thesis are threefold. The first aim of this thesis is to explore what attributable costs are associated with ACEs. Specifically, the thesis will ask if it is possible to calculate the lifetime costs of five of the most expensive diseases that are, at least partially, attributable to ACEs. The second aim of the thesis is to test the relationship between social/economic mobility and ACEs. Does having more ACEs as a child make it more likely that individuals will experience upward or downward social mobility later in life? Finally, the question will be asked if there are any enablers in terms of the promotion of resilience that aid social mobility in children who have experience of ACEs.

## **1.4 Research questions**

### **Part 1 (Chapters 2 & 3)**

- How possible is it to measure the portion attributable to ACEs of the cost of the diseases that have the highest expenditure with the available data?
- Where it is possible, to what extent are societal costs an increasing function of ACEs?

### **Part 2 (Chapters 4 & 5)**

- What is the relationship between having ACEs and upward or downward social mobility in terms of wealth in adulthood compared with in childhood?
- Can resilience (influenced by having a trusted adult) be used to explain this relationship?

## **1.5 Thesis structure**

The first part of this thesis gives a discussion around the costs of ACEs to society. It has been established that ACEs can have serious and lasting effects on individuals' life course health outcomes. So far, there has not been a concerted study looking specifically at the costs of not treating the health problems that can be caused by ACEs and this thesis will conclude by aiming to fill that gap in existing knowledge. The research question that will guide the systematic review in the second chapter is: what are the healthcare costs of the five main diseases, in terms of expenditure, in the UK and how much of these costs can be attributable to ACEs? Chapter 3 is an analytical chapter using a novel methodology to establish what are the lifetime costs associated with ACEs. The aim here will be to build a picture of the relationship between the occurrence of ACEs and healthcare costs, which will serve to guide our understanding of the costs associated with different ACE counts.

The recent Covid-19 pandemic has highlighted issues related to social inequalities with a disproportionate number of people from low socioeconomic status backgrounds dying from the virus. Professor Sir Michael Marmot has raised the issue of life expectancy levelling out and indeed going into retreat. In this context it is worth looking at the relationship between ACEs and living standards. This thesis investigates the relationship between ACEs and living standards by using social mobility as a proxy. Chapter 4 will give a description of the dataset to be used while chapter 5 will be the main analysis chapter and the main research question here is: is there a causal link between ACEs and social mobility and if so, what is the direction of this link?

## **1.6 History and definition of adverse childhood experiences (ACEs)**

Child abuse, it is argued, is a socially constructed notion. It is something that is made real by the way we construe our society (Stainton Roberts et al 1989). Our attitude, as a society, towards it has changed drastically over the past, say, 150 years. Indeed, it can be said, however shocking, that,

“...parents have always whipped, starved, locked up and raped their children, worked them mercilessly, sold them and abandoned them”. P10

Child maltreatment is nothing new, therefore. It was seen, in the century before last, that the right of parents to treat their children as they saw fit took precedence over the rights and welfare of the child. Indeed, the first cases of child maltreatment in the US to be taken to the law courts got there through legislation on dealing with cruelty to animals. Such was the importance placed on respecting the privacy of the family. By the end of the nineteenth century, society began to look at child maltreatment more seriously and was beginning to

put it before any considerations of the sanctity of family privacy. The Society for Prevention of Cruelty to Children was established in 1871 in New York and the National Society for the Prevention of Cruelty to Children in the UK in 1883. However, it is only recently that the 'sensibilities and outlook of our culture' (Kempe & Kempe 1978) has changed to provide us with the means of looking at child abuse and neglect as a social ill.

There have been many hard-hitting, high profile news items in the past few years on specific cases of child abuse and maltreatment. By the very nature of news sensationalism, these cases are highlighted and come across as extreme and disturbing. However, the occurrence of traumatic events in childhood is prevalent across society and there are many young children in the UK today suffering what has recently been termed as adverse childhood experiences (ACEs).

The term ACEs originated from the US with the work of Vincent Felliti and Robert Anda. Felliti ran an obesity clinic in San Diego and noticed that his patients were dropping out of the programme even though they were successfully losing weight. Perplexed by this he decided to look further into the problem and research into the background of his patients. He found that a high percentage of his patients had been abused as children, which led him to believe that they were using food as a coping mechanism to deal with their adverse experiences as children. Felliti and Anda (1998) formally identified ACEs as ten distinct experiences that cause trauma in childhood and can eventually affect life course health. The following is taken from the Center for Disease Control and Prevention's website and provides a succinct definition of each ACE:

## Abuse

**Emotional abuse:** A parent, stepparent, or adult living in your home swore at you, insulted you, put you down, or acted in a way that made you afraid that you might be physically hurt.

**Physical abuse:** A parent, stepparent, or adult living in your home pushed, grabbed, slapped, threw something at you, or hit you so hard that you had marks or were injured.

**Sexual abuse:** An adult, relative, family friend, or stranger who was at least 5 years older than you ever touched or fondled your body in a sexual way, made you touch his/her body in a sexual way, attempted to have any type of sexual intercourse with you.

## Neglect

**Emotional neglect:** Someone in your family helped you feel important or special, you felt loved, people in your family looked out for each other and felt close to each other, and your family was a source of strength and support<sup>1</sup>.

**Physical neglect:** There was someone to take care of you, protect you, and take you to the doctor if you needed it<sup>2</sup>, you didn't have enough to eat, your parents were too drunk or too high to take care of you, and you had to wear dirty clothes.

## Household Challenges

**Mother treated violently:** Your mother or stepmother was pushed, grabbed, slapped, had something thrown at her, kicked, bitten, hit with a fist, hit with something hard, repeatedly

---

<sup>1</sup> Items were reverse-scored to reflect the framing of the question.

<sup>2</sup> Items were reverse-scored to reflect the framing of the question.

hit for over at least a few minutes, or ever threatened or hurt by a knife or gun by your father (or stepfather) or mother's boyfriend.

**Household substance abuse:** A household member was a problem drinker or alcoholic or a household member used street drugs.

**Mental illness in household:** A household member was depressed or mentally ill or a household member attempted suicide.

**Parental separation or divorce:** Your parents were ever separated or divorced.

**Criminal household member:** A household member went to prison.

Anda, working at the Centre for Disease Control, and Felitti, who worked for health care firm Kaiser Permanente in Chicago, embarked upon a research study (Felitti, J, Anda, R, 1998) using information from 17,337 patient volunteers who had been asked about their adverse experience as children. They found that ACEs were prevalent with over 50% of the sample reporting having at least one ACE and a quarter reporting having two or more. They found a graded relationship between the number of ACEs and subsequent health problems. That is, the more ACEs a patient had the more likely they were to suffer from specific health problems. This graded relationship meant that ACEs were associated with such conditions as ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease. Compared to those with no ACEs those that had four or more were 4-12 times more likely to be alcoholics, to have drug problems, to have depression and to have attempted suicide. In addition, they were 2-4 times more likely to smoke, to have poor self-related health, have more than 50 sexual partners and have a sexually transmitted disease. They were also 1.4-



1.6 times more likely to be physically inactive or obese. Perhaps the most interesting finding from their study was that ACEs are inter-related. For example, for those with the ACE 'having a parent involved in substance abuse' they had a 62% chance of having the ACE 'mother treated violently'. It was shown that having multiple ACEs then increases the risk of multiple health problems, or co-morbidities, in later life (Felliti et al 1998).

The definition of ACEs used in UK studies is slightly different than the US based definition.

In the Welsh and English studies by Bellis et al (2015, 2014) they define ACEs in the following way:

**Child maltreatment:**

- Verbal abuse
- Physical abuse
- Sexual abuse

**Childhood household included:**

- Parental separation
- Domestic violence
- Mental illness
- Alcohol abuse
- Drug use
- Incarceration

The difference, therefore, between the US and UK definitions is that the UK definitions do not include the category for 'neglect' and that 'alcohol abuse' and 'drug use' were two

separate ACEs in the UK version. It is the UK version that will be used in this thesis for analysis purposes to be in line with the UK studies.

## **1.7 Theory**

ACEs can affect health during childhood (for example physical harm due to abuse) but also have lasting effects into adulthood. This is due to the stress resulting from ACEs changing the structure of the early brain, which can lead, indirectly, to ill health through health harming behaviour and directly through what is known as ‘biological embedding’, that is, “...through a direct biological pathway via alterations of physiological stress systems” (Solis et al 2015, pE739)

Shonkoff, Boyce and McEwen (2009) highlight three levels of stress. These are normative, routine, and toxic stress. Normative and routine stress constitutes positive stress that aids problem solving and coping skills. Such positive stress, such as a child experiencing the first day at school, is a normal part of growing up. It can be mitigated by parental care and does not last long. Exposure to ACEs leads to toxic stress. Toxic stress results from,

“...prolonged activation of the body’s stress response systems in the absence of the buffering protection of a supportive, adult relationship.” (Shonkoff 2012, p. 236)

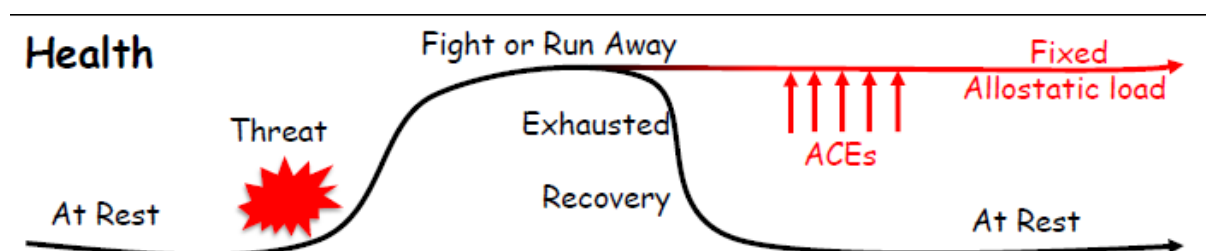
A popular example given is that of encountering a bear in a forest. Preservation and survival dictate that the individual will by-pass normal thinking and reasoning and enter a fight-flight-freeze mode. The problem arises when a child is subjected to a traumatic experience time after time and this mode is entered into too often resulting in detrimental effects on health.

Allostasis is the process through which the human body ‘maintains stability through change’. It activates adaptive responses to environmental and physiological changes (Danese 2012). It is the way the brain functions to deal with stress. According to Rosgoch (2011),

“...chronic activation (of stress management systems) results in over- or under-compensation, causing cascading effects within interconnected biological systems” (p.3)

The allostatic process is mediated through the integrated neural, endocrine, and immune systems. Within the neural system the amygdala is the part of the brain that generates cortisol and the prefrontal cortex (PFC) and the hippocampus can regulate levels. Toxic stress leads to a change in brain structure, which inhibits the PFC and hippocampus’s function leading to ‘allostatic overload’. The allostatic load refers to the wear and tear on the body because of cycles of allostasis. When exposed to stress, especially toxic stress, the brain, through the hypothalamic-pituitary-adrenomedullary (HPA) axis and sympathetic-adrenomedullary system, releases hormones such as adrenaline and cortisol, which are initially protective and work to get the body back to a resting state. The problem arises when one is exposed to repeated stress. Following a series of fight or flight responses these levels tend to stay high and tend not to return to a ‘resting’ level.

**Figure 1.2: Allostatic load because of toxic stress (Source: Bellis M.A. No Date)**



A study by Rosgoch et al (2011) attempted to measure the relationship between allostatic load and adverse health outcomes. According to Rosgoch et al (2011),

“...allostatic load and its consequent dysregulation of diverse brain and organ systems can increase the emergence of physical and mental disorders”. (P3)

However, a caveat to this was that they also recognised that ‘it cannot be concluded that the impact of maltreatment operates solely through allostatic load mechanisms, given the lack of relationship between maltreatment and allostatic load’. They further admitted that their allostatic load composite, the way they measured the allostatic load ‘did not adequately capture the diversity of ways that allostatic processes may be disrupted in maltreated children’ (p.15). This brings up some doubts as to whether maltreatment indeed leads to a fixed allostatic load however the authors admit that their measurement of allostatic load is lacking.

One of the other ways in which ACEs affects physical health is through the immune system. Studies have shown that violence victimization is associated with abnormal immune system functioning and elevated inflammation (Danese et al. 2007; Danese et al. 2011; Kiecolt-Glaser et al. 2011; Shirtcliff, Coe, & Pollak, 2009; Slopen et al. 2010; Surtees et al. 2003; Widom et al. 2012). Inflammation is how the body’s immune system attacks the antigens of microorganisms. It is triggered by ‘inducers’, or signals, that are eventually eliminated by the inflammatory response, which is then resolved, but once these inducers become chronic the inflammatory response cannot be resolved and becomes a chronic inflammatory state. This state does not differentiate between inducers and surrounding tissue and this collateral tissue damage can lead to diseases such as cardiovascular disease, type 2 diabetes and

dementia (Moffit 2013) and viral hepatitis, chronic obstructive pulmonary disease, liver cancer, asthma, autoimmune disease, poor dental health and depression (Shonkoff 2012).

ACEs can affect life expectancy directly through telomere erosion. Telomeres are 'caps' at the end of chromosomes that protect them. They are important in their role of aiding the replication of cells and their length determines their ability to do this. Sufficiently short telomeres lead to the arrest of cell replication, which is called senescence (Moffit 2013). There is an association between shortened telomeres and risk of morbidity and mortality (Cawthon et al. 2003; Ehrlenbach et al. 2009). There are many studies showing the link between shorter telomeres and risk factors for morbidity including smoking, obesity (Buxton et al 2011), schizophrenia (Yu et al 2008), mood disorders (Simon et al 2006) and psychosocial stress (Epel et al 2004). Recently there have been studies that link childhood stress with telomere length. Adults who reported ACEs had significantly shorter telomere lengths after controlling for potential confounders such as age, sex, BMI or smoking (Kananen et al. 2010; Kiecolt-Glaser et al. 2011; O'Donovan et al. 2011a; Tyrka et al. 2010).

## **1.8 Evidence**

In the ACE study (Felitti 1998) an association was found between a gradient of exposure to increasing numbers of ACEs with an index of health risk behaviours linked with adult disease and mortality. These were smoking, alcoholism, drug abuse, suicide attempts, sexual promiscuity, STDs, inactivity, and obesity. They also found a dose-response relationship of ACE exposure to heart disease, cancer, chronic bronchitis, hepatitis, skeletal fractures, and poor self-related health. Finally, they found a graded relationship, that is a proportional

one, between ACE factors and rates of psychotropic medications highlighting the link between ACEs and mental health problems.

While the ACE study demonstrated association between ACEs and later disease and mortality its main drawback was that it was a cross-sectional study interviewing adults at a certain time in their adulthood and asking them to remember facts about their childhood, which may be subject to recall bias. Widom et al (2012) used a longitudinal study that looked at documented cases of child maltreatment (hence eliminating recall bias) and following these individuals into adulthood. As Felitti (1998), they used measured health outcomes through a physical examination, which is more dependable than self-report. They found that after controlling for age, race and gender child maltreatment predicted above normal haemoglobin A1C, which is an indication of poor glycaemic control and poses a risk of diabetes. They also found that specific types of abuse and neglect were responsible for specific types of health outcomes. Physical abuse predicted an increased risk for malnutrition (OR = 2.39, 95% CI = 1.27, 4.49), above normal HbA1C (OR = 2.35, 95% CI = 0.95, 5.79) and an increased risk for above normal C-reactive protein which increases the risk of heart disease (OR = 1.88, 95% CI = 0.88, 3.99). With sexual abuse there were nonsignificant trends for HIV and hepatitis C but a significant increase in the risk of malnutrition (OR = 2.16, 95% CI = 1.02, 4.61). Neglect predicted above normal HbA1c (OR = 1.91; 95% CI = 1.10, 3.33).

Shin and Miller (2012) found a link between childhood maltreatment and obesity. They used data from the National Longitudinal Study of Adolescent Health (N = 8,471) and found

that 'Children who experienced neglect had a faster average rate of BMI growth over time compared to children who experienced no childhood maltreatment'.

A meta-analysis by Norman et al (2012) identified 124 studies that showed a relationship between child maltreatment and increased risk for a range of mental disorders, drug use, suicide attempts, sexually transmitted infections, and risky sexual behaviour.

Finally Afifi et al (2016) find an association between childhood maltreatment and an increased risk of a host of later-life health problems including asthma (OR = 1.1, 95% CI = 0.89, 1.3); arthritis (OR = 1.4, 95% CI = 1.3, 1.6); back problems (OR = 1.6, 95% CI = 1.4, 1.7); high blood pressure (OR = 1.1, 95% CI = 1.0, 1.3); migraine (OR = 1.9, 95% CI = 1.6, 2.2); COPD (OR = 1.3, 95% CI = 1.1, 1.7); diabetes (OR = 1.2, 95% CI = 0.998, 1.5); epilepsy (OR = 0.9, 95% CI = 0.5, 1.6); heart disease (OR = 1.2, 95% CI = 0.97, 1.4); cancer (OR = 1.3, 95% CI = 1.1, 1.5); stroke (OR = 1.4, 95% CI = 1.0, 2.0); bowel disease (OR = 1.8, 95% CI = 1.5, 2.2) and chronic fatigue syndrome (OR = 2.6, 95% CI = 1.7, 3.9). They also found that exposure to more than one type of maltreatment increased the odds of developing disease with the odds ratio of developing any physical condition having had experienced three types of maltreatment at (OR = 2.2, 95% CI = 1.4, 3.3) compared with (OR = 1.3, 95% CI = 1.1, 1.5) for one type of abuse.

As a final point in this section, it is worth making the distinction between ACEs as a deficit model compared to an assets model. Focusing on social issues through the lens of negativity can make it difficult to design policies to address ACEs. ACEs as a deficit model stipulates that individuals need support, therapy, trauma-informed interventions, and specialist provision. It can be argued however that *true* trauma informed philosophies are strength or assets based. According to a blog by Jessica Eaton (2019),

“The trauma-informed approach to trauma and suffering would be to support the human with the reactions, responses and consequences of being traumatised and harmed by others or by an event.”

Suffice to say that the ACEs approach as a predictive model of generally poor health outcomes has been criticised and it is worth bearing this in mind as we proceed with this thesis.

## **1.9 The extent of ACEs in England and Wales**

This section provides an outline of how prevalent ACEs are in our society by looking at each ACE separately and reporting the most recent statistics on their prevalence in England and Wales.

### **1.9.1 Abuse & Neglect**

There are no official figures on the number of cases of child abuse or neglect in England and Wales, however we do have figures on the number of children that have been identified as having support or protection due to abuse or neglect and we present this in the following tables.



**Table 1.1: Unadjusted incidence of children needing support for neglect and abuse in England and Wales 2017 to 2020**

	2017		2018		2019		2020	
	Wales	England	Wales	England	Wales	England	Wales	England
Category of abuse								
Neglect	1180	24,590	1,090	25,820	1,005	25,330	895	26,010
Physical abuse	325	3,950	355	4,120	285	4,170	190	3,820
Sexual abuse	125	2,260	115	2,180	120	2,230	70	1,970
Emotional abuse	1045	17,280	1,275	18,860	1,295	18,460	1,045	18,380
Neglect, physical abuse and sexual abuse	*	n/a	10	n/a	*	n/a	*	n/a
Neglect and physical abuse	95	n/a	80	n/a	65	n/a	75	n/a
Neglect and sexual abuse	20	n/a	30	n/a	30	n/a	25	n/a
Physical abuse and sexual abuse	10	n/a	10	n/a	10	n/a	5	n/a
Multiple		3,010		2,820		2,070		1,330
<b>Total</b>	<b>2,805</b>	<b>51,080</b>	<b>2,960</b>	<b>53,790</b>	<b>2,820</b>	<b>52,260</b>	<b>2,310</b>	<b>51,510</b>

Source: NSPCC (2021) Child protection register statistics England: 2017 – 2021 and NSPCC (2021a) Child protection register statistics Wales: 2016 – 2020

**Table 1.2: Incidence of children needing support for neglect and abuse per 10,000 child population**

	2017		2018		2019		2020	
	Wales	England	Wales	England	Wales	England	Wales	England
Category of abuse								
Neglect	22	24	21	25	19	25	17	25
Physical abuse	6	4	7	4	5	4	4	4
Sexual abuse	2	2	2	2	2	2	1	2
Emotional abuse	20	17	24	19	24	18	20	18
Multiple	2	3	2	3	2	2	2	1
<b>Total</b>	<b>53</b>	<b>51</b>	<b>56</b>	<b>53</b>	<b>53</b>	<b>51</b>	<b>44</b>	<b>50</b>

Source: Table 1.1 adjusted by ONS (2021) Population Estimates for UK

Table 1.1 gives the count of instances of children that need protection due to abuse or neglect by category of abuse and neglect. Obviously, these figures are not comparable due to total population difference between England and Wales so table 1.2 presents the incidence per 10,000 children in each population. We see that the incidence of abuse and neglect is consistently higher in Wales, apart from in 2020, with emotional abuse being the

biggest difference. The difference between the incidence of neglect has though widened between 2017 and 2020 while rates of sexual abuse are broadly similar.

### 1.9.2 Mother treated violently

This ACE's prevalence in the populations of England and Wales can be measured by recourse to figures on domestic violence, which are readily available. According to the Crime Survey of England and Wales (CSEW) in 2021 some 845,734 cases of domestic abuse-related crimes were reported, an increase of 6% on the previous year (ONS 2021). This follows on from previous increases and may reflect improved recording by the police and an increase in the number of cases reported.

### 1.9.3 Household substance abuse

The proportion of adults living in households with children who reported drug misuse in 2020/21 was 9.3% for single adults and 6.0% for cohabiting adults according to the CSEW. This was up from 6.9% and 6.0% respectively in 2014/15 (Home Office 2015 & 2021). In terms of alcohol misuse, let us define this as what the government refer to as 'binge drinking'. This is drinking 8 units or more for males and 6 units or more for females on any given day during the week.

**Table 1.3: Drinking habits, by sex and whether dependent children live in the household, Great Britain, 2017**

All persons aged 16 to 60					Percentages	
	Men		Women		All persons	
	Live with dependent children	Do not live with dependent children	Live with dependent children	Do not live with dependent children	Live with dependent children	Do not live with dependent children
As a proportion of the whole population						
Teetotal	18	18	25	19	22	18
Drank in the last week	64	60	46	57	54	58
Drank on at least five days in the last week	7	9	4	7	5	8
On heaviest drinking day in the last week...						
...exceeded 4/3 units	32	34	27	33	29	34
...exceeded 8/6 units	17	21	14	19	16	20
...exceeded 12/9 units	10	13	8	10	9	12

Source: ONS (2018) Drinking habits, by sex and whether dependent children live in the household, Great Britain, 2017

As seen in table 1.3 16% of those adults that live with dependent children are exposing their children to an ACE by taking part in binge drinking at least one day a week. While this is not the same as chronic alcoholism it can serve as a good proxy as to the prevalence of alcohol misuse among those with children.

#### 1.9.4 Mental illness in household

As we have previously described having a household member who has a mental illness is an ACE. Usually for ACE studies a mental illness is defined as suffering from depression or anxiety and this is the definition we will use here. According to the latest figures in 2015, published by the ONS the prevalence of anxiety and depression, the two main forms of

mental illness, in the UK was 4.5% and 4.2% respectively among those aged over 16 (ONS 2019).

### 1.9.5 Parental separation or divorce

Table 1.4 shows the number of couples that were divorced in England and Wales from 2010 to 2013 – the latest figures available. We see that there has been a steady decline in the incidence of divorce between these periods. Divorce rates may provide an underestimation of this type of ACE as the broader term ‘parental separation’ includes other types of separation. The table also shows the number of children affected by divorce and this has also fallen recently in general and across all age categories.

**Table 1.4: Number of couples divorced and number of children affected by divorce in England and Wales 2010 to 2013**

Year of divorce	number of couples divorced	Number of children aged under 16 by age-group			
		Total	0–4	5–10	11–15
2013	<b>114,720</b>	<b>94,864</b>	19,454	41,461	33,949
2012	<b>118,140</b>	<b>99,822</b>	20,533	43,353	35,936
2011	<b>117,558</b>	<b>100,760</b>	20,907	43,261	36,592
2010	<b>119,589</b>	<b>104,364</b>	21,921	44,635	37,808

Source: ONS (2015) Divorces in England and Wales: Children of Divorced Couples

### 1.9.6 Criminal household member

This ACE relates to any household member who has been incarcerated for any reason so let us present here figures on general incarceration in England and Wales. As of March 2021,

the total prison population in England and Wales was around 78,756 (House of Commons Library 2021). Not all these individuals will have children and no official record exists of children of prisoners as neither the courts, governments, nor local services ask routinely about them. However, research cited by the National Information Centre on Children of Offenders NICCO (2018) estimates that around 310,000 children in England and Wales are affected by parental imprisonment across England and Wales.

## **1.10 Methods used in part 1 (Chapters 2 & 3)**

### **1.10.1 Odds Ratios**

The systematic review in chapter 2 looks at two different areas. The first part of the systematic review looks at the literature on ACEs and diseases and reports odds ratios for the relationship between ACEs and these specific diseases. Odds ratios are a measure of association between exposure and outcome. The ratio is a measure of an outcome occurring given an exposure compared to when that exposure doesn't exist. Odds ratios of less than one can be interpreted as there being less likelihood of the outcome occurring given the exposure. Odds ratios greater than one mean that there is more likelihood of the outcome occurring given the exposure.

### **1.10.2 Cost of illness studies**

Secondly there is a review of articles on the cost of illness of five of the most prevalent diseases, in terms of expenditure, in the UK. According to Hanly et al (2015),

"If undertaken across several diseases, cost-of-illness studies can provide data on the public health needs of the population and help guide the allocation of governmental clinical, social care and research funds." (Hanly et al (2015), p.136)

Cost of illness studies are therefore seen as essential in trying to put a figure on the costs associated with diseases. They are a method used to measure the economic burden of a disease and costs are usually reported as direct health and non-health costs as well as indirect costs. Direct health costs include such items as cost of hospital care, medication, laboratory test and home care. Non health care direct costs can vary significantly across studies but may include such items as transportation and household amendments. Indirect costs are largely productivity losses due to the mortality or morbidity caused by the disease.

Cost of illness studies can be based on prevalence or incidence of the disease in question. The difference is that the prevalence approach considers all cases up to and including the point in time of the analysis and calculates yearly costs while the incidence approach considers all new cases each year and reports lifetime costs. Examples of both approaches have been found in this review.

Another methodological consideration to be aware of is the type of costing used in the study. The top-down approach looks at aggregate healthcare expenditures compartmentalised into the different diseases. A more popular method is the bottom-up approach which looks at healthcare resource use among a sample of patients and costs this according to official unit costs for each resource. (Chapko et al (2009))

The first part of the review looks at the odds ratios of ACEs in terms of how they are associated with the different diseases. Together with the cost of illness information the following chapter (Chapter 3) will use population attributable fraction (PAF) methodology to calculate how much of the cost of a disease can be attributed to ACEs. The following section explains more about the PAF methodology.

### 1.10.2 Attributable fraction methodology

Levin first proposed the idea of a population attributable fraction (PAF) in 1953. He built on work carried out by Doll where a PAF was published in 1951 using figures from his case-control study on smokers in London. Doll was able to attribute lung cancer incidence rates from his study to the incidence of smoking and he was able to “estimate the number of cases that would have been expected to occur if the entire population were non-smokers”. Therefore, in simple terms the method allows the researcher to find out how much of a disease can be prevented if we remove all the risk factors associated with that disease. Levin developed a novel way of deriving PAFs from rate ratios rather than rates and rate differences. As his explanation of how to derive a PAF was somewhat complex the derivation used by Leviton (given in Poole (2015)) a few decades later is given here.  $IR = I_1/I_0$  where  $I_1$  is the rate in the level of the higher rate while  $I_0$  is the rate in the lower level supposing that there is a binary exposure variable – higher exposure and lower exposure.  $I$  is the prevalence rate of the disease in the population and  $p$  is the proportion at risk. We can replace  $I$  with  $H$ ,  $R$  or  $P$ , if the outcome measure is a hazard, risk, or prevalence ratio. The overall rate  $I$  is a weighted average of the exposure specific rates  $I_1$  and  $I_0$  and can be expressed as:

$$I = pI_1 + (1 - p)I_0$$

This can be simplified to:

$$I = p(ID) + I_0$$

where  $ID = I_1 - I_0$  which is the rate difference between the two exposure levels. The population rate difference is:

$$PID = I - I_0 = p(ID)$$

The PAF is the PID expressed as a proportion of  $I$  which is:

$$PAF = \frac{I - I_0}{I}$$

Which can also be written, given that

$$I = p(ID) + I_0$$

as

$$PAF = \frac{p(ID)}{p(ID) + I_0}$$

Finally, if we divide this expression, numerator, and denominator by  $I_0$  we get the formula for PAF:

$$PAF = \frac{P(IR - 1)}{P(IR - 1) + 1}$$

where  $IR = I_1 / I_0$ .

1973 saw a major boost to the profile of the PAF with commentator Lilienfeld (1973) extolling its virtues in the American Public Health Association's First Wade Hampton Frost Lecture. The interpretation on the PAF was made clearer. "...the higher the PAF for a given cause, the higher the priority to be given to studying persons unexposed it in searching for additional causes. Conversely, the lower the PAF for a given cause, the higher the priority that should be accorded to studying exposed persons." (p149). The complement of a PAF is



the remaining percentage once the PAF percentage has been established. It is wrong to think of the complement as “equals the proportion of a population in which other causes are operating” however. Magnus and Beaglehole conducted a study in 2001 on coronary heart disease and suggested a PAF of 50% for smoking, hypertension, and blood lipids. It could be surmised that 50% of the disease was due to other causes and that “important undiscovered risk factors make up this apparent deficit, and that high priority should be given to further social, molecular, or other basic research”. However, they contended that if the PAF was 75% for these risk factors “whether another 25% exists, and how much of it we could expect to explain, are matters for debate.” In this light they argued for a reduced priority for further research for new causes. This can be juxtaposed with Lilienfeld’s argument that “the greater the PAF for a given cause, the greater the priority to be placed on searching for new risk factors among persons unexposed to that cause”. However, this argument runs out of steam, according to Poole, when the PAF reaches 100%. In this case “A more tenable perspective would be that the greater the PAF for a given cause or set of causes, the more advisable it is to look for new causes among exposed persons”

In 1981 Doll and Peto showed that PAF can sum to more than 100% if there is shared causal responsibility, that is, if a disease has more than one cause. Their famous study “The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today” made the case for not falling into the trap of ‘double counting’ and “adding together proportions that are not, in fact, mutually exclusive”. It may be commented upon that most diseases have a limited number of causes, even only one. Poole argues, however, that shared causal responsibility is not an “anomaly” but a “fact of etiologic life”.

To summarise therefore the population attributable fraction (PAF) methodology aims to establish how much of a disease is caused by certain risk factors. In the context of this thesis the amount of diseases caused by ACEs will be investigated and then multiplied by the cost of illness for that particular disease in order to arrive at the attributable cost of diseases that are partly caused by ACEs.

## **1.11 Methods used in Part 2 (Chapters 4 & 5)**

### **1.11.1 Cross sectional data**

The dataset used for Part 2, testing the hypothesis of the relationship between ACEs and social mobility, was supplied by the funder. It is a pooled cross-sectional dataset with n=13,130 and contains information on a number of survey questions asked by market research companies in England, Wales, Blackburn with Darwen and Southern England between 2012 and 2015. The question on wealth in childhood and in adulthood was used to measure social mobility. Respondents were asked to rank their wealth in childhood and adulthood on a Likert scale of one to ten, with one being very poor and ten being very wealthy. This question was asked in the Welsh and Southern English datasets only and so analysis has only been possible in these areas. The dataset also contains data on demographics, health harming behaviour, resource use and the different ACEs and ACE counts.

### 1.11.2 Logistic regression to identify associations

The association between ACEs and social mobility is firstly tested using a logistic regression. This type of regression is suitable when the dependent variable is binary, that is either zero or one. The data is firstly coded to give a binary variable of poor or affluent and then movement between these states is noted. For example, a person poor in childhood and affluent in adulthood will have an upward social mobility value of 1 while all other possible states (affluent to poor, stay poor, stay wealthy) will have a value of 0. This variable is then regressed on *ACE counts* to find out the relationship between ACEs and social mobility. The regression equation is given as:

$$Y_i = \alpha + \beta x_i + \varepsilon$$

Where Y=Social mobility, 1 – Became socially mobile by upward or downward mobility or 0 - stayed at the same level or became the opposite of the mobility in the first instance; and X = number of ACEs, 0,1,2-3,4+

It should be noted that looking at *ACE counts* may circumvent the fact that ACEs themselves are indeed complex. Each ACE might have a different effect and so a simple aggregation where ACEs are expressed as 0,1,2-3,4+ would fail to account for any combination effects and the added complexities emanating from these combinations. Briggs (2021) talks about synergy in this respect,

“...certain pairs of ACEs comprising the cumulative ACE score interact synergistically to significantly increase the overall risk beyond the sum (or product) of the contributions of each ACE to the outcome.” (Briggs et al 2021 p.243)

This idea relates to the recognition that many biological processes are ‘greater than the sum of their parts’ and this holistic view is at odds with the notion of ACEs as simple 0,1,2,3,4+ counts. Briggs (2021) also concedes that,

“The notion of a simple cumulative ACE score... implies that all of the traumatic experiences and aversive environments designated as ACEs make equivalent contributions to an individual’s risk for a given outcome.” (Briggs et al 2021 p.244)

And that, in fact, this is *not* the case. Let’s take these two points in turn. Firstly, producing ACEs counts and performing analysis on them does not consider the synergistic relationships that can occur between ACEs. For example, a child who suffers from two ACEs – physical abuse and a parent who is an alcoholic may suffer differently from another child who also has two ACEs – physical abuse and parents who are divorced. The two ACEs in each example may work together differently to produce different outcomes. This brings us to the second point – where ACEs ‘make equivalent contributions to an individual’s risk’. Each ACE is *not* equivalent in terms of its effect on outcomes and the count method of assigning ACEs does not consider this.

A caveat here to this proposed analysis would therefore be that these results should *not be seen as definitive* and that these limitations should be borne in mind when considering the results.

Interpreting the results involves looking at the odds ratio of the regressions. Odds ratios of less than one means that it is less likely for that social mobility to occur the more ACEs we have, while odds ratios of more than one signifies a higher likelihood of transition. For

example, if the odds ratio was higher than 1 for downward mobility it would mean that as we increase ACEs the likelihood of becoming downwardly socially mobile would increase.

## **1.14 Conclusion**

This introductory chapter has laid out the background to ACEs. It has discussed how, through various mechanisms, ACEs can have a significant effect on later life health outcomes. There is a gap in understanding the impact of ACEs on health and its costs and it has been identified that there is a paucity of literature on the effect of ACEs on social mobility. These two areas form the basis of this thesis. What links them is that socially mobile individuals often achieve higher levels of income and therefore a higher tax income for the government. This money can be reinvested into society to help tackle the causes and consequences of ACEs and identifying the cost of ACEs helps economic evaluation of interventions aimed at reducing the effects of ACEs. The next chapter presents a systematic review on the published evidence of cost of illness of the main diseases partly caused by ACEs and odds ratios (a statistic that gives an idea of the strength of association between two variables) on the attribution of ACEs to these diseases. This information will be the basis of the work carried out in chapter 3, that is to measure the attributable cost of ACEs. Chapter 5 will then bring this thesis together in that, having identified the costs of ACEs in the third chapter, we will then go on to measure what effect having ACEs has on social mobility – one of the main ways of improving society's living standards so that more money and resources are available to tackle the problems caused by ACEs and to spend addressing these costs brought about by ACEs.

## Part 1 – Exploring the costs of the highest expenditure diseases that are attributable to ACEs

## Chapter 2 – Systematic review of the odds ratios and cost of illness of diseases caused by ACEs

### 2.1 Background

Fang et al (2015a) have argued that calculating the economic burden of ACEs is important for several reasons. First it increases the awareness of the severity of ACEs, assists policy makers in funding decisions about developing preventive services to tackle ACEs and provides data for the economic evaluations of interventions aimed at reducing or preventing child maltreatment (Fang et al 2015a). In the context of this thesis it is important to calculate the costs associated with ACEs because it gives an idea of the outlay needed by government to tackle the problems caused by ACEs.

This chapter presents the findings of two PRISMA informed (PRISMA 2009)<sup>3</sup> systematic reviews to explore the link between ACEs and the top five life-course diseases in terms of expenditure: Mental illness, Circulatory disease, Cancer, Musculoskeletal disease and Genitourinary disease and to identify the costs of these diseases and the extent to which these diseases are associated with ACEs. This and the following chapter should be seen as context to the penultimate chapter that deals with the main hypothesis posited by this thesis. Namely that there is a relationship between having ACEs and upward or downward social mobility. The theoretical underpinning has been explored in Chapter 1 but let us reiterate it here. The idea is simple. That we have an increase in taxable income in the economy following a rise in living standards and that these monies can be channelled to

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<sup>3</sup> PRISMA (2020) guidelines were not available at the time of writing

improve outcomes associated with ACEs. To know how much of an outlay is required we have to conduct an exercise such as this one – to ascertain costs associated with ACEs.

## **2.2 Methodology**

This systematic review aims to gather the necessary information to help us put a lifetime cost on ACEs. It is divided into two parts of investigation. The first part of the review investigates odds and relative risk ratios in the way ACEs affect the diseases identified later in this review. The second part aims to gather the evidence base for the cost of illness of the five main diseases in the UK that can be said to be partly caused by ACEs. This will allow the calculation of lifetime costs attributable to different denominations of ACEs. The question of attribution is key here. It should be noted that this is not an exercise in finding the costs of specific diseases per se but rather the proportion of these costs that can be attributed to the presence of one or more ACEs.

The methodology we will be using, in the following chapter, to calculate the attributable cost of ACEs is population attributable fractions (PAF). This was first proposed by Levin (1953) and developed by Miettinen (1974) and others into what is known today as population attributable fractions. The idea is that there is seen to be a causal relationship between risk factors (ACEs) and adverse outcomes (disease). Then we can ask how much of the disease burden in any given population is eliminated if we eliminate the causal factors. That is, how much of the disease would be eliminated if we got rid of ACEs entirely. Put another way it can be thought of as how much of a given disease is caused by ACEs.

“PAF is defined as the fraction of all cases of a particular disease or other adverse condition in a population that is attributable to a specific exposure”



It is then possible to take the costs of a certain disease and calculate how much of that cost is attributable to ACEs by multiplying the PAF with the cost. The formula for PAF is as follows:

$$PAF = \frac{P(RR - 1)}{(1 + P(RR - 1))}$$

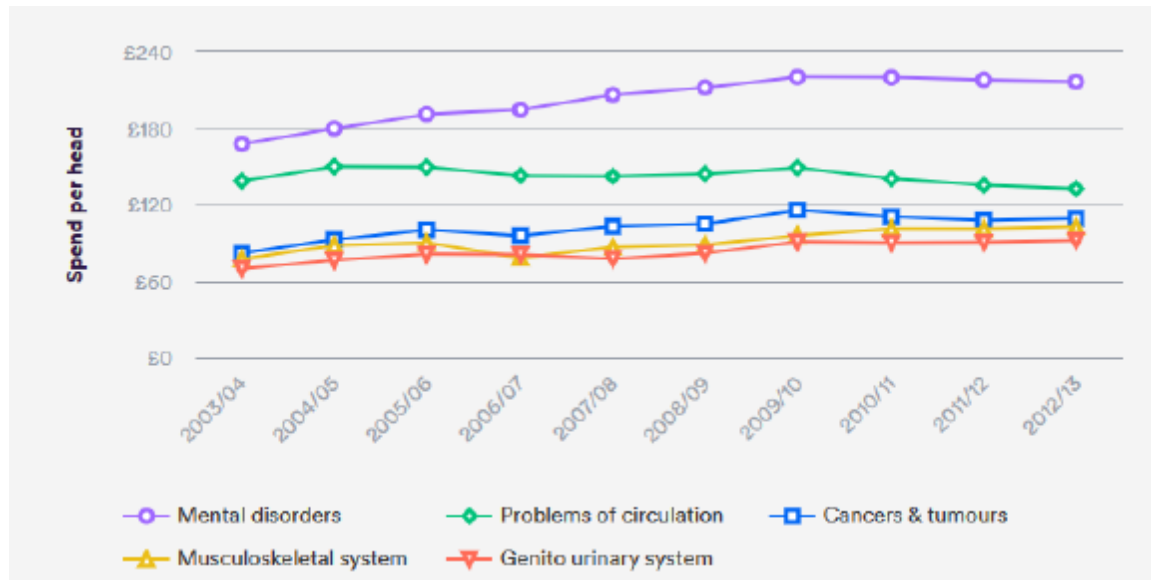
Where P = percentage of ACE endorsed in the sample and RR= relative risk ratio.

Usually in epidemiologic studies the relative risk or odds ratio is used to measure the strength of association between risk factors and outcomes. PAF goes a step further and takes the prevalence of the risk factor into account thus allowing for consideration of the importance of the risk factor. To populate this equation and to get estimates of PAF for different ACE counts we need information on the relative risk of diseases that can be partly attributed to ACEs. Further, we need information on the cost of diseases to calculate the cost that is attributable to ACEs. This is what this review seeks to achieve.

The main objective of this review was to provide evidence of the association between ACEs and the top five diseases in the UK in terms of expenditure. This was done by performing a meta-analysis of odds ratios that linked ACEs with these diseases. Another priority was to determine the lifetime costs associated with the diseases that were partly caused by ACEs.

According to the Nuffield Trust (2014) the main diseases, in terms of expenditure, in the UK are as follows: mental disorders, circulatory disorders, cancer, musculoskeletal disorders and genitourinary disorders.

Figure 2.1: Expenditure on diseases in the UK



Source: Nuffield Trust (2014)

The questions asked in this systematic review are as follows:

- What are the odds ratios of the five disease types that link ACEs with these diseases?
- What are the lifetime costs associated with diseases that are partly caused by ACEs?
- Where do these costs fall (i.e. NHS, society etc)?
- What is the proportion of costs that are direct (i.e. healthcare costs) and indirect (i.e. productivity losses)?
- Is it possible to take costs from international sources and extrapolate them to the UK?
- Is it possible to take costs from one type of disease/condition e.g. cancer and extrapolate them to other types of diseases/conditions to get the total cost of a particular disease/condition?

To address the systematic review questions the Campbell and Cochrane Economics Method Group (CCEMG) (Shemilt et al. 2008) design, methods and processes have been followed.

The systematic review is in two parts. The first part describes odds ratios for diseases partly caused by ACEs by searching for meta-analyses in this area; and the second part describes cost of illness studies for the five main diseases in terms of expenditure.

### 2.2.1 Inclusion and exclusion criteria – Part A of systematic review

Part A of the systematic review includes any study that discusses the odds ratios of how ACEs can partly cause those diseases. It excludes any Randomised Controlled Trials or other trials of interventions to reduce the prevalence of these diseases. Systematic reviews are included as they give an idea of other studies that may have been overlooked in the systematic search. The time frame for the studies considered is not limited as the

relationship between ACEs and the diseases is not considered to have changed much over time. Studies from areas outside Europe and the US are included in this review as the likelihood of ACEs causing these diseases is not considered to be vastly different based on geographical area. Grey literature was also searched including official costing documentation, local authority and charity reports etc. This was to reduce publication bias in the results.

### 2.2.2 Inclusion and exclusion criteria – Part B of systematic review

Part B of the systematic review includes any study that discusses the costs of those diseases identified in this chapter. It does not include any RCTs or other trial interventions to reduce the prevalence of these diseases but merely the economic cost of these diseases.

Systematic reviews are excluded due to the volume of information included in them. The time frame for the studies considered is not limited as cost figures are inflated to reflect current prices. Studies from areas outside Europe and the US are excluded as are those that are not in the English or Welsh languages. Grey literature was also searched as in part A of the review.

### 2.2.3 Types of outcome measures

Any evidence on the relative risk of those diseases due to the three categories of ACEs and the costs of diseases. The following are the outcomes we will consider under each part:

- Part A: Relative risk evidence: risk ratios, odds ratios; diseases/conditions: mental illness, circulatory, cancer, musculoskeletal, genitourinary; ACEs - emotional, physical, and sexual abuse; neglect; household dysfunction.

- Part B: Cost evidence: direct and indirect cost of illness, mental illness, circulatory, cancer, musculoskeletal, genitourinary; year of publication; cost year; methodology; time period; perspective

Figure 2.1 shows the systematic review flow chart. Both parts of the systematic review are guided by the CCEMG (Shemilt et al, 2008). The CCEMG framework has been used to create the search process and terms directly from the objectives. The databases, chosen for their relevance, are JSTOR, PubMed, Embase, Medline, Web of Science and PsycInfo. These databases were chosen due to their broad scope and high likelihood of containing papers related to this systematic search. Psycinfo was included as an important source of knowledge for mental health.

A Bangor University health sciences librarian was consulted to define the search terms, in terms of Medical Subject Heading (MeSH) keywords and to help identify relevant databases. These keywords were grouped, and groups of keywords were linked using Boolean operators (and, not, or). The search terms for part A of the systematic review identified were (Full search terms shown in Appendix 2):

- Adverse childhood experience AND disease type (e.g. cancer)

Search terms for part B were as follows:

- Cost of illness AND disease type (e.g., cancer)
- Burden of illness AND disease type (e.g., cancer)
- Cost\* of disease AND disease type (e.g., cancer)
- Economic burden of disease AND disease type (e.g., cancer)

The disease types of circulatory, musculoskeletal, and genitourinary did not generate any results when written in that form. It was decided then to use proxies to capture these diseases. The proxies used were 'stroke, myocardial infarction and heart disease' for circulatory; 'arthritis' for musculoskeletal and 'renal' for genitourinary.

Ancestral or hand searching was performed by searching the reference list of the chosen manuscripts. A search log was created to keep track of how the searching was conducted – this lists the search terms used and in which database they were used. This can enable the search to be replicated.

#### 2.2.4 Selection of studies

Two researchers independently screened and identified paper title and abstracts for their relevance. After the initial screening those articles considered relevant were obtained. These remaining studies were further scrutinised according to the inclusion/exclusion criteria by the two reviewers so that they were finally included/excluded.

### 2.3 Data Extraction

Data extraction forms designed for this review were used to extract data for inclusion in the results section of the review.

For the first part odds ratios, relative risk ratios, hazard ratios and prevalence ratios are extracted from meta-analyses and displayed in tables. Where papers only report odds ratios these will be converted to relative risk using the following formula (Grant 2014):

$$RR = OR / (1 - p + (p \times OR))$$

Where  $p$  is equal to the risk in the control group. The costs gleaned from part B will then be applied to the population attributable fraction calculated with information from part A to arrive at the costs of these diseases that are attributable to ACEs.

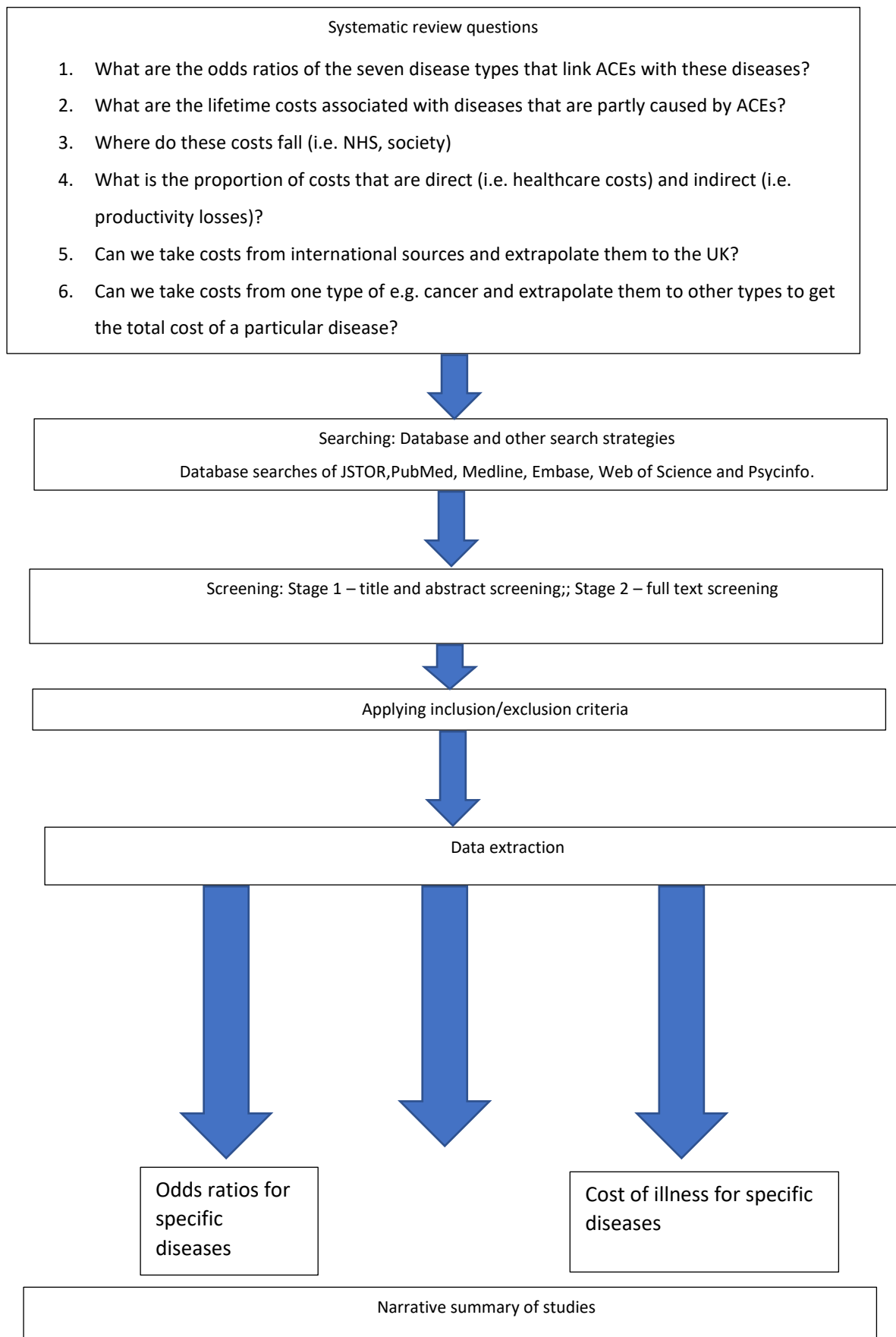
Cost of illness were extracted for part B from relevant studies and put into tables for comparison. The aim was to glean information from different countries and extrapolate to give an idea of what the spending would be in the UK for each disease.

“Extrapolation from one country where data is available to another where no data is available gives an indication of the burden of a disorder in the latter country.”

(Gustavsson et al 2011, p.725)

To this end any international data has been converted into GBP and inflated to 2020 figures.

**Figure 2.2: Systematic review flow chart**





## 2.4 Results – Part A

### 2.4.1 Part A of the systematic review

From the initial systematic search for papers, 5,114 papers were found after removing duplicates. After screening titles and abstracts 76 papers were agreed upon by both reviewers to be included in the full text screening. Following the screening of full texts 51 papers were included for data extraction. This process is shown in a PRISMA flow chart in Figure 2.3 below.

### 2.4.2 Cancer

Fourteen studies were found that reported odds/relative risk/hazard/prevalence ratios of cancer (Alcala et al 2017; Alcala et al 2017b; Amemiya et al 2019; Bellis et al 2014; Brown et al 2010; Felliti et al 1998; Brown et al 2013; Coker et al 2009; Fuller-Thompson et al 2003; Hyland et al 2013; Hu et al 2021; Hughes et al 2017; Morton et al 2012; Pettrucelli et al 2019). Odds ratios are shown for different types of ACEs and for different ACE counts. These range from 0.93 for physical abuse and colorectal cancer (Alcala et al 2017), which means that suffering from physical abuse makes it *less likely* that this will lead to colorectal cancer to 2.4 for sexual abuse and cervical cancer (Coker et al 2009) meaning that sufferers of sexual abuse are *more likely* to develop cervical cancer.

### 2.4.3 Circulatory disease

Seven studies were found for circulatory disease, which included heart disease, stroke and myocardial infarction (Wilson et al 2012; Campbell et al 2016; Dong et al 2004; Fuller-

Thompson et al 2012; Fuller-Thompson et al 2014; Jacquet-Smailovic et al 2021; White et al 2016) Two studies were found for stroke, which reported the odds ratio of emotional abuse affecting the likelihood of stroke at 1.097. (Wilson et al 2012) and an odds ratio of 2.56 for 4 ACEs or more in Campbell et al (2016) although this result was not statistically significant. Four studies discussed the odds ratios of ACEs affecting myocardial infarction ranging from 1.77 for the ACE of family member incarcerated in White et al (2016) to an odds ratio of 4.67 for four or more ACEs in Campbell et al (2016). Two studies gave information on heart disease which included Dong et al (2004) with an odds ratio of 3.6 for 7 to 8 ACEs and Fuller-Thompson (2010) which reported an odds ratio of 1.57 for the effect of physical abuse on heart disease.

#### 2.4.4 Mental health

Twenty-seven studies were included that discussed the odds/relative risk/hazard/prevalence ratios for mental health in terms of the likelihood that ACEs can partly cause certain mental illnesses. (Almeida et al 2011; Almuneef et al 2016; Almuneef et al 2017; Anda et al 2007; Al Shawi et al 2019; Bebbington et al 2004; Bielas et al 2016; Cambron et al 2014; Choi et al 2017; Crouch et al 2017; Fowler et al 2020; Fuller-Tompson et al 2012;; Hughes et al 2016; Hughes et al 2017; Lee et al 2013; Lu et al 2008; Mersky et al 2013; Oladeji et al 2010; Pettrucci et al 2019; Porter et al 2020; Raposo et al 2014; Rhee et al 2019; Roustit et al 2009; Sahle et al 2001; Subica et al 2013; Von Cheong et al 2017; Xiang et al 2020) The odds ratios reported range from 0.92 for the relationship between domestic violence and mental distress (Crouch et al., 2017) to 31.41 for the relationship between emotional abuse and borderline personality disorder (Porter et al., 2020).

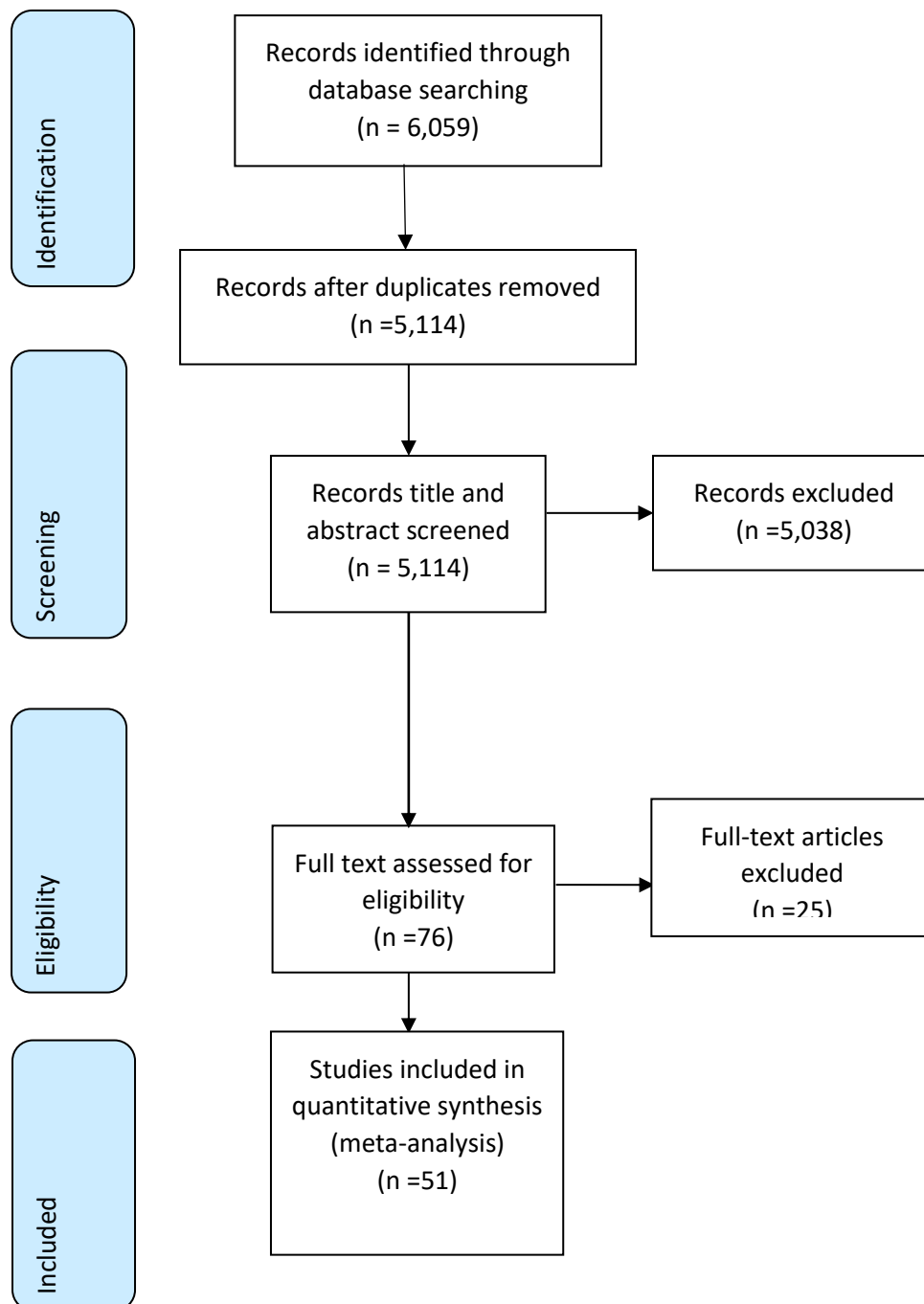
#### 2.4.5 Musculoskeletal disease

Two studies investigated the relationship between musculoskeletal disease, here defined by arthritis, and ACEs. The first (Baiden et al 2021) calculated the odds ratio for the relationship between physical abuse and arthritis to be 1.36 and sexual abuse to be 1.74 while Luiz et al (2018) reported the odds ratio of having four or more ACEs as 1.43.

#### 2.4.6 Genitourinary disease

There were no studies identified in the review that discussed the odds ratios of the relationship between ACEs and genitourinary disease.

Figure 2.3 : PRISMA 2009 Flow Diagram Part A



**Table 2.1: Odds ratios from systematic review**

	Author	Year	Disease	Country	Theme	ACE	Measure	Main outcome
Cancer	Alcala et al	2017	Colorectal cancer	US	Relationship between ACEs and CRC screening	Physical abuse	Odds Ratio	0.93
						Sexual abuse	Odds Ratio	1.18
						Emotional abuse	Odds Ratio	1.09
						Mentally ill	Odds Ratio	1.04
						Problem drinker	Odds Ratio	1.06
						Substance misuse	Odds Ratio	0.95
						Jailed	Odds Ratio	0.99
						Divorced	Odds Ratio	0.99
						Domestic violence	Odds Ratio	0.92
	Alcala et al	2017	Cancer	US	Relationship between ACEs and cancer	Physical abuse	Odds Ratio	1.31
						Sexual abuse	Odds Ratio	1.63
						Emotional abuse	Odds Ratio	1.34
						Mentally ill	Odds Ratio	1.36
						Problem drinker	Odds Ratio	1.22
						Substance misuse	Odds Ratio	1.52
						Divorced	Odds Ratio	1.08
	Amemiya et al	2019	Cancer	Japan	Relationship between ACEs and cancer	Any ACEs	Odds Ratio	1.26
						Number of ACEs	Odds Ratio	1.16
	Bellis et al	2014	Cancer	UK	Relationship between ACEs and 6 diseases inc cancer	All ACEs	Hazard ratio	2.38
	Brown et al	2010	Lung cancer	US	Relationship between ACEs and lung cancer	0	Risk ratio	1.00 (REFERENT )

					1	Risk ratio	0.73
					2	Risk ratio	1.48
					3	Risk ratio	3.10
					4 or 5	Risk ratio	2.55
					6,7,8	Risk ratio	3.18
Felliti et al	1998	Cancer	US	Relationship between ACEs and heart attack, cancer, stroke, COPD and diabetes	0	Odds Ratio	1.00
					1	Odds Ratio	1.20
					2	Odds Ratio	1.20
					3	Odds Ratio	1.00
					4 or more	Odds Ratio	1.90
Brown et al	2013	Cancer	US	Relationship between ACEs and cancer	Component 1	Odds Ratio	1.21
Coker et al	2009	Cervical cancer	US	Relationship between ACEs and cervical cancer	Sexual abuse	Odds Ratio	2.40
Fuller-Thompson et al	2005	Cancer	Canada	Relationship between ACEs and cancer	Physical abuse	Odds Ratio	1.45
					Parental unemployment	Odds Ratio	1.58
Hyland et al	2013	Cancer	Saudi Arabia	Relationship between ACEs and cancer	Physical abuse once a year	Risk ratio	3.60
					Physical abuse every 2/3 days	Risk ratio	3.66
					Psychological abuse once a year	Risk ratio	1.38
					Psychological abuse once every 2/3 days	Risk ratio	4.05
Hu et al	2021	Cancer	China	Relationship between ACEs and cancer	2 or 3 ACEs	Odds Ratio	1.35
					4 or more ACEs	Odds Ratio	2.17
Hughes et al	2017	Cancer	UK	Relationship between ACEs and various conditions including cancer	4 or more ACEs	Odds Ratio	2.31
Morton et al	2012	Cancer	US	Relationship between ACEs and cancer	Frequent psychological and physical abuse	Odds Ratio	Men - 3.558

	Pettrucelli et al	2019	Cancer	Various	Relationship between ACEs and various conditions including cancer	1	Odds Ratio	1.16
						2	Odds Ratio	1.09
						3	Odds Ratio	1.13
						4 or more ACEs	Odds Ratio	0.99
						Frequent psychological and physical abuse	Odds Ratio	Women - 2.184
Mental Health	Almeida et al	2011	Depression	Australia	Relationship between ACEs and depression	Physical abuse	Odds Ratio	3.25
						Sexual abuse	Odds Ratio	2.53
	Almuneef et al	2016	Depression	Kingdom of Saudi Arabia	Relationship between ACEs illnesses including depression	1	Odds Ratio	1.32
						2	Odds Ratio	2.11
						3	Odds Ratio	3.34
						4 or more	Odds Ratio	4.85
	Almuneef et al	2017	Depression	Kingdom of Saudi Arabia	Relationship between ACEs illnesses including depression	4 or more	Odds Ratio	7.00
	Anda et al	2007	Psychotropic medication	US	Relationship between ACE Score and prescriptions for psychotropic medications	0	Risk ratio	1.00 (referent)
						1	Risk ratio	1.3
							Risk ratio	1.6
						3	Risk ratio	1.6
	Al Shawi et al	2019	Depression	Iraq	Relationship between ACEs and depression	Emotional abuse	Odds Ratio	2.29
						Emotional neglect	Odds Ratio	2.78
						Physical abuse	Odds Ratio	1.71
						Physical neglect	Odds Ratio	2.13
						4	Risk ratio	2.3

					5+	Risk ratio	2.9
Bebbington et al	2004	Psychosis	UK	Relationship between ACEs and psychosis	Sexual abuse	Odds Ratio	15.47
					Violence in the home	Odds Ratio	8.97
Bielas et al	2016	Anxiety	Switzerland	Relationship between ACEs and anxiety in a cohort of young offenders	Total ACE score	Odds Ratio	1.68
		Depression		Relationship between ACEs and depression in a cohort of young offenders	Total ACE score	Odds Ratio	2.33
Cambron et al	2014	Anxiety	US	Relationship between ACEs and anxiety	0	Odds Ratio	1.00 (referent)
					1	Odds Ratio	2
					2	Odds Ratio	2.3
					3	Odds Ratio	3
		Bipolar		Relationship between ACEs and bipolar	0	Odds Ratio	1.00 (referent)
					1	Odds Ratio	2.4
					2	Odds Ratio	3.7
					3	Odds Ratio	7.1
Choi et al	2017	Depression	UK	Relationship between ACEs and depression	Psychological abuse	Odds Ratio	1.23
					Physical abuse	Odds Ratio	1.12
					Sexual abuse	Odds Ratio	1.12
					Emotional neglect	Odds Ratio	0.87
					Physical neglect	Odds Ratio	1.27
					Parental substance abuse	Odds Ratio	1
Crouch et al	2017	Mental distress	US	Relationship between ACEs and mental distress	Physical abuse only	Odds Ratio	1.82
					Domestic violence only	Odds Ratio	0.92
					Emotional abuse only	Odds Ratio	1.91
Fowler et al	2020	Depression	Ukraine	Relationship between ACEs and depression	1 or 2 ACEs	Odds Ratio	1.15
					3 or more ACEs	Odds Ratio	1.93



Fuller-Thompson et al	2012	Anxiety	US	Relationship between ACEs and anxiety	Physical abuse	Odds Ratio	1.61
							1.00 (REFERENT)
Hughes et al	2016	Mental illness	UK	Relationship between ACEs and low mental wellbeing	0	Odds Ratio	
					1	Odds Ratio	1.35
					2 to 3	Odds Ratio	1.946
					4+	Odds Ratio	3.856
Hughes et al	2017	Depression	UK	Relationship between ACEs and various conditions including depression	4 or more ACEs	Odds Ratio	4.40
Lee et al	2013	PTSD	US	Relationship between ACEs and mental health	Parental incarceration	Odds Ratio	1.88
		Anxiety				Odds Ratio	1.01
		Depression				Odds Ratio	1.56
Lu et al	2008	PTSD		Relationship between ACEs and mental health	All ACEs	Odds Ratio	1.18
		Psychiatric hospitalisation	US			Odds Ratio	1.12
Mersky et al	2013	Depression	US	Relationship between ACEs and mental health	0	Odds Ratio	1.00 (Referent)
					1	Odds Ratio	1.47
					2	Odds Ratio	2.01
					3 to 4	Odds Ratio	3.56
					5+	Odds Ratio	8.09
		Anxiety			0	Odds Ratio	1 (Referent)
					1	Odds Ratio	0.98
					2	Odds Ratio	2.29
					3 to 4	Odds Ratio	1.77
					5+	Odds Ratio	4.19

Oladeji et al	2010	Anxiety	Nigeria	Relationship between ACEs and mental health	2 or more	Odds Ratio	0.9
Pettrucci et al	2019	Depression	Various	Relationship between ACEs and various conditions including depression	1	Odds Ratio	1.64
					2	Odds Ratio	2.29
					3	Odds Ratio	3.02
					4 or more ACEs	Odds Ratio	4.78
Porter et al	2020	Borderline Personality Disorder	Various	Relationship between ACEs and BPD	Physical abuse	Odds Ratio	6.82
					Emotional abuse	Odds Ratio	31.41
					Sexual abuse	Odds Ratio	6.6
					Physical neglect	Odds Ratio	7.97
					Emotional neglect	Odds Ratio	22.97
Raposo et al	2014	Anxiety	US	Relationship between ACEs and mental health	All ACEs	Odds Ratio	1.48
		Personality disorder				Odds Ratio	2.11
Rhee et al	2019	Psychiatric disorder		Relationship between ACEs and psychiatric disorder	At least 1 ACE	Odds Ratio	2.11
Roustit et al	2009	Depression	France	Relationship between ACEs and various conditions including depression	Witnessing parental violence	Odds Ratio	2.01
					Sexual abuse	Odds Ratio	2.03
Sahle et al	2001	Depression	Various	Relationship between ACEs and mental health	Childhood maltreatment	Odds Ratio	2.02
		Anxiety			Childhood maltreatment	Odds Ratio	1.86
Subica et al	2013	Depression	US	Relationship between ACEs and depression	Physical abuse	Odds Ratio	1.41
					Sexual abuse	Odds Ratio	2.5
Von Cheong et al	2017	Depression	Ireland	Relationship between ACEs and depression	Any ACEs	Odds Ratio	2.85
Xiang et al	2020	Depression	US	Relationship between ACEs and depression	Physical abuse	Hazard Ratio	1.67

Circulatory	Wilson et al	2012	Stroke	US	Relationship between ACEs and myocardial infarction	Emotional neglect	Odds Ratio	1.097
	Campbell et al*	2016	Myocardial Infarction	US	Relationship between ACEs and 6 diseases inc myocardial infarction	0	Odds Ratio	1.00
						1	Odds Ratio	1.07
						2	Odds Ratio	1.12
						3	Odds Ratio	1.78
						4 or more	Odds Ratio	1.86
			Stroke	US		0	Odds Ratio	2.34
						1	Odds Ratio	2.73
						2	Odds Ratio	2.27
						3	Odds Ratio	2.69
						4 or more	Odds Ratio	2.56
	Dong et al	2004	Ischemic Heart Disease	US	Relationship between ACEs and IHD	1	Odds Ratio	1.10
						2	Odds Ratio	1.20
						3	Odds Ratio	1.60
						4	Odds Ratio	1.70
						5 to 6	Odds Ratio	2.00
						7 to 8	Odds Ratio	3.60
	Fuller-Thompson et al	2010	Heart Disease	Canada	Relationship between ACEs and heart disease	Physical abuse	Adjusted Odds Ratio	1.57
	Fuller-Thompson et al	2012	Myocardial Infarction	Canada	Relationship between ACEs and myocardial infarction	Sexual abuse	Odds Ratio	2.96
	Jacquet-Smailovic et al	2021	Myocardial Infarction	Various	Relationship between ACEs and Myocardial Infarction	Cumulative ACEs	Odds Ratio	1.88
	White et al	2016	Myocardial Infarction	US	Relationship between ACEs and Myocardial Infarction	Family member incarcerated	Odds Ratio	1.77

Musculoskel etal								
Baiden et al	2021	Arthritis	US	Relationship between ACEs and arthritis	Physical abuse	Adjusted Risk Ratio	1.36	
					Sexual abuse	Adjusted Risk Ratio	1.74	
Luiz et al	2018	Arthritis	US	Relationship between ACEs and arthritis	4 or more ACEs	Odds Ratio	1.43	

## 2.5 Results – Part B

### 2.5.1 Part B of the systematic review

In the initial systematic search 17,936 papers were found after removing duplicates.

Following the screening of titles and abstracts a total of 246 papers were considered for full text screening of which 76 papers were included for data extraction. This is shown in a PRISMA flow chart in figure 2.4 below. The median cost for all diseases was £17,539 per person per year. Costs have been converted from respective currencies to GBP using exchange rates that prevailed at the cost year of each study. They are then inflated from the cost year to 2020 prices, the latest available data at the time of writing, using the Bank of England inflation calculator. As well as costs reported in terms of per person per year the estimated lifetime costs were also calculated. This was done by multiplying the cost with ten and then the ten-year survival rates for each specific disease. Work will be done in the next chapter to extrapolate from specific disease types to whole diseases. Results have been stratified according to disease and are summarised below.

### 2.5.2 Cancer

Thirty two studies were found that reported costs of cancer (Andreas et al 2013; Babela et al 2020; Brodsky et al 2017; Broekx et al 2011; Cicin et al 2021; Damm et al 2012; Doran et al 2010; Seung et al 2017; Lingren et al 2002; Tilson et al 2008; Bencina et al 2011; Gustavsen et al 2020; Kontoudis et al 2014; Bending et al 2005; Jung et al 2011; Neves et al 2018; Ray et al 2010; Ekwueme et al 2016; Pettrucci et al 2008; Ekwueme et al 2011; Fourcade et al 2010; Tingstedt et al 2007; Hao et al 2016; Leal et al 2016; Sorensen et al 2007; Stokes et al

2008; Gerace et al 2017; Geenen et al 2017; Vallejo-Torres et al 2008; Yue et al 2020; Verleger et al 2018; Tinghog et al 2008). The highest annual cost was in the US for bladder cancer at £711,873 per person per year (Jung et al 2011) while the lowest cost was for prostate cancer, in Sweden, at £1,458 per person per year (Hao et al 2016).

### 2.5.3 Circulatory disease

Twenty-two studies looked at the costs of circulatory disease, defined here as heart disease, stroke and myocardial infarction. Costs for heart failure range from £1,904 to £84,998 per person per year (Biermann et al 2009; Czech et al 2013; Pavlusova et al 2018; Delgado et al 2014; Bungaard et al 2016; Stalhammar et al 2012; Baustein et al 2012 Kruse et al 2008). Only direct costs were reported in these studies (except Bungaard et al 2016) and these total costs would be higher if indirect costs were included.

For stroke fourteen studies were identified reporting costs that range from £1,629 to £149,087 (Alvarez-Sabine et al 2017; Asil et al 2008; Bottachi et al 2012; Girota et al 2016; Gloede et al 2020; Godwin et al 2010; Chinthammit et al 2012; Patel et al 2015; Jakobsen et al 2012; Demaerschalk 2012; Snozzi et al 2005; Lopez-Bastida et al 2012; Saka et al 2009; Smith et al 2012).

Only one study was found for the costs of myocardial infarction. It was produced by Baustein et al (2012) and reported an annual cost per person of £200,054

### 2.5.4 Mental health

Sixteen studies (Bode et al 2017; Sobocki et al 2007; Ekman et al 2013a; Ekman et al 2013b; Ekman et al 2013c; Neil et al 2014; Gustavsson et al 2011; Soetman et al 2008; Rovira et al

2012; Tanner et al 2020; Chevreul et al 2013; Pletscher et al 2014; Wagner et al 2014; Cai et al 2016; Pugliatti et al 2008) were included that discussed the costs of mental illness and psychotic disorders had the greatest per patient yearly cost at £87,018 (Neil et al 2010). The lowest cost was for brain disorders in Italy with a cost of £709 (Pugliatti et al 2008). However, it must be noted that this is a cost per citizen rather than the cost per case of brain disorder.

#### 2.5.5 Musculoskeletal disease

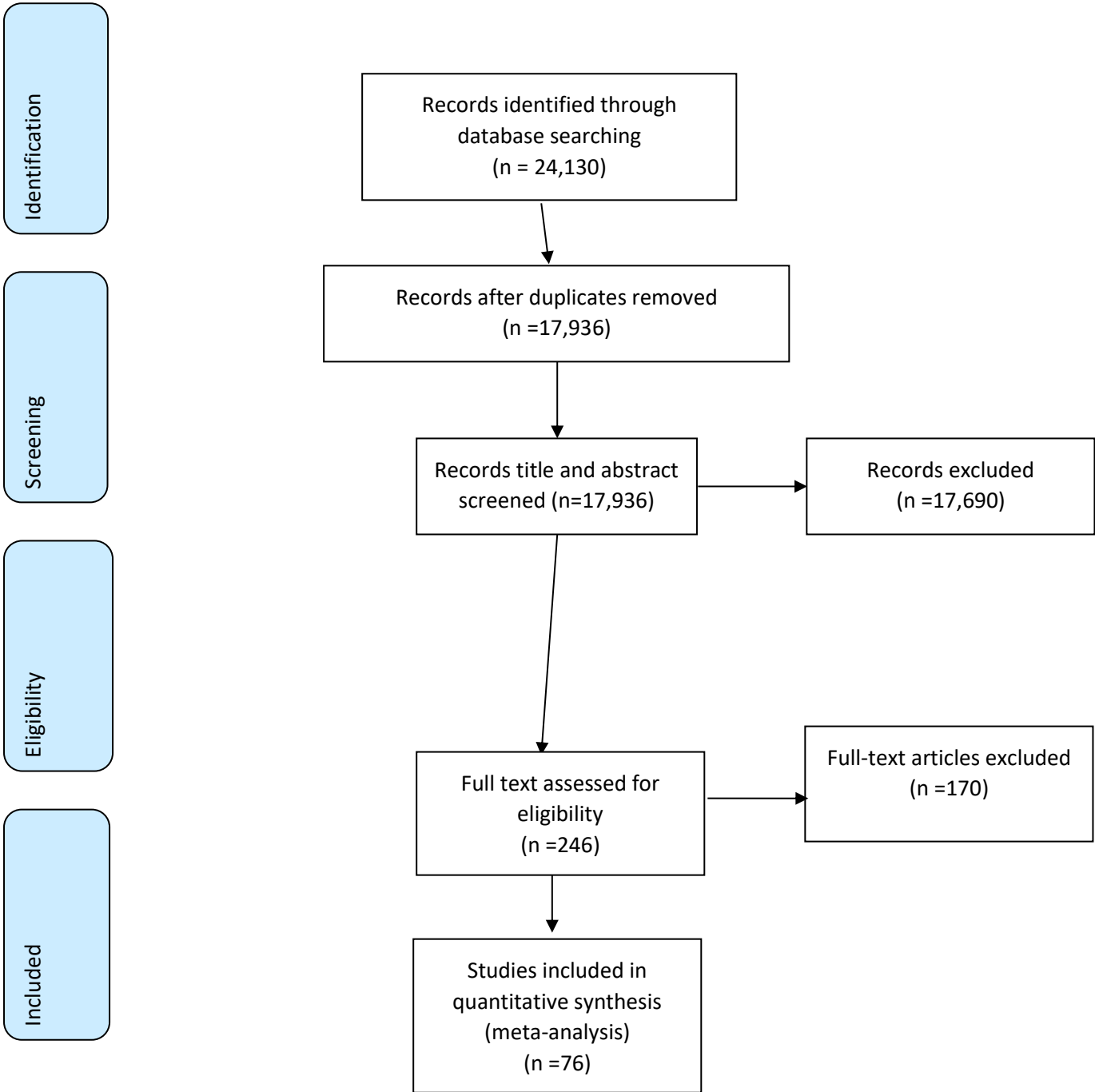
Six studies were found for the costs of arthritis. The costs ranged from £3,000 per patient per year for rheumatic disorders (van den Akker-van Marle 2012) to £33,508 for arthritis (Roodenrijs et al 2019).

#### 2.5.6 Genitourinary disease

No studies were found for the costs of genitourinary disease.



Figure 2.4: PRISMA 2009 Flow Diagram Part B





**Table 2.2: Cost of disease estimates from systematic review**

	Author	Cost Year	Disease	Currency	Country	Direct costs per person	Indirect costs per person	Costs per person	£	Inflated to 2020 (£)	Perspective	Lifetime Costs (£)
Cancer	Andreas et al	2013	Lung cancer	Euro	UK	8,377	1,414	9,791	8,314	9,744	n/a	9,744
	Babela et al	2020	Multiple Myeloma	Euro	Slovakia	155,645	127,611	283,256	252,060	252,060	Regulatory	2,192,922
	Brodsky et al	2017	Prostate cancer	Euro	Hungary	4,448	n/a	4,448	3,899	4,195	Payer	32,721
	Broekx et al	2011	Breast cancer	Euro	Belgium	12,037	95,419	107,456	70,814	127,434	Societal	968,498
	Cicin et al	2021	Lung cancer	Euro	Turkey	10,167	n/a	10,167	8,757	8,757	Payer	68,305
	Damm et al	2012	Colorectal cancer	Euro	Germany	8,750	n/a	8,750	7,098	8,572	n/a	66,862
			Breast cancer	Euro	Germany	4,300	n/a	4,300	3,488	4,212	n/a	32,011
			Prostate cancer	Euro	Germany	4,750	n/a	4,750	3,853	4,653	n/a	36,293
	Doran et al	2010	Melanoma	Australian Dollars	Australia	44,796	n/a	44,796	36,339	47,650	n/a	414,555
	Lidgren et al	2002	Breast cancer	SEK	Sweden	135,135	317,077	452,212	31,022	48,213	n/a	366,419
	Tilson et al	2008	Colorectal cancer	Euro	Ireland	39,607	n/a	39,607	31,531	8,038	n/a	45,817
				Canadian Dollars	Canada	76,816	n/a	76,816	45,989	49,476	Payer	
	Seung et al	2017	Lung cancer	Dollars	Canada	76,816	n/a	76,816	45,989	49,476	n/a	49,476
	Bencina et al	2011	Melanoma	Euro	Croatia	88-4333	n/a	2,123	1,843	2,146	Budget Holder	18,670
	Gustavsen et al	2020	Prostate cancer	US Dollars	US	188,928	n/a	188,928	167,198	174,062	n/a	154,915
	Kontoudis et al	2014	Melanoma	GBP	UK	31,123	1,427	32,550	32,550	37,268	Healthcare Provider	331,685
	Bending et al	2005	Colorectal cancer	GBP	England	20,117	n/a	20,117	20,117	28,698	n/a	163,579
	Jung et al	2011	Bladder cancer	US Dollars	US	658,055	n/a	658,055	571,148	711,873	n/a	327,462
	Neves et al	2018	Multiple Myeloma	Euro	Portugal	31,449	n/a	31,449	27,832	28,975	NHS	252,083
	Ray et al	2010	Melanoma	US Dollars	US	281,112	n/a	281,112	182,017	238,672	n/a	2,076,446
			Breast cancer	US Dollars	US	236,496	n/a	236,496	153,129	200,792	n/a	1,526,019
			Lung cancer	US Dollars	US	204,084	n/a	204,084	132,143	173,274	n/a	173,274
	Ekwueme et al	2016	Breast cancer	USD	US	n/a	2,293	2,293	1,467	1,606	n/a	12,206
	Petrucchi et al	2008	Multiple Myeloma	Euro	Italy	19,267	n/a	19,267	15,338	20,928	Hospital	182,074
	Ekwueme et al	2011	Cancer	USD	US	16,503	7,752	24,255	15,125	17,611	n/a	88,055

Circulatory Heart	Fourcade et al	2010	Prostate cancer	Euro	UK	3,682	n/a	3,682	2,931	3,736	Healthcare Payer	29,141
	Tingstedt et al	2007	Pancreatic Cancer	Euro	Sweden	16,066	n/a	16,066	10,997	15,604	n/a	7,802
	Hao et al	2016	Prostate cancer	Euro	Sweden	1,510	271	1,781	1,458	1,625	Societal	12,675
	Leal et al	2016	Bladder cancer	Euro	UK	32,615	29,313	61,928	50,236	56,673	n/a	283,365
			Cancer			19,209	38,585	57,793	46,882	52,890	n/a	264,450
	Sorensen et al	2007	Breast cancer	US Dollars	US	75,415	n/a	75,415	51,622	73,246	n/a	556,670
	Stokes et al	2008	Pancreatic Cancer	US Dollars	US	34,432	n/a	34,432	27,410	37,400	Payer	18,700
	Gerace et al	2017	Bladder cancer	Euro	Italy Netherlands	3,591	n/a	3,591	3,148	3,387	Societal	15,580
	Geenen et al	2017	Prostate cancer	Euro		17,931	n/a	17,931	14,461	16,557	n/a	129,145
	Vallejo-Torres et al	2008	Melanoma	GBP	England	2,607	n/a	2,607	2,607	3,557	n/a	30,946
	Yue et al	2020	Ovarian Cancer	US Dollars	US	13,566	n/a	13,566	8,239	9,433	n/a	33,016
			Uterine Cancer	US Dollars	US	6,852	n/a	6,852	4,162	4,765	n/a	34,308
			Cervical Cancer	US Dollars	US	2,312	n/a	2,312	1,404	1,607	n/a	8,196
	Verleger et al	2018	Lung cancer	GBP	England	17,761	n/a	17,761	17,761	18,490	n/a	18,490
	Tinghog et al	2008	Skin cancer	Euro	Sweden	2,074	1,636	3,710	2,545	362	Societal	3,149
	Biermann et al	2009	Heart failure	Euro	Germany	3,150	n/a	3,150	2,809	3,600	Societal Public payer	10,620
	Czech et al	2013	Heart failure	PLN	Poland Czech Republic	7,739	n/a	7,739	1,635	1,904	Healthcare System	5,617
	Pavlusova et al	2018	Heart failure	CZK		85,414	n/a	85,414	72,525	84,998		250,744
	Delgado et al	2014	Chronic heart failure	Euro	Spain	18,220	n/a	18,220	15,642	19,161	Societal	56,525
	Bungaard et al	2016	Heart failure	Euro	Denmark	11,926	5,113	17,039	13,952	15,548	n/a	45,867
	Stalhammar et al	2012	Heart failure	SEK	Sweden Czech Republic	72,613	n/a	72,613	58,904	71,133	n/#	209,842
	Baustein et al	2012	Myocardial Infarction	CZK		204,217	n/a	204,217	165,662	200,054	n/a	600,162
	Kruse et al	2008	Heart disease	Euro	Denmark	3,195	n/a	3,195	2,187	3,734	n/a	11,015
	Alvarez-Sabine et al	2017	Stroke	Euro	Spain	27,134	276	27,410	22,235	25,084	Societal	80,269
	Asil et al	2008	Stroke	US Dollars	Turkey	1,677	n/a	1,677	1,148	1,629	n/a Healthcare System	5,213
	Bottachi et al	2012	Stroke	Euro	Italy	9,044	n/a	9,044	7,337	8,860		28,352
	Girotra et al	2016	Stroke	US Dollars	US	4,317	n/a	4,317	3,197	3,563	n/a	11,402

Stroke	Gloede et al	2020	Stroke	US Dollars	Australia	5,207	n/a	5,207	4,059	4,059	Societal	12,989
	Godwiin et al	2010	Stroke	US Dollars	US	34,162	n/a	34,162	22,120	22,120	Insurer	70,784
	Chinthammit et al	2012	Stroke	US Dollars	US	18,796	n/a	18,796	11,863	14,326	n/a	45,843
	Patel et al	2015	Stroke	GBP	UK	45,409	n/a	45,409	45,409	51,481	Societal	164,739
	Jakobsen et al	2012	Stroke	US Dollars	Denmark	19,989	n/a	19,989	12,616	15,235	Societal	48,752
	Demaerschalk	2012	Stroke	US Dollars	US	74,353	n/a	74,353	46,928	56,670	n/a	181,344
	Lopez-Bastida et al	2004	Stroke	Euro	Canary Islands	15,691	1,926	17,617	11,957	17,539	Societal	56,125
	Snozzi et al	2005	Stroke	Euro	Switzerland	40,090	n/a	40,090	27,445	41,909	n/a	134,109
	Saka et al	2009	Stroke	GBP	UK	79,428	25,083	104,511	104,511	149,087	Societal	477,078
	Smith et al	2012	Stroke and transient ischemic attack	Euro	Ireland	12,186	5,065	17,251	11,808	15,652	Societal	50,086
Musculoskeletal	Roodenrijs et al	2019	Arthritis	Euro	Netherlands	37,605	n/a	37,605	33,013	33,508	n/a	268,064
	Klimes et al	2013	Arthritis	Euro	Czech Republic	8,968	2,307	11,275	9,574	11,221	n/a	89,768
	Eriksson et al	2015	Arthritis	Euro	Sweden	23,147	n/a	23,147	19,873	26,059	n/a	208,472
	Hamuryudan et al	2016	Arthritis	Euro	Turkey	4,954	2,802	7,756	6,732	8,391	n/a	67,128
	van den Akker-van Marle	2012	Rheumatic Disorders	Euro	Netherlands	2,665	n/a	2,665	2,288	3,000	Societal	24,000
	Turchetti et al	2013	Arthritis	Euro	Italy	13,595	n/a	13,595	11,800	14,707	n/a	117,656
Mental Health	Bode et al	2017	Borderline Personality Disorder	Euro	Germany	8,508	n/a	8,508	7,384	9,203	n/a	29,450
	Sobocki et al	2007	Depression	Euro	Sweden	1,900	3,600	5,500	3,765	5,371	Societal Primary Care	32,226
	Ekman et al	2013	Depression	Euro	Sweden	21,500	n/a	21,500	17,115	23,353		142,453
	Ekman et al	2013	Bipolar	Euro	Sweden	21,008	7,003	28,011	22,300	28,426	Societal	90,963
	Ekman et al	2013	Schizophrenia	Euro	Sweden	55,100	n/a	55,100	43,863	59,850	n/a	191,520
	Evensen et al	2012	Schizophrenia	US Dollars	Norway	106,000	n/a	106,000	66,902	80,791	n/a	258,531
	Neil et al	2014	Psychotic disorders	US Dollars	Australia	36,356	40,941	77,297	66,362	87,018	Societal	278,458
	Gustavsson et al	2011	Anxiety	Euro	UK	n/a	n/a	1,426	1,224	1,499	Societal	4,797
			Personality disorders			n/a	n/a	9,613	8,253	10,110	Societal	32,352
			Psychotic disorders			n/a	n/a	28,487	24,456	29,957	Societal	95,862

Soeteman et al	2008	Personality disorders	Euro	Netherlands	7,399	3,727	11,126	7,617	10,865	Societal	34,768
Rovira et al	2012	Anxiety	Euro	Spain	1,329	3,810	5,139	3,504	5,185	Societal	16,592
Tanner et al	2020	Depression	Canadian Dollars	Canada	8,244	n/a	8,244	12,152	19,080	Public Payer	116,388
Chevreur et al	2013	Mental disorders	Euro	France	1,642	2,033	3,675	2,516	3,334	Societal	10,669
Pletscher et al	2014	Schizophrenia	Euro	Switzerland	14,300	25,108	39,408	31,968	38,605	Societal	123,536
Wagner et al	2014	Personality disorders	Euro	Germany	28,026	n/a	28,026	24,060	31,549	n/a	100,957
Cai et al	2016	Depression	US Dollars	US	19,626	n/a	19,626	16,070	17,908	n/a	109,239
Pugliatti et al <sup>1</sup>	2008	Brain disorders	Euro	Italy	297	409	706	483	709	Societal	2,269

## 2.6 Discussion

This chapter reports the results of a systematic search for studies that consider the link between certain ACEs and subsequent disease and report this link in terms of a risk or odds ratio. This was done by searching for relevant studies in the different disease fields identified. In addition to this an estimate of the costs of diseases is also identified. The odds ratios varied in terms of how certain ACEs affect the likelihood of developing certain diseases. The highest odds ratio was 31.41 for the relationship between emotional abuse and borderline personality disorder (BPD), which provides evidence of a strong relationship and that the prevalence of BPD could be substantially lowered if effective interventions to tackle emotional abuse were successful. There were some odds ratios that were less than 1 which indicates that having that particular ACE reduces the odds of developing the disease. This was the case for domestic violence affecting mental distress at 0.92 (Crouch et al 2017), domestic violence affecting lung cancer at 0.92 (Alcala et al 2017) and having 1 ACE affecting lung cancer at 0.73 (Brown et al 2010). This may suggest that interventions aimed at reducing domestic violence, for example, may not lead to reductions in the disease prevalence. There is no clear association. However, it must be borne in mind that odds ratios do not necessarily infer causality and so it is not possible to say that for odds ratios less than one that ACEs protect against these diseases.

In terms of costs the evidence is as varied. The median lifetime cost per annum associated with having these diseases was £13,259 per person per year. Where costs for certain diseases from certain countries seem low it can usually be attributed to the fact that only direct hospital costs or indirect costs such as loss of productivity have been measured and not both types of costs.

The information gathered in this review will in theory then enable the calculation of the population attributable fractions (PAF) of certain ACEs as they relate to the disease in question. These PAFs, when applied to the total cost of diseases, will then allow, using the costs identified in this review, the estimation of the lifetime costs associated with ACEs which will be reported in the next chapter.

## **2.7 Conclusion**

Cost information is essential for conducting economic evaluations of health interventions to reduce the prevalence of ACEs. The aim of the next chapter is to describe the costs that are attributable to ACEs of the diseases identified here. It was decided that specific diseases should be investigated as they come from the 'biological embedding' described in chapter 1 or direct impacts of ACEs on physical health in adulthood. Diseases were looked at rather than health harming behaviour because there is an established literature on the topic of health harming behaviour (Bellis et al 2014). This review has provided the available published information on odds ratios and costs of diseases as they relate to ACEs.

However, there was some surprise as to the paucity of evidence especially in terms of the cost and the odds ratios for genitourinary disease. Further research is warranted as the search strategy will not have identified all relevant studies for each health condition. To do this would require very detailed search strategies for each health condition, and that is beyond the scope of this thesis. However, a more detailed search strategy may identify more cost of illness studies, and would allow us to account for differences in the costing methodologies used. Indeed, this may have a significant impact on the costs attributed to

ACEs. As mentioned, the next chapter uses the information in this systematic review to calculate the attributable costs of ACEs.

## Chapter 3 – The lifetime costs of ACEs to society

### 3.1 Introduction

The main purpose of this chapter is to find out if it is possible to put a financial cost to society on what we currently understand as adverse childhood experiences (ACEs).

The costs of ACEs, both in terms of the human cost to children of adversity per se, and costs to society are substantial. All types of ACEs have an effect on children as outlined in chapter 1 and the cost in terms of psychological scarring of surviving such things as abuse and neglect is substantial but not as amenable to measurement. Although outside the scope of this thesis this identification and measurement of direct costs to children of ACEs may be a topic for further research. It is more realistic to put financial costs on diseases that can be caused by ACEs. Chapter 2 presents the results of a systematic review aimed at gathering cost data for the five main disease types in the UK, in terms of expenditure, and odds or relative risk ratios for the link between these diseases and different ACEs. This chapter will use this information to arrive at an attributable cost for different ACE counts. The population attributable fraction method (PAF) is employed in order arrive at costs that are attributable to ACEs. Before proceeding it is worth referring to work done by Hughes et al (2020). They attempted the same kind of calculations and proceeded to measure the attributable costs of diseases that were partly caused by ACEs by using the PAF method together with using DALYs (disability adjusted life years) to estimate costs. My analysis could be seen to complement this work and offer a different method to look at the problem.



### 3.2 Methodology

First it is necessary to say what has been done to the cost information in the systematic review to give an estimate of lifetime costs associated with each disease. Survival rates from various sources were used. Firstly, the cost per year was multiplied by a factor of 10 as this is the length of time associated with the survival rates. Secondly, the 10-year figure was multiplied by the 10-year survival rate for each type of disease.

In accordance with Fang's (2015a) methodology this thesis will use population attributable fractions to calculate the costs associated with ACEs. Fang et al (2015a) use an incidence-based approach to estimate the lifetime cost per child maltreatment victim and aggregate lifetime costs for new child maltreatment cases in 2008.

Estimates of economic burden can take one of two approaches – prevalence and incidence-based. Prevalence-based approaches measures the direct and indirect costs that take place each year regardless of the onset of maltreatment. Prevalence refers to the number of cases of a given disease at a given point in time. Incidence however refers to the number of new cases in a given time period (usually 1 year). Incidence costs are more difficult to estimate as they need data on long- and short-term costs and consequences of ACEs. However, the incidence approach is more useful in economic evaluation of prevention initiatives to reduce ACEs as it considers lifetime costs.

The first objective is to measure the Population Attributable Fractions for ACEs and apply to the costs of different diseases. The formula for calculating population attributable fractions (Afifi 2008) is:

$$PAF = (P(RR-1))/(1+P(RR-1))$$

Where P = percentage of ACE endorsed in the sample and RR= relative risk ratio.

It is the proportion of the outcome that would be reduced if the exposure to that ACE was eliminated.

It is necessary to convert odds ratios to relative risk ratios and this can be done by accounting for the reference or zero ACEs group. Where papers only report odds ratios these will be converted to relative risk using the following formula (Grant 2014):

$$RR = OR / (1 - p + (p \times OR))$$

Where  $p$  is equal to the risk in the control group. It should be noted that only ACE *counts* can be converted into relative risk because we need a control group, which is usually zero ACEs and therefore the odds ratios for *specific* ACEs, such as neglect, cannot be converted in this way due to a lack of a control group.

### 3.2.1 Extrapolation

The estimates for relative risk and costs obtained from the two parts of the systematic review were incomplete and these gaps in the data need to be filled to do any meaningful analysis. This thesis proposes a novel approach to achieve this which can be broadly defined as performing an extrapolation of the data that *is* available or a *pro-rata* approach. For cancer, for example, we have cost information on twelve types, namely breast, lung, multiple myeloma, melanoma, skin, pancreatic, ovarian, uterine, cervical, colorectal, prostate and bladder cancer. A paper published by JAMA Oncology (2019) outlines the incidence of different types of cancer in the year 2017 according to the Global Burden of Disease so we can work out the percentage of each type of cancer's burden.

**Table 3.1: Lifetime costs for different types of cancer**

	%	Lifetime Costs
All Neoplasms	100.00	
Lip and oral cavity	1.59	.
Nasopharynx	0.45	.
Other pharynx	0.73	.
Oesophageal	1.93	.
Stomach	4.99	.
Colorectal	7.69	92,086
Liver	3.89	.
Gallbladder and biliary tract	0.86	.
Pancreatic	1.83	13,251
Larynx	0.86	.
Tracheal, bronchus and lung	8.83	63,858
Malignant skin melanoma	1.26	635,154
Non-melanoma skin cancer	31.29	3,149
Breast	8.01	576,970
Cervical	2.45	8,196
Uterine	1.66	34,308
Ovarian	1.17	33,016
Prostate	5.45	65,815
Testicular	0.29	.
Kidney	1.60	.
Bladder	1.94	305,413
Brain and nervous system	1.65	.
Thyroid	1.04	.
Mesothelioma	0.14	.
Hodgkinson's Lymphoma	0.41	.
Non-Hodgkinson Lymphoma	1.99	.
Multiple Myeloma	0.62	875,693
Other	2.92	.
Acute lymphoid	0.44	.
Chronic lymphoid	0.47	.
Acute myeloid	0.57	.
Chronic myeloid	0.16	.
Other	1.00	.

Part B of the systematic review provided the lifetime costs for breast, colorectal, prostate, bladder, melanoma, multiple myeloma, ovarian, cervical, uterine, pancreatic, lung and skin

cancer<sup>4</sup>. The total cost from the data that *is* available is £2,706,909 and this represents 72.2% of the cost. A 100% of the cost would therefore be:

$$\frac{2706909}{0.722} = 3,749,182$$

This can be inserted into the table above and costs for other cancers can be calculated by applying the incidence percentage figures to this total as follows:

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<sup>4</sup> Breast, pancreatic, bladder, lung multiple myeloma, melanoma ,prostate, colorectal, prostate cancer had costs from different studies – a simple average was taken

**Table 3.2: Extrapolated lifetime costs for different types of cancer**

	%	Lifetime Costs
All Neoplasms	100.00	
Lip and oral cavity	1.59	59,703
Nasopharynx	0.45	16,839
Other pharynx	0.73	27,402
Oesophageal	1.93	72,409
Stomach	4.99	186,916
Colorectal	7.69	92,086
Liver	3.89	145,889
Gallbladder and biliary tract	0.86	32,301
Pancreatic	1.83	13,251
Larynx	0.86	32,301
Tracheal, bronchus and lung	8.83	63,858
Malignant skin melanoma	1.26	635,154
Non-melanoma skin cancer	31.29	3,149
Breast	8.01	576,970
Cervical	2.45	8,196
Uterine	1.66	34,308
Ovarian	1.17	33,016
Prostate	5.45	65,815
Testicular	0.29	10,869
Kidney	1.60	60,162
Bladder	1.94	305,413
Brain and nervous system	1.65	61,999
Thyroid	1.04	39,036
Mesothelioma	0.14	5,358
Hodgkinson's Lymphoma	0.41	15,461
Non-Hodgkinson Lymphoma	1.99	74,705
Multiple Myeloma	0.62	875,693
Other	2.92	109,608
Acute lymphoid	0.44	16,533
Chronic lymphoid	0.47	17,452
Acute myeloid	0.57	21,432
Chronic myeloid	0.16	6,123
Other	1.00	37,659
Total		3,757,066 <sup>5</sup>
Average		113,850

<sup>5</sup> Total cost varies due to rounding

A simple average was then taken to give an estimate of lifetime costs for all cancers per individual. Thus, the average lifetime cost of cancer, per person can be said to equal £113,850.

The same exercise was done for mental health. The ten mental health problems identified in a report for the Global Burden of Disease Study (2022) can be categorised as follows: depressive disorders, anxiety disorders, bipolar disorder, schizophrenia, autism spectrum disorders, conduct disorder, attention-deficit hyperactivity disorder, eating disorders, idiopathic developmental intellectual disability, and a residual category of other mental disorders. The prevalence and those costs that *are* available from the systematic review are presented in table 3.3 below:

**Table 3.3: Lifetime costs for different types of mental illness**

	Incidence %	Lifetime Costs
Anxiety Disorders	29.1	10,694
Depressive Disorders	27.0	100,007
Other Mental Disorders	11.3	.
Idiopathic developmental intellectual disability	10.4	.
Attention-deficit hyperactivity disorder	8.2	.
Conduct Disorder	3.9	.
Bipolar Disorder	3.8	90,963
Autism Spectrum Disorder	2.7	.
Schizophrenia	2.3	191,196
Eating Disorders	1.3	.

To fill in the gaps in terms of costs the following methodology was employed. First, we totalled the costs available (£392,896<sup>6</sup>) and calculated the prevalence for these conditions (62%<sup>7</sup>). If £392,896 was 62% of the total, then the total would be:

<sup>6</sup> 36,164+32,237+92,083

<sup>7</sup> 15.1+0.3+0.4

$$\frac{392,896}{0.62} = \text{£}631,650$$

Costs for the other types of mental illness can be calculated by applying the prevalence percentage figures to this total as follows:

**Table 3.4: Extrapolated lifetime costs for different types of mental illness**

	Incidence %	Lifetime Costs
Anxiety Disorders	29.1	10,694
Depressive Disorders	27.0	100,007
Other Mental Disorders	11.3	71,485
Idiopathic developmental intellectual disability	10.4	65,629
Attention-deficit hyperactivity disorder	8.2	51,662
Conduct Disorder	3.9	24,458
Bipolar Disorder	3.8	90,963
Autism Spectrum Disorder	2.7	17,261
Schizophrenia	2.3	191,196
Eating Disorders	1.3	8,295

The average lifetime cost of mental illness was calculated as £63,165. The next disease category is circulatory diseases. These can be broken down to the following diseases as identified in the systematic review: myocardial infarction, heart disease and stroke. The extrapolation method used is not required here as we have costs for all these diseases

identified in the systematic review. It only remains therefore to list the cost and take an average to calculate the costs associated with circulatory disease:

**Table 3.5: Lifetime costs for different types of circulatory disease**

	Lifetime Costs (£)
Ischemic Heart Disease	88,951
Stroke	101,308
Myocardial Infarction	600,162

The average cost for circulatory diseases is calculated as £263,474.

Lastly there is musculoskeletal disease which we have defined for our purposes as arthritis.

The average lifetime cost of arthritis is calculated as £129,181. The genitourinary category of disease did not return any results in the systematic review and so will not be considered further.

Looking forward to the next section therefore the costs to be used in the PAF calculation are shown in the next final table:

**Table 3.6: Costs to be used in PAF calculations**

	Average Lifetime Cost (£)
Cancer	113,850
Mental Health	63,165
Circulatory	263,474
Musculoskeletal	129,181



## 3.3 Results

### 3.3.1 Cancer

If we take all cancers, we can apply the cost figure calculated in section one of this chapter to the PAFs calculated here. The following table shows the lifetime costs of cancer that are attributable to particular ACE counts. For each ACE count the PAF is multiplied by the average lifetime cost of all cancers which is £113,850 as seen in table 3.2. We do not show specific ACEs as it is not possible to calculate the relative risk ratio due to a lack of control group and further the odds ratios from Pettrucelli (2019) and Felliti (1998) were averaged to arrive at the odds ratios here:

**Table 3.7: Odds ratios and PAF for all cancers**

No of adverse childhood experiences	OR (95% CI)	All cancers		
		Prevalence	PAF (95% CI)	Cost
0	1.00	54.39	0.00	0
1	1.18	18.98	6.83	7,779
2	1.15	6.51	1.89	2,149
3	1.07	6.51	0.85	964
4+	1.45	13.61	12.11	13,791

To calculate the relative risk ratio for all cancers we take the risk of disease for cancer for those with zero ACEs from Bellis et al (2013). This is 2.9% and if inserted into the formula:

$$RR = \frac{OR}{1 - p + (p \times OR)}$$

gives the following table of relative risk ratios:

**Table 3.8: Odds ratios converted into relative risk ratios**

OR	RR
1	1
1.2	1.193
1.2	1.193
1	1
1.9	1.852

In fact, the difference between the odds ratios and the relative risk ratios is negligible and so our estimates for cost will not change from the previous table. It is seen here that average lifetime costs per person fall as we go from 1 to 2 ACEs but then rise again as a function of ACEs as the number of ACEs increase.

### 3.3.2 Mental health

The table below shows the PAFs for different types of mental illness. The highest PAF is for depression being caused by 4 or more ACEs. This is at 67.2%.

**Table 3.9: Odds ratios, relative risk ratios and PAF for different types of mental illness**

	Psychotropic Medication		
	RR (95% CI)	Prevalence	PAF (95% CI)
No of adverse childhood experiences			
0	1.00	54.39	0.00
1	1.30	18.98	11.39
2	1.60	6.51	7.81
3	1.60	6.51	7.81
4+	2.30	13.61	35.39

	Anxiety			
	OR (95% CI)	RR (95% CI)	Prevalence	PAF (95% CI)
No of adverse childhood experiences				
0	1.00	1.00	54.39	0.00
1	2.00	1.75	18.98	28.45
2	2.30	1.94	6.51	12.23
3	3.00	2.33	6.51	17.34
4+	n/a	n/a	13.61	n/a

	Bipolar			
	OR (95% CI)	RR (95% CI)	Prevalence	PAF (95% CI)
No of adverse childhood experiences				
0	1.00	1.00	54.39	0.00
1	2.40	2.00	18.98	37.93
2	3.70	2.67	6.51	21.72
3	7.10	3.79	6.51	36.32
4+	n/a	n/a	13.61	n/a

	Depression			
	OR (95% CI)	RR (95% CI)	Prevalence	PAF (95% CI)
No of adverse childhood experiences				
0	1.00	1.00	54.39	0.00
1	1.48	1.38	18.98	14.51
2	2.14	1.84	6.51	10.91
3	3.31	2.49	6.51	19.34
4+	5.91	3.47	13.61	67.21

The following table shows a summary of the population attributable fractions for each mental illness:

**Table 3.10: PAFs for different types of mental illness**

		PAFs			
		Psychotropic Medication	Anxiety	Bipolar	Depression
No of adverse childhood experiences					
0	0.00	0.00	0.00	0.00	0.00
1	11.39	28.45	37.93	14.51	
2	7.81	12.23	21.72	10.91	
3	7.81	17.34	36.32	19.34	
4+	35.39	n/a	n/a	67.21	

The next table shows the costs associated with each condition that are attributable to having a certain amount of ACEs:

**Table 3.11: Costs associated with different types of mental illness**

		Lifetime Costs (£)		
		Anxiety	Bipolar	Depression
No of adverse childhood experiences				
0	0	0	0	0
1	17970	23958	9167	
2	7725	13719	6888	
3	10953	22942	12218	
4+	n/a	n/a	42453	

What is striking about these two diseases is that the lifetime costs fall as we go from 1 to 2 ACEs but then rise again as a positive function of ACE counts. This may well be an anomaly of the data but would be an interesting trend worthy of comment if it was found to be systematic across all the disease types.

### 3.3.3. Circulatory disease

This trend, of falling and then rising costs can be seen with circulatory diseases as well.

Note the tables below showing costs falling as we go from 1 to 2 ACEs and then rising again as a function of ACEs.

**Table 3.12: Costs of associated with stroke**

	Stroke				
	OR (95% CI)	RR (95% CI)	Prevalence	PAF (95% CI)	Cost (£)
No of adverse childhood experiences					
0	1.00	1.00	54.39	0.00	0
1	1.35	1.29	18.98	10.84	10,982
2	1.22	1.18	6.51	2.38	2,410
3	1.64	1.50	6.51	6.54	6,626
4+	1.22	1.18	13.61	4.97	5,039

**Table 3.13: Costs associated with myocardial infarction**

	Myocardial Infarction				
	OR (95% CI)	RR (95% CI)	Prevalence	PAF (95% CI)	Cost (£)
No of adverse childhood experiences					
0	1.00	1.00	54.39	0.00	0
1	1.07	1.06	18.98	2.25	13,528
2	1.12	1.10	6.51	1.32	7,898
3	1.78	1.60	6.51	7.83	46,975
4+	1.86	1.66	13.61	17.86	107,176

**Table 3.14: Costs associated with heart disease**

	Heart Disease				
	OR (95% CI)	RR (95% CI)	Prevalence	PAF (95% CI)	Cost
No of adverse childhood experiences					
0	1.00	1.00	54.39	0.00	0
1	1.10	1.08	18.98	3.21	2,852
2	1.20	1.17	6.51	2.17	1,929
3	1.60	1.47	6.51	6.16	5,483
4+	1.70	1.55	13.61	14.84	13,199

### 3.3.4 Musculoskeletal and genitourinary disease

Unfortunately, the systematic review did not return any results as to the odds ratios of musculoskeletal nor genitourinary disease and therefore analysis of the costs was not possible.

## 3.4 Summary & Conclusion

The aim of this chapter was to appropriate costs against ACEs. Using the population attributable fraction methodology, it set out to measure lifetime costs to individuals of having certain amounts of ACEs that had an association with five specific diseases.

Unfortunately, as found in the systematic review, data, in terms of costs *and* odds ratios, was only available for three of the five disease types, namely cancer, mental health and circulatory disease. An estimation of lifetime costs for these three diseases was calculated using PAF methodology and it was discovered that in all cases the average lifetime cost per person fell as we went from 1 to 2 ACEs but otherwise costs were a positive function of the number of ACEs. Further research is needed to identify the odds ratios and costs associated with musculoskeletal and genitourinary diseases. Even though information on these diseases was not available, preventing any attempt to perform analysis, this chapter has been a useful exercise in demonstrating the methodology behind the possible calculation of costs associated with ACEs and further work may be carried out as and when this data becomes available.

During my time as research officer at CHEME I was involved in several reports for Public Health Wales, the main one writing a report “Transforming Young Lives across Wales: The

Economic Argument for Investing in Early Years” (2016). This report outlined the case for prevention in early years and how investing in children can produce economic benefits for society as a whole. I was offered access to a dataset hosted by Public Health Wales and discovered that there was a way of measuring social mobility using a certain variable in the dataset. It was decided that having established the costs associated with ACEs ways of paying for these costs would be worthy of investigation. It has been posited that one way of paying for these costs would be in extra tax revenue to government that came about due to an increase in mobility. The next part of the thesis will use the dataset mentioned to measure the effect ACEs have on social mobility, hence living standards and the ability of government to use extra tax revenues to deal with the costs of ACEs.

## PART 2 – Movin' on up? Exploring the relationship between ACEs and social mobility



## Chapter 4 – Data Description

To answer the research question, described in chapter one and concerning social mobility, this chapter sets out the data used. This chapter aims to introduce the dataset that will be used in this thesis and provide some descriptive statistics to identify any interesting trends shown in the data. The dataset that will be used is a combination of four datasets commissioned and funded by Public Health Wales, which were conducted in four different areas of the UK: Wales, England, Blackburn with Darwen, and south England (defined as Hertfordshire, Luton and Northamptonshire). The pooled dataset has information on 13,130 participants.

For all of these, market research companies were employed to collect the data.

### 4.1 England

In England the data was collected between April and July 2013 (Bellis et al 2014a). It used an established survey tool (Bellis et al 2013) that collected information on demographics, ACEs, and health harming behaviours (HHB). The ACE questions were based on the Center for Disease Control and Prevention's short ACE questionnaire and identified nine categories of ACEs (Anda et al 2010). These were physical, verbal, and sexual abuse; parental separation; exposure to domestic violence; and growing up in a household with mental illness, alcohol abuse, drug abuse, or incarceration. A pilot study (Bellis et al 2013) identified ACE prevalence in England and a sample of 4,000 was targeted. This was achieved by a random probability approach stratified by region and small area deprivation to get a sample representative of the wider English population. Two LSOAs were selected from each deprivation decile in each area. 16,000 households were initially sampled to accommodate

for non-compliance and non-response. The inclusion criteria for the study were that they were residence in a selected LSOA; age 18 to 69 years; and cognitive ability to participate in a face-to-face interview. After keeping those respondents that had complete information on all ACEs, age, sex, ethnicity, and IMD quintile a final sample size of 3,885 was achieved. The study was funded by HEFCE (the Higher Education Funding Council for England) and NHS public health observatory funding to Liverpool John Moores University.

## **4.2 Blackburn with Darwen**

In Blackburn with Darwen the data was collected in 2012 (Bellis et al 2014b). It was a random sample stratified by deprivation within the local authority area with households randomly selected from the postcode address file to represent the area's population. The ACE questionnaire was also based on Anda 2010's short ACE tool and the inclusion criteria was that they were resident in Blackburn with Darwen, age 18 to 70 years and cognitively able to complete the interview. To allow for non-compliance 3,000 households were selected to be interviewed. The final number of respondents was 1,763. The study was funded by Liverpool John Moores University and NHS R&D funds.

## **4.3 South England (Hertfordshire, Luton, and Northamptonshire)**

The data collection for this study (Ford et al 2016) was made in a response to the need to collect local data to inform local responses and realizing that there was some variability across population groups making comparisons difficult. In Hertfordshire, Luton and

Northamptonshire the data was taken as a random probability sample stratified by deprivation, ethnicity, and rural/urban areas. In total 5,621 respondents took part in the study but after removing those individuals without complete data for demographics and ACE count the final sample size was 5,454. The study was funded by participating local authorities and Public Health England and was conducted in between June and September 2015 by the Centre for Public Health at Liverpool John Moores University.

## **4.4 Wales**

Finally, in Wales the data was collected in 2015. It took a random quota sampling approach stratified by health board and deprivation and was funded by Public Health Wales. For the Welsh study a total of 4,127 households that were contacted met the inclusion criteria. Of these 2,028 completed the survey – a compliance rate of 49.14% (Bellis et al 2016).

## **4.5 Descriptive statistics**

### **4.5.1 Demographics by age groups**

This section breaks down demographic variables by age groups. In these data age is put into the following categories: 18-29, 30-39, 40-49, 50-59 and 60-69. It is possible to comment in a descriptive manner to highlight interesting features of the data. In this section row percentages are reported so that for any given variable percentages are reported for each category. For example, we see that 24.3 percent of males are in the 18-29 category and 23.9 percent of Blackburn with Darwen respondents are aged 18-29. We can say two things therefore: whether the category has an unusually high/low representation of some age

group or whether there is a relatively constant age distribution. Table 4.2 shows the age distribution of demographic variables with each variable broken down by age category.

For the survey areas there are two facets of the data worth commenting upon. In Blackburn with Darwen there is an under-representation of 50-59 year olds (14.4%) and an over-representation of 18-29 year olds (23.9%) while in Wales there is an over-representation of 18-29 year olds (30.4%) and an under-representation of 30-39 year olds (14.2%). For the other areas the age distribution is more or less constant. For males in the sample there is an over-representation of 18-29 year olds (24.3%) and an under-representation of 50-59 year olds (17.6%). For females there is an under-representation of 50-59 year olds (16.4%) and an over-representation of the 30-39 age group (22.3%). In terms of ethnicity there is an over representation of Asian people in the 30-39 group and an under representation of the 60-69 age groups. This may reflect the increasing level of immigration by Asian nationals into the UK over the past few years. This is reflected in the UK residency data where it is seen that there is a higher percentage of 18-29 year olds that have lived in the UK for less than 5 years (51.6%). Not surprisingly there is a high percentage of those that are single in the youngest (18-29) age group and this declines with age. Also, the age group with the highest percentage of 60-69 year olds is the widowers. Also declining with age are qualification levels indicating that people are becoming more qualified over time.

**Table 4.1: Demographic profile by age categories**

Survey Areas						
	18-29	30-39	40-49	50-59	60-69	
Blackburn with Darwen	23.9	22.2	22.0	14.4	17.4	100.0
England	21.0	19.9	20.5	18.0	20.7	100.0
Wales	30.4	14.2	17.8	17.5	20.2	100.0
Hertfordshire, Luton, and						
Northamptonshire	20.6	22.5	20.6	17.0	19.3	100.0
Sex						
Male	24.3	18.0	19.5	17.6	20.5	100.0
Female	21.4	22.3	21.1	16.4	18.7	100.0
Missing	12.2	14.3	8.2	24.5	40.8	100.0
Ethnicity						
White	21.7	18.4	20.0	18.1	21.8	100.0
Asian	26.6	31.6	21.4	11.1	9.4	100.0
Other	28.2	27.9	21.9	13.3	8.7	100.0
Missing	38.5	11.5	26.9	7.7	15.4	100.0
UK Residency						
From birth	22.9	18.2	20.1	17.7	21.1	100.0
Less than 5 years	51.6	31.1	12.1	3.4	1.8	100.0

5-10 years	33.0	43.5	14.9	6.5	2.1	100.0
Over 10 years	11.7	23.0	25.1	19.6	20.6	100.0
Missing	12.5	16.7	20.8	16.7	33.3	100.0

---

#### Relationship Status

Married/Civil partnership	7.3	23.3	25.5	20.7	23.2	100.0
Living with long term partner	36.8	33.5	16.8	9.7	3.2	100.0
Widowed	0.4	0.7	2.5	11.9	84.5	100.0
Separated	5.8	21.7	31.1	28.3	13.1	100.0
Divorced	1.8	8.3	25.0	34.7	30.2	100.0
Never married / single	60.4	15.0	11.5	7.3	5.8	100.0
Refused	15.4	15.4	23.1	19.2	26.9	100.0
Missing	23.5	23.5	23.5	11.8	17.6	100.0

---

#### Qualification Level

None	12.4	13.9	16.0	18.7	39.1	100.0
Secondary	26.2	19.0	22.3	18.1	14.5	100.0
College/Sixth Form	37.7	19.2	17.4	12.7	12.9	100.0
Higher Education	29.7	25.7	19.5	14.5	10.6	100.0
Professional/work related	14.5	23.5	23.2	18.7	20.2	100.0
Other	11.1	11.1	22.2	0.0	55.6	100.0
Missing	18.2	36.4	18.2	0.0	27.3	100.0

---

### 4.5.3 Diseases

**Table 4.2: Disease type in ACE dataset**

	n	%
Allergies	1735	23.68
Cancer	497	6.78
STI	236	3.22
Diabetes	678	9.25
Hypertension	1927	26.30
Angina	219	2.99
Heart Disease	294	4.01
Stroke	138	1.88
Asthma	660	9.01
Respiratory Disease	363	4.95
Liver Disease	102	1.39
Digestive Disease	478	6.52

Each respondent was asked whether they currently suffer from some type of disease. The highest rate for any disease was for hypertension, with 26.3% of those suffering a disease suffering from it. This was followed by allergies (23.68%) with the least prevalent being liver disease (1.39%).

#### 4.5.4 Diseases by age group

**Table 4.3: Percentages of disease type by age categories**

	18-29	30-39	40-49	50-59	60-69
Allergies	20.7	22.2	20.4	17.9	18.7
Cancer	2.4	6.4	12.9	23.1	55.1
STI	33.1	23.7	18.6	16.9	7.6
Diabetes	1.9	4.6	12.8	26.0	54.7
Hypertension	3.2	5.6	14.7	26.6	49.9
Angina	1.4	0.5	6.4	23.7	68.0
Heart Disease	1.4	3.7	9.9	24.1	60.9
Stroke	1.4	0.7	10.9	29.7	57.2
Asthma	20.5	18.9	20.5	21.8	18.3
Respiratory					
Disease	6.6	10.2	13.8	25.3	44.1
Liver Disease	4.9	11.8	23.5	25.5	34.3
Digestive Disease	7.5	13.4	22.6	22.2	34.3

If we breakdown diseases by age group, either positive or negative relationships are seen between certain diseases and age. For allergies there is, more or less, a constant age distribution, which is not surprising since we do not expect there to be a relationship between allergies and age. The same is true for asthma. However, there is a negative relationship, the problem is big in the younger age groups and gets better as they age, for



STI. There is a positive relationship, the problem gets worse over age, for cancer, diabetes, hypertension, angina, heart disease, stroke, respiratory disease, liver disease and digestive disease. These results are intuitive, and it gives confidence that the data is reliable.

#### 4.5.5 ACEs

**Table 4.4: Frequency of different types of ACE**

	n	%
Household Mental Illness	1554	11.84
Alcohol	1393	10.61
Drugs	528	4.02
Incarcerated	508	3.87
Divorce	2633	20.05
Witnessing Partner Violence	1946	14.82
Physical abuse	1054	8.03
Emotional abuse	2777	21.15
Sexual abuse	903	6.88
ACE Count		
0	7,172	54.62
1	2,588	19.71
2 to 3	2,055	15.65
4+	1,299	9.89

Table 4.4 above delineates the number of respondents with each individual ACE and the number with ACE counts of zero, 1, 2 to 3 and 4 or more ACEs. The ACEs with the most

incidence are emotional abuse (21.15%), divorce (20.05%) and witnessing partner violence (14.82%) while the ACE with the lowest incidence is having a parent who has been incarcerated. Over half the sample (54.62%) have never experienced ACEs while 9.89% have experienced 4 or more. Breaking down by age group however shows that the problem of Adverse Childhood Experiences is getting worse over time. As we look back, from the older age group to the younger nearly all ACEs have a higher incidence the younger the respondents. There is a negative relationship between ACEs and age which can be shown as the difference in incidence between the oldest age group and the youngest as shown in the last column of table 4.5. The biggest difference is with having a parent who takes drugs (-35.4).

**Table 4.5: Percentage of different types of ACE by age categories**

	18-29	30-39	40-49	50-59	60-69	Difference <sup>8</sup>
Household Mental Illness	26.4	21.0	21.6	16.9	14.1	-12.4
Alcohol	28.0	22.2	23.4	14.2	12.2	-15.8
Drugs	39.8	26.9	18.0	11.0	4.4	-35.4
Incarcerated	34.8	24.2	21.5	12.8	6.7	-28.1
Divorce	34.0	24.2	21.0	12.4	8.4	-25.6
Witnessing Partner						
Violence	23.8	22.1	22.6	15.5	15.9	-7.9
Physical abuse	20.7	20.1	23.1	17.6	18.6	-2.1
Emotional abuse	24.7	22.7	22.4	15.8	14.5	-10.2

<sup>8</sup> Difference between 60-69 and 18-29 groups

Sexual abuse		20.2	22.0	22.3	19.8	15.7	-4.4
ACE Count							
	0	20.6	19.4	19.9	18.1	22.1	1.5
	1	24.1	21.5	19.1	15.3	20.1	-4.1
	2 to 3	24.3	20.6	21.0	16.8	17.2	-7.1
	4+	28.9	23.4	23.9	14.8	9.1	-19.8

---

The starkest indication that the problem is getting worse however can be seen if the ACE counts are looked at. Young people, aged 18-29, are three times as likely to report 4 or more ACEs than older people aged 60-69 and they are generally less likely to report zero ACEs. It seems therefore that the problem of ACEs is not improving but getting worse over time and this is something that needs to be considered as the analysis gets under way.

#### 4.5.6 Demographics by ACE count

Finally, in this chapter, to delve further into the finding about ACE counts getting worse over time, the sample demographics are presented but broken down by ACE count. This will give an idea, for each category within each demographic variable the distribution of ACE counts within that category. For example, in Blackburn with Darwen 50.99% have no ACEs, while in Wales the figure is 53.55%.

The area with the highest percentage with 4 or more ACEs (13.86%) is Wales, the lowest being in England (8.31%). There are slightly more females with 4 or more ACEs (10.39%) than males (9.3%).

**Table 4.6: Demographics broken down by ACE count**

		ACE Count				
		0	1	2 to 3	4+	Missing
Survey	BwD	50.99	19.57	16.90	11.63	0.91
	England	53.62	22.68	15.39	8.31	0.00
	Wales	53.55	18.89	13.71	13.86	0.00
	Hertfordshire, Luton and North'shire	56.91	17.95	16.15	8.98	0.00
Sex	Male	54.18	20.11	16.31	9.30	0.10
	Female	54.97	19.35	15.15	10.39	0.14
	Missing	57.14	24.49	10.20	8.16	0.00
Age	18-29	49.60	20.95	16.79	12.59	0.07
	30-39	51.87	20.78	15.84	11.36	0.15
	40-49	53.43	18.52	16.20	11.62	0.22
	50-59	58.15	17.68	15.44	8.59	0.13
	60-69	61.48	20.16	13.75	4.58	0.04
Ethnicity	White	52.20	20.86	16.34	10.56	0.05
	Asian	72.56	12.76	9.76	4.44	0.48
	Other	54.76	16.82	17.08	10.82	0.52
	Missing	53.85	15.38	19.23	11.54	0.00
Deprivation						
Quintile	Wales					

1	57.60	14.74	11.56	16.10	0.00
2	48.48	23.60	13.45	14.47	0.00
3	55.98	20.10	11.96	11.96	0.00
4	54.74	18.42	15.79	11.05	0.00
5	50.71	18.10	15.95	15.24	0.00
England					
1	59.08	24.94	11.64	4.35	0.00
2	52.51	25.07	14.78	7.65	0.00
3	54.18	23.11	15.93	6.79	0.00
4	53.82	18.89	17.34	9.96	0.00
5	48.76	21.46	17.12	12.66	0.00

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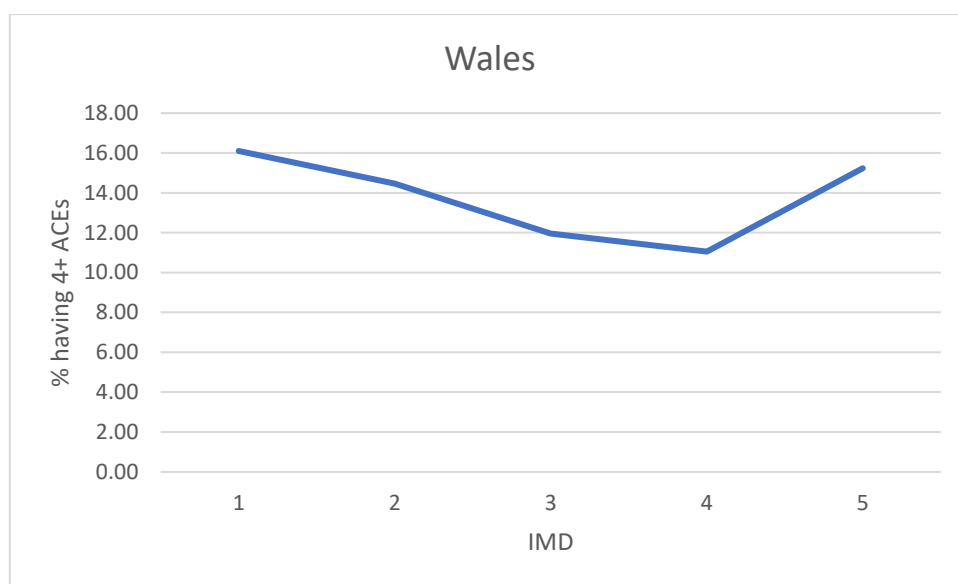
In terms of age our previous finding is borne out here, namely that the problem is getting worse over time. That is the youngest age group has the most respondents with 4 or more ACEs (12.59%) and the least with no ACEs (49.60%) while the oldest age group has the highest percentage with no ACEs (61.48%) and the lowest with 4 or more ACEs (4.58%).

In terms of ethnicity there is a higher percentage of Asian individuals with no ACEs (72.56%) than is the case with white people (52.20%) and much less people with 4 or more ACEs (4.44%).

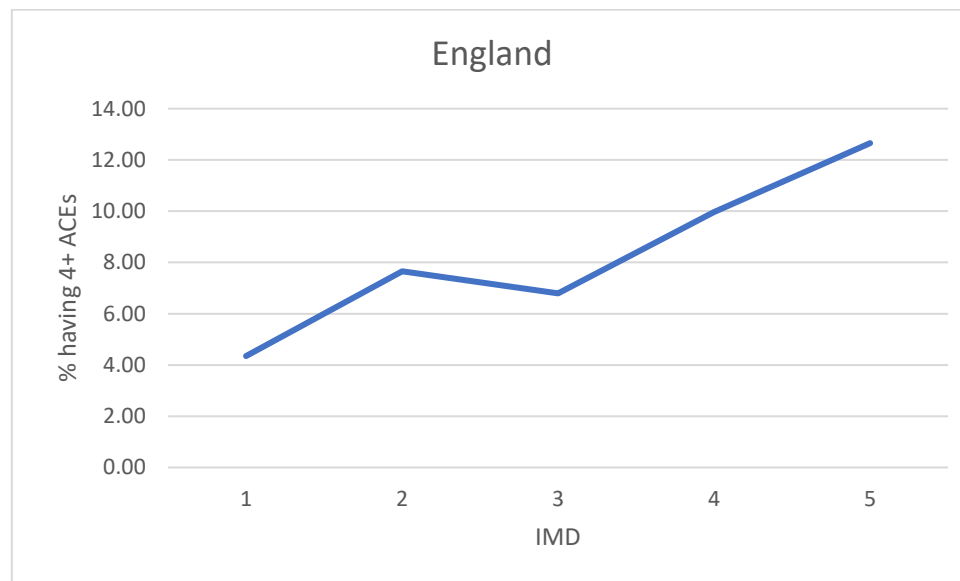
Finally, the relationship between deprivation and ACEs can be investigated. The data seems to suggest that there is a small difference in having 4 or more ACEs between the most deprived areas of Wales (15.2%) and England (12.7%) with a lower percentage of those in the most deprived areas having 4 or more ACEs in England. The distribution in England is

what we would expect, with ACE counts getting higher as deprivation increases (goes from 1 to 5). In Wales however, the U-shaped distribution is interesting and suggests that the very poorest communities have a lower number of ACEs than those in the middle of the distribution. Although care must be taken when looking at this issue due to small numbers as shown figures 4.3 and 4.4, which show the STATA output of cross tabbing index of deprivation with ACE counts, this finding has serious implications for policy and may shed new light on our thinking on the relationship between deprivation and ACEs.

**Figure 4.1: U shaped distribution of % having 4+ ACEs according to deprivation quintiles in Wales**



**Figure 4.2: Positive correlation of % having 4+ ACEs according to deprivation quintiles in England**



**Figure 4.3: STATA output for crosstabulation of Index of Multiple Deprivation and ACE counts in England**

IMD	ACE_Count_Categories				Total
	0	1	2	3	
1	462	195	91	34	782
2	398	190	112	58	758
3	415	177	122	52	766
4	416	146	134	77	773
5	392	173	139	102	806
Total	2,083	881	598	323	3,885

**Figure 4.4: STATA output for crosstabulation of Index of Multiple Deprivation and ACE counts in Wales**

walesDepri vation	ACE_Count_Categories				Total
	0	1	2	3	
1	254	65	51	71	441
2	191	93	53	57	394
3	220	79	47	47	393
4	208	70	60	42	380
5	213	76	67	64	420
Total	1,086	383	278	281	2,028

## Chapter 5 – Analysis of ACEs and social mobility using a large survey dataset

### 5.1 Chapter Summary

Chapters 2 and 3 provided a baseline cost of ACEs that could potentially be used when designing policy to combat the effects of ACEs. The information provided here gives an idea of the outlay that would be expected from government to pay for these diseases, part of which are caused by ACEs. The link between these chapters and this chapter is intuitive. Identify the costs and then look for ways to pay for this. One way that revenue could be generated would be through increased tax returns should the poorest in society witness an increase in their social status. This would then give rise to the ‘virtuous circle’ described in figure 1 whereby an increase in investment by government to improve resilience could potentially lead to those children flourishing and creating bigger economic benefits than the initial investment and thus generate further tax revenues for the government and more resources to invest back into the system.

This chapter is the main analysis chapter of this thesis. It provides some background material about social status and mobility before going on to discuss the literature on the relationship between adverse childhood experiences (ACEs) and social mobility. The methodology used for this analysis is then discussed before presenting the results of logistic and OLS regressions of the relationship between ACEs and social mobility. The chapter concludes by providing brief policy recommendations.



## **5.2 Introduction**

2020 has been a tumultuous year and the Covid-19 pandemic has starkly highlighted the deep inequalities in our society. People from disadvantaged backgrounds are disproportionately affected by the virus. It has been found that Covid-19 kills those from a deprived area at twice the rate of those in the most affluent (Nuffield Trust 2021). This reflects the more general link between deprivation or poverty and adverse health outcomes. Insofar as ACEs can lead to health problems in later life it would be interesting to look at how the occurrence of ACEs affects living standards. The costing chapter, chapter 3, gave an idea as to how much different ACEs are likely to cost society. This chapter investigates how society can generate benefits that could be used to offset some of these costs. It is in this spirit that this chapter looks at the relationship between ACEs in childhood and what happens to individuals' standard of living in adulthood and this is done by looking at social mobility.

## **5.3 Background to social mobility**

The definition of socioeconomic status (SES), according to the American Psychological Association (APA) is “the social standing or class of an individual or group” (APA, 2018). According to Metzler et al (2017) research has been conducted on the relationship between ACEs and health outcomes which also control for socio-economic indicators such as education, employment, and income. However, what has gained less attention is the role of ACEs as a potential determinant of life opportunities captured through education,

employment, and income. They claim that education, employment, and income are commonly used indicators of socio-economic status and are consistently correlated with health (Braveman et al 2010, Pickett 2001). Some studies have already been conducted looking into the connection between child abuse and neglect and later life education employment and income. However, these studies look only at child abuse and neglect and not the broader definition of ACEs that we define in this thesis.

Socioeconomic status has been conceptualized in many ways. Early studies conceptualized socioeconomic status as the occupation of the father (Taussig 1920) for example. Later works attempted to develop other instruments to measure socioeconomic status. These were more sophisticated techniques such as factor analysis or model-based approaches (NCES 2012). More recently there has been a consensus on how to measure socioeconomic status and this stipulates that it should be a composite variable measuring education, income, and occupation (Brese and Mirazchiyski 2013). However, according to Broer et al (2019) “The abstract nature of the concept of SES leaves some room for researchers to decide what proxy variables to use as SES measures” (p9). It is in this vein that I shall proceed and socioeconomic status, for the purpose of analysis in this chapter, will be wealth in adulthood.

Social mobility is the movement from one level of social or income status to another level over time. What economists call ‘intergenerational mobility’ measures the degree to which people’s social status changes between generations. Social mobility reflects “the extent to which parents influence the success of their children in later life or, on the flipside, the extent to which individuals can make it by virtue of their own talents, motivation and luck.” (Blanden 2005, p18). Another definition comes from Joslyn (1927), who states that

“movement from one social class to another, and the stratification of economic, occupational, and political groups with which this movement is associated” (p131). A paper for the Cabinet Office by Aldridge in 2004 defined social mobility as “...the movement or opportunities for movement between different social classes or occupational groups and the advantages and disadvantages that go with this, e.g., income” (p3). The best and simplest definition I have come across comes from Buscha (2018, p.167) which notes it as ‘the simple difference between an individual’s socio-economic position in adulthood and that of his or her parent(s) when the individual was a child.’

There are several types of social mobility. One can differentiate between horizontal and vertical mobility. Horizontal mobility occurs when a person moves occupation but still maintaining the same social class. An example of this would be a doctor giving up her practice and going to lecture in medicine. Vertical social mobility refers to a change in the occupational, political, or religious status of a person that causes a change in their societal position. Ascending or upward mobility means moving from a group in the lower socio-economic stratum into a higher societal position. Downward mobility occurs when individuals start life in an affluent background but later move down the social strata so that their position is lower as adults than they were as children.

The theoretical underpinnings of social mobility are often attributed to Russian-born American sociologist and political activist Pitirim Sorokin. In his book ‘Social and Cultural Mobility’ he argues that there is no such thing as a purely open or closed society and that the speed of social mobility depends on how developed the society in question is. People can move between different social status through different social interactions motivated by different factors in society to work towards a better standard of living.

Sorokin's (1959) theory however claims that social mobility can have negative and disruptive effects on individuals and society putting strain on individuals and leading to higher rates of "mental diseases and nervousness, psychoses, and neuroses" (Sorokin 1959:515). He talks about the 'weakening of social ties' that mobility brings in that people who are dynamic and move up or down the social ladder face disruption of the very things that make them 'belong' to the social group of their childhood. Something akin to this view is taken by Friedman (2014) who writes about social mobility being "exhausting and discomforting" (p. 362). Curl (2013) states that the upwardly mobile "express disdain for and struggle internally with some of the changes they have made and undergone . . . [they have] difficulty in maintaining connection with their families of origin and therefore feel distance from their roots and what once made them who they are' (p293). So upward mobility may not be as beneficial as one would think at face value. Chan in his 2018 work asks, 'Does intergenerational social mobility exact a toll on the well-being of individuals?' In this study Chan refers to Nikolaev and Burns (2014, p82) who say that "downward mobility . . . has a negative effect on the self-reported level of happiness and subjective health while upward mobility is associated with positive outcomes in subjective well-being" thus seeming to contradict Sorokin's dissociative thesis. This obviously is an area of great philosophical contention. For the purpose of this thesis, I will consider the more intuitive view of upward and downward social mobility in that upward mobility is more desirable than downward mobility.

It may be worth noting that there is another issue about if the disadvantaged have the freedom to choose whether to avail of opportunities for social mobility if the opportunities are very few. Is it a voluntary choice if they hesitate? The answer depends on whether an individual's choice set acting as an individual is the same as the individual's choice set acting

as a member of a collective, for example a social group. This is a point raised by G A Cohen.

What is freedom to choose? Consider the following example (Cohen 1983):

“Ten people are placed in a room the only exit from which is a huge and heavy locked door. At various distances from each lies a single heavy key. Whoever picks up this key -- and each is physically able, with varying degrees of effort, to do so -- and takes it to the door will find, after considerable application, a way to open the door and leave the room. But if he does so he alone will be able to leave it. Photoelectric devices installed by a jailer ensure that it will open only just enough to permit one exit. Then it will close, and no one inside the room will be able to open it again.” (Page 9)

Now suppose that

“...no one believes he will be able to secure the key in face of the capacity of the others to intervene (though no one would intervene, since, being so diffident, each also believes that he would be unable to remove the key from someone else.” (Page 10)

In one sense, it could be argued that all ten individuals have voluntarily *chosen* to remain in the room, a prison. They are free to choose to stay or leave. Cohen would argue, instead, that all ten individuals above are "collectively unfree". Following Cohen's line of reasoning, one might argue that the unemployed are collectively unfree to choose whether to be employed, if there are not enough jobs for all of them. The very poor or otherwise disadvantaged people living in their social setting, following Cohen's logic, may be collectively unfree to avail of opportunities for upwards social mobility if opportunities for movement are very few. Yet in the received theory of utility maximising individuals making work-leisure choice, there is no recognition of collective unfreedom. One might also argue that, under certain circumstances, the employed are also collectively unfree to make a choice either about particular jobs or about employment itself.

In any discussion about social mobility, it is useful to make the distinction between absolute and relative mobility. According to PEW (2012) absolute inter-generational mobility refers to whether the individual has more, or less, income, earnings, or wealth than their parents had at the same age. The coalition government of 2011 defined absolute mobility simply as “the extent to which people are able to do better than their parents” p.15 (HM Government 2011). Relative mobility refers to the person’s rank on the income, earnings or wealth ladder compared to their peers. So, if absolute social mobility is about increasing opportunities, relative social mobility is about how those opportunities are distributed. The most common argument in favour of improving relative social mobility is that it is a measure of fairness in society – of whether there are equal opportunities for individuals to gain rewards based on effort and talent. Indeed, the reasoning behind the coalition’s strategy back in 2011 was as follows:

“In a fair society what counts is not the school you went to or the jobs your parents did, but your ability and your ambition. In other words, fairness is about social mobility – the degree to which the patterns of advantage and disadvantage in one generation are passed onto the next. An unfair society is one in which the circumstances of a person’s birth determine the life they go on to lead” p.11. (HM Government 2011)

Social mobility matters for several reasons. First, an absence of social mobility implies an unequal society in terms of opportunity. Across political parties, equality of opportunity is a major aspiration. Second, in terms of economic efficiency and growth making the best use of resources means making the most of everyone’s talents. Lastly social mobility enables social cohesion with people believing that they can aspire to a better life for their children (Aldridge 2001).

So, what has happened to social mobility in the UK over recent years? The main source of information here is the Social Mobility Commission's 'State of the nation 2018-19: social mobility in Great Britain' report (SMC 2019). In it the authors define social mobility in terms of occupation. Their main finding is that social mobility has stagnated over the past four years. In terms of people remaining privileged it is found that the majority do remain in the privileged life into which they were born. Upward mobility, however, the chances of improving one's lot in life, faces obstacles, some of which lead to recommendations that need to be in place if upward social mobility is to be facilitated.

In terms of occupational mobility, the report finds that those from a privileged background are 80% more likely to gain employment in professional occupations than those from a working-class background. They find also that occupational social mobility has largely remained stagnant since 2014. Of those from professional backgrounds 60% were in professional jobs compared with 59% four years ago. There is also a class pay gap with those from a working-class background earning 24% less than their counterparts from a professional background. Indeed, even if working class people make it into professional employment, they still earn 17% less than their more privileged counterparts. This may suggest that there is a stubborn 'wealth effect' that follows those from a lower social status even if they are able to move up the ladder. In terms of unemployment and economic inactivity those from a poorer background still face higher unemployment and economic inactivity rates even though overall employment has increased (SMC 2019).

The story for Wales and Scotland is very interesting. The report finds that social mobility here is generally higher than for Great Britain as a whole. While the percentage of those from a privileged background who gain professional employment compared with those from

a working-class background is 80% in GB as a whole, as mentioned, the gap in Scotland is 70% and Wales 60%. So downward mobility is greater in Wales than in England, according to this measure.

From the perspectives of this thesis, we confine ourselves to income or, more precisely wealth, mobility. Also, we look at ideas of mobility in terms of poverty in childhood. For example, there were 600,000 children living in household in poverty in 2012 [HBAI 2012/13]. By 2017 there were 500,000 more children living in households below poverty level income. How many of those children in poverty from 2012 that have not become adults between the above two years continue to remain in households in poverty? The reason for asking that question is to ascertain the prospect for escape from child poverty except by becoming older and falling out of the count for children in poverty? Even if we find that there is some upward mobility for children out of child poverty through poor households becoming less poor, there is a second issue. The experience of child poverty being an adverse childhood experience, it may have an impact on future social mobility of these children (SMC 2019). Poverty relates to social mobility in that those in poverty now could come from a richer past and those in poverty earlier have now escaped from poverty having become richer. Then there would be perfect mobility. According to the report,

“Child poverty has an important influence on social mobility, as children living in poverty can often have worse health, worse education outcomes and start school developmentally behind their more advantaged peers.” (p.4)

This elucidates the link between ACEs and social mobility. Those from more disadvantaged backgrounds, economically, are more likely to suffer from poor health and a worse start in life. This is compounded by ACEs with ACEs associated with ill health in later life and one



would posit that the link between having several ACEs and starting life poor is strong and significant. However, there may be a route out of poverty for those people notwithstanding that they suffered from several ACEs as children and this chapter will examine how this may come about.

As a final point, it may be worth noting that data concerning social mobility in the UK largely comes from the LFS social mobility unit which is a cross sectional database. They recommend the inclusion of new questions to ascertain parental education and in particular region of origin. Currently it is argued there is no way of conducting robust regional analysis as the current survey only asks people about their current location rather than where they are from originally. It is interesting to see such an influential document (Friedman et al 2017) leaving out any discussion about ACEs and it is hoped that this thesis will contribute to the case for better data on social mobility and ACEs to be collected. It is seen that social mobility remains at the very top of the political agenda. Yet the UK has traditionally lacked a data source extensive enough to work out exactly where to target policy interventions intended to improve social mobility (SMC 2017).

## **5.4 What is already known about social mobility and ACEs**

Although we know that absolute social mobility is the norm in Britain with 43% of the general population experiencing upward mobility in 2014-15 and only 29% experiencing downward mobility in the same period measured by occupation (SMC 2017) relatively little is known about the link between social mobility and ACEs. This section reports the results of a rapid review into the evidence. No attempt at a full systematic review was made due to the prior knowledge that this is an emerging field and that peer reviewed articles discussing

the subject were scarce. However, a rapid review (Khangura 2012) using Google Scholar, with the search terms ACE social mobility, Childhood trauma social mobility, and Childhood maltreatment social mobility, produced a total of eight papers, which shall be discussed here.

The first paper to be discussed does not necessarily cover the link between ACEs and social mobility but rather the relationship between child health (which can be influenced by ACEs) and social mobility (Case & Paxson 2006). Here the authors claim a two-way relationship between childhood social status and health. They say that children from poor backgrounds are more likely to have health problems and at the same time children who have poor health are less likely to be economically successful later in life. They note that getting rid of income related disparities in health problems in childhood would not lead to a reduction in the disparities in income between richer and poorer adults. However, an improvement in child health would lead to a commensurate improvement in adult economic circumstances.

“The "double disadvantage" of low income and poor health may combine to prevent poor children from achieving economic success as they become adults.” (Case & Paxson 2006, p.152)

A critique of Case and Paxson 2006 might be that they fail to examine if low income in childhood and poor health in later life are correlated. They claim that poor health in childhood and poor health and low income in later life are correlated.

Some of the reasons why poor childhood health may limit economic success in adulthood include:

- Children with health problems tend to be less well educated

- Less healthy children become less healthy adults and therefore perform less well economically.

The challenge is to find programs and policies that work effectively against the causes of poor childhood health.

Although low income in childhood and poor health in later life are linked it may not be that low income causes poor health. Rather it may be that the relationship runs in the opposite direction and that poor childhood health causes low income in adulthood. This may be through such mechanisms as mothers of sick children being unable to work as they have to care for the child. Having posited this argument however the authors believe that it is not the case that poor childhood health causes low income in adulthood.

Waldfoegel (2004) discusses the link between the early years and social mobility. A great deal of inequality shows itself before children enter school signifying the importance of the early years in determining social mobility. Here the author specifies three influences on development in the early years: child endowment; parents and the home environment; and preschool care and education. Children start life with different endowments – such as health and temperament. These endowments occur due to nature (gene effects) and nurture (environmental effects) and can affect the child's development. Aspects of parental care are affected by income and financial hardship and the parent's endowments in terms of health, especially mental health, ability and so on. Research can tell us how much these influences matter and how amenable they are to policy intervention. It has been found that although parenting may be more important, interventions to improve non-parental care and education may be more effective. The main point of Waldfoegel's paper is how policies in the early years can promote more social mobility. Here social mobility is defined largely in

terms of cognitive attainment rather than income or wealth. The conclusion of this paper is that early years policies can affect social mobility and promote child well-being while at the same time promoting other societal goals such as social inclusion, poverty reduction, parental employment, parental choice, and gender equity.

Hardcastle et al (2018) look at the relationship between ACEs and later educational achievement. Although we define social mobility in this thesis in terms of wealth it can also be captured with educational achievement. This paper assesses the impact of ACEs and education on employment in adulthood. The authors found that those with  $\geq 4$  ACEs<sup>9</sup>, were significantly more likely to have no formal qualifications (AOR=2.18;  $P<0.001$ ). They also found that having  $\geq 4$  ACEs was significantly associated with later unemployment (AOR=2.52;  $P<0.001$ ) and long-term sickness and disability (AOR=3.94;  $P<0.001$ ). It is noted that currently it is unclear to what extent the relationship between ACEs and employment may be mediated by educational attainment. These were results of multivariate models. After the confounding effects of demographic variables and childhood affluence in the relationship between ACEs and educational outcomes were considered there remained a significant impact of those with  $\geq 4$  ACEs being twice as likely than those with no ACEs to have left education with no qualifications. The authors also looked at how childhood affluence affected the level of qualifications with the finding that affluence was independently associated with lower odds of achieving no qualifications. This is an interesting finding in the context of this chapter. If qualifications are a loose proxy for social status, with higher qualifications leading to enhanced job opportunities and higher wealth

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<sup>9</sup> These ACEs are the same as those defined by Felitti (1998): Physical Abuse, Sexual Abuse, Emotional Abuse, Neglect, Household mental illness/suicide, Household alcohol abuse, Household drug abuse, Household member incarcerated, Parents separated or divorced, and Witnessing partner violence

then it will be interesting to see if having more ACEs leads to an increase in downward social mobility.

Schurer et al's (2019) study highlights the effect of ACEs<sup>10</sup> on later life economic outcomes over and above that of socio-economic background. With each additional ACE they find a 9% earnings penalty, a 25% higher likelihood of being welfare dependent, and a 27% higher probability of subjective poverty at age 55. The authors use high quality cohort data from the National Child Development Study (NCDS) to explore the link between ACE and lifetime economic opportunities. This rich dataset provides the opportunity for researchers to build a composite measure of ACEs that depends on actual reporting of child maltreatment as it is a longitudinal dataset that tracks individuals from birth. Questions are asked at ages 7 and 16 and so the information gathered relates to the period in question rather than retrospectively asking questions on childhood trauma and so it is possible to have an objective measure of childhood adversity. They use a variance-decomposition approach that was developed in Heckman and Pinto (2013). They then "calculate the contribution of differences in observable characteristics, measured at a time when cohort members enter adulthood... (at age 17 in the UK)..., to the observed differences in earnings, welfare dependence, and subjective poverty between cohort members with high doses of ACEs (or other components of the ACE index) and cohort members without." The methodology employed here is a starting point for the methodology that will be used in this thesis, Although the data used in this thesis is cross-sectional and Schurer's data is longitudinal therefore some tweaks have to be made to our methodology. Further details will be provided

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<sup>10</sup> These ACEs are the same as those defined by Felitti (1998): Physical Abuse, Sexual Abuse, Emotional Abuse, Neglect, Household mental illness/suicide, Household alcohol abuse, Household drug abuse, Household member incarcerated, Parents separated or divorced, and Witnessing partner violence

in the methodology section of this chapter. Meanwhile the results provided in Schurer (2019) point towards those from an income poor background, measured by the income of the father, having a negative relationship between ACEs and adult earnings. The findings of this paper suggest that “some children from more disadvantaged families are not at risk of later-life disadvantage, and –crucially –that some children in better off families are very much at risk of later-life disadvantage”. This result may be useful to revisit when considering the findings of this thesis.

The main purpose of the study conducted by Powdhar (2019) was to determine whether socioeconomic status (SES) and access to care account for the relationship between ACEs and depression. The author found that higher number of reported ACEs<sup>11</sup> was associated with lower SES and less access to care, and these factors were associated with an increased risk of depression.

The mechanisms that link ACEs to poor lifetime health outcomes are not well understood (Font & Maguire-Jack 2015). Further the role of socio-economic conditions as a mediator of the link between ACEs and health risks have begun to be considered by researchers. Font and Maguire-Jack look at the relationship between adult socio-economic conditions such as marriage, divorce and separation, educational attainment, income and insurance status and the association of ACEs<sup>12</sup> with five health risks – depression, obesity, tobacco use, binge drinking, and self-reported sub-optimal health. They find that at high numbers of ACEs, 15–20% of the association between number of ACEs and adult health risks was attributable to

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<sup>11</sup> These ACEs are the same as those defined by Felitti (1998): Physical Abuse, Sexual Abuse, Emotional Abuse, Neglect, Household mental illness/suicide, Household alcohol abuse, Household drug abuse, Household member incarcerated, Parents separated or divorced, and Witnessing partner violence

<sup>12</sup> These ACEs are the same as those defined by Felitti (1998): Physical Abuse, Sexual Abuse, Emotional Abuse, Neglect, Household mental illness/suicide, Household alcohol abuse, Household drug abuse, Household member incarcerated, Parents separated or divorced, and Witnessing partner violence

socioeconomic conditions. Their main finding is that a higher number of ACEs is associated with lower levels of income, educational attainment, and marriage.

A small body of research has identified associations between maltreatment (as identified through child protection records) and income, earnings, and educational attainment in early and mid-adulthood (Font 2015). Currie and Widom (2010) is one such study. They used a prospective cohort design to compare cases of physical, sexual abuse and neglect with non-abused and non-neglected children during 1967-71 and followed into adulthood. Outcome measures of economic status and productivity were measured in 2003-2004 and it was found that adults who had a history of abuse/neglect had lower incomes, levels of education, employment, and fewer assets compared to the matched control children. In fact, there was a 14% gap between treatment individuals and controls in the probability of employment in middle age after controlling for background characteristics.

Pereira and Pinto (2017) look at the relationship between child maltreatment (in the forms of abuse and neglect) and economic outcomes at age 50. They employ the 1958 British birth cohort data to look at the relationship between child maltreatment and a host of outcomes including social mobility. Generally, the risk of a poor outcome increased by the number of maltreatment type. The types of maltreatment that were associated with social mobility were neglect and sexual abuse. Respondents in these categories were less likely to be upwardly mobile, as defined here by moving from manual to non-manual occupations, both between generations and within. The odds ratios here, for neglect and sexual abuse e.g., were 0.45 [95% CI, 0.39-0.53] and 0.61[95% CI, 0.50-0.74] respectively. They were also more likely to be downwardly socially mobile – to move from non-manual to manual

occupations (OR: 2.31 [95% CI, 1.56-3.41], and 2.11 [95% CI, 1.63–2.74]. They found no pattern for non-sexual abuse.

Their findings in general seem to say that “maltreated individuals grow up to experience socioeconomic disadvantage”. The rest of this chapter will aim to establish if this is true in the context of Wales and Southern England by looking at upward and downward mobility and their relationship with the number of ACEs individuals have in the survey that has been employed for this thesis.

## **5.5 Methodological approach**

### **5.5.1 Overview**

The research question to ask of the data is as follows: to what extent do ACEs affect social mobility? There are two variables in the data that would give an idea of social mobility: wealth in childhood (poor, affluent) and wealth in adulthood (poor, affluent). We begin by using a binary poor/affluent coding of the wealth data, whereby individuals were asked to rank their status on a one to ten Likert scale, and then a coding of ‘poor’ was given to rank 1 to 5 and ‘affluent’ given to 6 to 10. This is a quite crude and rather blunt instrument to perform any meaningful analysis but will give a feel of what the data is suggesting.

It will be possible to code the data to capture social mobility as follows:



**Figure 5.1: Coding wealth in childhood and wealth in adulthood variables**

		Adulthood	
Childhood		Poor	Affluent
	Poor	1	2
	Affluent	3	4

1 – Stay poor

2 – Go from poor to affluent (upward mobility)

3 – Go from affluent to poor (downward mobility)

4 – Stay affluent

Thus, it is possible to get transition probabilities of going from one state to another or staying in that state.

To obtain a more sophisticated model it is proposed that mobility should be classified by quartiles of wealth. This will add a certain richness to the data analysis whereby we not only look at social mobility as a binary 1 0 variable but also look at it in terms of quartiles and movement between them. The data on wealth in childhood and wealth in adulthood is scaled on a range from 1 to 10 with 1 being the poorest and 10 being the most affluent.

Using STATA, the data was coded as follows:

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recode wealthchild (1/3=1) (4/5=2) (6/8=3) (9/10=4), gen(wealthchild2)
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recode wealthadult (1/3=1) (4/5=2) (6/8=3) (9/10=4), gen (wealthadult2)
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**Figure 5.2: Coding the data into quartiles**

	Adulthood			
Childhood	Q1	Q2	Q3	Q4
Q1	Stay	Q1-q2	Q1-q3	Q1-q4
Q2	Q2-q1	Stay	Q2-q3	Q2-q4
Q3	Q3-q1	Q3-q2	Stay	Q3-q4
Q4	Q4-q1	Q4-q2	Q4-q3	Stay

So, there are sixteen states any individual can inhabit, and the diagonal of the matrix refers to those that stay in their socioeconomic position. To the left of the diagonal is downward social mobility and to the right upward social mobility.

It may be the case that looking at social mobility in terms of a binary variable or even quartiles could be construed as being too narrow. Later in the chapter we will look at another way of defining social mobility. The last part of this chapter will consider using a wider definition of social mobility and will use the wealth in adulthood and wealth in childhood in a different way. To this extent the social mobility variable will be adapted to be the difference between the wealth in adulthood score and the wealth in childhood score.

As mentioned at the beginning of this chapter the wealth variable asks respondents to rank their wealth on a Likert scale with 1 being very poor and 10 being very wealthy. If we take wealth in adulthood as the minuend and wealth in childhood as the subtrahend then we get the difference between wealth in adulthood and wealth in childhood. For example, if a respondent's wealth in childhood is 8 and wealth in adulthood is 5 we can say that there has been downward social mobility. Subtracting one from the other gives an 'index' of minus 3

(wealth in adulthood minus wealth in childhood) and so a minus score indicates downward mobility, a positive score denotes upward mobility and a zero signifies no change.

The idea is to find out what kind of association there is between having ACEs and upward or downward social mobility later in life. One would expect those with a high number of ACEs to experience downward social mobility later in life as the consequences of having ACEs have been identified as being detrimental to wellbeing and health and so maybe the ability to find well-paid employment. This was certainly what the literature review at the beginning of this chapter revealed. Conversely, one would expect those with a low number of ACEs to experience upward social mobility but not maybe to the same extent as those experiencing downward mobility. It will be relatively straightforward to perform a logistic regression with ACEs count as the RHS variable and mobility as a LHS variable while also controlling for other characteristics such as age, ethnicity, and location.

Ex ante I am looking to use a logistic regression to describe the relationship between the number of ACEs and socio-economic status. The main research question will be: does having more ACEs, that is abuse, neglect and household dysfunction, make it more or less likely to become socially mobile? The regression equation can be written as:

$$Y_i = \alpha + \beta x_i + \varepsilon$$

Where Y=Social mobility,

Assign value 1 – Became socially mobile by upward or downward mobility

Assign value 0 - stayed at the same level or became the opposite of the mobility in the first instance;

and X = number of ACEs, 0,1,2-3,4+

One will then be able to ascertain how much of the variation in social mobility can be accounted for by variation in ACEs. The reason this is interesting is that it would tell us the extent to which having ACEs affects the economy in terms of the living standards of people who suffer ACEs as children versus those who do not. It would enable a telling of a story as to how ACEs affect society showing us the mechanisms through which ACEs influence living standards.

## 5.6 Results

The following table gives an indication of general social mobility within the sample:

**Table 5.1: Social mobility – whole sample**

	Child	Adult
Poor	6,664	6,184
Affluent	756	1,237

There is upward mobility in the sample as a whole as the number of those poor as adults is lower than those poor as children and the number affluent as adults is higher than those affluent as children indicating a general upward mobility. The next table shows transition probabilities between different states of social status:

**Table 5.2: Transition probabilities – whole sample**

Transition	
Probability	
Poor to affluent	7.70%
Stay Poor	81.50%
Affluent to poor	1.30%
Stay affluent	8.80%

Clearly, there has been a shift towards being affluent if we take the sample as a whole. It is also possible to look at social mobility for those that have any ACEs. The ACE count category is coded into zero for no ACEs and 1 for any ACEs.

**Table 5.3: Transition probabilities – those with ACEs**

Transition	
probability	
Poor to affluent	8.17%
Stay poor	82.89%
Affluent to poor	1.43%
Stay [Affluent]	7.17%

But what is the relationship between social mobility and ACE *counts*? The ACEs categories will be used initially with the number of ACEs coded as 0, 1, 2-3, 4+. First, it is worth looking

at what other factors may influence social mobility apart from ACEs. The first regression to be run considers the effect of demographic variables on social mobility. Later ACEs will be added to this regression to ascertain the effect they have on mobility.

**Table 5.4: Logistic regression of demographics on upward social mobility**

Number of observations	7412					
				LR chi2(3)	87.930	
				Prob > chi2	0.000	
Log likelihood	-1983.059			Pseudo R <sup>2</sup>	0.022	
Upward Mobility	Odds Ratio	Std. Err.	z	P>z	[95% Conf. Interval]	
Gender	0.862	0.075	-1.700	0.090	0.727	1.023
Age Categories	1.297	0.040	8.430	0.000	1.221	1.378
Ethnicity	1.000	0.000	4.130	0.000	1.000	1.000
Constant	0.046	0.008	-17.670	0.000	0.033	0.065

**Table 5.5: Logistic regression of demographics on downward mobility**

Number of observations	7429					
				LR chi <sup>2</sup>	40.310	
				Prob > chi <sup>2</sup>	0.000	
Log likelihood = -505.66846				Pseudo R <sup>2</sup>	0.038	
Downward Mobility	Odds Ratio	Std. Err.	z	P>z	[95% Conf. Interval]	
Gender	1.058	0.216	0.270	0.784	0.709	1.578
Age categories	0.628	0.052	-5.580	0.000	0.534	0.740
Ethnicity	1.000	0.000	2.240	0.025	1.000	1.000
Constant	0.038	0.014	-8.880	0.000	0.019	0.079

As can be seen in Table 5.4 and 5.5 being female is associated with higher odds of becoming downwardly mobile (OR: 1.058; 95% CI: 0.709-1.578) and lower odds of becoming upwardly mobile (OR: 0.862; 95% CI: 0.727-1.023). However, these results are not statistically significant with a p value of 0.784 and 0.090 so we can argue that no effect was detected. In terms of age as people get older, they are less likely to become downwardly mobile (OR: 0.628; 95% CI: 0.534-0.740) and more likely to become upwardly mobile (OR: 1.297; 95% CI: 1.221-1.380). Ethnicity has parity in that individuals are neither more nor less likely to become mobile if they come from an ethnic minority.

Adding ACE counts into the regression gives some interesting results as table 5.6 below shows:

**Table 5.6: Logistic regression of ACE counts on upward mobility**

Number of observations	7412			LR chi <sup>2</sup>	96.120
				Prob > chi <sup>2</sup>	0.000
				Pseudo R <sup>2</sup>	0.024
Log likelihood = -1978.9654					
Upward Mobility	Odds Ratio	Std. Err.	Z	P>z	[95% Conf. Interval]
Gender	0.861	0.075	-1.710	0.088	0.726 1.022
Age Categories	1.311	0.041	8.680	0.000	1.233 1.393
Ethnicity	1.000	0.000	4.150	0.000	1.000 1.000
ACE Count Categories	1.127	0.046	2.900	0.004	1.039 1.221
Constant	0.041	0.007	-17.730	0.000	0.029 0.058

**Table 5.7: Logistic regression of ACE counts on downward mobility**

Number of Observations	7429			LR chi <sup>2</sup>	41.380
				Prob > chi <sup>2</sup>	0.000
				Pseudo R <sup>2</sup>	0.039
Log likelihood = -505.133					
Downward Mobility	Odds Ratio	Std. Err.	Z	P>z	[95% Conf. Interval]
Gender	1.058	0.216	0.280	0.782	0.709 1.579
Age Categories	0.624	0.052	-5.650	0.000	0.530 0.735
Ethnicity	1.000	0.000	2.280	0.023	1.000 1.000
ACE Count Categories	0.904	0.090	-1.020	0.309	0.744 1.098
Constant	0.042	0.016	-8.390	0.000	0.020 0.088

The first regression shows that an increase in ACE counts leads to an increase in the likelihood of being upwardly mobile (OR: 1.127; 95% CI: 1.039-1.221). This result, although significant at a 95% level, might seem counter intuitive – it is not expected that having more ACEs makes it more likely to move upwards socially. However, it *is* a statistically significant finding and the last section in this chapter examines why this may be the case.

Before proceeding, it is worth investigating what this relationship between upward social mobility and ACE counts looks like for Wales. If the analysis is done separately for Wales<sup>13</sup>, the results are as follows:

<sup>13</sup> The survey is randomised over the whole of England and Wales and so it may be misleading to draw separate conclusions for Wales



**Table 5.8: Logistic regression of ACE counts on upward mobility using only the Wales sample**

Number of Observations		2011			
				LR chi²	113.010
				Prob > chi²	0.000
Log likelihood = -1148.8308				Pseudo R²	0.047
Upward Mobility	Odds Ratio	Std. Err.	Z	P>z	[95% Conf. Interval]
Gender	0.967	0.098	-0.330	0.743	0.792 1.181
Age Categories	1.429	0.049	10.320	0.000	1.335 1.529
Ethnicity	1.000	0.000	0.540	0.592	1.000 1.000
ACE Count Categories	1.078	0.050	1.610	0.107	0.984 1.182
Constant	0.136	0.027	-9.930	0.000	0.091 0.201

The relationship between upward social mobility and ACE counts goes in the same direction as the sample as a whole with higher ACE counts leading to higher upward social mobility. However, the results are not statistically significant at 5% at the level of Wales and so it is not possible to make any claims regarding these results.

The following sections will offer a departure from the strict viewing taken so far of ACE counts and social mobility as a binary variable and investigate what happens when we loosen our definitions of ACE counts and social mobility. For example, if we take ACE counts as a *binary* variable with those with zero ACEs coded as zero and those with any ACEs coded as one a different picture emerges:

**Table 5.9: Regression results for upward mobility and ACEs as a binary variable**

Number of Observations	7429				LR chi <sup>2</sup>	1.350
					Prob > chi <sup>2</sup>	0.246
Log likelihood = -2027.7249					Pseudo R <sup>2</sup>	0.000
	Odds Ratio	Std. Err.	Z	P>z	[95% Conf. Interval]	
ACEs	1.106	0.096	1.160	0.245	0.933	1.312
Constant	0.080	0.005	-42.620	0.000	0.072	0.090

**Table 5.10: Regression results for downward mobility and ACEs as a binary variable**

Number of observations	7429				LR chi <sup>2</sup>	0.450
					Prob > chi <sup>2</sup>	0.502
Log likelihood = -525.59649					Pseudo R <sup>2</sup>	0.000
	Odds Ratio	Std. Err.	Z	P>z	[95% Conf. Interval]	
ACEs	1.146	0.232	0.670	0.501	0.770	1.705
Constant	0.013	0.002	-31.290	0.000	0.010	0.017

**Table 5.11: Regression results for staying poor and ACEs as a binary variable**

Number of observations	7429				LR chi <sup>2</sup>	4.370
					Prob > chi <sup>2</sup>	0.037
Log likelihood = -3517.5979					Pseudo R <sup>2</sup>	0.001
	Odds Ratio	Std. Err.	Z	P>z	[95% Conf. Interval]	
ACEs	1.136	0.069	2.090	0.037	1.008	1.280
Constant	4.266	0.169	36.660	0.000	3.948	4.611

**Table 5.12: Regression results for staying wealthy and ACEs as a binary variable**

Number of observations	7429				LR $\chi^2$	20.510
					Prob > $\chi^2$	0.000
Log likelihood = -2207.9914					Pseudo R <sup>2</sup>	0.005
	Odds Ratio	Std. Err.	Z	P>z	[95% Conf. Interval]	
ACEs	0.684	0.058	-4.470	0.000	0.579	0.808
Constant	0.113	0.006	-42.420	0.000	0.102	0.125

It is seen here that having ACEs is associated with a higher likelihood of experiencing upward social mobility *and* downward mobility but that the relationship is more pronounced with downward mobility (OR: 1.15 vs 1.11). However, these results are not statistically significant with p values above 0.05 and so we cannot make any claims regarding the effect of ACEs on social mobility using these definitions of ACEs and social mobility. The odds of staying poor while having ACEs is greater than one (OR:1.12; 95% CI: 1.056-1.189) signifying that there is a greater likelihood of staying poor if one has any ACEs while the odds of remaining affluent is lower than one (OR: 0.73; 95% CI: 0.109-0.132) meaning that staying affluent is less likely given those that have at least one ACE. These results are statistically significant.

So far social mobility has been a binary poor/affluent variable and ACEs have been treated as ACE *counts* and as binary 0 - no ACEs or 1 - at least 1 ACE. Individuals have moved up the socioeconomic ladder, moved down or stayed where they are. A more sophisticated approach would consider wealth *quartiles* giving more depth to the analysis.

**Table 5.13: Odds ratios for regression of at least 1 ACE on downward social mobility**  
(quartiles)

	Odds Ratio	Std. Err.	z	P> z	95% Confidence Interval	
q4 to q3	0.723	0.453	-0.520	0.605	0.211	2.471
q4 to q2	1	n/a	n/a	n/a	n/a	n/a
q4 to q1	n/a	n/a	n/a	n/a	n/a	n/a
q3 to q2	0.915	0.207	-0.390	0.694	0.587	1.425
q3 to q1	2.790	1.506	1.900	0.057	0.969	8.039
q2 to q1	2.809	0.760	3.810	0.000	1.652	4.774

These results show that movement between specific wealth quartiles produce different results. Movement from 0 to at least 1 ACE increases the odds of moving from the 2nd to the 1<sup>st</sup> quartile (OR: 2.80; 95% CI: 1.652-4.774) with this result displaying statistical significance. It seems therefore that there is some evidence to suggest that moving from 0 to at least 1 ACE is associated with some downward mobility, but this is dependent on where in the wealth distribution the individuals lie. Quartile 3 to 1 is just above the 0.05 threshold for the p-value. In terms of upward mobility, the next table shows the regression results for those moving from 0 to at least 1 ACE:

**Table 5.14: Odds ratios for the regression of at least 1 ACE on upward social mobility**  
(quartiles)

	Odds Ratio	Std. Err.	z	P> z	95% Confidence Interval	
q1 to q2	1.474	0.254	2.250	0.024	1.052	2.066
q1 to q3	1.655	0.302	2.760	0.006	1.157	2.367
q1 to q4	5.067	5.667	1.450	0.147	0.566	45.360
q2 to q3	0.979	0.097	-0.220	0.827	0.806	1.188
q2 to q4	0.422	0.344	-1.060	0.290	0.085	2.090
q3 to q4	0.631	0.224	-1.300	0.194	0.315	1.265

In terms of the effect of ACEs on upward social mobility having ACEs increases the likelihood of being upwardly socially mobile when people are moving from the very poor q1 category. There are statistically significant results for those that move from q1 to q2 and q1 to q3. This result seems to be saying that as the number of ACEs increases there is more likelihood of becoming upwardly socially mobile – a finding that supports the material result that we started with. For those that start at q2 and q3 there is a lower likelihood that they will become upwardly socially mobile, but the results are not statistically significant.

#### 5.6.1 Further analysis

This chapter so far has investigated the properties of considering social mobility as a) a binary variable and b) as quartiles. The last part of this chapter will consider using a wider definition of social mobility and will use wealth in adulthood and wealth in childhood in a different way. To this extent the social mobility variable will be adapted to be the difference between the wealth in adulthood score and the wealth in childhood score. As mentioned at the beginning of this chapter the wealth variable asks respondents to rank their wealth on a Likert scale with 1 being very poor and 10 being very wealthy. If we take wealth in adulthood as the minuend and wealth in childhood as the subtrahend then we get the difference between wealth in adulthood and wealth in childhood. For example, if a respondent's wealth in childhood is 8 and wealth in adulthood is 5 we can say that there has been downward social mobility. Subtracting one from the other gives an 'index' of minus 3 (wealth in adulthood minus wealth in childhood) and so a minus score indicates downward mobility, a positive score denotes upward mobility and a zero signifies no change. The regression chosen to perform this analysis has also changed. Since the dependent variable

is now continuous rather than binary, we can use a straightforward OLS regression assuming the errors are not related to the independent variable.

The following is the least squares<sup>14</sup> regression results from regressing the number of ACE counts on a non-binary measure of social mobility:

**Table 5.15: OLS regression of ACEs on continuous social mobility index**

Source	SS	df	MS			
Model	7475.40115	1	7475.40115	Number of obs =	7429	
Residual	1.7014e+09	7427	229080.189	F( 1, 7427) =	0.03	
				Prob > F =	0.8567	
				R-squared =	0.0000	
				Adj R-squared =	-0.0001	
Total	1.7014e+09	7428	229050.355	Root MSE =	478.62	

socialmobilityindex	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]	
ace_count_categories	-.9616045	5.323203	-0.18	0.857	-11.39659	9.473381
_cons	2.599393	7.00834	0.37	0.711	-11.13894	16.33773

As can be seen this gives us slightly different results to what has already been seen and it is apparent that the direction of travel is different here – the OLS coefficient is negative. In this regression we have that as we increase ACEs there is a commensurate reduction in social mobility. That is, there is more likelihood of *downward* social mobility. A result that appeals to our intuition and seems to be following the findings reported in the literature review. However, it must be noted that these results are not significant. We can therefore surmise that the final result of our analysis is a tenuous one and one that warrants further research.

It may also be a useful exercise to look at what happens to those not born in the UK to see if there are any differences as chapter 4 identifies a large proportion of young people (51.6%)

<sup>14</sup> As the dependent variable is now not a binary variable but a continuous range from -9 to +9

that had lived in the UK for less than 5 years in the two survey areas. Running a regression of 'UK residency' against social mobility with those born in the UK coded as 1 and those that have immigrated to the UK as 0 gives the following results:

**Table 5.16: OLS regression of UK residency status on social mobility index**

Source	SS	df	MS	Number of obs = 7429		
Model	32.7927592	1	32.7927592	F( 1, 7427) = 0.00		
Residual	1.7014e+09	7427	229081.191	Prob > F = 0.9905		
				R-squared = 0.0000		
				Adj R-squared = -0.0001		
Total	1.7014e+09	7428	229050.355	Root MSE = 478.62		

socialmobilityin~x	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]	
UK_Residency_Index	.0023428	.1958096	0.01	0.990	-.3814996	.3861851
_cons	1.823265	5.561927	0.33	0.743	-9.079688	12.72622

We see that there is no significant effect that can be identified here and so we must concede that the results are inconclusive as to any 'residency effect'.

There does not seem to be an effect of ACEs on social mobility therefore, but this depends on how we specify, econometrically, social mobility. To summarise there are three scenarios:

1. Social mobility as binary variable – having ACEs leads to upward social mobility. This is a counter intuitive finding but is statistically significant
2. Social mobility as quartiles – having ACEs has some effect on social mobility but this depends on where in the wealth distribution one starts.
3. Social mobility as a continuous 'index' – having ACEs leads to downward social mobility. This result is intuitive but not statistically significant.

As the analyst I have to make the choice between a result that is counter intuitive but statistically significant and a result that is intuitive but not statistically significant. This is a

difficult choice to make. However, model selection should not be based exclusively on the level of statistical significance of regression coefficients. Hendry (1980) explored the premise of econometrics as a science versus alchemy and his argument was entirely within the field of the above quandary. He specified a regression between money supply and prices and found that the relationship was intuitive but the statistical significance and the 'fit' of the regression was weak. Rather he re-specified the equation using a different explanatory variable. One which was exogenous, in which causality was from the explanatory to the dependent variable only, and that the explanatory variable was outside government control. He ran this regression and achieved excellent significance however the explanatory variable he used, deceptively, was cumulative rainfall! In the words of Hendry,

"It is meaningless to talk about "confirming" theories when spurious results are so easily obtained." (Hendry 1980, p. 395)

It remains the case, however, that in my thesis we still have two conflicting results. One can only suggest that this is a fruitful avenue of further research. A starting point, it is suggested, is to conduct a panel data survey. Although expensive, and it may be appropriate to conduct a Value of Information analysis to see if the gathering of this data would be worthwhile, a panel dataset would remove the limitation of imperfect recall as information on ACEs would be collected when the respondents were children and so one would be able to gather more reliable data about their ACEs. The other aim of further research would be to affirm what is going on with the conflicting evidence proposed by this thesis. It would be possible to look at whether the difference in statistical significance was itself statistically significant. Further, it is likely that the thesis has a mis-specified equation where explanatory variables may be detracting from significance levels of each other, or



there is a missing variable which is being proxied inappropriately. Von Tuzelmann (1968) solves a problem like this, but in time series and using economic history data. He claims that “...further study may reveal that some of the results obtained cannot satisfy closer analysis. It would be unusual for this situation to compel rejection of the results *in toto*, however. In many cases where the assumptions of the statistical procedure are shown to be invalid, there may be a range of techniques supplied by econometric theory to bypass the difficulty e.g., by transforming the variables” (p.176). Increasing the level of rigour, afforded by further research, may lead to further fruitful results or even new relationships. Indeed, it is suggested in Von Tuzelmann (1968) that a good way of approaching this situation is using instrumental variables. This technique uses a third variable, uncorrelated with disturbance errors in the dependent variable, to overcome the interdependence between the independent variable and the disturbance. This may prove a useful exercise in any further analysis. Suffice to say it was beyond the scope of this current thesis, but any further work should definitely look at a) instrumental variables b) conducting a panel survey and c) variable transformation.

Notwithstanding the earlier discussion about significance vs intuitiveness, and the need for further research, the main statistically significant finding in this chapter is that having more ACEs is associated with an increased chance of being upwardly socially mobile. This is a counter intuitive finding. It is worth, I believe, to examine why. As a way of addressing this counter-intuitiveness four explanations are offered here. One may be that children with low ACEs tend to be ‘under the radar’ of social services and not be picked up until they experience a higher number of ACEs. When this is the case, they may be picked up by social services and given the appropriate support that may help them combat their ACEs and go on to lead happy productive lives which may reflect the increase in upward social mobility

we see. The second point is concerned with the way respondents in the survey report their ACEs. One thing that stands out is that the experience of ACE in this data is based on recollection that may introduce noise into the data. Those that are more articulate may describe their memory of the past more clearly. If the survivors are represented in this group disproportionately, perhaps they appear more lucid, the results could be skewed to give the results we have got. Lucidity with which past experience is described may not necessarily be correlated with the accuracy with which the experience is described. Then that introduces noise that may affect the results. Third, adding ACEs together into categories (0,1,2,3,4+) may be problematic because that exercise assumes equivalence between ACEs. As discussed in chapter 1 the fact that we may have synergistic effects from adding ACEs means that any results that are shown have to be treated with caution and not be seen as definitive. Finally, another intuitive way of explaining this is through what is known as resilience. The next section explains exactly what is meant by resilience and how it relates to children growing up with ACEs. The following section will attempt to use this concept to explain why such counter intuitive results are found in the analysis of the data.

### 5.6.2 Resilience

There is a growing literature on resilience that harks back to the pioneering work of such commentators as Professor Sir Michael Rutter in the 1990s. It is a complex concept, one that attempts to describe the conditions under which individuals can thrive *despite* adversity. Its definition is varied but includes such wording as "...the ability to bounce back from adversity, frustration and misfortune" (Ledesma 2014, p1); and "...the capacity of a dynamic system to adapt successfully" (Masten 2014, p6). So, it is about the ability to adapt

to adverse circumstances and achieve good health and economic outcomes despite adversity and this can be done through numerous ways.

Michael Rutter's early work concentrated on the implications of resilience for family therapy. He began his pioneering 1999 paper by describing resilience as the "relative resistance to psychosocial risk experiences" (p119). He claims that children vary in their response to psychosocial risk and that both genetic and environmental influences are at play. Resilience is different from the concepts of social competence (Masten et al 1995), self-efficacy (Bandura 1995, 1997) or of positive mental health (Ryff and Singer 1998).

Indeed, the development of resilience is not necessarily related to positive experiences but rather may be strongly influenced by interpersonal relationships. Rutter referred to a study of children who came from Romanian orphanages and adopted in the UK. Although their adversarial background was the same the difference in outcomes for the children was startling with a wide variation in outcome measures.

Other commentators (Shastri 2013; King 2020) have remarked upon the idea of 'building blocks' that promote resilience. These come in the form of managing adversity, having some degree of control over the adversity, being involved and connected with family, friends, people in the community or online: the connection might involve an interest and hobby, sport, religion, music, walking or many other things, and finally above all else is the availability of at least one stable, caring, and supportive relationship between a child and an adult. That is, having a 'trusted adult' is vital to promote resilience. The notion of a trusted adult refers to a child having an adult in their lives that listens to them without judgement, without an agenda and whose sole purpose is to support and encourage positivity in the

child's life. They come with no expectations of how the child should behave, have clear boundaries, should be a good listener, and should be reliable and consistent.

A report by the National Scientific Council on the Developing Child (2015) states that,

“Whether the burdens come from the hardships of poverty, the challenges of parental substance abuse or serious mental illness, the stresses of war, the threats of recurrent violence or chronic neglect, or a combination of factors, the single most common finding is that children who end up doing well have had at least one stable and committed relationship with a supportive parent, caregiver, or other adult.” (NSCDC 2015; p3)

The pandemic has accentuated the importance of relationships with a ‘supported parent, caregiver or other adult’. These relationships provide a buffer against childhood adversity and help build key capacities such as the ability to regulate behaviour and to adapt to change enabling young people to thrive as adults. Further the *lack* of a ‘trusted adult’ can lead to a toxic stress response akin to ACEs themselves, as the NSCDC has stated,

“the development of healthy brain architecture is influenced by consistent, “serve and return<sup>15</sup>” interactions between young children and their primary caregivers. When these experiences are unavailable or repeatedly disrupted, the body perceives their absence as a serious threat, and activates its stress response systems.” (NSCDC 2015, p4)

This can lead to toxic stress and all the health implications described earlier in this thesis in section 1.7 of Chapter 1. However, the restoration of a trusted adult can turn this toxic stress back into tolerable stress and the balance is restored in individuals.

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<sup>15</sup> “Serve and return” interactions uses the analogy of a game of tennis and refers to when a child “serves” by reaching out for an interaction by, for example, making eye contact or speaking and the adult “returns” this by engaging with the child

The dataset used in this chapter contains a variable that relates to the existence of a 'trusted adult' in the lives of the respondents. It is a variable denoted by the values 0, 1 and 2 where zero is no trusted adult, 1 is a trusted adult some of the time and 2 is a trusted adult all of the time. Running a regression with the trusted adult variable as a regressor will provide us with an idea of how this variable affects mobility. The only limitation of using this method is that the quality of that trusted adult is not captured. Some individuals may have a better experience of having a trusted adult than others. This nuance in quality cannot be captured using the data we have and rather the trusted adult variable is reduced to a binary some of the time/all of the time outcome variable with no mention of the qualitative 'story' behind each individual's experience which may vary from individual to individual. However, this variable can be used as a measure of resilience against the exposure to the risk factors associated with ACEs and the next section examines this in more detail.

### 5.6.3 Trusted adult to explain counter intuitive ACEs finding

To test the relationship between having a trusted adult, ACEs, and upward social mobility a logistic regression was run with an interaction term. This was simply calculated by multiplying ACE counts with the trusted adult variable. This gave a variable which reflected having ACEs *and* having a trusted adult. The definition of ACEs as a binary 1 0 variable with 1 as having at least one ACE will be used here. The results are shown in the table below:

**Table 5.17: Logistic regression for upward mobility using an interaction term for trusted adult and ACEs as a binary variable**

Number of observations	7410					
				LR chi <sup>2</sup>	14.320	
				Prob > chi <sup>2</sup>	0.003	
Log likelihood = -2017.2296				Pseudo R <sup>2</sup>	0.004	
upward mobility	Odds Ratio	Std. Err.	z	P>z	[95% Conf. Interval]	
ACEs	0.999	0.096	-0.010	0.990	0.828	1.205
Trusted Adult	0.859	0.051	-2.550	0.011	0.764	0.965
Interaction	1.051	0.021	2.480	0.013	1.011	1.093
Constant	0.097	0.009	-26.310	0.000	0.081	0.115

The interaction variable can be interpreted as follows: having more ACEs *and* a trusted adult is associated with a higher chance of upward mobility (OR: 1.051; 95% CI: 1.011-1.093). This finding shows that individuals in the sample prove to have upward mobility *despite* having ACEs and that this can be explained by them having a trusted adult that help individuals mitigate against the harmful effect of ACEs. This result is statistically significant at the 95% level with a p value of 0.013.

## 5.7 Discussion

Upward social mobility can be seen as ‘improving one’s lot in life’ by going from one social class as a child to higher one in adulthood. ACEs may be a barrier to upward social mobility as the health effects of ACEs may be detrimental to one’s ability to earn in the labour market and so the ability to move upwardly into a higher social class may be hindered. General findings in the field of ACEs and social mobility reinforce this view as seen in the literature review in section 5.3. However, some of the findings in this thesis tend to go

against the conventional wisdom. Here it is the case that, if we look at ACE *counts* and define social mobility as a binary variable, there is a statistically significant higher likelihood that upward mobility occurs if there is more ACEs, a result that is counter intuitive. Looking at different ways of defining social mobility and ACEs somewhat alleviates this counter intuitiveness but doesn't get rid of it entirely. This paradoxical result may indeed be correct, but it cannot be confirmed without further investigation. For example, adding up the number of ACEs as done in the regressions above are based on a problematic assumption that the ACEs are similar. For example, being physically abused in childhood may be a class apart as an ACE than having parents who have divorced. Then there is another problem. Omitted variables can result in false signs and significance levels of coefficients for measures of ACEs that are used. It is a problem in these kinds of studies. Further research may have to be undertaken before devising policies based on these regressions. For example, the construction of a database, preferably a longitudinal cohort study containing questionnaires ensuring that the proxy variables for ACEs are indeed correctly specified. This kind of a study requires careful consideration and could prove expensive. However, a focussed study to examine the impact of some of the major childhood disadvantages is recommended before policy prescriptions are suggested.

A further attempt at defining social mobility was made in the final part of the results section. It was defined as wealth in adulthood minus wealth in childhood. This gave a non-binary continuous 'index' of social mobility with negative values indicating downward mobility, positive values indicating upward mobility and zero signifying no change. The results of running a regression on this index were inconclusive. Although the coefficient was negative, indicating an increase in ACEs led to downward mobility this was not statistically significant at the 95% level and so it had to be argued that no effect was found. The R

squared in this regression was very low (zero to four decimal places) and so any further research would have to bear this in mind and may want to look for alternative measures of social mobility as well as considering additional co-variables in models where these are available. Different ways of measuring social mobility (e.g. occupation, income) have been discussed in section 5.3 and any future research may want to use some of these methods if the data allows it. These alternative measures may provide very different results from those reported in this thesis.

This analysis chapter concludes by examining the relationship between ACEs, social mobility and having a trusted adult, which contributes to a notion of resilience. It is found that individuals in the sample achieve upward mobility *despite* having ACEs and this is because they have a trusted adult in their life which enables resilience to adversity and an opportunity to become upwardly mobile despite adversity.

Perhaps the main limitation of this chapter is that the analysis was restricted to the areas of Wales and Southern England. Although the areas Blackburn with Darwen and England as a whole were available the questions on wealth in childhood and adulthood were not asked in these areas and so analysis wasn't possible. Another limitation was the crudeness of the variable used to define social mobility. The questions on wealth asked the respondents to rank their wealth in childhood and adulthood on a scale of 1 to 10, with 1 being very poor to 10 being very wealthy. This means of measurement of someone's social status is very subjective and more detailed information on, perhaps, income would have been a more sophisticated way to measure social mobility.



## 5.8 Conclusion and brief policy recommendations

The results I have obtained are robust in the sense that they point clearly towards a case for undertaking further research, namely a longitudinal or panel survey, with a focus on collecting better proxy variables on ACE at childhood and impact (wealth, earnings, educational attainment) in adulthood. They are not robust in the sense of being able definitely to reject counter-intuitive results or definitely to rejoice over intuitively plausible coefficients.

In this chapter two issues have been addressed. The general relationship between ACEs and social mobility and more specifically what happens to social mobility when we have a trusted adult. To give some context to this finding a straightforward logistic regression was performed of the number of ACEs on different definitions of social mobility. Firstly, social status was defined as a binary variable (poor/affluent) with mobility occurring in four possible states: individuals had become upwardly socially mobile (going from poor to affluent), downwardly socially mobile (going from affluent to poor), had stayed poor or stayed wealthy. The results showed that, if we also code ACEs as a binary yes/no variable, having any ACEs is associated with a higher odds of downward mobility than is the case with upward mobility but these results were not statistically significant. There is also a higher odds of remaining poor and a lower odds of remaining wealthy. This result is sensible and appeals towards our intuition. However, if other definitions of mobility and ACE *counts* are used the results vary and start to become counter-intuitive. When using ACE counts there is a higher odds of becoming upwardly socially mobile with more ACEs. This may partly be explained by there being a greater level of social services intervention the more ACEs an individual has. Another explanation posited here is that the presence of a trusted adult

contributes to resilience and that people are becoming upwardly mobile *despite* having ACEs. This finding would point towards a policy recommendation of investing in interventions to increase and improve the role of trusted adults in our society. This recommendation will be developed further in the next chapter which will look at the policy environment around ACEs and possible further policy recommendations involved with specific interventions to reduce the effect of ACEs on the lives of individuals. The final policy recommendation is in terms of data collection. It is suggested that a longitudinal dataset is established to gather information about ACEs and social mobility. Although expensive this type of resource would be invaluable to future researchers that are looking at these relationships. It may be the case that, with this type of data, the counterintuitive results may either disappear or be explained better. There is definitely a need for further study in this area, but this chapter has provided a useful place to start from in looking at these issues.

## Chapter 6 – Conclusions and recommendations

### 6.1 Summary

Looking back, Felitti's (1998) pioneering work identified that ACEs were prevalent in society. Perhaps more prevalent than previously thought. Half of the study's participants had experienced at least one ACE with a further quarter having experienced two or more. The study's authors found a significant dose-response relationship between the number of ACE exposures and health risk factors (e.g., smoking, obesity, depression, sexually transmitted diseases), and with specific health outcomes (e.g., heart disease, cancer, skeletal fractures). The conclusion of the researchers were that ACEs were cumulative in their impact over individuals' lifetime and that they contributed to negative outcomes in terms of physical and mental health along with negative behavioural outcomes in adulthood and even premature death.

ACEs affect the development of the early brain, and this is done through what is called 'toxic' stress. Toxic stress, as set out in Chapter 1, is defined by Shonkoff (2012) as, "...prolonged activation of the body's stress response systems in the absence of the buffering protection of a supportive, adult relationship." (Shonkoff 2012, p. 236)

As children are exposed to abuse, neglect, and household dysfunction they experience this toxic stress, and this regulates how much cortisol is released in the brain triggering a fight-flight-freeze response. The crux here is that children who are repeatedly exposed to such stress end up with a continuously higher allostatic load and allostasis does not fall back to pre-abuse levels. There are four main ways in which ACEs can affect lifetime health.

Directly, by a process known as biological embedding which includes physical health being affected by changes in the early brain. And indirectly through health harming behaviour such as smoking, drug taking, alcoholism, obesity and so on. Other means of ACEs affecting health are through the immune system and telomere erosion.

The aim and purpose of this thesis was threefold: (1) to look at if it is possible to use available data to estimate the cost of ACEs; (2) to investigate the relationship between ACEs and standard of living through social mobility; and (3) to investigate whether this relationship can be mitigated by access to a 'trusted adult'.

It was noted that although there are many studies looking at the health effects of ACEs there is a paucity of studies that look specifically at costs. Chapter 2 was the beginning of this work and in this vein, a systematic review was conducted and presented gathering evidence on:

- a) The odds ratios of how much of these diseases are caused by ACEs
- b) The cost of illness of certain diseases that can be caused by ACEs

The five main diseases in terms of expenditure in the UK were looked at: cancer, mental health, circulatory disease, musculoskeletal disease and genitourinary disease. Insofar as ACEs have an association with these diseases it was considered a useful exercise to attempt to put a cost on these diseases and to calculate how much of this cost is attributable to ACEs. The results showed a wide variety of odds ratios for the different types of conditions as well as a range of costs pertaining to these diseases. There was a lack of data, especially for the costs of genitourinary disease and the odds ratios for both musculoskeletal and genitourinary disease. The aim, however, was not to establish odds ratios and costs per se

but rather to use this information to calculate the proportion of costs that could be attributed to ACEs, and this was the content of chapter 3.

Chapter 3 used populations attributable fractions (PAFs) to calculate the costs associated with ACEs in accordance with the methodology used in Fang (2015a). There were gaps in the data as identified in chapter 2, the systematic review, and these needed to be addressed. This thesis came up with a novel methodology to do this, which was based upon a basic extrapolation technique. The data on costs of diseases that *was* available was taken and the missing data was completed using extrapolation. For example, with cancer, there was data available for some types of cancer but not all. The costs for the types of cancer where there was no data were extrapolated based on the incidence of the types of cancer under scrutiny. Therefore, it was possible to calculate an overall cost for cancer which could be used later to calculate the cost of cancer associated with ACEs. It was calculated that the lifetime costs, per person, of all types of cancer was £113,850. The same methodology was conducted for mental illness, and it was calculated that the per person, lifetime costs of mental illness was £63,165.

There were some areas where data was not available. Further research is needed to identify the odds ratios associated with musculoskeletal and genitourinary disease as well as on the costs of genitourinary disease. The lifetime attributable cost of cancer for those with more than 4 ACEs was £13,791 while the attributable cost of depression for those with four or more ACEs was £42,453. The main finding was that costs reduced as we go from 1 to 2 ACEs before increasing again as a function of ACEs. This seems counter-intuitive as one would expect costs to be a positive function of ACEs. This may be an anomaly, or it may be a significant trend. Further work may be required to establish if this is the case.

With a view to exploring goals two and three of this thesis a precursor to chapter 5 was a data description chapter. This chapter went into detail to describe the dataset used for the thesis. It produced descriptive statistics and identified interesting trends within the data. The dataset covers four different areas of the UK: Blackburn with Darwen, Wales, England and South England defined by Hertfordshire, Luton and Northamptonshire and has information on 13,130 participants. Only two areas were used in the analysis – Wales and Southern England. This is because the variable ‘wealth in childhood’ and ‘wealth in adulthood’, the basis of our definition of social mobility, was only collected for these areas. The main analysis chapter was then written to investigate the relationship between social mobility and ACEs. Social *status* was defined according to the APA as “the social standing or class of an individual or group” while social *mobility* was defined as “the simple difference between an individual’s socio-economic position in adulthood and that of his or her parent(s) when the individual was a child.” (Buscha 2019, p156). There is a link between the costs of ACEs and living standards insofar as living standards largely decide the level of taxation available to invest in programmes to reduce ACEs. The costs associated with ACEs can therefore be, at least partially, offset by the economic benefits of upward social mobility and it is this concept that underpins this thesis. Namely that ACEs seem to lead to upward social mobility which in turn leads to increased tax revenues that can then be spent on combating the very diseases that are partially caused by ACEs. This cyclical motion within the system can be seen as creating a virtuous cycle out of a vicious cycle of ACEs leading to poor health and therefore lower income. The fact that, together with actioning a notion of a trusted adult, ACEs seem to lead to upward social mobility leads to this ‘virtuous cycle’ whereby improved wealth in general creates the funds necessary to tackle the problems caused by ACEs. This thesis does not argue *against* tackling the root causes and ACEs

themselves. Tackling ACEs and reducing their prevalence can have a positive economic impact in terms of lowering the incidence of some of the diseases partially caused by ACEs and hence lowering the expenditure on these diseases. But this thesis argues that there is an element of self-sufficiency here and that the process is in a way Laissez-Faire in that it is self-correcting with higher expenditure diseases being paid for by the gains in social mobility afforded by children having a trusted adult. This thesis suggests therefore that one way of reducing the effect of ACEs on society is to invest community resources in aiding and nurturing these 'trusted adults' within society because their influence can be substantial and help offset some of the negative impacts of ACEs on society in general. It is in this vein that this chapter was written to analyse the relationship firstly between ACEs and social mobility and secondly what happens to social mobility as we go from 0 to at least 1 ACE? When ACEs were defined as categories i.e. 0, 1, 2 to 3 4+ the results were counter intuitive with an increase in ACEs associated with an increase in upward social mobility and a reduction in downward social mobility. However, when we looked at ACEs as a binary yes/no variable this produced different results. There seemed to be an increase in downward social mobility as ACEs increased but simultaneously there was an increase in upward mobility but to a lesser extent than downward mobility. However, if the data is coded into quartiles, then some subtleties show themselves and it seems that there is some evidence to suggest that moving from 0 to at least 1 ACE is associated with some downward mobility, but this is dependent on where in the wealth distribution the individuals lie. Further analysis was undertaken that defined a new social mobility variable. This was calculated as the difference between the wealth in adulthood score and the wealth in childhood score. An index ranging from -10 to +10 was then created with negative values signifying downward mobility, positive values upward mobility and zero no change. It was

found that the relationship between this variable and ACEs was a negative one suggesting that an increase in ACEs led to an increase in downward social mobility. However, this result was not significant and so it should not be taken as definitive.

The main finding in this chapter however was that as ACEs increase there is a higher likelihood of upward social mobility. This counter intuitive finding was explained in four ways. First it was noted that individuals were not picked up by Social Services until they demonstrated that they had several ACEs and that this support mitigated against the harmful effect of ACEs. Secondly, there may be recall bias occurring here where individual respondents to the survey cannot elucidate their experiences correctly introducing a certain subjectivity into the data. Third, there may be an omitted variable process going on in the data whereby omitted variables can result in false signs and significance levels of coefficients for measures of ACE that are used. Lastly, by individuals having a 'trusted adult' in their lives that contributed to resilience against the effect of ACEs. A regression was run with the variables 'trusted adult' and ACE counts defined as an interaction term. This allowed the investigation to establish the 'trusted adult' variable as an explanatory variable as opposed to mere correlation.

An explanation for the main finding in this thesis was then posited. It was suggested that the tendency of the sample to become upwardly socially mobile with more ACEs could be explained by the role of a trusted adult in the lives of those individuals. A regression was run in order to ascertain this relationship and it was found that having a trusted adult in addition to more ACEs increased the likelihood of upward social mobility and so the presence of a trusted adult could indeed mitigate against the harmful effect of ACEs.



## 6.2 Policy context

There are no specific policy directives relating exclusively to ACEs as they are currently understood. Rather there is a wide range of policy orientation emanating from the UN Convention on the Rights of the Child (UNICEF 1989). This is the most comprehensive statement on the rights of the child and is the most widely endorsed human rights treaty relating to children in history. Article 3 of the convention states that “In all actions concerning children, whether undertaken by public or private social welfare institutions, courts of law, administrative authorities or legislative bodies, the best interests of the child shall be a primary consideration.” Thus, decision and policy makers should consider the well-being of children in every decision they make. Therefore, the rights and the welfare of children must be considered in every policy formulation at every level of government from economic, through to social, health, education and environmental policy making. In Wales this is covered by the 2015 Well-being of Future Generations Act which states the need that the welfare of children is built into policy making at every level.

It is perhaps article 19 and article 39 in the UN Convention that are most relevant to ACEs as they state respectively that a) children should be protected from all forms of violence and that “States Parties shall take all appropriate legislative, administrative, social and educational measures to protect the child from all forms of physical or mental violence, injury or abuse, neglect or negligent treatment, maltreatment or exploitation, including sexual abuse, while in the care of parent(s), legal guardian(s) or any other person who has the care of the child” and b) child victims have the right to be rehabilitated and that “States Parties shall take all appropriate measures to promote physical and psychological recovery

and social reintegration of a child victim of: any form of neglect, exploitation, or abuse; torture or any other form of cruel, inhuman or degrading treatment or punishment; or armed conflicts. Such recovery and reintegration shall take place in an environment which fosters the health, self-respect and dignity of the child.” It can be seen therefore that most specific policy relating to children is concerned with child protection (which includes intervention after the maltreatment has occurred) or prevention of child maltreatment.

### 6.2.1 European Union Policy

As a result of the Lisbon Treaty, it has been a specific EU objective to protect children from all forms of maltreatment (Dimitrova-Stull 2014). Child protection systems are mainly the responsibility of individual member states, but the EU takes an overarching role in keeping with its mandate to protect children. Its main functioning in this respect is to protect the rights of the child while the responsibility of protecting children lies with member states.

The European Commission laid out in its ‘Communication on Strategic Objectives 2005-2009’ that priority should be given to ‘effective protection of the rights of children, both against economic exploitation and all forms of abuse, with the Union acting as a beacon to the rest of the world’ (COM 2005). From this came both the Strategy on the Rights of the Child and the Agenda for the Rights of the Child. The Strategy on the Rights of the Child (2006) is based on specific objectives and is carried out through tangible measures. These measures include an EU wide single number telephone helpline for children (116 111), a missing child hotline (116 000) and the gathering of comparable EU-wide data.

The Agenda for the Rights of the Child (2011) was based around three guiding principles: making the rights of the child an integral part of the EUs fundamental rights policy; basing future policymaking on reliable data; and cooperating with stakeholders through the

European Forum on the Rights of the Child. There are four priority areas to the agenda, which are: child-friendly justice, vulnerable children, children in the EU's external action and child participation and awareness raising.

### 6.2.2 UK Policy

In the UK each devolved nation is responsible for its own policies and laws around safeguarding and child protection although these are based on similar principles in each country. The current state of play regarding child protection in the UK has come about after a series of high-profile incidents involving specific cases which can be described as follows and is taken from NSPCC (2016):

**1945** The first formal child death inquiry in England was the Curtis Committee Report into the death of Dennis O'Neill, who was killed at the age of 12 by his foster father.

**1973** The death of 7-year-old Maria Colwell led to the establishment of our modern child protection system.

**1984** Further changes were prompted partly by the inquiries into several other child deaths, including 4-year-old Jasmine Beckford.

**1989** The Children Act 1989 established the legislative framework for the current child protection system in England and Wales. The Children (Northern Ireland) Order 1995 and the Children (Scotland) Act 1995 set out the same for the other UK nations.

**2000** The death of 8-year-old Victoria Climbié led to Lord Laming's report which led to sweeping changes to the way children's services were structured in England and Wales.

**2002** The deaths of 10-year olds Holly Wells and Jessica Chapman in Soham led to the strengthening of legislation across the UK to protect children from adults who pose a risk to them.

Some of the most relevant laws to be passed by the Westminster government include the Children Act (2004). This strengthens the 1989 Children Act, which held central key principle was the paramount importance of the child's welfare. It created the role of children's commissioner for England, made local authorities appoint a director of children's services, responsible and accountable for delivery of services, and made sure that any injury to a child resulting in actual bodily harm could not be viewed as 'reasonable punishment'.

### 6.2.3 Policy in Wales

In Wales, child protection is the responsibility of the Welsh government. There are broad similarities between the Welsh and English child protection systems but in April 2016 the Social Services and Well-being (Wales) Act 2014 came into force meaning that Wales now has its own framework for social services. Any child protection issues that end up in court will still be treated according to English law, however.

In 2004 the Welsh government released the 'Children and young people: rights to action' framework which served as guidance for service providers to enhance the lives of children in Wales. It has seven core aims, which are all guided articles in the United Nations Convention on the Rights of the Child (UNCRC). These are, according to the NSPCC (2006), to:

- have a flying start in life.
- have a comprehensive range of education and learning opportunities.

- enjoy the best possible health and be free from abuse, victimisation, and exploitation.
- have access to play, leisure, sporting, and cultural activities.
- be listened to, treated with respect, and have their race and cultural identity recognised.
- have a safe home and a community which supports physical and emotional wellbeing.
- not be disadvantaged by poverty.

In addition, the Rights of Children and Young Persons (Wales) Measure 2011 made Wales the first nation in the UK to enshrine the UNCRC into law, making the rights of the child central to all policy and legislation in Wales.

### **6.3 Policy recommendations**

Currently there is a gap between what the science tells us about ACEs and the policies intended to reduce them or lessen their impact (NSCDC (2015)). Often the failure to succeed is seen as the fault of the individual, and economic, social and health policies fall short of attempting to create conditions under which resilience can develop. Removing the child from unsafe environments, for example, fails to see how improving the relationships within that environment and generating the building blocks of resilience can improve outcomes for those children. The role of a trusted adult is integral to this idea of resilience whereby children can undo some of the damage caused by ACEs by having someone to turn to. It is seen that merely the existence of a trusted adult is sometimes enough, or 'knowing that someone is there' should they need support even though actual support is not given at

that time, but it is the ‘knowing’ that is important and helps children control the toxic stress caused by ACEs.

Some poverty reduction policies fail to consider access to affordable, high-quality childcare when focusing on providing work for the parents. In this way they miss the chance of promoting both adult self-sufficiency and providing supportive experiences for their children. In this way these policies fail to see the link between huge economic costs to society of ACEs, social mobility and having a trusted adult.

The results posited in this thesis can be used to provide some policy recommendations around approaches to dealing with ACEs especially considering improving social mobility.

The main recommendation here is to design policies to enhance ‘serve and return’ interactions between children and their caregivers. These ‘serve and return’ interactions can take the form of communication and engagement between children and adults and the lack of these interactions can lead to the child developing toxic stress. It is important therefore to nurture these interactions although it may be difficult to design interventions that improve or increase the occurrence of these interactions. As stated in the NSCDC (2015) report,

“Recognizing the critical role of these interactive capabilities provides a strong incentive for developing new intervention strategies that explicitly target adult skill-building to improve the quality of adult-child relationships in order to improve life outcomes for vulnerable children.” (NSCDC (2015), p.9)

Finally, and perhaps the main policy recommendation, is that of investing in developing ‘external’ trusted adults. That is, identifying adults in the community that could perform the role and investing in developing this community resource so that children without the

presence of a trusted adult in their life can have access to the mitigating effects that a trusted adult would bring. This could be in different areas of life such as music and the creative arts in general and sport. An external trusted adult would be a well-known community figure that has a proven record of being a trusted adult to children. This resource could be tapped into to bring the benefits that a trusted adult can bring to a wider population of troubled children. Care must be taken however in designing these policies as the role of an external trusted adult could be exploited with those intent on harming children falsely getting into these roles. Sufficient vetting systems should be totally stringent, and it should not be possible for inappropriate adults to be in this role within communities.

To conclude, therefore, the area of ‘trusted adults’ can be seen as ripe for very interesting further research. It would be very useful to understand the societal costs and benefits of trusted adults in terms of perhaps looking at economic evaluations of interventions that promote the function of trusted adults in society. Another valuable addition to the literature would be a study on the availability of trusted adults and how to invest in fostering these people to become role models in communities. These research topics were beyond the scope of this thesis but would add a great deal to the debate.

## **6.4 Reflections on the thesis**

This thesis, in accordance with being ‘dispassionate’, only looks at the facts associated with ACEs and does not try to make any claims as to what the level of ACEs *should* be in society which would be the usual, normative, economic approach.

The existence of cost information can be very useful for conducting economic evaluation of interventions that may address the issues raised by children suffering ACEs. The cost side of any potential cost effectiveness, cost utility or cost benefit analysis is essential for providing reliable ICER estimates concerning any potential interventions. Another useful role of economic costs is to aid the government in terms of knowing how much of a problem ACEs posit. It gives an idea of the outlay needed by government to tackle the problems caused by ACEs. In the early stages of the PhD there were no studies that approached the subject of putting costs on diseases that were, partly, caused by ACEs and the first half of this thesis attempted to fill that gap in the knowledge by providing robust cost estimates that could then be used by government and in economic evaluation of interventions aimed at reducing the impacts of ACEs.

An exercise in identifying the attributable costs of ACEs was performed in this thesis asking the question if it was possible to put an attributable cost on ACEs. It was found that there was sufficient data for this to be possible for cancer, mental health and circulatory disease but the information was lacking to do the same for the other categories of disease, namely musculoskeletal and genitourinary disease. To build a fuller picture therefore of ACEs affecting the top five diseases in terms of their costs further research may be necessary.

One way of preventing ACEs from being passed down the generations is through social mobility. This thesis explored how individuals responded to a difficult start in life by looking at how they fared into adulthood in terms of their wealth in adulthood compared with the family's wealth when they were children. The thesis concludes that a relationship could be seen between having more ACEs and experiencing *upward* social mobility. That is, individuals seemed to be achieving upward social mobility *despite* having ACEs in their



childhood. This result ties in well with the context and concept underpinning the thesis.

Insofar as improved social mobility of children, *despite* ACEs, is the case, it is argued that this will lead to higher tax returns in future for government to use to invest in programmes a) to reduce the prevalence of ACEs and b) to improve resilience in children by investing in resources to develop ‘trusted adults’ within communities.

#### 6.4.1 Contribution to body of knowledge

In this thesis I have contributed to the body of knowledge in three ways:

- Have pushed back the frontiers of knowledge about the odds ratios and costs of diseases partly caused by ACEs by conducting a systematic review in this area.
- Have furthered the gathering of costs of ACEs.
- Have expanded the knowledge around the relationship between ACEs and social mobility.

### 6.5 Conclusion

In this thesis I have had to distinguish between a reductionist view and an empirical view of the world and I have used both approaches. Reductionist in terms of reducing the problem down to its constituent parts in order to perform analysis. And empirical in terms of that it has attempted to find the direction of travel in a hypothesised relationship and to establish if this is statistically significant. I have found that I can neither “reject the implausible”, nor “rejoice in the significant”. This ambiguity could be resolved with further study, and I believe that this thesis has provided the case for this further research. This thesis has clearly shown the dilemma that is present and has offered insights into how to resolve this dilemma.

It has been established that ACEs carry substantial costs both to individuals and to society. The measurement of these costs has gained little attention until very recently and when this thesis was begun there were no studies looking at the costs of ACEs to society. In this thesis I aim to contribute to the body of knowledge specifically in terms of the attributable cost of ACEs to the top five diseases in terms of expenditure in the UK. I've expounded this body of knowledge in the two systematic reviews that I have conducted. The work pointed out some areas where further research might be necessary where data on costs and/or odds ratios were not available and so for musculoskeletal and genitourinary disease the calculation of attributable costs was not possible. The thesis did find the lifetime costs, per person attributable to ACEs of Cancer, Mental Illness and Circulatory Disease which were £113,850, £63,165, and £41,805 respectively.

Also identified in this thesis was the relationship between social mobility and ACEs. It was established that further research is needed to examine in more detail what is happening in terms of the direction of travel of this relationship and the significance of the results. It has been proposed that a longitudinal study should be conducted which would give valuable data on ACEs as well as measures of wealth and income so that the contradiction in the results may be resolved.

Notwithstanding this it is seen that developing the role of the 'trusted adult' is seen as key in providing children with an element of resilience against the harmful effect of ACEs. This thesis argues that if the role of trusted adults can be maintained and developed then this can protect children against the possibility of downward social mobility and even lead to upward social mobility. That is, children can 'move on up' despite having several ACEs, if

given the right support with economic benefits both to themselves and the economy as a whole.

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## Appendix 1 – Systematic Review Protocol

**PROSPERO**  
International prospective register of systematic reviews

**NHS**  
National Institute for  
Health Research

**UNIVERSITY of York**  
Centre for Reviews and Dissemination

### Systematic review

#### 1. \* Review title.

Give the title of the review in English

Systematic review of evidence relating to the health economics of interventions to reduce the effects of adverse childhood experiences

#### 2. Original language title.

For reviews in languages other than English, give the title in the original language. This will be displayed with the English language title.

#### 3. \* Anticipated or actual start date.

Give the date the systematic review started or is expected to start.

03/10/2016

#### 4. \* Anticipated completion date.

Give the date by which the review is expected to be completed.

31/03/2017

#### 5. \* Stage of review at time of this submission.

Tick the boxes to show which review tasks have been started and which have been completed. Update this field each time any amendments are made to a published record.

**Reviews that have started data extraction (at the time of initial submission) are not eligible for inclusion in PROSPERO.** If there is later evidence that incorrect status and/or completion date has been supplied, the published PROSPERO record will be marked as retracted.

This field uses answers to initial screening questions. It cannot be edited until after registration.

The review has not yet started: Yes

**PROSPERO**  
International prospective register of systematic reviews



Review stage	Started	Completed
Preliminary searches	No	No
Piloting of the study selection process	No	No
Formal screening of search results against eligibility criteria	No	No
Data extraction	No	No
Risk of bias (quality) assessment	No	No
Data analysis	No	No

Provide any other relevant information about the stage of the review here.

It will be a chapter in my PhD thesis

It will be a chapter in my PhD thesis

**6. \* Named contact.**

The named contact is the guarantor for the accuracy of the information in the register record. This may be any member of the review team.

Mr Lloyd-Williams

**Email salutation (e.g. "Dr Smith" or "Joanne") for correspondence:**

**7. \* Named contact email.**

Give the electronic email address of the named contact.

huw.l.williams@bangor.ac.uk

**8. Named contact address**

Give the full institutional/organisational postal address for the named contact.

36 Stryd Dinorwig, Caernarfon, Gwynedd

**9. Named contact phone number.**

Give the telephone number for the named contact, including international dialling code.

+44 (0)1248382273

**10. \* Organisational affiliation of the review.**

Full title of the organisational affiliations for this review and website address if available. This field may be completed as 'None' if the review is not affiliated to any organisation.

Bangor University

**Organisation web address:**

**11. \* Review team members and their organisational affiliations.**

Give the personal details and the organisational affiliations of each member of the review team. Affiliation refers to groups or organisations to which review team members belong. **NOTE: email and country now MUST be entered for each person, unless you are amending a published record.**

Mr Huw Lloyd-Williams. Bangor University

**12. \* Funding sources/sponsors.**

Details of the individuals, organizations, groups, companies or other legal entities who have funded or sponsored the review.

Bangor University

**Grant number(s)**

State the funder, grant or award number and the date of award

**13. \* Conflicts of interest.**

List actual or perceived conflicts of interest (financial or academic).

None

**14. Collaborators.**

Give the name and affiliation of any individuals or organisations who are working on the review but who are not listed as review team members. **NOTE: email and country must be completed for each person, unless you are amending a published record.**

**15. \* Review question.**

State the review question(s) clearly and precisely. It may be appropriate to break very broad questions down into a series of related more specific questions. Questions may be framed or refined using PI(E)COS or similar where relevant.

To systematically review the evidence on the health economics of interventions to reduce the effects of adverse childhood experiences

**16. \* Searches.**

State the sources that will be searched (e.g. Medline). Give the search dates, and any restrictions (e.g. language or publication date). Do NOT enter the full search strategy (it may be provided as a link or attachment below.)

CINAHL, MEDLINE, PubMed Central Open Access, Cochrane Library (EED, Dare, HTA), ASSIA, PROQUEST.

Exclusions:

Adults 18 years of age

#### 17. URL to search strategy.

Upload a file with your search strategy, or an example of a search strategy for a specific database, (including the keywords) in pdf or word format. In doing so you are consenting to the file being made publicly accessible. Or provide a URL or link to the strategy. Do NOT provide links to your search **results**.

Alternatively, upload your search strategy to CRD in pdf format. Please note that by doing so you are consenting to the file being made publicly accessible.

Yes I give permission for this file to be made publicly available

#### 18. \* Condition or domain being studied.

Give a short description of the disease, condition or healthcare domain being studied in your systematic review.

Adverse childhood experiences (ACEs)

#### 19. \* Participants/population.

Specify the participants or populations being studied in the review. The preferred format includes details of both inclusion and exclusion criteria.

Inclusion: Children who have suffered ACEs 18 years of age

Exclusion: Adults 18 years of age

#### 20. \* Intervention(s), exposure(s).

Give full and clear descriptions or definitions of the interventions or the exposures to be reviewed. The preferred format includes details of both inclusion and exclusion criteria.

Interventions aimed at reducing the effect of adverse childhood experiences. For example: Family Support, Child Support and Education, Increasing identification of child maltreatment, Community and societal interventions.

#### 21. \* Comparator(s)/control.

Where relevant, give details of the alternatives against which the intervention/exposure will be compared (e.g. another intervention or a non-exposed control group). The preferred format includes details of both inclusion and exclusion criteria.

Usual care

#### 22. \* Types of study to be included.

Give details of the study designs (e.g. RCT) that are eligible for inclusion in the review. The preferred format includes both inclusion and exclusion criteria. If there are no restrictions on the types of study, this should be stated.

Economic evaluations, clinical studies

### 23. Context.

Give summary details of the setting or other relevant characteristics, which help define the inclusion or exclusion criteria.

### 24. \* Main outcome(s).

Give the pre-specified main (most important) outcomes of the review, including details of how the outcome is defined and measured and when these measurement are made, if these are part of the review inclusion criteria.

ACE scores

#### \* Measures of effect

Please specify the effect measure(s) for you main outcome(s) e.g. relative risks, odds ratios, risk difference, and/or 'number needed to treat.

### 25. \* Additional outcome(s).

List the pre-specified additional outcomes of the review, with a similar level of detail to that required for main outcomes. Where there are no additional outcomes please state 'None' or 'Not applicable' as appropriate to the review

None

#### \* Measures of effect

Please specify the effect measure(s) for you additional outcome(s) e.g. relative risks, odds ratios, risk difference, and/or 'number needed to treat.

### 26. \* Data extraction (selection and coding).

Describe how studies will be selected for inclusion. State what data will be extracted or obtained. State how this will be done and recorded.

### 27. \* Risk of bias (quality) assessment.

State which characteristics of the studies will be assessed and/or any formal risk of bias/quality assessment tools that will be used.

Risk of bias will be assessed using Cochrane's 'Domain Based Evaluation' tool.

### 28. \* Strategy for data synthesis.

Describe the methods you plan to use to synthesise data. This **must not be generic text** but should be **specific to your review** and describe how the proposed approach will be applied to your data. If meta-analysis is planned, describe the models to be used, methods to explore statistical heterogeneity, and software package to be used.

A quantitative synthesis is proposed for the cost data. Also a narrative synthesis is planned.

**29. \* Analysis of subgroups or subsets.**

State any planned investigation of 'subgroups'. Be clear and specific about which type of study or participant will be included in each group or covariate investigated. State the planned analytic approach.

None planned

**30. \* Type and method of review.**

Select the type of review, review method and health area from the lists below.

**Type of review**

Cost effectiveness

No

Diagnostic

No

Epidemiologic

No

Individual patient data (IPD) meta-analysis

No

Intervention

No

Meta-analysis

No

Methodology

No

Narrative synthesis

No

Network meta-analysis

No

Pre-clinical

No

Prevention

No

Prognostic

No

Prospective meta-analysis (PMA)

No

Review of reviews

No

Service delivery

No

Synthesis of qualitative studies

No

Systematic review

Yes



Other  
No

**Health area of the review**

Alcohol/substance misuse/abuse  
No

Blood and immune system  
No

Cancer  
No

Cardiovascular  
No

Care of the elderly  
No

Child health  
No

Complementary therapies  
No

COVID-19  
No

Crime and justice  
No

Dental  
No

Digestive system  
No

Ear, nose and throat  
No

Education  
No

Endocrine and metabolic disorders  
No

Eye disorders  
No

General interest  
No

Genetics  
No

Health inequalities/health equity  
No

Infections and infestations  
No

**PROSPERO**  
**International prospective register of systematic reviews**

International development

No

Mental health and behavioural conditions

No

Musculoskeletal

No

Neurological

No

Nursing

No

Obstetrics and gynaecology

No

Oral health

No

Palliative care

No

Perioperative care

No

Physiotherapy

No

Pregnancy and childbirth

No

Public health (including social determinants of health)

No

Rehabilitation

No

Respiratory disorders

No

Service delivery

No

Skin disorders

No

Social care

No

Surgery

No

Tropical Medicine

No

Urological

No

Wounds, injuries and accidents

No

Violence and abuse

No

### 31. Language.

Select each language individually to add it to the list below, use the bin icon to remove any added in error.  
English

There is an English language summary.

### 32. \* Country.

Select the country in which the review is being carried out. For multi-national collaborations select all the countries involved.

Wales

### 33. Other registration details.

Name any other organisation where the systematic review title or protocol is registered (e.g. Campbell, or The Joanna Briggs Institute) together with any unique identification number assigned by them. If extracted data will be stored and made available through a repository such as the Systematic Review Data Repository (SRDR), details and a link should be included here. If none, leave blank.

### 34. Reference and/or URL for published protocol.

If the protocol for this review is published provide details (authors, title and journal details, preferably in Vancouver format)

Add web link to the published protocol.

Or, upload your published protocol here in pdf format. Note that the upload will be publicly accessible.

Yes I give permission for this file to be made publicly available

Please note that the information required in the PROSPERO registration form must be completed in full even if access to a protocol is given.

### 35. Dissemination plans.

Do you intend to publish the review on completion?

Yes

Give brief details of plans for communicating review findings.?

### 36. Keywords.

Give words or phrases that best describe the review. Separate keywords with a semicolon or new line. Keywords help PROSPERO users find your review (keywords do not appear in the public record but are included in searches). Be as specific and precise as possible. Avoid acronyms and abbreviations unless these are in wide use.

### 37. Details of any existing review of the same topic by the same authors.

If you are registering an update of an existing review give details of the earlier versions and include a full bibliographic reference, if available.

### 38. \* Current review status.

**PROSPERO**  
**International prospective register of systematic reviews**

Update review status when the review is completed and when it is published. New registrations must be ongoing.

Please provide anticipated publication date

Review\_Ongoing

**39. Any additional information.**

Provide any other information relevant to the registration of this review.

**40. Details of final report/publication(s) or preprints if available.**

Leave empty until publication details are available OR you have a link to a preprint. List authors, title and journal details preferably in Vancouver format.

Give the link to the published review or preprint.

## Appendix 2 – Systematic Review Search Terms

Search terms:

The five most expensive diseases were cancer, mental health, circulatory, musculoskeletal, and genitourinary. Proxies were used for circulatory (heart disease, stroke, and myocardial infarction), musculoskeletal (arthritis) and genitourinary (renal) diseases as seen below:

Search terms – Part 1:

1. Adverse childhood experience AND Cancer
2. Adverse childhood experience AND Mental Health
3. Adverse childhood experience AND Heart Disease
4. Adverse childhood experience AND Stroke
5. Adverse childhood experience AND Myocardial Infarction
6. Adverse childhood experience AND Arthritis
7. Adverse childhood experience AND Renal

Search terms – Part 2:

1. Cost of illness AND Cancer
2. Cost of illness AND Mental Health
3. Cost of illness AND Heart Disease
4. Cost of illness AND Stroke
5. Cost of illness AND Myocardial Infarction
6. Cost of illness AND Arthritis
7. Cost of illness AND Renal
8. Burden of illness AND Cancer
9. Burden of illness AND Mental Health

10. Burden of illness AND Heart Disease
11. Burden of illness AND Stroke
12. Burden of illness AND Myocardial Infarction
13. Burden of illness AND Arthritis
14. Burden of illness AND Renal
15. Cost\* of disease AND Cancer
16. Cost\* of disease AND Mental Health
17. Cost\* of disease AND Heart Disease
18. Cost\* of disease AND Stroke
19. Cost\* of disease AND Myocardial Infarction
20. Cost\* of disease AND Arthritis
21. Cost\* of disease AND Renal
22. Economic burden of disease AND Cancer
23. Economic burden of disease AND Mental Health
24. Economic burden of disease AND Heart Disease
25. Economic burden of disease AND Stroke
26. Economic burden of disease AND Myocardial Infarction
27. Economic burden of disease AND Arthritis
28. Economic burden of disease AND Renal

## Appendix 3: STATA code

```
name: <unnamed>
log: C:\Users\hbp808\OneDrive - Bangor University\Desktop\ACE PhD\Analy
> sis\2020 Final Analysis\Log1.smcl
log type: smcl
opened on: 30 Apr 2021, 13:33:51

.
. keep id survey gender age_cats ethnicity imd walesdeprivation relationshipsta
> tus employmentstatus collegequals trustedadult higheredquals professionalqual
> s noquals qualificationlevel wealthchild wealthadult cancer stroke respirator
> ydisease liverdisease ace_hmi ace_alcohol ace_drugs ace_incarcerated ace_sepc
> ivorce ace_ipv ace_physicalabuse ace_emotionalabuse ace_touchyousex ace_emoti
> onalabuseyn ace_touchthemsex ace_forcedsex acesexualabuse acesexualabuseyn ac
> e_total_new ace_count_categories

.
. ****To get wealth in childhood and adulthood as a binary poor/affluent variat
> le*****
.
.
.
. recode wealthchild (1/5=1) (6/10=2), gen (wealthchildbinary)
(6720 differences between wealthchild and wealthchildbinary)

. recode wealthadult (1/5=1) (6/10=2), gen (wealthadultbinary)
(7209 differences between wealthadult and wealthadultbinary)

.
. *****Drop observations without wealth variables*****
.
. drop if wealthadult == .
(5,676 observations deleted)

. drop if wealthchild == .
(25 observations deleted)

.
. ****To get social mobility as a binary variable*****
.
. gen upward_mob = 1 if wealthchildbinary ==1 & wealthadultbinary==2
(6,852 missing values generated)

. replace upward_mob = 0 if wealthchildbinary==2 & wealthadultbinary==1
(99 real changes made)

. replace upward_mob = 0 if wealthchildbinary==1 & wealthadultbinary==1
(6,080 real changes made)

. replace upward_mob = 0 if wealthchildbinary==2 & wealthadultbinary==2
(656 real changes made)

.
. gen downward_mob = 1 if wealthchildbinary==2 & wealthadultbinary==1
(7,330 missing values generated)

. replace downward_mob = 0 if downward_mob==.
(7,330 real changes made)

.
. gen stay_poor = 1 if wealthchildbinary==1 & wealthadultbinary==1
(1,349 missing values generated)

. replace stay_poor = 0 if stay_poor==.
(1,349 real changes made)

.
. gen stay_wealthy = 1 if wealthchildbinary==2 & wealthadultbinary==2
(6,773 missing values generated)

. replace stay_wealthy = 0 if stay_wealthy==.
(6,773 real changes made)
```

```

. ****To get wealth in childhood and adulthood as quartile****
.
. recode wealthchild (1/3=1) (4/5=2) (6/8=3) (9/10=4),gen(wealthchild2)
(6717 differences between wealthchild and wealthchild2)

. recode wealthadult (1/3=1)(4/5=2) (6/8=3) (9/10=4), gen (wealthadult2)
(7188 differences between wealthadult and wealthadult2)

.
.
.
. *****To get social mobility in quartiles*****
.
. *****Stay poor and upward mobility*****
.
.
. gen q1_stay =1 if wealthchild2==1 & wealthadult2==1
(1,913 missing values generated)

. replace q1_stay = 0 if q1_stay==.
(1,913 real changes made)

. gen q1_q2 =1 if wealthchild2==1 & wealthadult2==2
(7,291 missing values generated)

. replace q1_q2 = 0 if q1_q2==.
(7,291 real changes made)

. gen q1_q3 =1 if wealthchild2==1 & wealthadult2==3
(7,305 missing values generated)

. replace q1_q3 = 0 if q1_q3==.
(7,305 real changes made)

. gen q1_q4 =1 if wealthchild2==1 & wealthadult2==4
(7,424 missing values generated)

. replace q1_q4 = 0 if q1_q4==.
(7,424 real changes made)

```



```

. *****Middle two caegories*****
. gen q2_stay =1 if wealthchild2==2 & wealthadult2==2
(7,067 missing values generated)

. replace q2_stay = 0 if q2_stay==.
(7,067 real changes made)

. gen q2_q1 = 1 if wealthchild2==2 & wealthadult2==1
(7,365 missing values generated)

. replace q2_q1 = 0 if q2_q1==.
(7,365 real changes made)

. gen q2_q3 = 1 if wealthchild2==2 & wealthadult2==3
(6,989 missing values generated)

. replace q2_q3 = 0 if q2_q3==.
(6,989 real changes made)

. gen q2_q4 = 1 if wealthchild2==2 & wealthadult2==4
(7,421 missing values generated)

. replace q2_q4 = 0 if q2_q4==.
(7,421 real changes made)

.
. gen q3_stay =1 if wealthchild2==3 & wealthadult2==3
(6,840 missing values generated)

. replace q3_stay = 0 if q3_stay==.
(6,840 real changes made)

. gen q3_q2=1 if wealthchild2==3 & wealthadult2==2
(7,348 missing values generated)

. replace q3_q2 = 0 if q3_q2==.
(7,348 real changes made)

. gen q3_q1=1 if wealthchild2==3 & wealthadult2==1
(7,413 missing values generated)

. replace q3_q1 = 0 if q3_q1==.
(7,413 real changes made)

. gen q3_q4=1 if wealthchild2==3 & wealthadult2==4
(7,393 missing values generated)

. replace q3_q4 = 0 if q3_q4==.
(7,393 real changes made)

.
. *****Stay affluent and downward mobility*****
.
. gen q4_stay=1 if wealthchild2==4 & wealthadult2==4
(7,409 missing values generated)

. replace q4_stay = 0 if q4_stay==.
(7,409 real changes made)

. gen q4_q2 =1 if wealthchild2==4 & wealthadult2==2
(7,427 missing values generated)

. replace q4_q2 = 0 if q4_q2==.
(7,427 real changes made)

. gen q4_q3 =1 if wealthchild2==4 & wealthadult2==3
(7,418 missing values generated)

. replace q4_q3 = 0 if q4_q3==.
(7,418 real changes made)

. gen q4_q1 =1 if wealthchild2==4 & wealthadult2==1
(7,429 missing values generated)

. replace q4_q1 = 0 if q4_q1==.
(7,429 real changes made)

```

```
. *****Creating a binary ACE variable where 0=no ACEs and 1= at least one
> ACE*****
.
. gen acebinary = 1 if ace_count_categories>=1
(4,150 missing values generated)

. replace acebinary = 0 if acebinary==.
(4,150 real changes made)
```

```
. *****Regressions*****
.
. logistic upward_mob gender age_cats ethnicity
```

```
Logistic regression               Number of obs   =      7,412
                                LR chi2(3)        =      87.93
                                Prob > chi2        =     0.0000
Log likelihood = -1983.0592       Pseudo R2      =     0.0217
```

upward_mob	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
gender	.8623351	.0752868	-1.70	0.090	.7267103	1.023271
age_cats	1.297068	.040023	8.43	0.000	1.220949	1.377932
ethnicity	1.00018	.0000436	4.13	0.000	1.000094	1.000265
_cons	.046406	.0080619	-17.67	0.000	.033014	.0652303

Note: \_cons estimates baseline odds.

```
. logistic downward_mob gender age_cats ethnicity
```

```
Logistic regression               Number of obs   =      7,429
                                LR chi2(3)        =      40.31
                                Prob > chi2        =     0.0000
Log likelihood = -505.66846       Pseudo R2      =     0.0383
```

downward_mob	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
gender	1.057611	.2157759	0.27	0.784	.7090262	1.577574
age_cats	.6282558	.0522982	-5.58	0.000	.5336783	.7395943
ethnicity	1.000169	.0000753	2.24	0.025	1.000021	1.000316
_cons	.0381775	.0140442	-8.88	0.000	.0185643	.0785117

Note: \_cons estimates baseline odds.

```
. logistic upward_mob gender age_cats ethnicity ace_count_categories
```

```
Logistic regression               Number of obs   =      7,412
                                LR chi2(4)        =      96.12
                                Prob > chi2        =     0.0000
Log likelihood = -1978.9654       Pseudo R2      =     0.0237
```

> -	upward_mob	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]
> ]						
> -	gender	.861445	.0752532	-1.71	0.088	.7258873 1.02231
> 8	age_cats	1.310773	.0408497	8.68	0.000	1.233105 1.39333
> 2	ethnicity	1.00018	.0000434	4.15	0.000	1.000095 1.00026
> 5	ace_count_categories	1.126565	.0462922	2.90	0.004	1.039391 1.22104
> 9	_cons	.0406877	.0073467	-17.73	0.000	.0285606 .057964
> 1						

> -  
Note: \_cons estimates baseline odds.

```
. logistic downward_mob gender age_cats ethnicity ace_count_categories

Logistic regression                                Number of obs   =    7,429
                                                    LR chi2(4)      =    41.38
                                                    Prob > chi2     =    0.0000
Log likelihood =   -505.133                      Pseudo R2      =    0.0393
```

	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
downward_mob						
gender	1.058218	.2159276	0.28	0.782	.7093963	1.57856
age_cats	.6241316	.0520529	-5.65	0.000	.5300117	.734965
ethnicity	1.000171	.0000752	2.28	0.023	1.000024	1.00031
ace_count_cat	.9038479	.0897781	-1.02	0.309	.7439548	1.09816
_cons	.0419463	.0158579	-8.39	0.000	.0199938	.088002

Note: \_cons estimates baseline odds.

```
. logistic upward_mob gender age_cats ethnicity ace_count_categories if survey=
> =3
```

```
Logistic regression                                Number of obs   =    2,011
                                                    LR chi2(4)      =   113.01
                                                    Prob > chi2     =    0.0000
Log likelihood = -1148.8308                      Pseudo R2      =    0.0469
```

	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
upward_mob						
gender	.9671639	.0984223	-0.33	0.743	.7922798	1.18065
age_cats	1.429054	.0494403	10.32	0.000	1.335365	1.52931
ethnicity	1.000024	.000044	0.54	0.592	.9999374	1.0001
ace_count_cat	1.078279	.050456	1.61	0.107	.9837863	1.18184
_cons	.135508	.0272619	-9.93	0.000	.0913519	.201007

Note: \_cons estimates baseline odds.

```
. logistic upward_mob acebinary
```

```
Logistic regression                                Number of obs   =    7,412
                                                    LR chi2(1)      =    1.41
                                                    Prob > chi2     =    0.2359
Log likelihood = -2026.3207                      Pseudo R2      =    0.0003
```

upward_mob	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
acebinary	1.108716	.0964117	1.19	0.235	.9349787	1.314738
_cons	.0805737	.0047648	-42.59	0.000	.0717558	.0904751

Note: \_cons estimates baseline odds.

```
. logistic downward_mob acebinary
```

```
Logistic regression                                Number of obs   =    7,429
                                                    LR chi2(1)      =    0.45
                                                    Prob > chi2     =    0.5018
Log likelihood = -525.59649                      Pseudo R2      =    0.0004
```

downward_mob	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
acebinary	1.146028	.2322249	0.67	0.501	.7703928	1.704818
_cons	.0126891	.0017708	-31.29	0.000	.0096526	.0166809

Note: \_cons estimates baseline odds.

```
. logistic stay_poor acebinary
```

```
Logistic regression               Number of obs   =    7,429
                                LR chi2(1)         =     4.37
                                Prob > chi2         =    0.0366
Log likelihood = -3517.5979       Pseudo R2        =    0.0006
```

stay_poor	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
acebinary	1.135573	.069232	2.09	0.037	1.007674	1.279705
_cons	4.266497	.1688626	36.66	0.000	3.948044	4.610638

Note: \_cons estimates baseline odds.

```
. logistic stay_wealthy acebinary
```

```
Logistic regression               Number of obs   =    7,429
                                LR chi2(1)         =    20.51
                                Prob > chi2         =    0.0000
Log likelihood = -2207.9914       Pseudo R2        =    0.0046
```

stay_wealthy	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
acebinary	.6838069	.0581328	-4.47	0.000	.5788551	.8077875
_cons	.1128989	.0058047	-42.42	0.000	.1020764	.1248688

Note: \_cons estimates baseline odds.

```
.
. gen interaction = ace_count_categories*trustedadult
(2 missing values generated)
```

```
. logistic upward_mob acebinary trustedadult interaction
```

```
Logistic regression               Number of obs   =    7,410
                                LR chi2(3)         =    14.32
                                Prob > chi2         =    0.0025
Log likelihood = -2017.2296       Pseudo R2        =    0.0035
```

upward_mob	Odds Ratio	Std. Err.	z	P> z	[95% Conf. Interval]	
acebinary	.9987988	.0957548	-0.01	0.990	.8277007	1.205265
trustedadult	.85897	.0511097	-2.55	0.011	.7644173	.9652181
interaction	1.051042	.0210782	2.48	0.013	1.010531	1.093177
_cons	.0965359	.0085778	-26.31	0.000	.0811063	.1149007

Note: \_cons estimates baseline odds.

Note: 38 failures and 0 successes completely determined.

```
.
.
.
. translate "C:\Users\hbp808\OneDrive - Bangor University\Desktop\ACE PhD\Analy
> sis\2020 Final Analysis\Log1.smcl" "C:\Users\hbp808\OneDrive - Bangor Univers
> ity\Desktop\ACE PhD\Analysis\2020 Final Analysis\Log1.pdf", replace
(file C:\Users\hbp808\OneDrive - Bangor University\Desktop\ACE PhD\Analysis\202
> 0 Final Analysis\Log1.pdf written in PDF format)

.
. save "C:\Users\hbp808\OneDrive - Bangor University\Desktop\ACE PhD\Analysis\2
> 020 Final Analysis\ACE Dataset Social Mobility Analysis Version 4 ", replace
file C:\Users\hbp808\OneDrive - Bangor University\Desktop\ACE PhD\Analysis\202
> 0 Final Analysis\ACE Dataset Social Mobility Analysis Version 4 .dta saved

.
. log close
    name: <unnamed>
    log: C:\Users\hbp808\OneDrive - Bangor University\Desktop\ACE PhD\Analy
> sis\2020 Final Analysis\Log1.smcl
    log type: smcl
    closed on: 30 Apr 2021, 13:34:01
```