# A MULTIDISCIPLINARY INVESTIGATION INTO SOCIOECONOMIC VARIATION IN BEHAVIOUR

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#### ABSTRACT

Socioeconomic differences in behaviour are widely documented, but are not yet well understood. I propose that they can be better understood by using concepts from evolutionary theory. Evolutionary theory predicts that mortality risk should be important in determining life history traits such as the scheduling of growth and reproduction. An extension of this concept is that mortality risk should influence the degree to which people value benefits and costs in the present versus those in the future. Thus, many socioeconomic differences in behaviour may represent differences in time perspective, generated by inequalities in mortality risk. This raises the question of what cues evolved psychological mechanisms rely on when forming their estimates of personal mortality risk. I first report a test of the hypothesis that the deaths of others are used as a cue to mortality risk. The results showed that experiences of close bereavement are associated with steeper future discounting and earlier ideal, and actual, reproductive timing (Chapter 2). I then report the results of two experimental tests of whether the ages of others might be used as an indicator of local mortality rates. Manipulating the age profiles of sets of faces viewed in laboratory experiments did not have a clear effect on future discounting or reported ideal reproductive timing (Chapter 3). I move on to testing the hypothesis that the controllability of mortality risks should be most important for behaviour. The results of a correlational study showed that perceived extrinsic mortality risk mediated the association between socioeconomic status and effort spent looking after health (Chapter 4). I then report three experiments that demonstrate that priming participants to feel that prevailing sources of mortality risk are, or are not, controllable alters a simple health behaviour - the choice of a healthy food reward (Chapter 5). Finally, I review the bigger picture of socioeconomic differences in behaviour. I explain how the lack of control associated with lower socioeconomic status may lead to present-oriented behaviour in a range of domains - a phenomenon that I have called the Behavioural Constellation of Deprivation. I highlight some principles from evolutionary theoretical models that can deepen our understanding of how socioeconomic inequalities can become amplified and embedded. I discuss mechanisms by which extrinsic mortality risk may influence behaviour. I then review the evidence in support of my position, highlighting the fact that many researchers working from different perspectives have converged on control and time perspective as explanations for socioeconomic differences in behaviour. I finish by discussing the wider implications of my thesis and some of the related questions which could be answered in future research (Chapter 6 & 7).

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# **Chapter 1. Introduction**

#### 1.1 The context of my thesis

Socioeconomic inequalities in outcomes such as health and mortality are an issue of concern to policy makers and to society as a whole. Furthermore, the paradox of the persistence of health inequalities in modern welfare states is considered an enduring puzzle (Mackenbach 2012). Thus, the literature is replete with efforts to understand the forces that generate and perpetuate health inequalities. Evidence suggests that much of the disparity in health and mortality is the result of socioeconomic variation in behaviour (Pampel et al. 2010). Why the people in society who face the most challenging life circumstances should respond to them with behaviours that exaggerate their problems is another unresolved paradox (Haushofer & Fehr 2014). Furthermore, evidence suggests that this paradox is not restricted to health behaviour. Financial, reproductive and even environmental behaviours also differ with socioeconomic status (see Chapter 6 for a review). The work that I have included in this thesis uses concepts from evolutionary theory to explain socioeconomic variation in behaviour. Specifically, it examines the extent to which behaviours are influenced by mortality risk - a key factor in evolutionary models of life histories and ageing.

#### **1.2** How I arrived at my thesis question

I developed an interest in the evolutionary behavioural sciences during my undergraduate degree in Zoology. I was impressed with the explanatory power that the evolutionary perspective offered for understanding behaviour. This led me transfer to a degree in Zoology with Evolutionary Psychology. After graduating, I spent several years working in policy and communication. Whilst working for the Department of Health, I became interested in using an evolutionary perspective to understand the apparent societal effects of economic inequality, which were heavily publicised and debated at the time (Wilkinson & Pickett 2009; Saunders & Evans 2010). Epidemiologists reported that economic inequality was associated with a range of health and social outcomes including, trust, mental illness, life expectancy, infant mortality, obesity, educational performance, teenage births, homicides, imprisonment and social mobility (Wilkinson & Pickett 2009). However, this correlational evidence could not confirm a causal effect of economic inequality. I believed that an evolutionary perspective might shed light on the problem. I continued to work for the

Department of Health, whilst studying part-time for an MSc in Evolutionary Psychology. My MSc thesis investigated the effect of cues to inequality on future discounting - an attempt to test whether there might be a direct psychological effect of economic inequality.

Authors such as Wilkinson and Picket had proposed that economic inequality has a direct psychological impact, generating poor health and social outcomes through stress (Wilkinson & Pickett, 2009). However, I came to hypothesise that some of the outcomes associated with economic inequality could be explained, not as direct responses to that inequality, but as responses to the differing mortality rates experienced by those of lower socioeconomic status - a hypothesis inspired by life history theory.

My thesis does not tackle the question of what behaviours should result from having low relative status. This is an interesting question for further investigation (see section 7.4). However, my thesis does begin to address the hypothesis that life expectancy, which varies with socioeconomic status, should influence behaviour.

### 1.3 The progression of my ideas

The chapters in this thesis are ordered in such a way as to represent the maturation of my thinking about the role of mortality risk in socioeconomic differences in behaviour.

The first chapters consider some of the environmental cues that evolved psychological mechanisms might use to gauge mortality risk. First, I report a test of the hypothesis that the deaths of others, especially those to whom one feels close, might be used as a cue to mortality risk (Chapter 2). Second, I report the results of an investigation into whether the ages of others might be used as an indicator of local mortality rates (Chapter 3). These investigations yielded some interesting results. However, they tested cues to mortality risk that could not convey one important detail – the controllability of the mortality risks in question.

It is important to make the distinction between *extrinsic* mortality risk, which cannot be reduced by individual behaviour, and *intrinsic* mortality risk, which can. A theoretical model by Nettle (2010b) made the prediction that extrinsic, but not

intrinsic mortality risk should reduce the optimal investment in health behaviour. This intrinsic-extrinsic distinction is important: To say that poor health behaviour is the result of having relatively high mortality risk is circular, given that health behaviour contributes to mortality risk. However, to say that mortality risks beyond individual control disincentivise healthy behaviour is not circular. By definition, the extrinsic portion of a person's mortality risk cannot be a result of their behaviour.

Thus, Chapters 4 and 5 focus on the hypothesis that it should be the controllability of mortality risk that is most important for behaviour. I report several tests of this hypothesis. The first is a correlational study examining perceived extrinsic mortality risk and its association with reported effort spent looking after health (Chapter 4). I then report three experiments in which I primed participants to feel that prevailing sources of mortality risk were, or were not, controllable and tested the effect of this on a simple health behaviour (Chapter 5).

The final chapter pulls together much of my learning into a review, which examines the bigger picture of socioeconomic differences in behaviour. It explains how the lack of control that accompanies lower socioeconomic status, may lead to present-oriented behaviour in a range of domains. It then discusses how principles from evolutionary theoretical models can deepen our understanding of the physiological and psychological embedding of poverty (Chapter 6).

# 1.4 Other work I have undertaken during my doctoral registration

During the course of my doctoral studies, I have undertaken other work that is relevant to the question of socioeconomic differences in behaviour. This work is not included in my thesis, either because I am not the primary author, or because the work is not directly relevant to the theme of the thesis. However, I have included this work in the appendix, because it was done alongside my thesis research and has informed my current thinking on socioeconomic differences in behaviour. This additional work includes:

 A book chapter on socioeconomic gradients in health behaviour (appendix 9.1)

- 2. A paper on local norms of cheating and third party punishment in an affluent, versus a deprived neighbourhood (appendix 9.2)
- 3. A paper reporting an experiment in which participants that spent time in an affluent or deprived neighbourhood rapidly adopted the trust and paranoia profiles of the residents of those neighbourhoods (appendix 9.3).

# Chapter 2. Death and the Time of your Life: Experiences of Close Bereavement are Associated with Steeper Financial Future Discounting and Earlier Reproduction

Like chapters 4 and 5, this chapter is a published paper. I have not altered it for inclusion in this thesis, except in order to refer to other relevant material within the thesis. The citation information for the publication is as follows:

Pepper, G. V. & Nettle, D. (2013). Death and the time of your life: experiences of close bereavement are associated with steeper financial future discounting and earlier reproduction. *Evolution and Human Behavior*, 34, pp.433–439.

# 2.1 Abstract

Evolutionarily-based theories predict that people should adopt a faster life history strategy when their mortality risk is high. However, this raises the question of what cues evolved psychological mechanisms rely on when forming their estimates of personal mortality risk. In a sample of 600 North Americans, we examined associations between ideal or actual reproductive timing and two possible cues to mortality risk: 1) the total number of people a person knew who had died (death exposure); and 2) the number of those people to whom they felt close (bereavement). We also took a measure of financial future discounting, in order to establish whether experiences of death or bereavement are associated with a more general shortening of time horizons. We found that a greater number of bereavements were robustly associated with a lower ideal age at first birth, or an increased hazard of an actual first birth at any given age and with steeper future discounting. We did not find significant associations between any of these outcomes and overall death exposure. This suggests that the deaths of people with whom one is close may be a more salient cue for the calibration of reproductive and financial time horizons than the deaths of more distant acquaintances.

# 2.2 Introduction

A prediction commonly made in human behavioural ecology is that when the risk of mortality is high, people should start to reproduce earlier in their lives, at the expense of other investments (Chisholm et al. 1993; Nettle 2011; Nettle et al. 2011; Wilson & Daly 1997). Evidence suggests that ages at first birth are indeed

lower in human populations where mortality rates are high (Bulled & Sosis 2010; Low et al. 2008; Nettle et al. 2011; Nettle 2010a; Quinlan 2010), and that birth rates can increase following a sudden and salient increase in local mortality (Rodgers et al. 2005). However, relatively little is known about exactly which cues to mortality are important in shaping people's reproductive decisions in real-world settings. Moreover, it is not currently clear whether cues to mortality influence reproductive decision-making in a domain-specific way, or cause a shortening of psychological time horizons more generally. Several authors have predicted a general shortening of time horizons in response to mortality risk (Hill et al. 2008; Kruger et al. 2008; Wilson & Daly 1997; Daly & Wilson 2005). This would include a preference for smaller rewards that will be received sooner rather than larger ones to be received later (future discounting). Thus, we might expect both earlier reproduction and steeper future discounting to occur in response to cues indicative of local mortality rates (Griskevicius, Tybur, et al. 2011; Wilson & Daly 1997). Here, after reviewing some of the relevant literatures, we examine the associations between ideal and actual ages at first birth, future discounting and two potential environmental cues to mortality risk: 1) overall exposure to death and 2) close bereavements, in a survey of 600 North Americans.

#### 2.2.1 Mortality risk and initiation of reproduction

Models of the evolution of life histories predict that species facing high mortality rates should start to reproduce at a younger age (Stearns 1992), and this prediction is borne out by comparative evidence (see Harvey & Zammuto, 1985). An extension of this concept within human behavioural ecology is the idea that humans have evolved the capacity to ontogenetically calibrate their reproductive strategies in response to local mortality risk (e.g. Chisholm, Ellison, Evans, *et al.*, 1993; Lawson & Mace, 2011; Nettle, Coall & Dickins, 2011). As mortality risk increases, the benefits of earlier reproduction become greater. Earlier reproduction both increases the likelihood of reproducing (before death), and maximises the length of time for which the parent will be available to provide care for the child. Conversely, where mortality risk is lower, the benefits of delaying reproduction become greater. Delay allows for greater somatic development or the accrual of resources that could subsequently be invested in children. Thus, people who have a lower mortality risk and the ability to accrue resources or

improve their condition should delay the initiation of reproduction. Meanwhile, those faced with high mortality risks and low resource gathering potential should reproduce as sooner. The evidence suggests that this is what people do. Across countries, there is a strong association between mortality rates and age at first birth (Bulled & Sosis 2010; Low et al. 2008; Low et al. 2013). The same patterns can be seen among individuals within countries (Nettle 2010a; Quinlan 2010; Wilson & Daly 1997). However, we know little about the environmental cues that trigger these changes in reproductive strategy in humans. A handful of studies have begun to investigate such cues. For example, one study found that girls who perceive that they live in an unsafe environment have higher odds of becoming teen mothers than girls who believe that their environment is safe (Johns 2010). Some psychological experiments have also demonstrated that mortality primes influence participants' attitudes to reproduction, including their ideal ages at first birth (Griskevicius, Delton, et al. 2011; Mathews & Sear 2008). However, the artificial cues used in such experiments may not be those that are of importance to real world behaviour. Thus, we used observational data to explore which experiences were most strongly associated with reproductive schedules. We predicted that greater exposure to death and bereavement would be associated with earlier ideal or actual ages at first birth.

#### 2.2.2 Mortality risk and future discounting

Altering reproductive strategy in line with cues to mortality risk may be a domainspecific response restricted to reproductive motivations, or it may be part of a more general shift in time horizons. In the same way that it makes adaptive sense to have children at an earlier age if mortality risk is high, it may make sense to prioritise immediate rewards and costs over delayed ones (Wilson & Daly 1997). If the risk of death is high, the odds of being alive to receive future rewards are reduced. Future discounting is the tendency to choose smaller–sooner rewards over later–larger ones. It is conceptually aligned with time horizons and is often used as a measure of them (Adams 2009c; Daugherty & Brase 2010; Teuscher & Mitchell 2011). Some authors have proposed that changes in time horizon are a mechanism for functional developmental adaptation to uncertain environments (Hill et al. 2008; Kruger et al. 2008). One feature of such uncertain environments could be high mortality risk. However, there is still much to be learned about how

cues to mortality are related to future discounting. One laboratory experiment demonstrated that people who reported low childhood socioeconomic status (SES) and were exposed to mortality primes discounted the future more steeply than those who were not exposed to mortality primes (Griskevicius, Tybur, et al. 2011). Exposure to violence has been found to be associated with future discounting (Ramos et al. 2013) and earthquake survivors discount future rewards more steeply than controls (Li et al. 2012). Evidence from health psychology suggests that bereavement may be a trigger for impulsive behaviours (e.g. Stroebe, Schut & Stroebe, 2007). For example, young people who lose their parents suddenly and unexpectedly perform more health risk behaviours than controls (Hamdan et al. 2012). If exposure to death or bereavement triggers a shortening of time horizons, then this could help to explain the association between bereavement and impulsive behaviours. However, to our knowledge associations between general exposure to death, bereavement and future discounting have not yet been examined. We examined them and predicted that greater exposure to death and bereavement would be associated with steeper future discounting.

# 2.2.3 Exposure to death and close bereavements as cues to mortality risk

There has been limited research into the relative importance of environmental cues to personal mortality risk. However, there is some evidence regarding the types of cue that might be important. Exposure to violence is known to be associated with future discounting (Ramos et al. 2013) and with health-risk behaviours among adolescents - including early initiation of sexual intercourse (Berenson et al. 2001). Perceived environmental risk is a predictor of teen motherhood (Johns et al. 2011). Experimental mortality risk priming both increases future discounting (Callan et al. 2009; Griskevicius, Tybur, et al. 2011; Mathews & Sear 2008).

One very simple possible cue to mortality risk may be the number of deaths to which one is exposed. If these are a reflection of rates of mortality in one's environment, they may be a good indicator of one's own mortality risk. People may behave according to a simple rule of thumb such as, "each time someone

you know dies, shorten your time horizons a bit". However, deaths of close friends or relatives may be more important still. Relatives will share one's genes and therefore are likely to have similar vulnerabilities to disease (Manolio et al. 2009). They are also likely to share one's environment, which may be the source of the mortality risk. Similarly, close friends are likely to share one's environment. They are also more likely to share other characteristics, such as age, gender or personal habits, than mere acquaintances. Such shared characteristics may make their vulnerability to mortality risks a good reflection of one's own. Therefore, overall exposure to death may act as a mortality cue, but the deaths of people with whom one identifies closely may be given a greater weight than the deaths of others.

# 2.2.4 Predictions

In the current study, we tested associations between exposure to death (number of a person's acquaintances who died), close bereavements (number of people a person felt close to who died) and ideal and actual ages at first birth, as well as future discounting. We predicted: 1) that both exposure to death and close bereavement would be associated with lower ideal and actual ages at first birth and steeper future discounting, and; 2) that the effect of close bereavements would be greater than the effect of overall exposure to death.

### 2.3 Methods

The Newcastle University Faculty of Medical Sciences Ethics Committee approved our study. Six hundred North American volunteers were surveyed anonymously online using the SocialSci survey platform [www.socialsci.com]. Our sample had previously been recruited by SocialSci to take part in surveys via this platform. SocialSci recruit using a distributed online advertising network, print media and live recruitment. They award Amazon credit to respondents for taking part in their surveys. Our respondents completed an electronic consent form before proceeding. They were asked for their age, gender and gross annual income (\$USD). We asked if they had children and asked them for their ideal or actual ages at first birth (as appropriate - see below). We measured future discounting using a series of monetary choice tasks (below). After collecting these outcome measures, we asked about recent exposure to deaths and close

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bereavements and took a subjective measure of SES (below). The questionnaire can be seen in appendix 9.4.

#### 2.3.1 Ideal and actual ages at first birth

Respondents were asked whether they had children. If they had children, we asked, "How old were you when your first child was born?" If they did not have children, we asked, "What would be your ideal age to start having children?" Respondents selected their ideal and actual ages at first birth from a drop-down menu with choices ranging from 16 to 45 years of age. Prior studies have shown that reported ideal age at first birth is a strong predictor of subsequent actual age at first birth (Nettle et al. 2009). Therefore, we were confident that ideal age at parenthood would be a good indicator of reproductive strategy for the currently childless participants.

#### 2.3.2 Future discounting

Respondents were offered a series of 20 hypothetical choices between a larger monetary reward "in a year's time" (the delayed reward) and a smaller monetary reward "today" (the immediate reward). The delayed rewards were held constant at \$100, while the immediate rewards ranged from \$1 to \$99. The range of k parameters (k expresses the point of indifference between immediate and delayed rewards) represented by these choices were between 0.271232 and 0.000027 (where k = (A-V)/(VD), A is the amount of the delayed reward, V is the present subjective value of the delayed reward and D is the delay). This covers a slightly larger range of k than can normally be expected in similar populations (Kirby & Marakovic 1996; Kirby et al. 1999). To encourage consistent answers, the immediate reward choices were arranged in ascending order from \$1 to \$99 with both the delayed reward choices and the delay period held constant.

#### 2.3.3 Exposure to death and close bereavements

To avoid any priming effects (Mathews & Sear 2008), we asked questions about deaths at the end of the survey. We asked participants whether anyone they knew had died in the past 5 years. Those who said yes were then asked, "How many people that you know have died in the past five years?" This was our measure of exposure to death. We then asked, "How many of those people did you feel you were very close to?" We will refer to this measure as the number of close

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bereavements. We asked about deaths in the past 5 years rather than deaths over a longer period because we felt that our participants were more likely to remember recent deaths accurately. In addition, deaths that are more recent should be a better assay of current mortality risk than deaths in the more distant past.

### 2.3.4 Subjective SES measure

Respondents were asked to complete a subjective measure of SES taken from prior studies by Griskevicius et al. (Griskevicius, Delton, et al. 2011; Griskevicius, Tybur, et al. 2011). Respondents were asked to rate their agreement on a scale from one (strongly disagree) to seven (strongly agree) with the statements: a) "I don't worry too much about paying my bills"; b) "I have enough money to buy things I want", and; c) "I don't think I'll have to worry about money too much in the future." The three responses correlated well with one another (r = 0.53-0.63, p =0.001) and were therefore summed to give an overall subjective SES score. It was important to control for SES because, as explained above, resource availability should influence reproductive scheduling in tandem with mortality risk (Nettle 2010a). In addition, lower SES individuals are known to discount future rewards more steeply than higher SES individuals (Adams & Nettle 2009; Adams 2009b). We used this subjective SES measure alongside the more objective measure of income, because we wanted to be able to include younger respondents (who could be, or could become, teen parents) in the analysis. For younger respondents, measures such as income or education are not a good reflection of SES, because younger people are often still financially dependent upon parents and have not yet completed their education. Meanwhile, measures such as parental income are often inaccurately reported (Boyce et al. 2006) and cannot be easily compared with the incomes of the older respondents in the sample.

### 2.4 Analysis

Statistical tests were run in SPSS version 19.0. Age, sex, income (square root transformed) and subjective SES score were controlled in all models. We used a general linear model (GLM) to test associations between exposure to death and bereavement and ideal age at first birth for those participants who had not yet

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had children. We then tested the associations between exposure to death and close bereavements and actual age at first birth separately: We used Cox regression to assess the proportional hazard of a first birth at any given age, based on exposure to death and close bereavements, with sex, income and SES controlled in the model. We used a GLM to test associations between exposure to death and future discounting for all participants, again with age, sex, income and SES controlled. We used Pearson correlations to assess the relationship between future discounting and ideal and actual ages at first birth.

### 2.5 Results

#### 2.5.1 Descriptive Statistics

Of the 600 respondents, 262 (44%) were male, 336 (56%) were female and two did not report their sex. Respondent ages ranged from 13 to 72 years (see Table 1 for descriptive statistics). Four hundred eighty-one (80%) of our respondents had been exposed to one or more deaths in the prior 5 years. One hundred twenty-three (21%) of the sample had children. Subjective SES scores ranged from the minimum possible score of 3 to the maximum possible score of 21. The highest number of deaths reported was 30, with the mean being close to two. The highest number of close bereavements reported was 28, with a mean close to one. Ideal ages (non-parents) and actual ages (parents) at first birth had similar ranges (Table 1).

Table 1. Descriptive statistics for Chapter 2 : age, income, SES, death exposure, close bereavements, age at first birth, ideal age at first birth and future discounting.

	Range	Minimum	Maximum	Mean	Standard Deviation
Age	59.00 13.00		72.00	27.16	9.86
Income (\$USD)	1500000.00	0.00	1500000.00	40035.53	87842.72
SES	18.00	3.00	21.00	11.30	4.84
Death exposure	30.00	0.00	30.00	2.41	2.80
Close bereavements	28.00	0.00	0.00 28.00		1.63
Age at first birth	27.00	16.00	43.00	25.49	5.36
Ideal age at first birth	29.00	16.00	45.00	29.37	4.39
Future discounting	20.00	0.00 (k<0.27123)	20.00 (k≥0.00003)	8.74	5.38

Discount parameter, k = (A-V)/(VD), where A = delayed reward, V = immediate reward and D = delay.

# 2.5.2 Exposure to death and bereavement and ideal ages at first birth

There was no association between death exposure and ideal age at first birth (Table 2). However, the number of close bereavements was significantly associated with ideal age at first birth, even with age, sex, income and SES controlled. The negative parameter value (B = -0.46) indicates that a larger number of close bereavements was associated with an earlier ideal age at first birth. In addition to the effect of close bereavements, there were sex differences in ideal age at first birth, with males reporting a slightly higher mean ideal age than females. Ideal ages at first birth were also slightly higher in older respondents.

	F ratio	р	В	Standard error [B]	Lower bound (95% CI)	Upper Bound (95% CI)
Age	15.35	0.00*	0.12	0.03	0.06	0.17
Sex <sup>†</sup>	4.48	0.03*	0.82	0.39	0.06	1.58
Income	0.22	0.64	0.00	0.00	-0.01	0.00
SES	1.40	0.24	-0.05	0.04	-0.13	0.03
Death exposure	0.05	0.82	-0.02	0.10	-0.21	0.17
Bereavement	4.28	0.04*	-0.46	0.22	-0.89	-0.02

Table 2. GLM results for Chapter 2: ideal age at first birth with age, sex, income, SES, death exposure and bereavements in the model.

Being female and having reported a greater number of bereavements were associated with earlier ideal ages at first birth.

df = 1, error = 452, p = significance.

\*  $p \le 0.05$ . † The reference category is female, so the ideal age at first birth is later for males.

# 2.5.3 Exposure to death and bereavement and actual ages at first birth

Of the 600 respondents, 123 (20%) of the respondents had children and 477 (79%) did not (censored cases in the Cox regression). Thirteen cases had missing values for deaths or bereavement. As predicted, experiences of close bereavement were associated with an increased hazard of having a first child at any given age (Table 3, Figure 1). One or two bereavements did not significantly increase the hazard of having had a child at a given age relative to those who reported no bereavements. However, there was a significantly greater hazard of a first birth at a given age for those reporting 3–4 or 5+ bereavements relative to those reporting no bereavements. Indeed, the hazard of a first birth at each age roughly doubled with each level of bereavement (Figure 1). Being male was associated with a decreased hazard of having a first child at a given age. Total death exposure did not affect the hazard of a first birth (Table 3). This result mirrors our finding for ideal ages at first birth in the childless participants.

	Hazard	Lower Cl	Upper Cl	р
Sex <sup>†</sup>	0.586	0.394	0.873	0.009*
Income	1.003	1.001	1.004	0.000*
SES	0.963	0.927	1.000	0.050*
Death exposure	1.006	0.948	1.067	0.848
Bereavements <sup>††</sup>				0.002*
1-2 bereavements <sup>††</sup>	1.351	0.880	2.073	0.169
3-4 bereavements <sup>++</sup>	2.546	1.164	5.568	0.019*
5+ bereavements <sup>††</sup>	5.442	2.228	13.292	0.000*

Table 3. Cox regression results for Chapter 2: hazards of having a first child at each age, given sex, income, SES, death exposure and level of close bereavement.

CI = 95% confidence interval, p = significance. Level of reported bereavement was associated with age at first birth, even with sex, income, SES and more general death exposure controlled (see also Figure 1).

\*p  $\leq$  0.05. † The reference category is female, so the hazard of having a first child at each time point is lower for males. †† The reference category is no bereavement, so the hazard of having a first child at each time point was greater for respondents who reported 3–4 or 5+ bereavements, than for those who reported no bereavements. However, the hazard for respondents who reported 1–2 bereavements was not significantly greater than those who reported none.



Figure 1. Cumulative hazards of having a first child at a given age for four levels of close bereavement: no bereavement; 1–2 bereavements; 3–4 bereavements; & 5+ bereavements. Sex, income, SES & more general death exposure are controlled (see Table 3).

# 2.5.4 Exposure to death and bereavement and financial future discounting

Even with age, sex, income, SES and more general death exposure controlled, the number of close bereavements was associated with financial future discounting (Table 4). The effect of bereavements was in the predicted direction, with a higher number of bereavements being associated with a higher future discounting score. That is, respondents who reported a larger number of close bereavements tended to select smaller sooner rewards rather than later larger ones. Subjective SES was also a predictor of future discounting, with higher SES being associated with a lower future discounting score. That is, people of higher SES tended to prefer to wait for later larger rewards.

	F ratio	р	В	Standard error [B]	Lower bound (95% CI)	Upper Bound (95% CI)
Age	0.17	0.68	0.01	0.03	-0.04	0.06
Sex	0.02	0.90	0.06	0.45	-0.83	0.94
Income	0.04	0.83	0.00	0.00	-0.01	0.00
SES	10.16	0.00*	-0.15	0.05	-0.24	-0.06
Death exposure	3.02	0.08	-0.18	0.10	-0.38	0.02
Bereavement	6.63	0.01*	0.45	0.18	0.11	0.79

Table 4. GLM results for future discounting with age, sex, income, SES, death exposure and bereavements in the model.

SES and reported number of bereavements were significant predictors of future discounting score. A higher future discounting score indicates a greater tendency to choose a smaller sconer reward over a later larger one. A greater number of reported bereavements were associated with a greater tendency to choose a smaller sconer reward over a later larger one. A lower SES score was associated with a greater tendency to choose a smaller sconer reward over a later larger one. A lower SES score was associated with a greater tendency to choose a smaller sconer reward over a later larger one. A lower SES score was associated with a greater tendency to choose a smaller sconer reward over a later larger one. df = 1, error = 571, p = significance. \* p ≤ 0.05.

# 2.5.5 Future discounting and ideal and actual ages at first birth

Pearson correlations revealed an association between future discounting and both ideal and actual ages at first birth (Table 5). Respondents who discounted the future more steeply had lower ideal and actual ages at first birth. The association between future discounting and actual age at first birth was stronger than the association between discounting and ideal age at first birth.

	Age at first birth	Ideal age at first birth	Age at first birth and ideal age at first birth (combined)
n	123	477	600
Future discounting	310*	158*	227*

Table 5. Correlations between future discounting scores and ideal or actual ages at first birth.

A higher future discounting score indicates a greater tendency to choose a smaller sooner reward over a later larger one. Thus, a greater tendency to choose smaller-sooner rewards over later larger ones is associated with earlier ideal and actual ages at first birth. n = sample size. \*p ≤ 0.05

#### 2.6 Discussion

We predicted: 1) that both exposure to death and close bereavement would be associated with lower ideal and actual ages at first birth and steeper financial future discounting, and; 2) that the effect of close bereavements would be greater than the effect of overall exposure to death. Part of prediction 1) was supported by the data. We found that a greater number of reported bereavements were associated with a lower ideal age at first birth, an increased hazard of a first birth at any given age, and steeper future discounting. This finding held true, even after controlling for age, sex, income and SES. However, we did not find significant associations between general exposure to deaths and ideal or actual ages at first birth. In support of prediction 2, our findings suggest that experiences of close bereavement, more so than exposure to deaths in general, act as a cue to mortality risk. This could be because the deaths of people with whom one is close are a better indicator of one's own mortality risk than the deaths of more distant acquaintances.

To our knowledge, there has been no prior research examining bereavement as a cue that might affect reproductive timing. Prior studies have examined the association between mortality rates and ages at first birth within and between countries (Bulled & Sosis 2010; Low et al. 2008; Nettle 2010a; Nettle et al. 2011; Wilson & Daly 1997). Others have demonstrated that early life stress and other cues to a harsh environment are associated with faster life history strategies (e.g. Chisholm, Quinlivan, Petersen, et al., 2005; Chisholm, Ellison, Evans, et al., 1993). However, this is the first study we know of that has investigated how personal experiences may act as environmental cues to mortality risk and trigger differences in life history strategy. This study bridges a gap between the demographic findings that show associations between mortality and ages at first birth (e.g. Low, Hazel, Parker, et al., 2008) and the experimental studies that find priming effects of mortality (Griskevicius, Delton, et al. 2011). It gives us additional information about what sort of cues ought to be most important for life history strategies in real populations. Furthermore, our results tell us that laboratory studies using mortality priming might only be expected to produce

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small effects. The nature of cues – for example the person whose death the participant is primed with – will be important.

As well as suggesting that bereavement may exert an influence on reproductive decision-making, our results suggest that it affects future discounting, and thus time horizons, in a more general way. This finding converges with those from the public health literature, which show associations between bereavement and impulsive health risk behaviours (Hamdan et al. 2012). It also confirms the predictions of Wilson and Daly (1997), Kruger et al. (2008) and Hill et al. (2008), that steeper future discounting across a range of domains may be part of a suite of psychological adjustments that produce a faster life-history strategy.

Although our income and subjective SES measures were not associated with ideal age at first birth, they did predict actual age at first birth and the subjective SES measure predicted financial future discounting. Prior research has documented associations between income and future discounting (e.g. DeWit, Flory, Acheson, *et al.*, 2007). However, in our sample, close bereavements and subjective SES, but not incomes, were associated with future discounting score. The lack of an association between income and future discounting may have been due – as previously discussed – to the fact that income is not a good measure of SES for younger participants. In addition, the subjective SES score captured more fine grained aspects of resource availability, such as disposable income (2.3.4, b) and financial stability (2.3.4, c).

It should be noted that we did not find an association between age and future discounting. Prior studies have found that younger individuals discount the future more steeply than older ones (e.g. Green, Fry & Myerson, 1994), while others have not supported such findings (e.g. Green, Myerson, Lichtman, *et al.*, 1996). This may have been because income is a confounding factor in some studies. For example, the former study (Green et al. 1994) compared discount rates in children, young adults and older adults, but did not control for income or other SES measures. The latter study (Green et al. 1996), which found no association between age and future discounting, compared 30-year-olds with incomematched 70-year-olds. We also found no sex differences in future discounting in

our sample. Perhaps this is not surprising, as support for gender differences in prior studies has been mixed. Studies have found that: women discount the future more steeply than men (Reynolds et al. 2006); that men discount future rewards more steeply than women (Kirby & Marakovic 1996), and that there is no significant sex difference (Harrison et al. 2002; Wilson & Daly 2004).

There are some limitations to our data set. It was an opportunity sample and so was not population representative. In addition, respondents who already had children when surveyed may have had them before the deaths that we recorded with our 5-year death exposure measure. Therefore, we cannot conclude that the bereavements captured by our questions resulted in the lower ages at first birth. However, our data is cross sectional and it is not generally possible to infer causality in such data anyway. Furthermore, it is possible that, even after controlling for age, those respondents who reported greater exposure to death and bereavement in the 5 years prior to questioning had experienced similar levels of bereavement in their earlier years.

In general, it is difficult to draw conclusions about causality on the basis of correlational data. However, the relationships we report here are robust to control for age, sex, income and subjective SES measures. Furthermore, the results are consistent with findings from laboratory mortality priming experiments and may reflect the way in which such mortality cues produce effects in the real world (Callan et al. 2009; Griskevicius, Delton, et al. 2011; Griskevicius, Tybur, et al. 2011). Nevertheless, further investigations using longitudinal data and experimental work are warranted in order to address the causality issue.

Finally, the structure of our survey only permitted respondents to select an ideal age at first birth. It did not allow them to state that they did not desire children at all. This is a limitation because we will be unaware of respondents who do not wish to have children and perhaps should have been treated differently in our analyses.

The fact that we found an effect of bereavement, but not of death exposure may tell us something about the psychological mechanisms involved in processing
cues to personal mortality risk. Statistics about deaths have been found to have a lesser emotional impact when they cite large numbers (Slovic 2007) and they appear to motivate different decisions about the value of lives (Fetherstonhaugh et al. 1997). If people use some sort of availability heuristic (Tversky & Kahneman 1973) to assess their own mortality risk, then detailed knowledge of individual deaths may distort responses to risk of mortality by the same cause. For example, Sunstein (2003) discusses how the use of availability heuristics could lead to probability neglect. He explains that in the aftermath of a terrorist attack, repeat attacks can be more readily imagined (availability heuristic), and so people tend to over-estimate the likelihood that they will happen. This leads people to react with a fear which is out of proportion to the risk of such an event occurring. If close bereavements lead to a particular cause of death being more readily imagined, then they may skew perceptions of the risk of death due to that cause. However, it is possible that a mechanism more complex than an availability heuristic is at work. For example, people may calculate fatality proportions rather than fatality frequencies. That is, for a person with a smaller social network, each death may have a greater weight than for a person with a larger social network. Future studies might address this by collecting measures of social network size alongside measures of exposure to death and bereavement.

The nature of the causes of death will also be important. There is evidence to suggest that extrinsic mortality risk (risk of death to circumstances beyond the individual's control) is important in determining behaviour (Nettle 2010b; Pepper & Nettle 2014b). We therefore suggest that individuals who experience close bereavements due to extrinsic causes will have shorter time horizons than those whose bereavements are due to intrinsic causes. Future studies should investigate this possibility in more detail.

In conclusion, our results suggest that close bereavements act as a cue to mortality risk, triggering an accelerated life history strategy. The sheer number of deaths a person reported did not show significant effects. This may be because the deaths of a person with whom one is close are a better reflection of one's own mortality risk due to shared genes and or environment. Furthermore, we found

that the number of close bereavements reported was associated with financial future discounting. This indicates that cues to personal mortality risk may provoke a more general shortening of time horizons. Thus, the response may not be unique to the reproductive domain.

# Chapter 3. On the face of it: faces as cues to local mortality rates

#### 3.1 Abstract

Evolutionarily-based theories predict that people should adopt a faster life history strategy when their mortality risk is high, a prediction borne out by evidence. We might also expect people to be less willing to forgo a smallersooner reward for a larger-later one (future discounting), if their mortality risk is high, since they are less likely to live to receive the later reward. However, little is known about what cues evolved psychological mechanisms rely on when forming their estimates of personal mortality risk. One possible cue to mortality risk is the ages of others in the local environment. If others appear to be living to a good age, this might lead one to, unconsciously, calculate that prospects for longevity are good. We tested this hypothesis in two studies. In study 1, participants viewed either an "older" set of faces in which 40% of images were of people over the age of 50, or a "younger" set of faces in which 20% of images were of people over the age of 50. We then measured participants' ideal age at first birth, future discounting and subjective life expectancy. Study 1 revealed a possible effect of the manipulation on future discounting. However, because the effect was small, the study was replicated. Study 2 used more divergent image sets in which either 50% of images were of people over the age of 50, or 10% of the images were of people over the age of 50. Study 2 did not replicate the finding of study 1. This may have been because the result of study 1 was spurious, or it may have been due to unintentional variation in stimulus features other than age between studies 1 and 2. Having the images rated for health confirmed that there was unintentional variation in the apparent health of the images used in studies 1 and 2. Therefore, although it is likely that result seen in study 1 was spurious, it is not possible to draw any firm conclusions.

## 3.2 Introduction

Socioeconomic differences in behaviour are pervasive and well documented, but their causes are not yet well understood. There are a several attitudes and behaviours associated with socioeconomic status (SES), variations in which could be explained, at least in part, as an adaptive response to local mortality

rates (see Chapter 6 for a review). These include reproductive timing, which can be used as an indicator of life history strategy, and the value placed upon future outcomes (future discounting), which may be a more general response to the fact that mortality risk limits the likelihood of receiving future rewards. We have already found associations between these variables and experiences of bereavement (Chapter 2, Pepper & Nettle, 2013), suggesting that people may use the deaths of others as a cue to their own mortality risk. Experiences of bereavement cannot be experimentally manipulated in order to assess their causal effects. However, other potential cues to life expectancy can be. Here we report an investigation of whether measures of intended reproductive timing and future discounting are sensitive to a potential cue to life expectancy - the distribution of the ages of others.

#### 3.2.1 The ages of others as a cue to life expectancy

Evidence suggests that people are able to predict their own life expectancies with a reasonable degree of accuracy (Lawlor et al. 2003; Delavande & Rohwedder 2011; Adams et al. 2014). However, little is known about the information that people use to make such assessments.

One possible environmental cue to life expectancy, which may vary with the socioeconomic composition of a neighbourhood, is the age-profile of the people encountered there. Nettle, Coyne & Colléony (2012) compared two neighbourhoods in the same city – one deprived and one affluent. People over the age of 60 were observed on the streets less often in the deprived than the affluent neighbourhood. Nettle, Coyne & Colléony (2012) argued that regular exposure to this "visual diet" that included fewer older adults and more young adults may lead individuals to unconsciously calculate that they are unlikely to live to old age themselves. They suggested that this might be one of a range of cues that trigger fast life history strategies in deprived neighbourhoods. The studies reported here aimed to test this idea by manipulating anticipated survival using a visual diet of older, or younger, faces.

#### 3.2.2 Life expectancy and timing of reproduction

Evolutionary life history theory predicts that species facing high mortality rates should begin their reproductive lives sooner than those in low mortality environments (Stearns 1992), a prediction borne out by comparative evidence (see Harvey & Zammuto, 1985). This concept has been extended by human behavioural ecologists to generate the theory that humans have evolved the capacity to ontogenetically calibrate their reproductive strategies in response to local mortality risk (Chisholm et al. 1993; Lawson & Mace 2011; Nettle et al. 2011). As mortality risk increases, earlier reproduction becomes a more adaptive choice: People should act to maximise their likelihood of successfully reproducing before death as well as the length of time during which they are alive to care for their children. Conversely, in an environment of low mortality risk, it may be adaptive to delay reproduction: Delay allows for greater somatic development and the accrual of resources that could subsequently be invested in children. Thus, people who face few mortality risks and have the ability to accrue resources or improve their condition should delay reproduction. Meanwhile, individuals in high mortality environments with low resource gathering potential should start reproducing sooner. This prediction is also supported by empirical evidence. Across countries, there are strong associations between mortality rates and age at first birth (Bulled & Sosis 2010; Low et al. 2013; Low et al. 2008). Similar patterns can be seen among individuals within countries (Nettle 2010a; Quinlan 2010; Daly & Wilson 1997). Experimental mortality primes also influence participants' attitudes to reproduction, including their ideal ages at first birth (Griskevicius, Delton, et al. 2011; Mathews & Sear 2008; Wisman & Goldenberg 2005).

Although evidence supports the idea that life history strategies are calibrated in response to mortality risk, little is known about what environmental cues to trigger these calibrations. One study found that girls who perceive their neighbourhoods to be unsafe more often become teen mothers than those who feel that their environment is safer (Johns 2010). Another study found that fertility in Oklahoma increased after the Oklahoma City bombing in 1995 (Rodgers et al. 2005). We have found that experiences of close bereavement

are associated with earlier ideal and actual ages at first birth (Chapter 2, Pepper & Nettle, 2013). However, these studies were correlational and cannot confirm causal relationships. In this chapter, we report an experimental test of the effect of a potential cue to life expectancy (the ages of others) on ideal age at first birth.

## 3.2.3 Life expectancy and the value of future rewards

In the same way that it makes adaptive sense to have children at an earlier age if mortality risk is high, it makes sense to prioritise immediate rewards and costs over delayed ones (Wilson & Daly 1997; Daly & Wilson 2005). When mortality risk is high, the odds of being alive to receive future rewards are reduced.

Future discounting is the tendency to choose smaller-sooner rewards over later-larger ones and is often used as a measure of time preference (Adams 2009c; Daugherty & Brase 2010; Teuscher & Mitchell 2011). Some authors have proposed that changes in time preference are a mechanism for functional developmental adaptation to uncertain environments (Hill et al. 2008; Kruger et al. 2008). High unpredictable or uncontrollable mortality risk may be a feature of such environments.

There is some evidence supporting a relationship between mortality and future discounting. Exposure to violence has been found to be associated with future discounting (Ramos et al. 2013). Earthquake survivors discount future rewards more steeply than controls (Li et al. 2012). Evidence suggests that bereavements trigger impulsive behaviours, which are conceptually linked to future discounting (e.g. Stroebe, Schut & Stroebe, 2007). In addition, we have now found that experiences of close bereavement are associated with a greater tendency to discount future rewards (Chapter 2, Pepper & Nettle, 2013). These findings are correlational and cannot confirm causal links. However, one experiment has demonstrated that, people who reported lower childhood SES and were exposed to a newspaper article reporting random acts of violence, discounted the future more steeply than those who were not exposed to mortality primes (Griskevicius, Tybur, et al. 2011).

## 3.2.4 Predictions

We hypothesised that measures of future discounting, ideal age at first birth and subjective life expectancy would vary in response to the visual diet that participants were exposed to. We predicted that:

1. Viewing a higher proportion of older faces would increase participants' subjective life expectancies relative to those who viewed a lower proportion of older faces,

2. Viewing a higher proportion of older faces would increase participants' ideal ages at first birth relative those who viewed a lower proportion of older faces, and,

3. Viewing a higher proportion of older faces would decrease participants' tendencies to discount future rewards (to choose smaller-sooner over largerlater) relative to those who viewed a lower proportion of older faces.

## 3.3 Study 1

As outlined above, the existing evidence suggests that cues to mortality risk should affect a range of behaviours including reproductive scheduling and the tendency to prioritise immediate rewards over delayed ones. The distribution of the ages of others in one's environment may act as one such cue. In study 1, we carried out an experimental test of whether a visual diet of older, or younger, faces would affect subjective life expectancy, ideal age at first birth, or future discounting.

## 3.4 Study 1 Methods

## 3.4.1 Sample

We recruited 40 males and 39 females from the Faculty for Medical Sciences Buildings at Newcastle University. Their ages ranged from 18 to 48 years (mean = 22.20, s.d. = 4.62). Participant ages (t<sub>77</sub> = -1.87, p=0.065), sex ( $\chi^2$  = 0.62, df = 1, p = 0.43) and childhood postcode deprivation scores (t<sub>56</sub> = -0.44, p=0.67) did not differ significantly across treatment groups. Ethical approval for the study was given by the Faculty of Medical Sciences Ethics Committee

(ref 00455). All participants gave informed consent, were debriefed and had the opportunity to ask questions.

## 3.4.2 Procedure

All data were collected on a computer in a controlled laboratory environment. We recruited participants individually, face-to-face. We briefed them about the procedure before they agreed to take part. They were told that the experiment was about personality and perception. We informed them that they would see a series of faces on a screen, be asked to decide whether these were male or female and then fill out a personality questionnaire. They were offered a chocolate bar as a reward for participating.

We programmed the experiment using Qualtrics Labs, Inc. software, Version [2012] of the Qualtrics Research Suite, <u>http://www.qualtrics.com</u>. Participants viewed a series of 50 faces on a computer screen. Their ostensible task was to move each face into a box to indicate whether they thought it was a male or female face. This task was given to ensure that they paid sufficient attention to the faces for any priming to take effect. Participants were randomly assigned to treatments by Qualtrics. They were either presented with an "older" set of faces, or a "younger" set of faces (see stimuli, section 3.4.3). Participants then answered a series of questions described as a personality questionnaire (see measures, section 3.4.4). Finally, they were asked whether they thought they could guess what the study was about. This was to ensure that results were not due to any demand characteristic.

## 3.4.3 Stimuli

The photographs used were taken from a set of images from the Centre for Vital Longevity database (Minear & Park 2004). We used a subset of the images, which were black and white and had previously been rated for age, mood, familiarity and picture quality (Kennedy et al. 2009).

Image sets were constructed such that either 40% of the images were of people over the age of 50 (older faces), or 20% of images were of people over the age of 50 (younger faces). These proportions reflect skewed versions of

the distribution of over 50s in the UK population: 35% of the UK population are over 50 years of age (22.7 million in 64.1 million – data from Office for National Statistics website, www.ons.gov.uk). We manipulated the image sets based on the actual ages of the people in them, rather than their rated ages. However, the actual and rated ages of the faces in the stimulus sets were highly correlated at 0.97 (p<0.001). Images were selected so that face ages varied, without any difference between the sets in mood, familiarity or picture quality. Paired t-tests confirmed that both the actual and rated age profiles of the image sets used in the treatments differed as intended (age:  $t_{49} = 2.29$ , p = 0.026, rated age:  $t_{49}$  = 2.37, p = 0.022). The mood profiles of the image sets were not significantly different ( $t_{49} = 1.16$ , p = 0.252). Rated familiarity ( $t_{49} = 1.16$ , p = 0.252). 0.46, p = 0.647), rated memorability ( $t_{49} = 0.41$ , p = 0.687) and rated image quality ( $t_{49} = -0.26$ , p = 0.796) were also the same across treatments. There were 25 male faces and 25 female faces in each image set. Only images of Caucasian individuals were used. In order to allow the images to fit easily into the "drag and drop" boxes on the screen, all images were reduced to 60% of their original size (300 x 450 pixels).

#### 3.4.4 Measures

The outcome measures are explained in detail in this section. However the questionnaire is also included in the appendix (section 9.5). The questionnaire asked participants for basic demographic information – gender, age, and postcode at age 15. Postcode at age 15 was used to generate a deprivation score for participants' home addresses using the Office for National Statistics' Indices of Multiple Deprivation (IMD; Mclennan, Barnes, Noble, *et al.*, 2011). The IMD identify deprived areas of the country by combining a range of economic and social indicators into a single score. These scores are considered to be a useful objective measure of an individual resident's socioeconomic status (Danesh et al. 1999). Many of the participants were undergraduate university students, so postcode at age 15 was more likely to be an accurate indicator of their socioeconomic status than the postcode for their term-time address. Since the data were collected in 2011 and the mean participant age was 22 years, we used IMD scores from 2004, when the

majority of participants would have been 15 years old (data release options were 2004, 2007 and 2011). The IMD scores used were at the level of the lower layer super output area (LSOA), the smallest UK geographies unit for which IMD data are available.

Age, gender and postcode at age 15 were requested at the end of the questionnaire, because participants would not vary their answers to these questions in response to the prime. The other questions, answers to which might have varied in response to the prime, were presented in randomized order immediately after the participants had finished categorising the faces. The randomized order of the questions was intended to counterbalance any order effects. It was also intended to ensure that the absence of an effect for any outcome variable could not be attributed to the prime "wearing off" during the time taken to complete the questionnaire. The outcome measures collected through the questionnaire were: ideal age at first birth (AFB), future discounting (FD) and participants' subjective odds of living beyond the age of 75 (subjective life expectancy).

#### 3.4.4.1 Ideal age at first birth

Ideal AFB was measured simply by asking participants "What would be your ideal age to start having children, if you were to do so?" This question was tailored to the age of the participants, who were largely students between 18 and 25 years of age and were unlikely to already have children.

## 3.4.4.2 Future discounting

Participants were offered a series of 20 hypothetical choices between a larger reward "in a year's time" (delayed reward) and a smaller reward "today" (immediate reward). The delayed rewards were held constant at £100, while the immediate rewards ranged from £1-99. The range of *k* parameters (expressing the point of indifference between present and future rewards) represented by these choices were between 0.271232 and 0.000027. Where k = (A-V)/(VD), with A being the amount of the future reward, V being the present subjective value of the delayed reward and D being the delay. This covers a slightly larger range of k than could be expected, based on

undergraduate samples in other studies (Kirby & Marakovic 1996). The immediate reward choices were arranged in ascending order from £1 to £99 with both the delayed reward choices and the delay held constant. This was done in order to encourage consistent answers. For convenience, the measure of future discounting used in analysis was not k, but simply the number of times a participant chose the immediate reward above the delayed alternative. A table in the appendix (section 9.6) shows the list of the choices presented and the k values those choices represent.

#### 3.4.4.3 Subjective Life Expectancy

I used a measure of anticipated survival from the English Longitudinal Study of Ageing (Taylor et al. 2007; Adams & Nettle 2009) as a manipulation check – to test whether the face based primes had any measurable effect on subjective life expectancy. We asked the question, "What do you think the chances are that you will live to be 75 or more?" Participants were asked to move a sliding bar on a 100-point scale, with 0 being 'No chance' and 100 being 'Definitely'.

#### 3.5 Study 1 Analysis

Analysis was performed using SPSS 19. All of the outcome variables in the sample violated the assumption of univariate normality (a prerequisite for multivariate normality) for one or both of the treatment groups (see appendix 9.7 for a table of Kolmogorov-Smirnov statistics). Neither Levene's test (see appendix 9.8) nor Box's test were significant ( $F_{18, 8963} = 1.228$ , p=0.228) and General Linear Modelling (GLM) is thought to be robust to non-normality (even to out-perform comparable non-parametric tests) when the assumption of homogeneity of covariance is not violated (Box & Andersen 1955; Finch 2005). Thus, we used a GLM, with age, sex and postcode deprivation score included as covariates. However, Box's test may be prone to type II errors when samples deviate from multivariate normality (Field 2005). Future discounting data frequently violate the assumption of normality, necessitating non-parametric analysis (Rachlin et al. 1991; Bickel et al. 1999). Thus, the data were also analysed using Mann-Whitney tests.

## 3.6 Study 1 Results

The overall GLM, which included ideal AFB, FD and subjective life expectancy as outcome variables showed no significant effect of treatment ( $F_{3,50} = 1.62$ , p = 0.196). The univariate models showed a small ( $\eta_p^2 = 0.086$ ) but significant effect of treatment on FD score (Figure 2), but not ideal AFB or subjective life expectancy (Table 7). There was also an effect of age on ideal AFB, with older participants reporting a later ideal age to begin reproduction (Table 7). Mann-Whitney tests supported the significant effect of treatment on FD score, but not ideal AFB or subjective life expectancy (Table 7).

None of the participants guessed the purpose of the experiment. We were therefore reasonably confident that the study was not affected by demand characteristics.



Figure 2. Mean future discounting scores for participants who viewed older faces (40% over 50 years old) or younger faces (20% over 50 years old) in Study 1. A higher future discounting score indicates a greater preference for immediate rewards over delayed ones. Error bars represent 95% confidence intervals.

Effect of treatment on:	Mann-Whitney U	Ζ	р
Ideal age at first birth	718	-0.61	0.540
Future discounting score	546	-2.30	0.022*
Subjective life	765	0.88	0.883

Table 6. Mann-Whitney results for treatment effect in study 1.

 $p = significance (*p \le 0.05)$ 

		F ratio	Р	В	Standard error [B]	Lower bound (95% CI)	Upper Bound (95% CI)
Ideal age at first birth	Age	5.13	0.027*	0.20	0.09	0.02	0.37
	Sex <sup>†</sup>	0.10	0.752	-1.49	1.18	-3.87	1.25
	IMD Score	2.35	0.132	-0.06	0.04	-0.14	0.02
	Treatment	0.27	0.607	-1.33	1.28	-3.90	1.25
	Age	3.71	0.059	-0.28	0.15	-0.57	0.02
Future discounting score	Sex <sup>†</sup>	0.29	0.59	1.77	1.99	-2.23	5.76
	IMD Score	1.35	0.251	0.08	0.07	-0.06	0.21
	Treatment	4.92	0.031*	-1.95	2.15	-6.27	2.38
Subjective life expectancy	Age	0.62	0.434	-0.31	0.39	-1.11	0.49
	Sex <sup>†</sup>	0.33	0.567	-2.24	5.49	-13.26	8.78
	IMD Score	0.00	0.981	0.00	0.18	-0.36	0.37
	Treatment	0.14	0.709	1.28	5.94	-10.65	13.20

Df = 1, error = 54, p = significance (\*p  $\leq$  0.05), †The reference category is female

## 3.7 Study 1 Discussion

The results of study 1 suggested that participants who were exposed to a higher proportion of older faces discounted the future less steeply, as predicted. That is, they chose smaller-sooner rewards over later-larger ones less often. However, there was no change in the other variables predicted to respond to the prime and the overall GLM did not show a significant effect of treatment. It was only when FD score was examined in a univariate model or using a Mann-Whitney test that the effect could be seen. If the prime was successful and the hypothesis correct, it is surprising that other variables, such as subjective life expectancy or AFB did not change along with the FD scores. This makes it difficult to determine whether the prime had the expected effect, or whether the apparent difference in FD scores was due to a type 1 error. We therefore went on to replicate study 1, using more heavily skewed proportions of older, and younger, faces in the image sets.

## 3.8 Study 2

Study 2 replicated the method used in study 1, but used more extreme age profiles for the stimuli. If the effect seen in study 1 was genuine, then we predicted that using increased proportions of older and younger faces in the stimuli would amplify the intended cue and thereby the effect.

An extra outcome measure was also added: Participants' choice between fruit and chocolate as a reward for taking part in the study. This was done to investigate whether the potential priming effect seen in study 1 would influence a health-related behaviour – snack choice. Evidence supports an association between time perspective and health behaviour. For example, people who are less future oriented are more often overweight and are more likely to smoke (Weller et al. 2008; Adams 2009c; Adams & Nettle 2009). We predicted that the participants who viewed the set of images containing a greater proportion of older faces would be more likely to choose a healthy food reward and less likely to discount future monetary rewards.

## 3.9 Study 2 Methods

## 3.9.1 Sample

To match the sample size to that of study 1, 40 males and 41 females were recruited within the Faculty for Medical Sciences Buildings at Newcastle University. Participant ages ranged from 18 to 36 years (mean = 20.96, s.d. = 3.00). Postcode deprivation

scores ranged from 2.5 to 63.7 (of a possible 0.53 – 87.80, mean = 14.32, s.d. = 12.64).

As in study 1, participants were randomly allocated into one of the two treatment conditions. Participant age ( $t_{79} = -1.60$ , p = 0.112), sex ( $\chi^2 = 1.58$ , df = 1, p = 0.262) and postcode deprivation scores ( $t_{50} = 1.04$ , p = 0.304) did not differ significantly across treatment groups.

As with study 1, all participants gave informed consent, were debriefed and had the opportunity to ask questions.

#### 3.9.2 Procedure

The procedure was as reported for study 1, but with one addition: The participants were offered a choice between chocolate and fruit as a reward for taking part in the experiment. This was done to investigate whether the priming effect found in study 1, if it were replicated, would extend to a health behaviour – snack choice (see measures section for details). Participants were offered the choice of food at the end of each session before they were debriefed.

#### 3.9.3 Stimuli

As with study 1, the photographs used were taken from the Centre for Vital Longevity database (Minear & Park 2004; Kennedy et al. 2009). However, the proportions of older and younger faces used to construct the image sets were more extreme: In the older set of faces, 50% of images were of people over the age of 50. In the younger set, only 10% of the images were of people over the age of 50. Paired t-tests confirmed that the age profiles of the images in the treatments differed as intended ( $t_{49} = 4.46$ , p < 0.001). Meanwhile, the mood profiles of the image sets ( $t_{49} = 1.30$ , p = 0.197), their rated familiarity ( $t_{49} = 1.09$ , p = 0.278), image quality ( $t_{49} = 0.39$ , p = 0.700), and memorability ( $t_{49} = 1.23$ , p = 0.224) did not differ across treatments. As with study 1, there were 25 male faces and 25 female faces in each image set and only Caucasian faces were used.

#### 3.9.4 Measures

The measures were identical to those reported for study 1 except for the additional measure of snack choice: Participants were offered a choice between fruit or

chocolate as a reward for taking part in the experiment. There was a selection of 2 kinds of fruit and 2 kinds of chocolate, to reduce the likelihood of participants basing their choice on their dislike of a particular brand of chocolate or type of fruit. The fruits on offer were always apples and clementines. The chocolate bars were either Aero or KitKat bars (for 33 participants) or Toffee Crisp or KitKat (for 48 participants).

## 3.10 Study 2 Analysis

As in study 1, all of the outcome variables in the sample violated the assumption of univariate normality for one or both of the treatment groups (see appendix 9.9 for Kolmogorov-Smirnov statistics). They also violated the assumption of equality of error variances for subjective life expectancy, though not for the other variables (see appendix 9.10 for results of Levene's test). In this case, Box's test was significant ( $F_{18, 6229} = 1.87$ , p = 0.014), making it more appropriate to use a non-parametric test. However, to maintain consistency with study 1, we employed both GLM and Mann-Whitney tests. As in study 1, we included age, sex and postcode deprivation score as covariates in the GLM. We used a chi-square test to assess whether food reward choice differed between treatments.

## 3.11 Study 2 Results

The results of study 1 were not replicated. The overall model showed no significant effect of treatment on ideal AFB, FD or subjective life expectancy ( $F_{3,44} = 0.34$ , p = 0.796) and the univariate models also showed no effect of treatment on FD, ideal AFB or subjective life expectancy (Table 8). Mann-Whitney results supported the same conclusion (Table 9). There was a significant effect of sex on ideal age at first birth, with males preferring to wait longer before their first birth (Table 8, male mean = 29.5 years, female mean = 27.2 years).

Only 5 participants refused food rewards. 55 participants selected chocolate and 21 participants selected fruit. When the participants who took no reward were included in the analysis, there was no statistically significant difference in food choice between treatment groups, ( $\chi^2$  (2, n = 81) = 2.70, p = 0.278). This result did not change when those participants who refused a food reward were excluded from the analysis ( $\chi^2$  (1, n = 76) = 2.70, p = 0.123). However, there was a trend towards taking fruit more often in the younger faces treatment: 19% of the participants chose fruit over

chocolate after the older faces treatment, while 35% chose fruit over chocolate after the younger faces (Figure 3). This trend may not have reached significance because it did not represent a genuine effect, or the study may have lacked the power to detect an effect. Post-hoc power analysis using G\*Power 3.1.7 (Faul et al. 2007) indicated that, assuming a population effect size equal to that seen in the sample ( $\phi$ = 0.183, n = 81, df = 2) the study had power of 0.294. Based on this observed effect size, a sample of 462 would be required to achieve power of 0.95. Arguably, it is tautological to estimate sample size using an observed effect from an underpowered study (O'Keefe 2007). However, we are not aware of any studies from which an effect of independent interest could be drawn.

		F ratio	р	В	Standard error [B]	Lower bound (95% CI)	Upper Bound (95% CI)
Ideal age at first birth	Age	1.04	0.313	-0.12	0.12	-0.35	0.11
	Sex <sup>†</sup>	16.08	0.000**	2.21	0.92	0.35	4.07
	IMD Score	1.99	0.165	-0.04	0.03	-0.09	0.02
	Treatment	0.90	0.347	0.36	0.85	-1.35	2.06
Future discounting score	Age	1.49	0.229	-0.31	0.25	-0.81	0.20
	Sex <sup>†</sup>	0.71	0.404	-0.01	2.02	-4.07	4.05
	IMD Score	0.02	0.885	-0.01	0.06	-0.12	0.10
	Treatment	0.17	0.680	-0.57	1.85	-4.29	3.15
Subjective life expectancy	Age	0.88	0.354	0.84	0.89	-0.96	2.63
	Sex <sup>†</sup>	3.00	0.090	-5.04	7.17	-19.47	9.39
	IMD Score	1.70	0.198	0.26	0.20	-0.14	0.65
	Treatment	0.00	0.966	2.91	6.56	-10.30	16.12

Df = 3, error = 44, p = significance (\*p  $\leq$  0.05, \*\*p  $\leq$  0.01), †The reference category is female

Effect of treatment on:	Mann-Whitney U	Ζ	р				
Ideal age at first birth	656	-1.38	0.167				
Future discounting score	737	-0.59	0.553				
Subjective life expectancy	682	-1.12	0.265				
p = significance							

Table 9. Mann-Whitney results for treatment effect study 2.



Figure 3. Frequencies of food reward choice among participants who viewed older faces (50% over 50 years old) or younger faces (10% over 50 years old) in Study 2.

#### 3.12 Study 2 Discussion

The findings of study 2 did not replicate those of study 1. It is may be that the effect found in study 1 was a type 1 error, or that the studies were underpowered. However, since some of the stimulus images differed between studies 1 and 2, it is also possible that some variable other than age was incidentally altered. Given that people often display different rates of physiological ageing ("the progressive loss of function accompanied by decreasing fertility and increasing mortality with advancing age", Kirkwood & Austad, 2000) for their chronological age, it is possible that perceptions of age and health can be confounded. Cues suggesting that the local population are unhealthy might act counter to cues suggesting that people are living a long time. If there were differences in the health cues present in the faces used in studies 1 and 2, then the age and health cues might have cancelled one another out. In order to test this, we had the face images used in both studies rated for apparent health (see section 3.13).

There was no significant effect of treatment on the additional variable that was included in study 2 - the choice of food reward. Though not statistically significant, there was a tendency for participants to take fruit more often after having viewed the image sets containing a higher proportion of younger faces (Figure 3). This runs contrary to my prediction, which was that there would be an increase in preference for fruit in the older faces treatment: We had hypothesised that, if viewing a larger proportion of older faces led participants to unconsciously calculate that they would live longer themselves, they would be more willing to invest in their future health. However, given that the result was not significant and the study was likely underpowered, we cannot draw any firm conclusions regarding this apparent trend.

#### 3.13 Study 3 - Rating the study images for apparent health

As discussed above, it is possible that age and health cues were confounded in the images used in studies 1 and 2. Many of the images of older people, or those who were rated as older, might also have been perceived as unhealthy. If participants viewing the older faces were also receiving cues suggesting that people are in poor condition, this would have the opposite effect to that which was intended. Participants in the older faces group may have unconsciously

calculated that they were in an environment with greater morbidity. That is, rather than seeing older faces and thinking that they too were likely to live a long time, participants may have simply increased their estimates of their risk of morbidity. We collected ratings of perceived health for the images in order to test this possibility.

## 3.14 Study 3 Methods

## 3.14.1 Sample

90 raters were recruited online through mailing lists and networks and via the online social networking site, Facebook, <u>http://www.facebook.com</u>. All raters remained anonymous as we did not collect any demographic information.

## 3.14.2 Procedure

Participants read online information and debrief text. Informed consent was assumed within the participant information page, which read, "Your participation is voluntary and you are free to stop at any time without giving a reason. By continuing to the next screen you give your consent for your ratings to be used in our research."

To avoid fatigue, participants were not asked to rate the full set of 180 faces. They were asked to rate a randomized subset of 45 images. Participants paced their own answers and moved onto the next image when ready by pressing a forward arrow button.

## 3.14.3 Stimuli

We used all of the 180 images that were standardized by Kennedy, Hope & Raz, (2009) from the database at <u>http://agingmind.utdallas.edu/facedb</u> (Minear & Park 2004; Kennedy et al. 2009). Online face ratings tasks were created using Qualtrics Labs, Inc. software, Version [2012] of the Qualtrics Research Suite, <u>http://www.qualtrics.com</u>. In all cases, participants were presented with a single screen per image. The images were displayed at their original size (300 x 450 pixels).

#### 3.14.4 Measure

Each image the participants were asked to rate was presented with the text, "How healthy does this person look? Please move the slider below to tell us how healthy you think they are where 0 is 'not at all healthy' and 100 is 'in perfect health' ". Participants moved a sliding bar on a scale to indicate their answer.

#### 3.15 Study 3 Results

There was good consistency among health ratings for the images (Cronbach's  $\alpha$  = 0.83). For the subset of images used in studies 1 and 2, rated health was strongly associated with both actual age (r<sub>70</sub> = -0.73, p < 0.001) and rated age (r<sub>70</sub> = -0.76, p < 0.001). There were also weaker correlations between rated health and picture quality (r<sub>70</sub> = 0.30, p < 0.001) and rated memorability (r<sub>70</sub> = -0.32, p = 0.006).

The image sets used in the older and younger profile sets in study 1 did not differ significantly in their rated health ( $t_{49} = -1.39$ , p = 0.170, r = 0.123, Table 10). However, health ratings for the images used in the treatments in study 2 did differ significantly ( $t_{49} = -2.29$ , p = 0.026, r = 0.249, Table 10).

Study	Treatment group	Mean	Standard deviation	N
Study 1	Older faces	65.04	11.30	50.00
	Younger faces	67.76	10.85	50.00
Study 2	Older faces	63.30	11.97	50.00
	Younger faces	69.08	10.74	50.00

Table 10. Means and standard deviations for the older and younger facesimage sets from studies 1 and 2.

#### 3.16 Study 3 Discussion

The health ratings for the face images suggest that perceived health and age were confounded in the image sets used in studies 1 & 2. Since age and rated health were highly correlated, varying the age profiles of the image sets also created unintended variation in their apparent health. This variation in the apparent health profiles of the faces was two times greater in study 2 than in study 1 (Table 10). This difference may explain why study 2 did not replicate the result of study 1. However, it is difficult to draw any strong conclusion regarding this possibility. To confirm whether this explanation holds, the experiments would need to be repeated using images that vary on age but not health and on health but not age.

The health ratings we collected for the images by Kennedy, Hope & Raz (2009) revealed some associations between rated health and other perceived aspects of the images. There was a small positive correlation between rated health and picture quality. This suggests that raters may, to some extent, confuse the quality of the individual with that of the image. Participants also rated the healthier looking faces as being less memorable. If healthier faces are indeed less memorable, this may indicate an attentional bias towards faces containing cues to ill health, which could have adaptive significance for disease avoidance (Neuberg et al. 2011).

## 3.17 Overall Discussion

Although there appeared to be an effect of the age profiles of the stimuli on future discounting in study 1, this effect was not replicated in study 2. Unfortunately it is not possible to ascertain whether this is because the effect seen in study 1 was spurious, or because the effect was dampened by unintentional variation in the health profiles of the images used in study 2. However, the fact that the effect seen in study 1 was seen in study 1 was small and was not significant in the overall GLM lends weight to the former explanation.

There are a number of possible explanations for the lack of conclusive results for the studies reported. Firstly, age distributions of faces in the environment may not be used to estimate life expectancy. It is possible that, because the ages of those whom one regularly encounters can be skewed by causes other than mortality (e.g. social segregation), they are not a reliable indicator of personal life expectancy.

Alternatively, images on a screen may not be an ecologically valid cue to life expectancy. Perhaps our participants did not consider the people whose faces they viewed to be relevant to their own prospects. It is likely that the life expectancies of people with whom one identifies (family, peers and community members) are a better reflection of one's own prospects, due to shared genes, characteristics or environments. Indeed, we have found that the number of close bereavements someone had suffered, but not the total number of deaths they had been exposed to, was associated with both ideal and actual ages at first birth and future discounting (Chapter 2). This suggests that the deaths of people with whom one is close may be a better indicator of one's own mortality risk than the deaths of more distant acquaintances.

It is also likely that exposure to a brief artificial cue in the laboratory is trivial when considered against the background of a lifetime of exposure to cues signalling a contrary outcome. If a participant has a stable anticipation of their life expectancy, built on years of cues suggesting that they will live a long time, then viewing images of younger faces in the laboratory will do little to dampen that impression. Indeed, for our student sample, being surrounded by a high proportion of younger

faces is likely to be the norm. That is, our younger faces manipulation may not be very different from what students encounter in their day-to-day environments.

It is possible that the result in study 1 represented a real effect, which was cancelled out by unintentional variation in the apparent health (or other aspects) of the faces used in study 2. We used real face images, rather than digitally morphed or averaged ones, because we felt that they offered a more ecologically valid cue. However, this meant a lack of control over the cues contained within the faces, leading to confounds between age and health. Future studies might be improved by the use of averaged faces in which age, but not health is varied (Burton et al. 2005).

Finally, it is possible that the age distribution of faces in a person's environment has no effect because faces cannot convey the most pertinent aspect of mortality risk – whether or not it can be prevented by individual action. A behavioural ecological model by Nettle (2010b) predicted that it should be whether or not mortality risk is extrinsic (beyond individual control) which matters most for health behaviour. This principle could also be applied to other behaviours which involve a present-future trade-off – including future discounting and reproductive scheduling (Chapter 2). If this is the case, then it would be more appropriate to examine life expectancy cues that can convey information about the controllability of mortality risks. We begin to address the question of the importance of controllability of mortality risks in Chapters 4 and 5 (Pepper & Nettle, 2014b, 2014a).

## Chapter 4. Perceived Extrinsic Mortality Risk And Reported Effort In Looking After Health: Testing A Behavioural Ecological Prediction

Like chapters 2 and 5, this chapter is a published paper. I have not altered it for inclusion in this thesis, except in order to refer to other relevant material within the thesis. The citation information for the publication is as follows:

Pepper, G. V, & Nettle, D. (2014). Perceived extrinsic mortality risk and reported effort in looking after health: Testing a behavioural ecological prediction. *Human Nature*, 25(3), 378–392.

## 4.1 Abstract

Socioeconomic gradients in health behavior are pervasive and well documented. Yet, there is little consensus on their causes. Behavioral ecological theory predicts that, if people of lower socioeconomic position (SEP) perceive greater personal extrinsic mortality risk than those of higher SEP, they should disinvest in their future health. We surveyed North American adults for reported effort in looking after health, perceived extrinsic and intrinsic mortality risks, and measures of SEP. We examined the relationships between these variables and found that lower subjective SEP predicted lower reported health effort. Lower subjective SEP was also associated with higher perceived extrinsic mortality risk, which in turn predicted lower reported health effort. The effect of subjective SEP on reported health effort was completely mediated by perceived extrinsic mortality risk. Our findings indicate that perceived extrinsic mortality risk may be a key factor underlying SEP gradients in motivation to invest in future health.

## 4.2 Introduction

Socioeconomic gradients in health outcomes are pervasive and well documented (Adler & Ostrove 1999; Melchior et al. 2011); people of lower SEP have shorter life expectancies and shorter healthy life expectancies than those of higher SEP (Crimmins & Saito 2001; Liao et al. 1999; Phelan et al. 2010; Wilkinson 1992). Evidence suggests that socioeconomic differences in health behavior account for up to half of the socioeconomic health gradient (Mokdad et al. 2004; Stringhini et al. 2010). People of lower SEP are more likely to smoke or to drink excessively than those of higher SEP (Harrell et al. 1998; Pridemore 2011), and are less likely

to take part in regular physical activity (McLaren 2007; Wardle et al. 2002). They are also less likely to adhere to treatment programs, even when there is no financial cost to doing so (Barr et al. 2002; Goldman & Smith 2002). The reasons for this SEP gradient in health behaviors have become an enduring point of debate across a range of disciplines including epidemiology, public health, health psychology, sociology and behavioural economics (Pampel et al. 2010).

Many nuanced explanations for SEP gradients in health behavior have been put forward, but there is currently little consensus across disciplines regarding their causes (Cutler & Lleras-Muney 2010; Pampel et al. 2010; Pepper & Nettle 2014c). Some explanations are based on the idea that people of lower SEP face constraints which people of higher SEP do not. These explanations posit that a lack of resources (a fundamental component of SEP) or a lack of specific health knowledge (potentially related to the education component of SEP) constraints people's ability to protect their health.

The first constraint-based explanation, that people of lower SEP lack the resources to "purchase" health (Darmon & Drewnowski 2008), cannot be a considered a complete one because it does not apply to some of the most common health damaging behaviors: Smoking, poor diet, physical inactivity, and alcohol consumption are major behavioral causes of mortality. Indeed, they were reported to have been the leading causes of death in the United States in the year 2000 (Mokdad et al. 2004). For at least two out of these four behaviors (smoking and alcohol consumption), the unhealthy option (consumption) is more financially costly than the healthy one (abstinence). Thus, the people who can least afford to spend money are spending money on behaviors that damage their health.

The second constraint-based explanation is that the socioeconomic gradient in health behavior is the result of socioeconomic differences in specific health knowledge (e.g., Siahpush, McNeill, Hammond, *et al.*, 2006). However, providing specific health information does not change behavior equally among high and low SEP individuals. For example, in the UK between 2003 and 2008 there was extensive government investment in public health information campaigns. Buck and Frosini (2012) examined how four behaviors (smoking, excessive alcohol

use, poor diet, and low levels of physical activity) changed during this time. They found that high SEP individuals dramatically reduced their levels of unhealthy behavior during the public health campaign period, whereas low SEP individuals did not. Receiving specific health information may have improved behavior in individuals already motivated to invest in health, while failing to change behavior in others.

Other studies have found that knowledge of the harms of smoking or the importance of exercise, for example, is widespread and differs minimally by SEP (Layte & Whelan 2009; Pampel et al. 2010). This raises the possibility that there is greater incentive for higher SEP individuals to invest in protecting their health than there is for individuals of lower SEP. In support of this, evidence suggests that desire to quit smoking and use of cessation tools do not differ across social class, whereas quitting success does (Kotz & West 2009). This implies that there is an SEP gradient in motivation to quit, rather than in knowledge that smoking is bad for health. We have given just a few examples here, but there is a wide range of evidence demonstrating SEP differences in response to health interventions (see White, Adams & Heywood, 2009).

If constraint-based explanations are incomplete, then we must turn to alternatives. We have argued elsewhere that most of the explanations put forward in the non-evolutionary literature are proximate ones that are potentially consilient with a single ultimate explanation, which we will now discuss (Pepper & Nettle 2014c). This ultimate explanation does not necessarily conflict with other explanations invoking proximate mechanisms such as stress, social networks, or efficacy and agency. It is simply a different level of explanation.

In previous work, we presented a theoretical model to explain SEP gradients in health behavior using an adaptive framework from behavioral ecology (Nettle 2010b). Following a long tradition in evolutionary biology (Medawar 1952; Williams 1957), the model divided the risk of mortality into two components: an extrinsic component, which remains the same regardless of the behavioral decisions that the individual makes, and an intrinsic component, which reflects how much effort the individual invests in preventing the health risks that can be mitigated. The model assumed that health-protecting investments are costly, in

the sense that the time and energy devoted to them must be taken away from other activities that individuals value. (There is a trade-off between investing in health behavior and investing in other adaptively relevant activities.)

The results of the model showed that as the extrinsic component of mortality risk increases, the optimal investment in protective health behavior decreases. Under conditions of high extrinsic mortality, the value of health-protecting investments is reduced, since even if one makes them, one may well be killed by something extrinsic anyway. Thus, people facing higher extrinsic mortality risks should reduce their investment in preventative health behavior and reallocate their investment toward other things. If people of lower SEP perceive that they face increased extrinsic mortality risk relative to people of higher SEP, then reduced investment of energy in long-term health could be an adaptively patterned response to the perceived environment, rather than a result of constraints or mistakes.

The model we have just described, though specific to health behaviors, is derived from life history theory. Models of the evolution of life histories predict that adaptively relevant behaviors such as reproductive scheduling and parental investment should be sensitive to mortality rates (see Stearns, 1992), and this prediction is borne out by comparative evidence (e.g., Harvey & Zammuto, 1985). An extension of this concept within human behavioral ecology is the idea that humans have evolved the capacity to ontogenetically calibrate their reproductive strategies in response to local mortality risk (e.g., Chisholm, Ellison, Evans, *et al.*, 1993; Lawson & Mace, 2011; Nettle, Coall & Dickins, 2011; Wilson & Daly, 1997). Indeed, empirical work has demonstrated associations between mortality rates and indicators of life history strategy (Low et al. 2008; Nettle et al. 2011; Quinlan 2010). However, nothing has been done specifically to investigate the associations between perceived extrinsic mortality risk and motivation to invest in health.

Here we focus on *perceived* extrinsic mortality risk. We do so because, although evidence suggests that people of lower SEP are generally exposed to greater risk of extrinsic mortality (e.g. Bolte, Tamburlini & Kohlhuber, 2010; Soskolne & Mano, 2010), we do not know that they perceive this to be the case. We look for SEP

differences in reported effort in looking after health and safety in general because, as discussed above, SEP differences in health behavior may reflect SEP differences in motivation to look after health, rather than constraints in their ability to protect their health.

In this paper, we report our findings from a survey of North American adults which included questions designed to test our hypothesis — that perceived extrinsic mortality risk would mediate the relationship between SEP and effort in looking after health. We collected measures of income, subjective SEP, effort made in looking after health, and perceived risk of mortality. Perceived risk of mortality, or its inverse, subjective life expectancy, has been widely studied (Dunkel et al. 2010; Krupp 2012), but we introduced a novel method to discriminate the extrinsic component of perceived mortality from the intrinsic component.

Based on the evolutionary model described above (Nettle 2010b), we made the following predictions:

1. Lower SEP will be associated with greater perceived *extrinsic* mortality risk, rather than perceived *intrinsic* mortality risk.

2. Greater perceived *extrinsic* mortality risk will be associated with lower reported effort in looking after health.

3. The relationship between SEP and reported effort in looking after health will be mediated by perceived *extrinsic* mortality risk.

## 4.3 Methods

## 4.3.1 Data Collection

The study was approved by the Newcastle University Faculty of Medical Sciences Ethics Committee. 600 North American volunteers were surveyed anonymously online using the SocialSci survey platform (www.socialsci.com). Respondents had previously signed up to take part in surveys via this platform. SocialSci recruits using a distributed online advertising network, print media, and live recruitment. They award Amazon (www.amazon.com) credit to respondents for taking part in their surveys. Respondents completed an electronic consent form before proceeding. They were then asked for basic demographic information:

age, gender, and gross annual income. Following this, we collected measures of reported effort spent looking after health, perceived risk of mortality, and subjective SEP. (This data was collected as part of the same survey referred to in Chapter 2.)

#### 4.3.2 Measures of SEP

We measured SEP in two different ways. First, we asked respondents to enter their gross annual income in US\$ into a free-text box. This measure was captured at the beginning of the survey along with age and gender. A free-text box was used to avoid unintentional priming effects that could be elicited by using income brackets (Haisley et al. 2008). Income was square-root transformed for analysis. Respondents were also asked to complete a subjective measure of SEP taken from prior studies (Griskevicius, Tybur, et al. 2011; Griskevicius, Delton, et al. 2011). This was done at the end of the survey, to avoid priming effects. They were asked to rate their agreement on a scale from 1 (strongly disagree) to 7 (strongly agree) with the statements: (a) "I don't worry too much about paying my bills"; (b) "I have enough money to buy things I want," and (c) "I don't think I'll have to worry about money too much in the future." The three responses correlated well with one another (r = 0.56 - 0.68, p < 0.01) and hence we summed them to give an overall subjective SEP score. The income and subjective SEP measures were correlated with one another (r = 0.30, p < 0.01), but not so highly as to treat them as equivalent. Income and subjective SEP were therefore entered separately into all our analyses.

#### 4.3.3 Reported Effort in Looking after Health

As a measure of motivation to invest in health, respondents were asked, "How much effort do you make to look after your health and ensure your safety these days? 0 is 'no effort at all' and 100 is 'the maximum effort you could make.'" This question was asked before the questions used to determine perceived risk of mortality. Questions about general motivation to protect health have been found to be predictive of a range of health behaviors (e.g., Becker, Drachman & Kirscht, 1972; Mirotznik, Ginzler, Zagon, *et al.*, 1998). We used a single general question about motivation because responses to multiple questions about individual health behaviors often need to be summed to show the same effect as a single, more

general motivation question in relation to actual behavior (e.g., Becker, Maiman, Kirscht, *et al.*, 1977; Mirotznik, Ginzler, Zagon, *et al.*, 1998; Mirotznik, Feldman & Stein, 1995).

## 4.3.4 Perceived Risk of Extrinsic and Intrinsic Mortality

We created two novel survey items to separate out the extrinsic and intrinsic components of perceived mortality risk. We asked, "If you made the maximum effort you could make to look after your health and ensure your safety, what do you think the chances would be that you would live to be 75 or more? 0 is 'no chance' and 100 is 'definitely.'" The extrinsic component of subjective mortality risk (henceforth perceived extrinsic mortality risk) is 100 minus this response. It is the portion of perceived mortality risk that the individual believes they cannot reduce via health effort. We then asked respondents, "If you made no effort at all to look after your health and ensure your safety, what do you think the chances would be that you would live to be 75 or more? Again, 0 is 'no chance' and 100 is 'definitely.'" Our perceived intrinsic mortality risk variable was the difference between the preceding question and this one. That is, it is the portion of overall perceived mortality risk which the respondent believes they are able to reduce via health effort. The relationship between our original measures and these variables is illustrated in Figure 4. We have also illustrated the predicted relationship between perceived mortality risks and subjective SEP in Figure 5A.

## 4.4 Analysis

We excluded 138 respondents who were under the age of 21 since measures of income and subjective SEP are likely to be unstable in participants younger than this age. We also excluded 22 individuals who spent less than 2 min completing the survey, the minimum possible time to engage with the questions established by piloting; 1 individual whose reported income was more than 10 standard deviations above the mean; and 1 individual whose sex was missing. (We give details of the effect of these exclusions in the results section.) This left a final sample of 438 respondents. We tested our three predictions using General Linear Models (GLM) in SPSS version 19.0, with age and sex as control variables in all cases. For prediction 3, we tested the statistical significance of mediation with a Sobel test (Preacher & Hayes 2004).



Figure 4. Schematic of our measures of perceived extrinsic and intrinsic mortality risk. The perceived extrinsic risk is the difference between 100% and the perceived chances of surviving to age 75 with maximum effort in looking after health. It is the portion of perceived mortality risk that the individual believes they cannot reduce via health effort. The perceived intrinsic risk is the difference between the perceived chances of living to 75 with maximum effort in looking after health, and with minimum effort in looking after health. It is the portion of perceived mortality risk which the individual believes they can reduce via health effort.

#### 4.5 Results

The raw data are downloadable as an online supplement to this paper. Of the 438 respondents included in the analysis, 184 were male and 254 were female. Ages ranged from 21 to 72 years (mean = 30.11 years, SD = 9.65). Reported personal annual incomes ranged from \$0 to \$250,000 (untransformed mean = \$39,307, SD = \$38,888). Subjective SEP ranged from the minimum possible score of 3 to the maximum possible score of 21 (mean = 11.11, SD = 4.90).

## 4.5.1 Prediction 1 Lower SEP will be associated with greater perceived extrinsic mortality risk, rather than perceived intrinsic mortality risk.

We ran a multivariate GLM with perceived extrinsic and intrinsic mortality risk as the outcome variables, and income, subjective SEP, age, and sex as the predictors. Subjective SEP was negatively associated with perceived extrinsic mortality ( $F_{1, 433} = 6.97$ , p < 0.01). That is, higher subjective SEP was associated with lower perceived extrinsic mortality (B = -0.83, s.e. [B] = 0.31). Income was not associated with perceived extrinsic mortality risk ( $F_{1,433} = 1.34$ , p = 0.25). Neither subjective SEP ( $F_{1, 433} = 0.86$ , p = 0.36) nor income ( $F_{1,433} = 0.18$ , p = 0.67) were significantly associated with perceived intrinsic mortality risk. (See Table 11 for full model results). Thus, for subjective SEP but not for income, our results conformed to the pattern we predicted (Figure 5B).

## 4.5.2 Prediction 2 Greater perceived extrinsic mortality risk will be associated with lower reported effort in looking after health.

In a GLM with reported effort looking after health as the outcome variable and perceived extrinsic and intrinsic mortality risk along with age and sex as the predictors, both perceived extrinsic ( $F_{1,433} = 244.13$ , p < 0.01) and perceived intrinsic ( $F_{1,433} = 5.42$ , p = 0.020) mortality risk were significantly associated with reported effort looking after health. Both associations were negative, with higher perceived mortality risk associated with lower reported effort (extrinsic: B = -0.64, s.e.[B] = 0.04; intrinsic: B = -0.10, s.e.[B] = 0.04). However, the association of reported health effort with perceived extrinsic mortality risk was much stronger than that with perceived intrinsic mortality risk. Perceived extrinsic mortality risk explained a substantial fraction of the variation not accounted for by other variables ( $n_P^2 = 0.362$ ), and perceived intrinsic mortality risk explained very little

of the variation not accounted for by other variables ( $\eta_p^2 = 0.012$ ). (See Table 12 for full model results.)



Figure 5. A: Predicted relationship between SEP and perceived mortality risk (arbitrary units). We predicted that it would be the perceived extrinsic mortality risk rather than the perceived intrinsic mortality risk that would show a social gradient. B: A plot of the observed relationship between our subjective SEP measure and perceived mortality with subjective SEP split into quartiles.
Predictor	Outcome	В	Standard error [B]	F ratio	Р	Lower Bound	Upper Bound	Effect size
Age	Perceived extrinsic	0.23	0.17	1.96	0.162	-0.09	0.56	0.005
Income		-0.02	0.02	1.34	0.248	-0.05	0.01	0.003
Subjective SEP		-0.83	0.31	6.97	0.009*	-1.45	-0.21	0.016
Sex		-2.01	2.95	0.46	0.497	-7.81	3.79	0.001
Age	Perceived intrinsic	-0.31	0.15	4.02	0.046*	-0.61	-0.01	0.009
Income		0.01	0.02	0.18	0.670	-0.02	0.04	0.000
Subjective SEP		0.27	0.29	0.86	0.355	-0.30	0.84	0.002
Sex		2.49	2.72	0.84	0.361	-2.86	7.84	0.002

Table 11. Results of a GLM predicting perceived extrinsic mortality risk (top) and perceived intrinsic mortality risk (bottom) from age, income, subjective SEP and sex.

Df = 1, error = 433, \*p < 0.05, reference category for sex is female, effect size =  $\eta_p^2$ 

	В	Standard error [B]	F ratio	р	Lower Bound	Upper Bound	Effect size
Subjective SEP	0.05	0.21	0.06	0.803	-0.37	0.47	0.000
Income	-0.00	0.01	0.09	0.769	-0.03	0.02	0.000
Perceived Extrinsic Mortality	-0.64	0.04	244.13	0.000*	-0.72	-0.56	0.362
Perceived Intrinsic Mortality	-0.10	0.04	5.42	0.020*	-0.19	-0.02	0.012
Age	0.09	0.11	0.69	0.407	-0.13	0.32	0.002
Sex	-3.49	1.99	3.08	0.080	-7.40	0.42	0.007

Table 12. Results of a GLM predicting reported health effort from subjective SEP, income, perceived extrinsic mortality risk, perceived intrinsic mortality risk, age, and sex.

Df = 1, error = 433, \*p < 0.05, reference category for sex is female, effect size =  $\eta_p^2$ 

# 4.5.3 Prediction 3 The relationship between SEP and reported effort in looking after health will be mediated by perceived extrinsic mortality risk.

To test prediction 3, we followed the steps laid out by Baron and Kenny (1986) for detecting mediation effects. We could not test for mediation of the association between income and reported health effort because the former was not a predictor of the latter.

However, subjective SEP was a significant predictor of reported effort in looking after health, with age, sex, and income controlled (F1,433 = 3.94, p = 0.048, B = 0.56, s.e.[B] = 0.28; see Table 13 for full model results). We had already established that subjective SEP was a predictor of extrinsic mortality perception (see prediction 1). To test for mediation, we added perceived extrinsic mortality to the GLM predicting reported effort in looking after health from age, sex, subjective SEP, and income (this is the model in Table 12). We found that the relationship between subjective SEP and health behavior was no longer significant (F1,433 = 0.06, p = 0.803) because perceived extrinsic mortality (F1,433 = 244.13, p < 0.01) explained the variation that was explained by SEP in Model 1 (Table 14). This suggests complete mediation (Baron & Kenny 1986), a conclusion supported by a significant Sobel test (z = 2.65, p < 0.01).

	В	Standard Error [B]	F ratio	р	Lower Bound (95% CI)	Upper Bound (95% CI)	Effect size
Subjective SEP	0.56	0.28	3.94	0.048*	0.01	1.11	0.009
Income	0.01	0.02	0.32	0.574	-0.02	0.04	0.001
Age	-0.02	0.15	0.03	0.873	-0.32	0.27	0.000
Sex	-2.46	2.63	0.88	0.349	-7.63	2.70	0.002

Table 13. Results of a GLM predicting reported effort in looking after health from subjective SEP, income, age, and sex

df = 1, error = 433, \*p<0.05, reference category for sex is female, effect size =  $\eta_p^2$ 

		В	Standard error [B]	F ratio	р	Lower Bound (95% Cl)	Upper Bound (95% CI)	Effect size
Model 1	Subjective SEP as a predictor of health effort†	0.56	0.28	3.94	0.048*	0.01	1.11	0.009
Model 2	Subjective SEP as a predictor of perceived extrinsic mortality†	-0.83	0.31	6.97	0.009*	-1.45	-0.21	0.016
Model 3	Perceived extrinsic mortality as a predictor of health effort with Subjective SEP controlled†	-0.64	0.04	244.13	0.000*	-0.72	-0.56	0.362
	Subjective SEP as a predictor of health effort with extrinsic mortality controlled†	0.05	0.21	0.06	0.803	-0.37	0.47	0.000
	Sobel Z	Standard error	р					
Mediation	2.65	0.20	0.008*					

# Table 14. A summary of the models (Table 11, Table 12, & Table 13) used to examine the mediation of the relationship between subjective SEP and reported effort in looking after health by perceived extrinsic mortality risk.

†Age, sex, and income are also controlled in all models, df = 1, error = 433, \*p < 0.05, effect size =  $\eta_p^2$ 

# 4.5.4 Effects on our Results of Including Participants Under the Age of 21

In the analyses reported above, we excluded 138 respondents who were under the age of 21 because we felt that personal income would not be an accurate reflection of their actual SEP, and parental income measures are often inaccurately reported (Boyce et al. 2006). Indeed, the correlation of income with subjective SEP increased from r = 0.20 (p < 0.01) to r = 0.30 (p < 0.01) after these younger participants were excluded. Nonetheless, for completeness, we also present the results of the analyses without any data excluded. They are as follows: The association between subjective SEP and reported health effort (controlling for age, sex, and income) was no longer significant when participants under 21 were included ( $F_{1,559} = 2.51$ , p = 0.114,  $\eta_p^2 = 0.004$ ). This means that we were unable to test for mediation. Nevertheless, with the younger participants included, perceived extrinsic mortality (controlling for age, sex, income, subjective SEP, and perceived intrinsic mortality) remained a significant predictor of reported health effort, with a moderate effect size ( $F_{1,559} = 318.20$ , p < 0.01,  $\eta_{\rm P}^2 = 0.364$ ). Despite the fact that we were unable to formally test for mediation, this result supports our hypothesis that perceived extrinsic mortality risk, which is often associated with SEP, is potentially a better predictor of health effort than SEP per se.

#### 4.6 Discussion

Our previously presented theoretical model (Nettle 2010b) led us to predict that perceptions of high extrinsic mortality risk would trigger psychological mechanisms that cause disinvestment in preventative health measures. We argued that this might explain the socioeconomic gradient in health behavior, if people of lower SEP are, or perceive themselves to be, at greater risk of extrinsic mortality (Nettle 2010b; Pepper & Nettle 2014c). Here, we collected survey data to test this hypothesis. We found a socioeconomic gradient in perceived mortality risk, with greater perceived risk among those of lower subjective SEP. Separating out the extrinsic and intrinsic components of this risk showed that it was the extrinsic component of perceived risk that increased as subjective SEP decreased, with no gradient in the intrinsic component (Figure 5B). Perceived extrinsic mortality risk was strongly negatively associated with reported effort in

looking after health, whereas perceived intrinsic mortality risk was only weakly associated with it (Table 12).

We found that our subjective measure of SEP, but not gross annual income, was positively associated with reported effort in looking after health. However, this association was completely mediated by perceived extrinsic mortality risk. This suggests that people of lower subjective SEP may be less motivated to look after their health, but only because they perceive themselves to be subject to mortality risks that are beyond their control. These results are consistent with previous empirical findings that people of lower SEP tend to be more fatalistic about their health outcomes and have a greater belief in the influence of chance on their health than those of higher SEP (Wardle & Steptoe 2003). However, they also demonstrate the benefits of taking an adaptively informed approach to understand variation in human behavior in the sphere of health. Our a priori theoretical model (Nettle 2010b), based on the behavioral ecological literature, suggested the potential importance of distinguishing extrinsic from intrinsic mortality, and it also predicted that extrinsic mortality motivated people to reduce their effort in looking after their health.

There are a number of limitations to the current study. We used an opportunity sample recruited through an existing online participant pool. It would be desirable to investigate whether the same patterns are found in population-representative samples. Our main SEP measures were income and a self-report scale. Income reporting in surveys is often inaccurate; disposable income, though more complex to assess, may be a better predictor of behavior (Moore et al. 2000; Winkler et al. 2006). The subjective SEP measure we used did capture more fine-grained aspects of resource availability, such as disposable income and financial stability that would not be captured simply by asking people for their gross annual income. However, this was a self-report measure of SEP: Although it was simple to administer, its relationship to more objective factors such as education and occupational status has not been explored here. To address this it would be ideal for a measure of perceived extrinsic mortality risk to be included in large health surveys in which respondents' SEPs are well characterized.

The socioeconomic gradient in reported health effort was only detectable in our sample with the participants under the age of 21 excluded from analysis. However, the existence of socioeconomic gradients in health behavior is extremely well documented in previous literature, and the null association in our sample without exclusions may simply reflect the instability of self-reported income and subjective SEP in participants who are not yet financially independent. Even with large samples, studies on the relationship between SEP and health behaviors often find small effects, especially when they use individual health behaviors rather than composite measures (e.g. Friestad & Klepp, 2006; Halleröd & Gustafsson, 2011; Hanson & Chen, 2007). So, though associations between SEP and health behavior are reliably uncovered in a variety of studies, effect sizes in individual studies tend to be small. Our findings suggest that this may be because a third variable — extrinsic mortality risk — accounts for much of the relationship between SEP and health behavior. In our data, the effect size for the relationship between perceived extrinsic mortality risk and reported health effort ( $\eta_p^2 = 0.362$ ) was substantially greater than for the association between our subjective SEP measure and reported health effort ( $\eta_p^2 = 0.009$ ). Indeed, the relationship between subjective SEP and reported health effort was entirely extinguished when perceived extrinsic mortality risk was added to the model (Table 14). This may be because our measures of SEP were not comprehensive. However, it may also be because SEP-related differences in perceived extrinsic mortality risk have greater power to explain differences in health effort than SEP per se. This possibility should be investigated further.

Our findings have potential implications in applied settings. They suggest that people of lower SEP may not make less effort to look after their health whimsically or through ignorance. Rather, they perceive that whatever they do, there is a relatively high chance that they will be killed by something that they can do nothing about, so they follow a behavioral strategy of investing more of their energy in other things. Improving our understanding of what shapes perceived extrinsic mortality risk, and how to alter it, could therefore increase the efficacy of public health interventions.

As discussed in the introduction, our predictions about SEP gradients in health effort were derived from life history theory. Models of the evolution of life histories

predict that behaviors such as health effort, reproductive scheduling, and parental investment should be sensitive to mortality risk. However, they also predict that if extrinsic mortality risk is high, we might expect a more general shift in time horizons (Hill et al. 2008; Kruger et al. 2008; Wilson & Daly 1997). That is, we might predict an increased tendency to prioritize immediate rewards and costs above delayed ones because, when risk of death is higher, the odds of being alive to receive future rewards are lower. There is some evidence that changes in time horizons occur in response to perceived mortality risk. When experimentally exposed to mortality primes, people who reported having low childhood SEP have been found to discount the future more steeply than those who were exposed to control primes (Griskevicius, Tybur, et al. 2011). People who reported suffering a greater number of recent close bereavements have been found to discount the future more steeply than those who had suffered fewer (Chapter 2, Pepper & Nettle, 2013). Exposure to violence has been found to be associated with future discounting (Ramos et al. 2013), and earthquake survivors have been found to discount future rewards more steeply than controls (Li et al. 2012). Given the large body of literature linking time perspective and related concepts, such as delay discounting and impulsivity, to health behavior (Adams 2009c; Adams & Nettle 2009; Beenstock et al. 2011), it is plausible that perceived extrinsic mortality risk affects both time horizons and motivation towards health effort, thus accounting for the associations found between them. However, to our knowledge, there have been no direct tests of the impact of extrinsic mortality cues on health behaviors. It is important that such tests be developed.

The research presented here focused on perceived extrinsic mortality risk. However, relatively little is known about the environmental cues that produce these perceptions. Cues might include exposure to violent crime or knowing people who have died from circumstances beyond their control. Indeed, evidence suggests that fear of crime and experiences of bereavement are associated with poor health (Chandola 2001; Stafford et al. 2007; Stroebe et al. 2007). It would be useful to understand to what extent such cues contribute to a person's perceived extrinsic mortality risk and whether qualitative differences between cues are important.

It would also be useful to know how accurate people's perceptions of mortality risk are. Some epidemiological evidence suggests that actual as well as perceived extrinsic mortality risk is higher in low-SEP communities (Bolte et al. 2010; Soskolne & Mano 2010). Although there may be a veridical basis to these perceptions, they may be inflated by media scare stories or by exaggerated accounts from peers. If this is the case, then something as simple as correcting people's perceptions may be enough to improve their health behaviors. However, this is not to understate the fundamental importance of public action to tackle the sources of extrinsic mortality that differentially affect those of lower SEP. Not only would making low-SEP neighborhoods and workplaces safer have the primary benefit of reducing extrinsic mortality, it could also produce a secondary benefit of improved health behaviors. This would have the overall effect of reducing socioeconomic inequalities in health.

# Chapter 5. Out Of Control Mortality Matters: The Effect Of Perceived Uncontrollable Mortality Risk On A Health-Related Decision

Like chapters 2 and 4, this chapter is a published paper. I have not altered it for inclusion in this thesis, except in order to refer to other relevant material within the thesis. The citation information for the publication is as follows:

Pepper, G. V, & Nettle, D. (2014). Out of control mortality matters: the effect of perceived uncontrollable mortality risk on a health-related decision. *PeerJ*, 2(e459), 1–24.

# 5.1 Abstract

Prior evidence from the public health literature suggests that both control beliefs and perceived threats to life are important for health behaviour. Our previously presented theoretical model generated the more specific hypothesis that uncontrollable, but not controllable, personal mortality risk should alter the payoff from investment in health protection behaviours. We carried out three experiments to test whether altering the perceived controllability of mortality risk would affect a health-related decision. Experiment 1 demonstrated that a mortality prime could be used to alter a health related decision: the choice between a healthier food reward (fruit) and an unhealthy alternative (chocolate). Experiment 2 demonstrated that it is the controllability of the mortality risk being primed that generates the effect, rather than mortality risk per se. Experiment 3 showed that the effect could be seen in a surreptitious experiment that was not explicitly health related. Our results suggest that perceptions about the controllability of mortality risk may be an important factor in people's healthrelated decisions. Thus, techniques for adjusting perceptions about mortality risk could be important tools for use in health interventions. More importantly, tackling those sources of mortality that people perceive to be uncontrollable could have a dual purpose: making neighbourhoods and workplaces safer would have the primary benefit of reducing uncontrollable mortality risk, which could lead to a secondary benefit from improved health behaviours.

#### 5.2 Introduction

It is important to understand what factors influence health behaviour. Some of the leading causes of death in developed countries result from preventable unhealthy behaviours such as inactivity, poor diet, smoking and alcohol consumption (Mokdad et al. 2004). Such preventable behaviours also cause a substantial burden on healthcare systems. For example, obesity-related health problems, such as type 2 diabetes and heart disease, are becoming a major issue in the UK, with 61% of adults and 30% of children in England being overweight or obese. Such obesity and overweight related health problems are estimated to cost the NHS over £5 billion a year (Department of Health Report 2011).

A substantial research effort has been made towards improving the efficacy of health messages to promote behaviour change. One of the key ideas to emerge from this research has been that perceived control and efficacy should influence health behaviour. Health Locus of Control describes the extent to which a person believes that their health is determined by the actions of individuals, rather than by chance, and whether the locus of that control is internal (a result of their own actions) or external (resulting from the actions of others). Prior findings suggest that Health Locus of Control is important both for health outcomes (e.g., Burker, Evon, Galanko, *et al.*, 2005; Poortinga, Dunstan & Fone, 2008) and for health behaviours (Reitzel et al. 2013; Wardle & Steptoe 2003).

Other research themes focus on the effects of mortality salience and perceived threat on health behaviour. Terror Management Theory (Greenberg et al. 1986) proposes that people have a fear of death which causes anxiety or terror when they are made aware of their vulnerability. It suggests that, when people are made to think about their mortality (a condition known as mortality salience) they will attempt to buffer their anxieties and to suppress conscious thoughts of death. Goldenberg and Arndt (2008) extended Terror Management Theory to create the Terror Management Health Model for behavioural health promotion. They proposed that conscious thoughts about death (as elicited by many fear appeals) would trigger behavioural responses (in this case, health improving behaviour) aimed at reducing the threat, and thus the accompanying fear of death. They proposed that when thoughts about death are unconscious, people should act

not to reduce the threat to their life, but to direct their efforts to maintaining a sense of meaning and self-esteem.

The fear appeal literature combines elements of control with those of threat. (Fear appeals are messages intended to persuade people to change their behaviour by inducing fear regarding health threats.) Theoretical frameworks used in the fear appeal literature (e.g., Extended Parallel Process Models and Protection Motivation Theory - comprehensively reviewed by Witte & Allen, 2000) emphasise the importance of efficacy in eliciting behaviour change. In general, these theories suggest that if there is a strong threat to health and a highly effective solution is available, then people will act to use that solution. However, if messages offer threats without suggesting that there are effective solutions, behaviour change will not occur. That is, these models state that threat serves to motivate people towards possible solutions, but that if people do not feel that the solutions will be effective, they are unlikely to act (Goei et al. 2010; Lewis et al. 2013; Witte & Allen 2000).

#### 5.2.1 The uncontrollable mortality risk hypothesis

Similarly, our previously presented theoretical model (Nettle 2010b) combined elements of control and threat to life. It suggested that differences in health behaviour could be explained by differential exposure to uncontrollable mortality risk: the Uncontrollable Mortality Risk Hypothesis. The hypothesis suggests that people who are likely to be killed by factors beyond their control should be less motivated to invest effort in looking after their future health. This makes intuitive sense when you consider that people who are exposed to high uncontrollable mortality risk are less likely to survive to reap the rewards of their healthy behaviour, which are likely to be garnered in the far future. To give a caricatured example, there is little point in investing in a healthier diet when you feel you could be killed by an erupting volcano at any moment. We previously tested predictions from this hypothesis using survey data (Pepper & Nettle, 2014b, Chapter 4). We found that people who perceived a higher portion of their personal mortality risk to be beyond their control were less motivated to invest effort in looking after their personal mortality risk to be beyond their control were less motivated to invest effort in looking after their health.

Our hypothesis differs from theories in the fear appeal literature, since these focus on the controllability of the specific aspects of health which are being communicated and not on the controllability of mortality risk more generally. For example, they predict that the belief that you can control your risk of diabetes by modifying your diet will affect your motivation to eat healthily. By comparison, our hypothesis predicts that perceived control over mortality risk should alter motivation towards healthy behaviour - even when the healthy behaviour is not a recommended response to that risk. For example, if you believe you are unable to control your risk of falling victim to a volcanic eruption, you should be less inclined to eat healthily. A healthy diet is not a recommended response to reduce the threat posed by a volcano and yet, we should expect the controllability of one risk to influence the payoff to investing in mitigating the other.

Our hypothesis also takes a different perspective to Health Locus of Control studies, which tend to implicitly assume that Health Locus of Control is a stable individual trait, rather than a flexible response to information from the environment. By comparison, behaviour as a response to environmental cues is a key assumption of the Uncontrollable Mortality Risk Hypothesis. Finally, while Terror Management Theory emphasises the importance of mortality per se, our hypothesis suggests that it is the controllability or the mortality risk that should be important.

In summary, a range of theories emphasize the importance of mortality salience and control for the behavioural responses to health messages. Our Uncontrollable Mortality Risk Hypothesis specifically predicts that cueing mortality risk per se will not affect health behaviours, but rather, that it will be the controllability of the mortality risk that influences the decision to behave healthily.

Here, we present three experiments testing this prediction. The first was a test of whether mortality primes can be used to influence a health-related decision — the choice between a healthy food reward and an unhealthy one. The second experiment used the same method but with primes that separated out the effects of controllability from those of mortality priming. That is, we tested whether there is an effect of mortality salience per se, or whether it is the controllability of mortality risk which is important. The third study aimed to rule out the possibility

that the results of the first two studies were due to demand characteristics; the participants did not know that they were taking part in an experiment and health was never explicitly mentioned.

# 5.3 Experiment 1: the effect of uncontrollable mortality on a health-related decision

Experiment 1 tested whether an uncontrollable mortality prime would affect a simple health-related decision: the choice between a reward of fruit (the healthy option) and chocolate (the unhealthy option). For this proof-of-concept experiment, we chose primes that we expected to produce the most extreme results. One prime suggested that causes of death were uncontrollable, and that people sharing the participant's demographics were dying younger than average (uncontrollable short life prime). The other prime suggested both that causes of death were controllable and that people sharing the participant's demographics the participant's demographics were living longer than average (controllable long life prime). We predicted that participants would report stronger intentions towards healthy behaviour and be more likely to choose fruit in the controllable long life treatment than in the uncontrollable short life treatment.

# 5.4 Methods, materials and analysis

All of our experiments (1, 2 & 3) received ethical approval from the Newcastle University Faculty of Medical Sciences ethics committee. Participants for experiments 1 and 2 were recruited using the Crowdflower crowdsourcing platform (http://crowdflower.com). Participants followed a link to the experiment, which was generated using Qualtrics Labs, Inc. software, Version [2013] of the Qualtrics Research Suite, <u>http://www.qualtrics.com</u>. Participants were presented with an information screen which contained statements about ethics and privacy and provided contact details for the experimenters. The introduction to the study explained that it was about life expectancy differences within the UK (see questionnaire in appendix 9.11). This included a link to a news article about Public Health England's Longer Lives website (http://longerlives.phe.org.uk/), which provides a map of the regions of England, ranked by rates of premature mortality. Since experiment 1 was launched on 2<sup>nd</sup> July 2013, less than a month after this map had been headline news, it made a timely cover story for the experiment. Participants completed an electronic consent form.

We needed to ensure that our participants were from the UK, because the primes were based on UK postcode statistics. Thus, participants were filtered through a location check using their Internet Protocol address (IP address) and an explicit question about whether they were resident in the UK. Participant location information (based on IP address) and reported postcode were triangulated with self-reported UK residency to assess the reliability of the data. Consistency of location reporting was used as an inclusion criterion (see appendix 9.12).

Participants moved on to a screen which asked for their age, gender and current postcode. After giving this information, all participants were presented with a "loading" animation, timed to auto-progress after 12s. The message under the animation read, "Thanks for submitting your information. It may take a while to match it to health data for people of your age and gender in your postcode area. Please wait a few moments". This loading screen was designed to create the impression that the demographic information given by participants was being used to look up real information about life expectancies for people who shared their characteristics. Participants then were randomly allocated to one of the primes.

In each prime, the message fed back to the participant used dynamically generated content to display a message tailored with the age, gender and postcode which had been entered previously. This was done to make the participants feel as though the information about their mortality risk was personal to them.

# 5.4.1 Uncontrollable short life prime

The uncontrollable short life priming screen read as follows: "Statistics indicate that, on average, [age] year-old [male/female]s in your postcode area [(postcode)] die 13 years younger than [male/female]s of the same age in the rest of the UK. The reasons for this are unclear and may be due to factors beyond individual control, such as traffic accidents and air pollution. We want to understand more about why this is happening. Please answer the following questions about your health".

#### 5.4.2 Controllable long life prime

The controllable long life priming screen read: "Statistics indicate that, on average, [age] year-old [male/female]s in your postcode area [(postcode)] live 13 years longer than [male/female]s of the same age in the rest of the UK. The reasons for this are unclear and may be due to individual behaviours, such as diet and exercise habits. We want to understand more about why this is happening. Please answer the following questions about your health."

#### 5.4.3 Outcome variables

Following the priming screen, participants moved on to the health behaviour questions. They were asked to answer some simple scale-based (0-100) questions about their intended health behaviour over the coming week (see appendix 9.11 for full questionnaire). We refer to the answers to these as selfreported health intentions. The first was a general question about the effort the participant intended to put into looking after their health. The second question was about whether the participant intended to eat the recommended 5 portions of fruit and vegetables a day. The third question was about whether the participant would do a recommended level of exercise. The final question was about how much alcohol the participant intended to consume. After the questionnaire was completed, participants were moved onto a screen, which was ostensibly separate to the questionnaire. They were thanked for taking part in the study and told that, as an extra thank you for taking part, they could opt to be entered into a prize draw. They were asked to select the prize which they would prefer to win. The options were an organic fruit box worth £11, or chocolate collection box worth £11. This was our behavioural outcome measure - their choice between a healthier prize (fruit) and an unhealthy one (chocolate). After choosing their reward, participants moved on to a debrief screen, which made it clear that the feedback given about life expectancies in their area had been false (debrief text is included in the questionnaire shown in appendix 9.11)

#### 5.4.4 Covariates

The age and gender that the participants entered at the beginning of the experiment were used as covariates. Their postcode was used to generate a deprivation score for their current residential neighbourhood. This was done

using the Office for National Statistics' Indices of Multiple Deprivation (IMD; Mclennan, Barnes, Noble, *et al.*, 2011). The IMD identify the most deprived areas of the country by combining a range of economic and social indicators into a single score. Areas can be identified by their IMD rank, which is considered to be a useful objective measure of an individual resident's socioeconomic status (Danesh et al. 1999). We used the statistics for the lower layer super output areas — LSOAs.

Finally, we used the lengths of time that the participant spent on the participant information screen and the priming screen as covariates. We did this because participants who spent more time reading the cover story and feedback information may have believed the cover story to a greater extent and thus may have been more strongly primed.

# 5.5 Analysis

All analysis was carried out in SPSS version 19. We excluded data from participants whose self-reported location was not consistent with our location checks (see section 9.12). The effects of our covariates on reported health intentions were assessed using a GLM. This was done so that any covariates that had a significant effect on self-reported health intentions could be controlled for in our main statistical model.

The effects of treatment on reward choice were evaluated using binary logistic regression. As in the GLM, we first assessed which, if any, of the covariates had an effect on reward choice in order to include them in the main model as needed. The data for all experiments reported in this paper can be accessed as part of the Supplemental Information.

# 5.6 Results

# 5.6.1 Descriptive statistics

35 participants were randomly allocated to the controllable long life treatment and 37 to the uncontrollable short life treatment. 39 participants were male and 33 were female. Ages ranged from 19 to 69 years. Time spent on the information page ranged from 0 to 199s, with a mean of 20s. Time spent on the priming pages ranged from 9 to 138s, with a mean of 22s. Participants' neighbourhood IMD

scores ranged from 3.64 to 65.40 (of a possible 0.53-87.80) with a mean of 23.88.

There was no significant difference in the ages of the participants across treatments ( $t_{70} = -0.50$ , p = 0.62). There was also no difference between treatments in the time spent on the information page ( $t_{69} = 0.70$ , p = 0.48) or the priming page ( $t_{69} = 1.09$ , p = 0.28). The IMD score of participants' postcodes did not vary across treatments ( $t_{61} = -0.59$ , p = 0.558). There was no difference in the distribution of the sexes of participants across treatments (Fisher's exact, p = 0.35).

#### 5.6.2 Main results

There was no effect of any of our covariates on self-reported health intentions. Thus, the covariates were not included in the main model (Table 15). There was also no effect of treatment on the self-reported health intentions (Table 15 & Table 16).

None of the covariates showed an effect on choice of fruit, rather than chocolate, as a reward. However, there was an effect of treatment on reward choice (Table 17). Of the participants in the uncontrollable short life treatment, 31% (n = 10) chose fruit as a reward. In the controllable long life treatment, 57% (n = 20) of the participants chose fruit (Figure 6, Table 17).

Table 15. GLM results for experiment 1. GLM results showing the effect of the covariates (model 1) and the controllable long life and uncontrollable short life treatments (model 2) on self-reported health intentions.

Model 1: Covariates	F	р	$\eta_p^2$
Age	1.44	0.238	0.115
Sex <sup>b</sup>	0.72	0.585	0.061
IMD score	0.37	0.828	0.033
Time on info page	1.65	0.178	0.131
Time on priming page	1.58	0.196	0.126

Model 2: Model for	F	р	$\eta_p^2$
Treatment	1.47	0.223	0.093

#### Notes.

a. df = 4, error = 44, p = significance ( $*p \le 0.05$ ).

b. The reference category is female.

c. df = 4, error = 57, p = significance ( $*p \le 0.05$ ).

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 Table 16. Means for experiment 1. Means and standard deviations for self-reported health intentions in the controllable long life and uncontrollable short life treatments.

Reported health intention	Treatment	Mean (standard deviation)
Effort in looking after health	Uncontrollable short life	62.67 (26.72)
	Controllable long life	67.93 (20.96)
Intention to eat 5 portions of fruit and veg per day	Uncontrollable short life	47.94 (34.29)
	Controllable long life	63.17 (26.80)
Intention to exercise three times over the coming week	Uncontrollable short life	60.70 (33.82)
	Controllable long life	56.03 (31.85)
Intended units of alcohol intake over the coming week	Uncontrollable short life	5.69 (7.08)
	Controllable long life	8.03 (16.18)

Table 17. Binary logistic regression results for experiment 1. Binary logistic regression results showing the effect of the covariates (model 1) on the odds ratios for selecting fruit over chocolate and the effect of the controllable long life prime compared with the uncontrollable short life prime (model 2).

Model 1: Covariates only	Odds ratio (lower CI – upper CI)	р
Sex <sup>a</sup>	1.64 (0.54-5.01)	0.383
Age	1.01 (0.97-1.06)	0.653
Neighbourhood deprivation score	1.00 (0.96-1.03)	0.896
Time spent on information page	1.00 (0.97-1.04)	0.790
Time spent on priming page	0.96 (0.91-1.01)	0.128

Model 2: Model for treatment effect	Odds ratio (lower CI – upper CI)	р
Treatment	2.93 (1.08-8.00)	0.036*

Notes.

CI = 95% confidence interval,  $p = \text{significance } (*p \le 0.05)$ .

a The reference category is female.



Figure 6. Fruit and chocolate choice in experiment 1. The percentage of participants who chose fruit or chocolate rewards after exposure to either a controllable long life prime or uncontrollable short life prime.

#### 5.7 Experiment 1 discussion

Contrary to our prediction, the results of experiment 1 demonstrated no effect of our primes on self-reported health intentions. However, there was an effect of our primes on a health-related decision — the choice of fruit versus chocolate. The effect of treatment on reward choice was notable. The proportion of participants who chose fruit went up from 31% in the uncontrollable short life prime to 57% in the controllable long life treatment (an 84% relative increase). The fact that there was an effect of the prime on the behavioural measure but not the self-report measures suggests that the priming may produce an implicit, automatic response, rather than an explicit, reasoned one. This is interesting, given that prior evidence suggests that a number of health-related decisions involve implicit, automatic processes (Gibbons et al. 2009; Sheeran et al. 2013).

Several aspects of experiment 1 needed improving upon. The experiment had no control condition, so we could not say what the baseline preferences with no priming would be. Our design also did not separate the effects of priming mortality per se from those of controllability, since our two primes differed in both these dimensions. Finally, it is possible that the effect seen in experiment 1 was actually a normative one: in the uncontrollable short life condition, the health behaviour of others was not mentioned. Meanwhile, in the controllable condition, the health behaviour of others was described. Social norms are thought to influence health behaviour (Ball et al. 2010; Wood et al. 2012), and it is possible that our participants were automatically conforming to the norms described in the primes. It was important to rule out this potential confound. Thus, in experiment 2, we added a control treatment, and designed new primes which separated the effect of mortality salience from that of controllability. Since the norms contained in the two controllable treatments were opposing, this also addressed the potential of a confounding normative effect.

# 5.8 Experiment 2: separating the effects of mortality priming from those of controllability priming

Our second online experiment built upon our first. We added a control condition in which participants entered their demographic data and postcode, but received no feedback about life expectancy for people of their demographic. We also separated out the life expectancy component of the message (whether it

suggested that people were living for more or less time than others) from the controllability of the causes of mortality. Thus, there were five conditions: uncontrollable short life, uncontrollable long life, controllable short life, controllable long life and a control condition. Our Uncontrollable Mortality Risk Hypothesis (see Introduction) predicts that the controllability of the primed mortality risk should be more important than whether or not mortality per se is made salient. Thus, we hypothesized that participants in the two controllable treatments would be more likely to choose fruit than participants in the uncontrollable treatments, regardless of whether the prime suggested that people were living longer or dying younger. In light of the result of experiment 1, we expected that we might see no effect of treatment on self-reported health intentions.

# 5.8.1 Methods and materials

As in experiment 1, participants were recruited using Crowdflower and followed a link to a Qualtrics-based experiment. The experiment was launched on August 14, 2013. The participant information, consent form and location check screens were the same as those used in experiment 1 (see appendices 9.11 and 9.12). Again, participants entered their demographic information, saw a "loading" animation, and then were randomly allocated to one of the treatments. While the primes in experiment 1 were personalised to age, gender and postcode, experiment 2 primes were only personalised by postcode. In addition, the reference frames were changed. We did this in order to test a form of words which would not involve deceit, because in our later field study (experiment 3, see section 5.12), there would be no opportunity to debrief participants. This meant shifting the reference frame (either the same residential area in the year 2000, or other UK regions in the present), so that deceit was not necessary (because it is true that people in Tyne & Wear are living longer than they were in the year 2000, but also, not as long as others in the UK — see experiment 3, section 5.12).

# 5.8.2 Control condition

In the control condition, there was no feedback after the participant entered their information. They simply waited for 12s at the loading screen and then saw the

message, "Thanks for submitting your basic information. Please answer the following questions about your health".

#### 5.8.3 Uncontrollable short life prime

The uncontrollable short life prime consisted of a message saying that people living in the participant's postcode area were dying younger than people in other parts of England. The reasons given for this were beyond the participant's control — in this case, high rates of violent crime and traffic accidents: "Statistics indicate that, on average, people in your postcode area [(postcode)] die younger than people in other parts of England. This seems to be because there are higher rates of traffic accidents and violent crime than in other areas. Please answer the following questions about your health".

# 5.8.4 Uncontrollable long life prime

The uncontrollable long life prime said that people living in the participant's postcode area, were now living longer than they had in the year 2000. Again, the reasons given were beyond individual control: "Statistics indicate that, on average, people in your postcode area [(postcode)] are living longer now than they were in the year 2000. This seems to be because of improvements in road safety and reductions in violent crime. Please answer the following questions about your health".

# 5.8.5 Controllable short life prime

The controllable short life prime stated that people living in the participant's postcode area, were dying younger than people in other parts of England. This time reasons given were within individual control — in this case, individual health behaviours: "Statistics indicate that, on average, people in your postcode area [(postcode)] die younger than people in other parts of England. The reasons for this are unclear, but it may be due to individual behaviours, such as diet and exercise habits. We want to understand more about why this is happening. Please answer the following questions about your health".

#### 5.8.6 Controllable long life prime

The controllable long life prime consisted of a message saying that people living in the participant's postcode area, were now living longer than they had in the

year 2000. Again, the reasons given were controllable: "Statistics indicate that, on average, people in your postcode area [(postcode)] are living longer now than they were in the year 2000. The reasons for this are unclear, but it may be due to individual behaviours, such as diet and exercise habits. We want to understand more about why this is happening. Please answer the following questions about your health".

# 5.8.7 Outcome variables

The outcome variables were the same as those used in experiment 1.

# 5.8.8 Covariates

As in experiment 1, age, gender, postcode IMD score and time spent on the information and priming pages were used as covariates.

# 5.8.9 Exclusions

The exclusion criteria were the same as those used in experiment 1 (see appendix 9.12).

# 5.9 Analysis

As in experiment 1, the effects of our covariates on reported health intentions were assessed using a GLM, so that any that had a significant effect could be included in the main model. We also used custom contrasts to investigate whether there were differences between the uncontrollable and controllable treatments and between the long and short life treatments. As in experiment 1, the effects of treatment on reward choice were tested using binary logistic regression. Again, we first assessed whether any covariates had an effect on reward choice, so that they could be included in our model. We ran a factorial treatment model, which contrasted the effects of the controllable treatments with the uncontrollable and the long life treatments with the short life ones.

# 5.10 Results

# 5.10.1 Descriptive statistics

There were 35 participants in the control treatment, 59 in the uncontrollable short life treatment, 44 in the uncontrollable long life treatment, 31 in the controllable short life treatment and 26 in the controllable long life treatment. There were 117

male participants and 78 female. Ages ranged from 18 to 73 years. Time spent on the information page ranged from 1 to 1,402s, with a mean of 102s. Time spent on the priming pages ranged from 0 to 448s, with a mean of 19s. IMD scores ranged from 3.15 to 87.80 (of a possible 0.53 - 87.80) with a mean of 25.84. There was no significant difference in the ages of the participants across treatments (F<sub>4,190</sub> = 1.20, p = 0.31). There was no difference between treatments in the time spent on the information page (F<sub>4,184</sub> = 0.69, p = 0.60) or the priming page (F<sub>4,186</sub> = 1.78, p = 0.13). There was also no significant difference in the IMD score of participants' postcodes across the treatments (F<sub>4,170</sub> = 0.99, p = 0.414). The distribution of the sexes of the participants was not significantly different across treatments (Fisher's exact, p = 0.13).

#### 5.10.2 Main results

In our covariates only model, there was an effect of sex on self-reported health intentions. Specifically, there was an effect of sex on intention to exercise (Table 18), with males having a greater intention to exercise than females (male mean = 70.34, s.e. = 2.97; female mean = 58.13, s.e. = 3.50). Thus, sex was included in the main model. However, as in experiment 1, there was no effect of treatment on self-reported health intentions (Table 18, Table 19). There were also no significant differences in reported health intentions when we compared controllable with uncontrollable or long life with short life conditions using custom contrasts (Table 20).

Table 18. GLM results for experiment 2. GLM results for the effect of covariates on health intentions (model 1) and the adjusted model for treatment plus sex, which had a significant effect in the first model (model 2).

Model 1: Covariates	F	р	$\eta_p^2$
Age	1.05	0.384	0.040
Sex	3.30	0.014*	0.116
IMD score	1.22	0.305	0.046
Time on info page	0.35	0.844	0.014
Time on priming page	0.50	0.735	0.019

Model 2: Model for	F	Р	$\eta_p^2$	df	df error
Treatment	1.01	0.437	0.032	12	363
Sex	4.92	0.001*	0.142	4	119

Notes.

a. df = 4, error = 101, p = significance ( $*p \le 0.05$ ).

b. p = significance (\*p  $\leq$  0.05).

Self-reported intentions	Treatment	Mean (s.d.)
Effort in looking after health	Control	67.24 (24.14)
	Uncontrollable long life	67.63 (21.91)
	Uncontrollable short life	62.53 (21.57)
	Controllable long life	65.4 (28.40)
	Controllable short life	60.26 (26.29)
Intention to eat 5 portions	Control	50.84 (31.13)
of huit and veg per day	Uncontrollable long life	60.94 (27.67)
	Uncontrollable short life	52.4 (29.20)
	Controllable long life	67.73 (25.88)
	Controllable short life	57.17 (31.96)
Intention to exercise three	Control	60.6 (33.99)
times over coming week	Uncontrollable long life	69.13 (29.92)
	Uncontrollable short life	66.53 (30.76)
	Controllable long life	57.40 (38.94)
	Controllable short life	62.52 (31.41)
Intended units of alcohol	Control	6.64 (9.84)
Intake over coming week	Uncontrollable long life	6.88 (7.75)
	Uncontrollable short life	5.55 (9.82)
	Controllable long life	3.07 (3.90)
	Controllable short life	3.13 (5.83)

Table 19. Means and standard deviations for self-reported health intentions in<br/>experiment 2.

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Table 20. Custom contrast results for experiment 2. Results of custom contrasts between controllable and uncontrollable, and short andlong life treatments for self-reported health intentions.

Custom contrast of controllable versus uncontrollable conditions	Sum of Squares	Mean Square	F	р
Effort in looking after health	101.41	101.41	0.18	0.672
Intention to eat 5 portions of fruit and veg per day	26.53	26.53	0.03	0.861
Intention to exercise three times over the coming week	1022.65	1022.65	0.99	0.322
Intended units of alcohol intake over the coming week	63.45	63.45	0.68	0.410
Custom contrast of long life versus short life conditions	Sum of Squares	Mean Square	F	р
Effort in looking after health	1266.21	1266.21	2.25	0.135
Intention to eat 5 portions of fruit and veg per day	1528.08	1528.08	1.77	0.185
Intention to exercise three times over the coming week	323.19	323.19	0.31	0.577
Intended units of alcohol intake over the coming week	64.55	64.55	0.70	0.406

Df = 1, p = significance (\*p  $\leq$  0.05).

None of the covariates in the covariates only model had an effect on choice of fruit as a reward (Table 21). Thus, no covariates were included in the main model. There was an effect of treatment on reward choice. Participants in the controllable treatments were more likely to choose fruit than participants in the uncontrollable treatments, or in the control (Table 21, Figure 7). However, there was no difference in food choice between the short and long life primes (Table 21, Figure 7). That is, there was an effect of the controllability of the mortality risk that was primed. The effect was of a similar magnitude to that seen in experiment 1. In the control treatment, 55% (n = 18) chose fruit. In the uncontrollable treatments 51% and 51% (uncontrollable long life, n = 21 and uncontrollable short life, n = 29) of participants chose fruit. In the controllable treatments, 71 and 75% (controllable long life, n = 15, controllable short life, n = 20) of the participants choose fruit.



Figure 7. Fruit and chocolate choice in experiment 2. The percentage of participants who chose fruit or chocolate rewards in response to controllable or uncontrollable, long or short life primes and the control condition of experiment

Table 21. Binary logistic regression results for experiment 2. *Binary logistic* regression results showing the effect of covariates and of treatments on the odds of selecting fruit over chocolate.

Model 1: Covariates only	Odds ratio (lower CI –upper CI)	р
Sex <sup>a</sup>	0.68 (0.30-1.50)	0.340
Age	1.03 (0.99-1.07)	0.125
Neighbourhood deprivation score	1.00 (0.98-1.03)	0.978
Time spent on information page	1.03 (0.99-1.06)	0.134
Time spent on priming page	1.00 (0.99-1.01)	0.470

Model 2: Model for treatment effect	Odds ratio (lower CI –upper CI)	р
Controllable vs. uncontrollable	2.59 (1.22-5.47)	0.013*
Long life vs. short life	1.06 (0.54-2.10)	0.862

CI = 95% confidence interval,  $p = \text{significance} (*p \le 0.05)$ 

a. The reference category is female.

# 5.11 Experiment 2 discussion

Experiment 2 parsed the effects of controllability from those of long and short life primes. The results showed that people were more likely to choose fruit over chocolate in the controllable, but not the uncontrollable treatments, regardless of whether they were told they were likely to have longer, or shorter life spans. The result in the experimental control treatment looked similar to those in the uncontrollable treatments (Figure 7). This suggests that, at least for the sample of participants in experiment 2, the "default" reward preference was akin to the preference under conditions of uncontrollable mortality.

As in experiment 1, there was no effect of treatment on self-reported intentions, but there was an effect on reward choice. As discussed for experiment 1, this suggests an implicit or automatic decision process, rather than an explicit or reasoned one.

The results of experiment 2 helped us to rule out the possibility that the effect seen in experiment 1 was a normative one. In experiment 1, in the uncontrollable short life condition, the health behaviour of others was not mentioned. Yet, in the controllable long life condition, it was the health behaviour of others in the participants' demographic that was suggested to be the cause of their longevity. This might have elicited a social norms effect by suggesting that others of the same demographic were living healthy lives. Norms are thought to play a role in influencing health behaviour (Ball et al. 2010; Wood et al. 2012). Thus, it was important that we use experiment 2 to rule out the possibility of a normative effect. In experiment 2, in the controllable mortality condition, the norm was that people were dying younger because of poor health habits. The selection of fruit still increased in this condition, relative to the uncontrollable and control conditions, suggesting that the result of experiment 1 was not due to a normative effect.

Although experiment 2 parsed the effects of controllability from those of long and short life primes and also ruled out the possibility of a normative effect, another potential confound remained: there may have been a demand effect, because both experiments 1 and 2 were explicitly health related. In order to rule this out, we ran a third experiment in the field.

# 5.12 Experiment 3: a replication of the controllability priming effect in a surreptitious field experiment

This field experiment built upon our online experiments. We ran it as a surreptitious experiment in order to remove any demand characteristics. This also allowed us to test whether the effect could be seen in a real-world setting. The study took place in a busy shopping centre in the Tyne and Wear area. Participants were told that they were taking part in a public opinion survey run by Newcastle University in exchange for being entered into a prize draw. Rather than our participants giving their details and receiving feedback about the average person of their demographic, we primed them using a question on the polling card. The questions suggested that people in Tyne and Wear are living longer, either due to uncontrollable causes, or due to controllable ones. That is, the primes were both long life primes, but the controllability of the causes was different. We hypothesised that, as in experiments 1 and 2, participants in the uncontrollable treatment.

#### 5.13 Methods

#### 5.13.1 Recruitment

Participants were recruited at a large shopping centre in the Tyne and Wear area. Data were collected over two weekends in November 2013, with the first run of data collection running from Friday to Sunday and the second on a Saturday and Sunday (five days in total). The experimenter stood next to a pop-up stand with two large polling boxes and the prize draw cards. The pop-up stand and the cards gave instructions for participating. The experimenter also explained the entry procedure verbally. Participants were asked to complete a polling card with their name, address and date of birth. They were then asked to circle their answer to a multiple choice question (the prime — see details below) and to place their card into a polling box. The main incentive to participants were told that they would all be entered for the chance to win this main prize. As "bonus" prizes there were ten organic fruit boxes and ten chocolate collection boxes to be won. Participants had to indicate which of these they would prefer to win, by posting their card into the relevant polling box. The primes were presented alternately at the polling

stand in two hour slots, which were counterbalanced across the 50h during which data were collected.

#### 5.13.2 Covariates

Age was calculated from the date of birth entered on the polling cards. As in the two online experiments, postcode IMD score was also used.

# 5.13.3 Primes

We used two primes, both longevity-focussed, but differing in their controllability. In the uncontrollable condition, participants were asked to answer the following multiple choice question: "Recent statistics show that people in Tyne and Wear are living longer now than they were in the year 2000. Why do you think this is? (A) Because there are fewer traffic accidents. (B) Because there is less violent crime. (C) Both: there are fewer traffic accidents and less violent crime". This question was designed to imply that the most important local sources of mortality were things beyond individual control.

In the controllable condition, participants were asked to answer a different multiple choice question: "Recent statistics show that people in Tyne and Wear are living longer now than they were in the year 2000. Why do you think this is? (A) Because people have more control over the kind of healthcare they receive. (B) Because people are looking after themselves better. (C) Both: people have more control over their care and are looking after themselves better". This question was intended to imply that the most important local sources of mortality were things within individual control. (An electronic copy of the prize draw card can be found in appendix 9.13.)

# 5.13.4 Outcome variable

The outcome variable was our participants' choice of bonus prize. As in experiments 1 and 2, this could be either an organic fruit box worth £11 or a chocolate collection box worth £11.

# 5.13.5 Analysis

As in experiments 1 and 2, the effects of treatment on reward choice were evaluated using binary logistic regression. In model 1 we assessed the effects of
the covariates, so that any that had a significant effect could be included in the model for treatment effect (model 2).

### 5.14 Results

## 5.14.1 Descriptive statistics

There were 121 participants in the uncontrollable treatment, and 116 in the controllable treatment. Ages ranged from 15 to 87 years. IMD scores ranged from 3.75 to 74.48 (of a possible 0.53 - 87.80) with a mean of 27.91. There was no significant difference in the ages of the participants across treatments ( $t_{229} = -0.78$ , p = 0.43). There was also no significant difference in the IMD score of participants' postcodes across the treatments ( $t_{227} = -0.16$ , p = 0.875).

## 5.14.2 Main results

Neither age, nor neighbourhood IMD score had any effect in the covariates only model. Thus, they were not included in the main model (Table 22). There appeared to be an effect of treatment on tendency to choose fruit, as a reward. Of the participants in the uncontrollable treatment, 22% (n = 27) chose fruit as a reward. In the controllable treatment, 34% (n = 39) of participants chose fruit, a 54% relative increase (Figure 8). However, the result of the binary logistic regression was marginally non-significant (p = 0.054, Table 22).



Figure 8. Fruit and chocolate choice in experiment 3. The percentage of participants who chose fruit or chocolate rewards in response to controllable or uncontrollable long life primes.

Table 22. Binary logistic regression results for experiment 3. Adjusted model showing the odds of selecting fruit over chocolate by experimental treatment with the uncontrollable treatment as the reference category.

Model 1 – covariates only	Odds ratio (lower CI – upper CI)	р
Age	1.01 (1.00-1.03)	0.177
Neighbourhood deprivation score	1.00 (0.98-1.02)	0.825
Model 2 – model for treatment effect	Odds ratio (lower CI – upper CI)	р
Treatment	1.76 (0.99-3.14)	0.054

Notes.

CI = 95% confidence interval,  $p = \text{significance} (*p \le 0.05)$ .

#### 5.15 Experiment 3 discussion

Our field experiment replicated the pattern seen in our online experiments, although the effect was marginally non-significant. This may have been due to a lack of power to detect the effect, which was smaller than in the other studies (odds ratios: experiment 1 = 2.93; experiment 2 = 2.59; experiment 3 = 1.76). However, given that qualitatively similar results were found for all three studies, we can be more confident that the statistically marginal result of experiment 3 represents a real effect (Moonesinghe et al. 2007). Future experiments should use larger samples to ensure adequate power.

There were some ways in which the effects seen in experiments 1 and 2 may have been diluted in experiment 3. The uncontrolled nature of the experimental environment allowed unpredicted participant behaviours. For example, some participants (n = 13) filled out the question card and then handed the card to a child or spouse, allowing them to choose the prize (invariably the children chose chocolate). Once the cards were in the polling boxes, they could not be traced, so these participants could not be identified or excluded from the analysis. If participants had not allowed those who accompanied them to choose the prizes, the effect might have been larger, but unfortunately it is not possible to confirm this.

Similarly, the fact that the experiment took place in a large shopping centre during November may have influenced the results. Many participants were at the centre to do their Christmas shopping. When selecting chocolate, some participants (number not noted) made comments such as, "I would choose fruit for myself, but chocolate will make a good Christmas present for someone". Thus, the effect might have been diluted in this experiment, but not in the online experiments, which were carried out earlier in the year.

There was one other minor issue with the field experiment (3). The experimenter was not blind to the treatments. However, the online experiments (1 and 2) were double-blind, since the treatments were randomly allocated by Qualtrics, and, as we have seen, the results were comparable.

The fact that the observed effect was replicable in a surreptitious experiment goes some way towards ruling out the possibility of a demand effect. Participants were

not aware that they were taking part in an experiment, or that it was related to health behaviour.

Finally, the result of experiment 3 demonstrates that the effect seen in the online experiments can be translated into a real world setting. This suggests that enhancing people's sense of control over sources of mortality and ill health could be an effective way of improving real world health behaviours.

### 5.16 Overall discussion

The results of our online and field experiments lend support to the Uncontrollable Mortality Risk Hypothesis. They suggest that perceptions about the controllability of mortality risk may have an important influence on health behaviours. Experiment 1 was the first, to our knowledge, to demonstrate an effect of uncontrollable mortality priming on a health-related decision. Experiment 2 was the first to separate out the effects of uncontrollable and controllable mortality primes on a health-related decision. Experiment 3 replicated the main effect of the first two experiments in a surreptitious experiment, suggesting that the effect seen in the first two experiments was not due to any demand characteristic.

While our experimental treatments affected participant behaviour, there was no effect on our participants' self-reported intentions (experiments 1 and 2). This implies that the decision to take fruit as a reward may have involved implicit and automatic processes (occurring without explicit reasoning see Evans, 2003), even when health was made salient. That is, people may not consciously calculate their degree of control over their mortality risk and then decide whether to choose a healthy or unhealthy reward. Previous research shows that a number of health behaviours seem to involve implicit processes and there have been calls to examine the role of implicit processes in health behaviour more closely (Gibbons et al. 2009; Sheeran et al. 2013).

In our introduction, we outlined theoretical perspectives that shared features of the Uncontrollable Mortality Risk Hypothesis. Although our experiments were not designed to test the predictions of the alternative hypotheses outlined in our introduction, we can still discuss our results in their context.

Our results may help to shed light on the associations between Health Locus of Control and health behaviour (Reitzel et al. 2013; Wardle & Steptoe 2003). When people feel that they have low control in general (external control beliefs), they are likely to believe that they have little control over their mortality risk. If so, investing effort, time or money in controlling what little they can, would have a lower payoff than for others who feel that they have more control over their mortality risk (internal control beliefs).

The Extended Parallel Process Model states that messages depicting threats will be acted upon to the extent that the available solutions are seen to be effective (Witte & Allen 2000). It proposes that a threat must have severe consequences in order to gain people's attention and motivate them to act. In addition to this, the recommended action must be perceived to be highly effective for this motivation to be translated into behavioural change. However, our result suggests that a threat does not need to be overt for an effect to be seen. In our experiments, there were no dramatic fear appeals. We simply mentioned that people of the participant's demographic were either living longer (or not) than average and manipulated the causes to be more or less controllable. In experiment 3, health was barely mentioned and no health advice was given. Nonetheless, we saw a switch to a healthier reward choice. This is likely to be because the choice was between two foods which are widely known to be healthy (fruit) and unhealthy (chocolate). No further health information was needed. This demonstrates that fear appeals may not be necessary to motivate behaviour change. In some cases, where the healthy choice is widely known to be so (e.g., to not smoke), recommended health actions may not be needed. It may be enough simply to reduce perceived (or better still, actual) uncontrollable mortality risks. Indeed, the fact that uncontrollable mortality risk alters the likely payoff of investing in health, could help to explain why interventions intended to improve health behaviours simply by giving information have been ineffective (e.g., Buck & Frosini, 2012; Downs, Wisdom, Wansink, et al., 2013). Merely giving information could be insufficient to change motivation (Pepper & Nettle 2014c; White et al. 2009), especially when the information given only pertains to risks already perceived as controllable and does nothing to reduce the severity of any uncontrollable risks perceived.

If the effects of our primes were implicit and automatic, as they appeared to be, this would contradict the predictions of the Terror Management Health Model. The Terror Management Health Model predicts that people should act in a health oriented way when explicitly primed, but not when the mortality salience is implicit (Goldenberg & Arndt 2008). In addition, in the treatments where participants were told they would live longer than average, it could be reasoned that mortality is made more distant, rather than salient. However, we still saw an effect in these treatments, based on whether the causes of mortality were controllable, rather than upon whether premature mortality was emphasised.

More research on the effects of uncontrollable mortality risk is needed. If mortality controllability priming could be used to increase motivation towards healthy behaviours, then it is important to test it in new populations and situations and to learn more about when it works. For example, our primes were effective in a situation where people were being offered a food reward free-of-charge. However, the situation may be different when people are paying for the food themselves. Our reward options were binary (fruit versus chocolate). Results may be different if there is a range of options to choose from — especially if the options are less obviously healthy and unhealthy ones. Furthermore, the experiments we have run so far have only examined food choice. We do not currently know whether such primes can be used to influence other health-related decisions.

Finally, although this is beyond the scope of the hypothesis, it is possible that control over factors other than mortality risk may influence health behaviour. Future experiments could include additional treatments, which prime the controllability of risks unrelated to mortality, such as the risks of becoming unemployed or becoming a victim of theft.

It is also important to learn more about perceptions of the controllability of common mortality risks. Understanding where perceptions come from could help policy makers to influence any sources of information which lead to misconceptions. For example, if media scare stories bias perceptions of uncontrollable mortality risk, then increasing awareness of this issue among journalists and calling for increased journalistic responsibility would be important.

The effect of controllability may go beyond health behaviour. It is possible that the controllability of mortality risk influences a range of behaviours involving trade-offs between costs and rewards in the present and those in the future. When the risk of death is high (and cannot be mitigated), the odds of being alive to receive future rewards are reduced. Thus, people who believe they have a high and uncontrollable risk of mortality should be less future-oriented than those who believe that they can control their mortality risk. There is some support for this idea in the existing literature. Differences in time perspective have been shown to be associated with a variety of health behaviours (Adams & Nettle 2009; Adams & White 2009; Adams 2009b; Adams 2009a; Adams 2009c), and with differences in reproductive scheduling (Daly & Wilson, 2005; Kruger, Reischl & Zimmerman, 2008; Pepper & Nettle, 2013 - Chapter 2). There is also evidence to suggest that differences in time perspective could be caused by exposure to signals of mortality risk. For example, future discounting has been found to be steeper in people who had experienced a larger number of recent bereavements (Pepper & Nettle, 2013, Chapter 2) and in recent earthquake survivors, compared to controls (Li et al. 2012).

The results of our experiments support the idea that perceptions about the controllability of mortality risk may be an important factor influencing people's health-related decisions. This finding is congruent with other evidence about the importance of Health Locus of Control for health (Burker et al. 2005; Holt et al. 2000; Poortinga et al. 2008; Wardle & Steptoe 2003; Williams-Piehota et al. 2004) and the influence of mortality priming on behaviour (Griskevicius, Tybur, et al. 2011; Griskevicius, Delton, et al. 2011; Mathews & Sear 2008). However, our Uncontrollable Mortality Risk Hypothesis is subtly different to other perspectives in the health literature and the results of our experiments suggest that the difference may be a crucial one. Adjusting perceptions about the controllability of mortality risk could become an important tool in health interventions. Our findings also emphasise the importance of tackling sources of mortality which are beyond individual control. Making neighbourhoods and work places safer would have the primary benefit of reducing mortality risks beyond individual control, but could also lead to improved health behaviours.

## Chapter 6. The Behavioural Constellation of Deprivation: Variation in Behaviour, Caused by Inequalities in the Promise of Tomorrow?

This chapter is a draft of a paper that I have written to draw together some of the main ideas of this thesis. I plan to submit a version of it for publication and would be grateful for your comments and suggestions for improvement.

## 6.1 Abstract

Socioeconomic differences in behaviour are pervasive and well documented, but their causes are not yet well understood. Here, we make the case that there is a clustering of behaviours associated with socioeconomic status, which we call the behavioural constellation of deprivation. We hypothesise that the relatively limited control associated with lower socioeconomic status curtails the extent to which people can expect to receive future rewards, leading to more present oriented behaviour in a range of domains. We illustrate this idea using the specific factor of extrinsic mortality risk, an important factor in evolutionary theoretical models. We emphasise the idea that the present oriented behaviours of the constellation are a logical response to structural and ecological factors, rather than pathology or a failure of willpower. We highlight some principles from evolutionary theoretical models that can deepen our understanding of how socioeconomic inequalities can become amplified and embedded. These principles are that: 1) Small initial disparities can lead to larger eventual inequalities, 2) Feedback loops can operate to embed early life circumstances, 3) Constraints can breed further constraints, and 4) Feedback loops can operate over generations. We discuss mechanisms by which extrinsic mortality risk may influence behaviour. We then review how our perspective fits with other findings about control and time perspective. Finally, we discuss the implications of our perspective for research and policy.

### 6.2 Introduction

Socioeconomic inequalities in life outcomes, such as health and life expectancy, are an issue of concern to policy makers and to society as a whole. The public health literature is replete with efforts to understand the forces that generate and perpetuate health inequalities. This literature shows that, to some extent, it is people's behaviour that generates much of the socioeconomic disparity in health and mortality (Pampel et al. 2010). Why the people in society who face the most challenging life circumstances should respond to them with behaviours that exaggerate their problems is an unresolved paradox. Furthermore, evidence suggests that this paradox is not restricted to health behaviour. In a recent review of financial behaviour, Haushofer and Fehr (2014) argued that "poverty may have particular psychological consequences that can lead to economic behaviors that make it difficult to escape poverty."

There have been thorough reviews of socioeconomic gradients in individual types of behaviour. For example, financial, health and even environmental behaviour have been examined (Haushofer & Fehr 2014; Pampel et al. 2010; Gifford & Nilsson 2014). However, these papers address the literature in behavioural silos. They do not ask questions as to why all of these behaviours should be simultaneously socioeconomically patterned. This paper aims to address that gap in the literature. We first make the case that there is a clustering of behaviours associated with socioeconomic status (SES) - the behavioural constellation of deprivation (BCD, section 6.3). We then explain this clustering of behaviour by:

- 1. Establishing it as a logical response to having limited control over the future outcomes of investments made in the present (section 6.4),
- 2. Illustrating how one specific uncontrollable factor, extrinsic mortality risk, should lead people to devalue the future (section 6.4.3),
- Examining the ways in which the BCD can cause deprivation to become embedded and amplified through additive routes and feedback loops (section 6.4.6),
- 4. Discussing the mechanisms by which limited control over future outcomes may cause the BCD (section 6.5).

We go on to discuss how our perspective converges with, and differs from, others attempts to understand socioeconomic differences in behaviour in terms of control and time perspective (section 6.6). Finally, we highlight some key implications of our perspective for policy and future research (section 6.7).

### 6.3 The Behavioural Constellation of Deprivation

In this section we present a cluster of behaviours that have been consistently found to vary with SES, our BCD. At first glance, the behaviours seem varied and unrelated, but we will argue that they have a common theme – that of balancing costs and benefits in the present, with those that may be encountered in the future.

People of lower SES tend to incur more debt, save less for the future and invest less in education than those of higher SES (Lea et al. 1993; Livingstone & Lunt 1992; White 1982; Chowdry et al. 2011; Blanden & Gregg 2004; Sirin 2005). They have children sooner - an effect most visible at its extreme with the consistent socioeconomic patterning of teen pregnancies (e.g. Smith, 1993; Imamura, Tucker, Hannaford, *et al.*, 2007; Johns, 2010; Nettle, 2010a). They also tend to invest less in their children – not only financially, but also through other efforts such as breastfeeding, reading to children and taking and interest in their education (Nettle 2010a; Kohlhuber et al. 2008; Kiernan & Huerta 2008; Hango 2007). People of lower SES tend to be less concerned about the environment and exhibit less environmentally friendly behaviour than those of higher SES (for a review see Gifford & Nilsson, 2014).

Research has consistently uncovered socioeconomic gradients in a range of health behaviours. People of lower SES have poorer diets and are less physically active than those of higher SES (McLaren 2007; Wardle et al. 2002; Everson et al. 2002; Brennan et al. 2009; Mobley et al. 2006; Droomers et al. 1998). They are more likely to use illicit drugs and to drink excessive amounts of alcohol (Boyle & Offord 1986; Daniel et al. 2009; Legleye et al. 2011; Droomers et al. 1999; Mäkelä 1999; Méjean et al. 2013). They also smoke more and have greater difficulty in quitting smoking (Harrell et al. 1998; Melotti et al. 2011; Legleye et al. 2011; Kotz & West 2009). This clustering of unhealthy behaviour is an enduring

conundrum in public health and is thought to contribute substantially to socioeconomic inequalities in health and mortality (Pampel et al. 2010).

## 6.4 Understanding the Behavioural Constellation of Deprivation

All of the BCD behaviours that we have outlined above entail trade-offs between the present and future. For example, the decision to save money, rather than spend it immediately, prioritises future needs and wants over present ones. Putting time, effort and money into getting an education, may yield future rewards, such as a better paid job. However, resources invested in getting an education cannot be spent on other endeavours that may be more immediately rewarding. To invest in a child's wellbeing or education, is to invest in the future of that child. However, those resources cannot be invested in other things, including other children. Many environmental issues entail deferred consequences. For example, we will not immediately see the damage done by our carbon emissions. Thus, many actions undertaken to mitigate environmental harm involve effort made in the present, to reduce negative consequences in the future. Similarly, healthy behaviour in the present often involves forgoing an activity that is rewarding in the short term, such as eating sugary foods, to prevent potentially detrimental health effects in the future. It might also involve investing time, money or energy in doing exercise that can feel unpleasant in the present, but will repay health dividends in the future.

There are myriad concepts in the literature related to the idea of a trade-off between costs and benefits in the present and future. For clarity, we have defined these terms in the glossary (below). For simplicity, we use the term "time preference" to refer to related concepts and measures such as future discounting, consideration of future consequences, impulsivity and future orientation. Measures of time preference have been related to many BCD behaviours and we review this literature later in section 6.6. At this point, it should be sufficient to say that, the BCD may be a result of socioeconomic differences in the trade-off between present and future. Support for this idea is reflected in the way that attitudes and perceptions vary with SES: People of lower SES have been found to be more impulsive, less future oriented and more pessimistic about their

futures than those of higher SES (DeWit et al. 2007; Adams & White 2009; Robb et al. 2009).

Why might there be socioeconomic differences in time preference? The literature presents a variety of perspectives on the question. Some view impulsivity as the result of "deficient inhibitory processes", implying that impulsivity is pathology (Dalley et al. 2011; Bari & Robbins 2013). Others suggest that stress and negative affect cause "short-sighted decision-making", implying that present oriented decisions are the result of poor judgement or impaired cognition brought on by stress (Haushofer & Fehr 2014). By contrast, we argue that socioeconomic differences in time preference may represent a logical response to factors associated with SES.

One of these factors may be personal control. People of lower SES report a lesser sense of personal control (Kraus et al. 2009; Bobak et al. 1998; Wardle & Steptoe 2003). In section 6.4.2, we will argue that this is because factors that are, by definition, a part of being lower SES, limit personal control, restricting the ability to ensure that investments in the future pay off.

### 6.4.1 Glossary

**Socioeconomic Status (SES)** – refers to ranking in a social and economic hierarchy and is usually measured by one or more factors including education, occupation, income and personal wealth.

**Behavioural constellation of deprivation (BCD)** – the cluster of behaviours associated with socioeconomic status, described in this paper (section 6.3).

**Extrinsic mortality risk** – is the part of a person's risk of death that *cannot* be influenced by their investment in healthy behaviour or physiological repair. It is the portion of total mortality risk that is not intrinsic.

**Intrinsic mortality risk** – is the part of a person's risk of death that *can* be influenced by their investment in healthy behaviour or physiological repair. It is the portion of total mortality risk that is not extrinsic.

**Impulsivity** – has been described in various ways. For example, impulsivity has been defined as a tendency to act with less forethought than others with equal ability and knowledge. It has also been defined as the propensity to have rapid, unplanned reactions to stimuli without considering the negative consequences of these reactions.

**Generativity** – refers to the belief that one's actions have future consequences.

**Time preference** - describes how an individual's preference for an outcome varies as a function of the time to that outcome.

**Time perspective** – describes the extent to which a person's focus on past, present and future experiences influences their decision making in the present.

**Future discounting** – is the tendency to choose smaller-sooner rewards over later-larger ones. Future discounting is also referred to as **delay discounting** and is often used as **a measure of time preference**. The inverse of future discounting is referred to as the **ability to delay gratification**.

**Future orientation** – describes the extent to which a person focuses on future outcomes.

**Consideration of Future Consequences (CFC)** – describes the extent to which a person's consideration of future outcomes influences their behaviour in the present.

**Locus of control** – describes the extent to which a person believes that their life outcomes are determined by their actions, rather than by chance. The locus of control can be described as internal (a result of their own actions) or external (resulting from the actions of others).

**Health locus of control** – is the same as the concept of locus of control, but is applied specifically to health outcomes. Note that the **Multidimensional Health Locus of Control Scale (MHLC)**, a commonly used measure of health locus of control, does not measure perceived control over mortality risk.

**Self-efficacy** – describes the extent to which a person believes in their own ability to complete a task. This is also referred to as **perceived behavioural control**.

**Ontogeny** – is the developmental lifespan of an organism.

**Ontogenetic calibration** - is the process of an individual adapting to its environment during the course of development.

**Ultimate explanations** – address the question of *why* something should be. They usually involve identifying the evolutionary (adaptive) function of trait or behaviour. **Proximate explanations** – address the question of *how* something happens. They usually involve identifying physiological or psychological mechanisms that produce a trait or behaviour.

#### 6.4.2 Socioeconomic status and personal control

How might SES relate to someone's degree of personal control? People of lower SES are, by definition, poorer and less educated than those of higher SES (Braveman et al. 2005). This lack of knowledge and wealth may limit the control that people of lower SES have over their lives (Infurna et al. 2011). Education provides knowledge that can increase control through the ability to tackle problems, while wealth enhances the ability to purchase solutions to problems. For example, residents in a deprived community may face a range of hazards such as unsafe housing or violent crime. They are less able to control their exposure to such hazards, if they cannot afford to move to a safer neighbourhood or to pay for repairs to their housing. Furthermore, a lack of education may mean that they are not familiar with their legal rights and local governance systems, limiting their ability to mitigate hazards by appealing to the relevant authorities. This idea is supported by several decades' worth of studies demonstrating associations between income, education or occupational grade and both perceived and actual personal control (Turner & Noh 1983; Umberson 1993; Ross & Wu 1995; Mirowsky et al. 1996; Lachman & Weaver 1998; Bosma et al. 1999; Gilmore et al. 2002; Lundberg et al. 2007; Poortinga et al. 2008; Kiecolt et al. 2009; Lee et al. 2009; Infurna et al. 2011).

## 6.4.3 Control and the promise of the future – the specific example of control over mortality risk

Limited control may include a restricted ability to ensure that returns on investments made in the present, for a payoff in the future, will be received. The most extreme example of a factor that limits the payoffs of investments for the future is death. A risk of death that is beyond one's control can be considered to be an extrinsic mortality risk (see glossary - section 6.4.1). Let us consider the role of extrinsic mortality risk in SES differences in health behaviour. If people of lower SES feel that they are likely to be killed by something they cannot control, it would be logical for them to invest (relatively) less effort in looking after their health (the part of their mortality risk that one cannot influence becomes larger, the odds of living long enough to see the rewards of healthy living become diminished.

A simplified example of the logic is as follows: If you live in a neighbourhood beset by violent crime, your risk of being a victim of homicide is relatively high. Again, if you are poor and cannot afford to move to a better neighbourhood, this risk is beyond your control. Under such circumstances, there may seem little point in quitting smoking or eating healthily, since you may not live to see the benefits of these actions. A quote from a young offender from Atlanta illustrates the severity of this problem in some deprived neighbourhoods; "...Where I'm from you never know if you gonna live one minute to the next. It's like a war out there. People die every day. You can go to sleep and hear gunshots all night man, all night..." (Brezina et al. 2009). This may seem exaggerated, but evidence shows that there are strong SES gradients in mortality due to homicide (Cubbin et al. 2000; Shaw et al. 2005; Redelings et al. 2010), assault and other violent crimes (Leyland & Dundas 2010; Markowitz 2003).

Furthermore, violent crime is not the only factor that might make mortality risk less controllable for the poor. Even when health risk behaviours are considered, low income populations still suffer an elevated risk of mortality relative to higher income populations (Lantz et al. 1998). This suggests that lower SES individuals face mortality risks that do not result from their behaviour – they are extrinsic. A systematic review by Bolte, Tamburlini and Kohlhuber (2010) examined environmental inequalities among children in Europe, offering examples of specific risks to which the poor are more exposed. They found that lower SES children suffer from multiple and cumulative exposures to health hazards including; traffic-related air pollution, noise, lead, environmental tobacco smoke, inadequate housing and unsafe residential conditions.

At first glance, it may seem that the levels of extrinsic mortality risk associated with deprivation in developed nations cannot be sufficient to cause meaningful differences in behaviour. However, (Nettle 2010b) used a mathematical model to make the case that increases in uncontrollable mortality at low absolute rates (1-3%), could be expected to lead to marked shifts in health behaviour. Although inequalities in control over exposure to hazards need not be great to have detectable effects on health behaviour, there are marked inequalities in mortality by certain causes. For example, in the UK between 1996 and 2000, people living in the poorest 10% of neighbourhoods were more than 5.7 times more likely to

be murdered than those living in the wealthiest 10% (Shaw et al. 2005). Given the conclusion of Nettle's (2010b) model, we might expect such inequalities to generate substantial SES differences in health behaviour.

In support of the idea that limited control should cause disinvestment in health, we have found that lower SES people perceive a greater portion of their mortality risk as being extrinsic and that this is associated with reduced health effort (Pepper & Nettle, 2014b, Chapter 4). Lawlor, Frankel, Shaw, *et al.* (2003) put forward a similar hypothesis. They examined trends in smoking prevalence among the different social classes over time (1948-99). They found that, once the health risks of smoking became widely known, there were marked decreases in smoking in the upper social classes, but not in the lower classes. They suggested that this was because the lower social classes were still suffering a substantial burden from non-smoking-related morbidity and premature mortality that reduced the incentive to forgo the otherwise appealing activity of smoking. Indeed, this idea is supported by evidence that smoking is more prevalent amongst occupational groups who are more exposed to hazards in the workplace and less prevalent among those who are exposed to fewer hazards at work (Sterling & Weinkam 1990).

It is not only health behaviour that should change in response to extrinsic mortality risk. People who have a limited ability to ensure their own longevity should operate on a shorter time scale with respect to a range of outcomes (Daly & Wilson 2005). The evidence suggests that they do. People living under conditions of high extrinsic mortality have children sooner than those living under conditions of low extrinsic mortality (Wilson & Daly 1997; Quinlan 2010; Störmer & Lummaa 2014). Across countries, there are strong associations between mortality rates and ages at first birth (Bulled & Sosis 2010; Low et al. 2013; Low et al. 2008). Similar patterns can be seen among individuals within countries (Nettle 2010a; Quinlan 2010; Wilson & Daly 1997). There are also associations between parental investment and mortality risk (Quinlan 2007). Experimental evidence shows that mortality primes can influence attitudes towards reproduction, including ideal ages at first birth (Griskevicius, Delton, et al. 2011; Mathews & Sear 2008). One study even examined several behaviours from the BCD simultaneously. It showed that the scheduling of marital and reproductive

behaviours, as well as investment in and attainment of education, were associated with life expectancy (Krupp 2012).

Mortality risk also appears to influence the extent to which people value financial future outcomes. Exposure to violence is associated with future discounting (Ramos et al. 2013). Earthquake survivors discount future rewards more steeply than controls (Li et al. 2012), and experiences of close bereavement are associated with greater future discounting (Pepper & Nettle, 2013, Chapter 2). In an experiment by (Griskevicius, Tybur, et al. 2011), participants who reported lower childhood SES and were exposed to a newspaper article reporting random acts of violence, discounted future rewards more steeply than controls.

# 6.4.4 Control and the promise of the future – other extrinsic factors may matter

We have made the case that there is a behavioural constellation associated with deprivation, which is characterised by a tendency to prioritise proximate outcomes above distant ones. We have suggested that people of lower SES prioritise the present because they are less able to ensure that they will receive future outcomes. This illustrates the link between SES, control and time preference. We have used extrinsic mortality risk as an illustrative example, in part because mortality is the most definitive future limiting factor. Moreover, extrinsic mortality risk has been extensively studied in evolutionary theoretical models, principles from which can be used to deepen our understanding of socioeconomic differences in behaviour (see section 6.4.6).

Nonetheless, it is important to note that, although we have emphasised the role of extrinsic mortality risk, socioeconomic differences in control over other future limiting factors may also be important. For example, deprived neighbourhoods have lower levels of trust, cooperation, and social capital (Schroeder, Pepper & Nettle, 2014 - see apendix 9.2; Drukker & van Os, 2003; Drukker, Kaplan, Feron, *et al.*, 2003; Hill, Jobling, Pollet, *et al.*, 2014). This may result in their residents feeling less able to rely on others to deliver on their promises of future rewards. They should therefore be less willing to accept a delay, which contains an inherent risk that the future reward will not be received. This idea is supported by experimental evidence. After having interacted with an experimenter who failed

to deliver on a promise, children were less willing to wait for a larger reward than those who had interacted with a reliable experimenter (Kidd et al. 2013).

In summary, a combination of future limiting factors, including extrinsic mortality risk, may account for the BCD, which is characterised by the prioritisation of present over future. Many of our examples have involved the expected effect of future limiting factors on the willingness to wait for *rewards*. However, it should also be noted that, the same principle applies to *negative outcomes*. People should be less worried about accruing debt, if they believe there is a limited chance that they will ever have to repay it. Similarly, they should be less concerned about indulging in activities that are rewarding in the short term, and damaging in the long term, if they think that they may not be around to see the negative consequences of those actions in the future (Daly & Wilson 2005).

#### 6.4.5 Extrinsic mortality risk in evolutionary models

Evolutionary theoretical models have comprehensively examined extrinsic mortality risk as a factor in ageing and life histories (Medawar 1952; Stearns 1992). Models of ageing identify it as a factor that limits the energetic investment that should be made in physiological repair (Kirkwood 1977; Kirkwood & Austad 2000). They also predict earlier reproduction in response to extrinsic mortality risk (Kirkwood & Rose 1991; Westendorp & Kirkwood 1998). These predictions are supported by empirical evidence: Mammals that suffer high levels of natural mortality mature earlier, start reproducing sooner, have shorter gestation periods and give birth to larger litters of smaller offspring (Harvey & Zammuto 1985; Promislow & Harvey 1990). Experimental evolution studies show that, if adult mortality rates are manipulated, shorter lifespans and earlier peak fecundity evolve (Stearns et al. 2000).

Most models of ageing and life histories examine how the strategies of organisms should evolve over generations. However, the logic of these models inspired the prediction that people should ontogenetically calibrate their behavioural investments in health in response to extrinsic mortality (Nettle 2010b). That is, we assume that natural selection has endowed organisms with the ability to adjust their behaviours in response to their environments (adaptation within a lifetime). This assumption is supported by evidence that humans calibrate their

reproductive strategies in response to local mortality risk (e.g. Chisholm, Ellison, Evans, *et al.*, 1993; Lawson & Mace, 2011; Nettle, 2011; Nettle, Coall & Dickins, 2011; Low, Hazel, Parker, *et al.*, 2008).

We have recently found support for the idea that people may alter their *behavioural* investments in health in response to extrinsic mortality risk (Pepper & Nettle 2014b; Pepper & Nettle 2014a). This prediction was made based on evolutionary theory. However, another implication of the theory is that *physiological* investment in health may be calibrated within an individual's lifetime, based on rates of extrinsic mortality (Cichoń 1997). That is, exposure to extrinsic mortality may dictate rates of physiological ageing. This relates to the question of health inequalities, because it has been proposed that people of differing SES may age at different rates (Adams & White 2004). Thus, SES differences in exposure to extrinsic mortality risk may drive SES differences in ageing.

In summary, evolutionary biological models have made the case that extrinsic mortality risk should determine rates of ageing due to physiological disinvestment in future health (Kirkwood 1977; Kirkwood & Austad 2000). They have also demonstrated that investments may be calibrated within an individual's lifespan, explaining differential rates of ageing (Cichoń 1997). We argue that behavioural investments in the future may be similarly determined by extrinsic mortality risk (Nettle, 2010b; Pepper & Nettle, 2014b - Chapter 4, Pepper & Nettle, 2014a - Chapter 5, Pepper & Nettle, 2014c - appendix 9.1). If extrinsic mortality risk does trigger a double disinvestment in future health, through both behavioural and physiological pathways, then this could generate a composite effect. However, there are other routes by which inequalities might become magnified. In section 6.4.6, we outline some principles from evolutionary models that can help us to understand how health inequalities are perpetuated.

# 6.4.6 Principles from evolutionary models and their relevance to socioeconomic inequalities

The dual disinvestment in the future, described in section 6.4.5, could lead to marked disparities in health outcomes from initially small differences in exposure to extrinsic morality risk. However, there need not be double disinvestment for

this to occur. If there are initial inequalities in exposure to extrinsic mortality risk, these will become summed with the intrinsic mortality risk generated by disinvestment in health, to give a larger total mortality risk (Figure 9, Nettle, 2010b).





We have outlined two additive routes by which small initial differences in exposure to extrinsic mortality risk may be amplified, generating larger eventual disparities in mortality: 1) double disinvestment through both physiological and behavioural pathways and, 2) the combined effect of extrinsic mortality risk and the intrinsic risk it causes via behavioural and physiological disinvestment. Disparities may also become amplified as a result of feedback loops. A simple example is as follows. Let us assume that unhealthy behaviours do some amount of irreparable damage. Once this damage is done it is, technically, extrinsic. That is, damage done in the past cannot be reversed by healthy behaviour in the present. This irreparable damage, like other sources of extrinsic mortality risk, limits the benefit of healthy behaviour, which leads to more unhealthy behaviour, which does more damage. Thus, healthy behaviour is further disincentivised and the cycle compounds itself (Figure 10). Given such a dynamic, one could take

two identical individuals, start their lives in environments with differing levels of extrinsic mortality risk, move them into identical environments, and still see diverging outcomes (Figure 11). Such positive feedback loops are often identified in theoretical models (e.g. Sozou & Seymour, 2003; Luttbeg & Sih, 2010).



Figure 10. The hypothesised feedback loop between extrinsic mortality risk, intrinsic mortality risk (resulting from behavioural and physiological disinvestments in health) and total mortality risk. Extrinsic mortality risk alters the optimum behavioural and physiological investments in health. Any level of disinvestment in health increases the total mortality risk. Assuming that disinvestments in health leave irreparable damage, they will feed back into extrinsic mortality risk, increasing it and continuing the feedback loop.



Figure 11. Illustrative model showing how the effects of early life exposure to extrinsic mortality risk may become embedded over time. Two theoretical individuals start out in environments with differing levels of extrinsic mortality risk. One faces low extrinsic risk (dotted line) and the other relatively high extrinsic risk (solid line). Initially, their risks are entirely environmental. That is, the risk of mortality that the individuals face is entirely due to external causes. At this initial stage, they decide to make different levels of investment in their future health, based on their externally generated risk of extrinsic mortality. Damage due to any disinvestment at the time of each investment decision is assumed to be irreparable. At the second decision point, the individual who started in the high risk environment has invested less in health than the individual who started in the low risk environment, thereby sustaining more irreparable damage. Both individuals then find themselves in an environment with the same level of external extrinsic mortality risk. At this point, the only difference between them is the level of investment that they initially made in their health – their internal extrinsic mortality risk. This difference, entirely the result of investment decisions made based on their initial environments, influences the optimum investment in health at future time points. Thus, the original difference between the two individuals becomes amplified, so that they experience different levels of overall mortality risk, despite having been in the same environment for the majority of their lifespan.

Of course, Figure 11 shows an oversimplified model, which makes some unrealistic assumptions. However, it illustrates how inequalities in early life might become embedded to the point that later intervention has little impact in terms of closing the life expectancy gap. This is important, given that much evidence supports the idea that early life circumstances are important for determining health in later life (e.g. Case, Fertig & Paxson, 2005; Palloni, Milesi, White, *et al.*,

2009; Blackwell, Hayward & Crimmins, 2001; Haas, 2008; Miller, Chen & Parker, 2011; Nettle, 2014; Aizer & Currie, 2014). Data even show associations between early life circumstances and biomarkers of ageing. For example, adult blood DNA methylation profiles have been found to be more strongly associated with childhood than adult SES (Borghol et al. 2011) and childhood exposure to violence is associated with greater telomere erosion (Shalev et al. 2012).

Another principle from theoretical models that can be applied to the question of the BCD is that of constraint. Individuals who start out in a poor state, economically or physiologically, may appear to make illogical choices, when in fact they are "making the best of a bad job" (Luttbeg & Sih 2010). In theoretical models of adaptive behavioural syndromes, individuals who started off in a better state always did better than those who started in poorer states, even though all individuals were making optimum decisions given their starting points (Luttbeg & Sih 2010). This emphasises the fallacy of assuming that the logical choice is the same for all individuals. What is optimal for one individual might be suboptimal for another. The concept of making the best of a bad lot is important for our two hypothetical individuals in Figure 11. Although their adult environments are identical, they may still display different health behaviours and experience different health outcomes, because they had different early life experiences. Their initial decisions, which were optimal given the constraints they faced at the time, alter what is optimal for them to do later on, relative to those who had a better start: Constraints breed constraints.

The feedback loops that we have described can also be amplified over generations. Those who start out in poor conditions may do worse overall than those who don't and can pass this disadvantage onto their children, further exacerbating the cycle. A recent review by Aizer and Currie (2014) summarised the data in support of this. They found that maternal disadvantage translated to poorer child health through a range of mechanisms including poor maternal health, poor maternal health behaviour and exposure to harmful environmental factors.

We have reviewed a number of principles from evolutionary models of ageing (the result of physiological disinvestment in future health) that could be applied to

the problem of individual differences in health behaviour (behavioural disinvestments in future health). First and foremost, we have emphasised the idea that extrinsic mortality risk should reduce investment in future outcomes. We have also reviewed the ideas that small initial differences can lead to large eventual disparities, and that feedback loops are at work, and can operate intergenerationally. These principles can help us to understand how socioeconomic inequalities in health and longevity can become embedded and amplified through differing rates of ageing and unhealthy behaviours. The differences in life expectancy that are generated through these additive pathways and feedback loops may drive the BCD.

## 6.5 The mechanisms by which extrinsic mortality risk may influence behaviour

We have used insights from evolutionary models to explain why the BCD should exist in populations of people who have limited control over their future prospects (section 6.4.3). Another central feature of the evolutionary approach is that it makes the distinction between ultimate and proximate causes of behaviour (Mayr 1961; Tinbergen 1963). Ultimate explanations are about why a behaviour should occur in a given population and environment, given the payoffs to that behaviour in that environment. As such, our explanation is an ultimate one. However, ultimate explanations do not preclude proximate ones, which are about how behaviour is generated. For example, they might identify the psychological or neural mechanisms involved. From an evolutionary perspective, these proximate and ultimate explanations are complementary (Scott-Phillips et al. 2011).

So what proximate mechanisms might underlie the BCD? Perhaps people are conscious of their own future prospects and deliberately alter their behaviour to reflect them? In a study of low-income American teen mothers, Geronimus, (1996) found that, despite the stigma attached to teen motherhood, the young women seemed to be choosing to have children sooner. They seemed to perceive that women should have children sooner because their health would not be good enough to withstand pregnancy and motherhood later on. This contrasts with the common perception that teen pregnancies are the result of whim or ignorance. Bolland (2003) found that young people in deprived urban

neighbourhoods, who did not expect to live long, saw little point in planning for their futures, and tended to engage in risky behaviour, such as substance abuse.

What of the possibility that people adjust their behaviour in response to mortality cues without any conscious reflection? In experimental tests, we found that, if people were primed to feel that prevailing mortality risks were controllable, they were more likely to choose a healthy snack than an unhealthy one. However, those participants who chose a healthier snack did not report a greater intention to eat healthily than participants who did not (Pepper & Nettle, 2014a, Chapter 5). This suggests that the effect may be due to an implicit, automatic response, rather than an explicit, reasoned one. This is consistent with prior evidence suggesting that some health-related decisions involve implicit, automatic processes (Gibbons et al. 2009; Sheeran et al. 2013). Another interesting implication of this finding is, that a BCD behaviour can be altered using a brief manipulation. Thus, although unhealthy behaviours may be partly driven by embedded beliefs, behaviour remains relatively malleable, with people responding immediately to new information about their prospects.

What of the idea that people may act impulsively because impulsive behaviours are a lower class norm? We have argued that this may be one of the proximate mechanisms that exacerbate behaviour (Pepper & Nettle 2014c). For example, peers may support healthy behaviour, or encourage unhealthy behaviour in social settings (Christakis & Fowler 2007; Christakis & Fowler 2008). Once established, SES differences in behaviour may be further perpetuated by class norms. However, this does not explain why lower, or higher, SES groups initiate those patterns of behaviour in the first place. For this, an ultimate explanation is required.

In conclusion, multiple proximate mechanisms may act in concert. People may make deliberate, reflective choices, based on their perceived future prospects, or their responses may be automatic and unconscious. People may also learn about their own life prospects from others, adopting the social norms of their communities. Yet none of these mechanisms are mutually exclusive. They may all be proximate ways in which the BCD comes about, as a result of the ultimate cause – extrinsic mortality risk (and other extrinsic future-limiting factors).

#### 6.6 How our explanation fits with other findings

As outlined in section 6.4.2, being low SES, by definition, means having limited wealth and education. We argue that this means that lower SES people have restricted control over future-limiting factors, including the most definitive of future limiting factors - extrinsic mortality risk. This should lead them, simultaneously, to have lower perceived control, a greater tendency to discount future rewards, and to display many of the more present-oriented behaviours in the BCD. That is, low perceived personal control, future discounting and BCD behaviours should be correlated. We arrived at this prediction from the perspective of evolutionary theory. However, researchers working from myriad perspectives have converged on the finding that control and time preference are associated with BCD behaviours. We shall now review some of this evidence.

The consumer behaviour literature has explored the role time perspective in financial behaviour. Perhaps unsurprisingly, future orientation increases the tendency to save for the future (Jacobs-Lawson & Hershey 2005; Howlett et al. 2008). Measures of perceived control, such as fatalism and locus of control, are also associated with the tendency to save for the future. Specifically, people who are more fatalistic, or perceive themselves to have less control over the future, less often save for the future (Shapiro & Wu 2011; Perry & Morris 2005). This can also have an impact at the household level. Households in which the reference person has a higher degree of perceived control save more in absolute terms, but also as a percentage of their income (Cobb-Clark et al. 2013).

Measures of time preference are also associated with educational attainment. Future discounting is negatively associated with both high school and college grades (Kirby et al. 2005; Duckworth & Seligman 2006; Lee et al. 2012). Similarly, being future oriented is associated with better academic engagement and performance in high school students (Brown & Jones 2004). There have even been experimental interventions, aimed at increasing future orientation in order to improve educational and career outcomes in high school and college students (Marko & Savickas 1998). Similarly, locus of control has been related to educational outcomes. Children with greater perceived personal control show

better educational attainment, independent of other factors, such as SES and their parents' level of interest in their education (Juan David Barón 2009; Flouri 2006). The related, but distinct concept of self-efficacy has also been found to predict students' educational engagement, aspirations and attainment (Zimmerman 2000).

There is a scarcity of literature on the association between time preference and reproductive timing. However, we have found that people who had experienced a greater number of bereavements (a potential cue to mortality risk) had earlier ideal and actual ages at first birth and also discounted future financial rewards more steeply (Pepper & Nettle 2013). The literature on control beliefs and reproductive timing similarly is sparse. One study found that adolescents who had a child, or reported trying to have one, also reported greater hopelessness, including agreement with the statement "I do not expect to live a very long life" (Bolland 2003). There is also some evidence regarding locus of control and risky sexual behaviour. Having an internal locus of control has been related to increased contraceptive use and a decreased likelihood of becoming an unmarried parent (Wallston & Wallston 1978).

The role of time preference in environmental behaviour has not been widely examined. However, the available evidence suggests that future oriented people are more likely to conserve water and to use public transport (Corral-Verdugo & Pinheiro 2006; Joireman et al. 2004). People who score highly for generativity, the belief that one's actions have future consequences, also report more eco-friendly consumer behaviour and environmentally friendly intentions (Urien & Kilbourne 2011). People who have greater perceived personal control also tend to be more environmentally friendly. Those with a more internal locus of control report stronger pro-environmental intentions and behaviour, and less environmentally harmful behaviour (Fielding & Head 2012). They have also been found to be more willing to purchase products with environmentally friendly packaging (Schwepker & Cornwell 1991).

There is more literature on the links between time preference and health behaviour. Adams (2009c) has reviewed evidence in support of the idea that a

greater future time preference decreases the likelihood of being a smoker and increases success in quitting smoking. Several studies have found that measures of time preference including future discounting and consideration of future consequences are associated with eating behaviours, body mass index and being overweight or obese (Price et al. 2013; Weller et al. 2008; Borghans & Golsteyn 2006; Adams & White 2009; Adams & Nettle 2009). One study found that measures of delay discounting and time perspective predicted reported tobacco, alcohol and drug use, exercise frequency, eating breakfast and use of seatbelts (Daugherty & Brase 2010). Another study found that delay discounting was a weak predictor of body mass index, smoking, and exercise behaviours. However, it was a stronger predictor when health behaviours were aggregated; suggesting that delay discounting may predict a cluster of health behaviours, rather than any single health behaviour (Chabris et al. 2008).

As a result of the associations between locus of control and health behaviour (Wallston & Wallston 1978), the concept of the locus of control has been extended to create health locus of control (Wallston & Wallston 1981). This has generated a burgeoning literature on the subject. People with a greater belief in the influence of chance on health do less sporting activity, attend fewer dental check-ups, and less frequently participate in health courses, or otherwise seek out health information. Meanwhile, those who have an internal health locus of control consume less alcohol, smoke less and are more likely to adhere to medical regimens (Grotz et al. 2011; O'Hea et al. 2005; Leong et al. 2004). There is also a large volume of literature on the association between perceived control and health outcomes more generally. After examining decades' worth of evidence from the Whitehall Studies, Marmot (2004) concluded that, "Autonomy – how much control you have over your life – and the opportunities you have for full social engagement and participation are crucial for health, well-being and longevity".

#### 6.7 The implications of our perspective

We have introduced the BCD, a cluster of behaviours associated with SES. We have established that BCD behaviours are characterised by disinvestment in the

future - a logical response to having a limited ability to ensure returns on investments in future outcomes. We have emphasised that small initial disparities can lead to larger eventual inequalities through additive routes and feedback loops, including intergenerational transmission. And, we have discussed the mechanisms by which restricted control over future limiting factors might generate the BCD behaviours, making the distinction between proximate and ultimate categories of explanation.

How should all of this change our approach to the question of socioeconomic differences in behaviour? A key implication of our perspective is that concepts such as locus of control and time preference should be viewed, not as fixed traits, but as adaptive responses that reflect a person's environment and future prospects. Thus, rather than attempting to train people to be more future oriented (as in Marko & Savickas, 1998), it may be better to focus on those factors that cause them to be present oriented in the first place. For example, tackling sources of extrinsic mortality may, not only reduce extrinsic mortality risk, but it may also improve BCD behaviours such as health behaviour and investment in education. This conclusion echoes that of Geronimus (1996), who wrote on the matter of teen pregnancy, "...as a matter of social policy, focusing on teen pregnancy prevention as the solution to persistent poverty may be the modern-day equivalent to suggesting that those without bread can eat cake. Instead or in addition, policy approaches that would offer poor women and men real reasons to expect to live predictable, long lives deserve a prominent position on the policy agenda."

That said we have reason to believe that interventions that adjust perceptions might also be a fruitful avenue of investigation. As discussed previously, we have found that priming people to believe that prevailing mortality risks are controllable made them more likely to choose a healthy snack reward (Pepper & Nettle, 2014a, Chapter 5). An implication of this is that, although we might expect the effects of deprivation to be somewhat entrenched (Figure 11), behaviour appears to remain plastic – at least to some extent. However, we do not know the extent to which improvements in a person's situation can compensate for past experience and damage. More research is needed to determine the extent to

which behaviours remain malleable over the life course. This could inform the development of interventions based around adjusting perceptions, but could also answer important questions about the reversibility of the effects of early life adversity.

The reversibility of the effects of early life circumstances on health is an important area for future research. We have showed that the effects of one's initial disadvantages can remain visible (relative to others who have not suffered those disadvantages), even after circumstances improve (Figure 11). However, we do not know to what extent the effects of initial disadvantage can be erased by bestowing later advantages. It is possible that there is a point of no return, after which the effects of early life circumstances cannot be reversed. Alternatively, it may be possible to "catch up" in later life by overcompensating with behavioural and physiological investments in health.

Another important question is that of the accuracy of perceptions. Little is known about the extent to which people's perceptions reflect their objective situations. It is possible that, in the case of extrinsic mortality risk, perceptions may become skewed as a result of media scare stories or exaggerated tales from peers (Sunstein 2003). If this is the case, simply working to correct misperceptions about risk may be enough to change behaviours in those whose perceptions are skewed. Conversely, people's perceptions may fairly accurately reflect their life chances (Mirowsky & Ross 2000; Lima-Costa et al. 2012). In this case, it might be considered unethical to adjust perceptions and it would be better to focus on tackling sources of extrinsic mortality risk and improving people's future prospects.

Another implication of our perspective is that we might expect control over mortality risk (and other future limiting outcomes) to be stronger predictors of BCD behaviours than SES itself. For this hypothesis to be tested, good measures of control over mortality risk would be needed. Though it is relatively easy to measure *perceived* control over mortality risk (Pepper & Nettle 2014b), it is more difficult to find objective measures. However, one could argue that the portion of a person's mortality risk not due to health behaviour is extrinsic. This can be

calculated (e.g. Lantz, House, Lepkowski, *et al.*, 1998) and tested for associations with BCD behaviours.

Finally, we have shown that small initial disparities can lead to larger eventual inequalities (section 6.4.6). This helps to shed some light on the paradox of the persistence of health inequalities in modern welfare states (Mackenbach 2012). Even in the absence of abject poverty, smaller relative disadvantages may generate noticeable relative differences in outcomes such as healthy life expectancy. An important question for future research will be to pinpoint the specific disadvantages that generate these differences, so that they can be addressed.

#### 6.8 Conclusion

We have introduced a behavioural phenomenon associated with socioeconomic status, which we call the behavioural constellation of deprivation (BCD). We have established that these behaviours are characterised by disinvestment in the future, which we view as a logical response to having a limited ability to ensure returns on investments in future outcomes. We have also discussed the evolutionary theoretical models that inspired our perspective. We have outlined how key principles from these models can help us to understand the dynamics of the BCD. These principles are that: 1) Small initial disparities can lead to larger eventual inequalities, 2) Feedback loops can operate to embed early life circumstances, 3) Constraints can breed further constraints, and 4) Feedback loops can operate over generations. We have discussed the mechanisms by which restricted control over future limiting factors might generate the BCD behaviours, making the distinction between proximate and ultimate types of explanation. We have reviewed literature from other fields, which has converged on similar conclusions regarding the roles of perceived control and the future in explaining behaviours from the BCD. Finally, we have highlighted some of the key implications of our perspective for policy and future research.

## Chapter 7. Summary, implications and future research

## 7.1 A summary of my thesis

The chapters in this thesis have explored the ways in which mortality risk may lead to socioeconomic differences in behaviour. Chapters 2 and 3 examined the cues that people might use to estimate their personal mortality risk. Chapter 2 showed that bereavements, but not overall death exposure, predicted steeper future discounting and a lower ideal or actual age at first birth. This suggests that the deaths of people with whom one is close may be a more salient cue for the calibration of time horizons than the deaths of more distant acquaintances.

Chapter 3 tested the hypothesis that people use the ages of others in the local environment as a cue to mortality risk. In two experiments, participants viewed either an "older" or a "younger" set of faces. We then measured participants' ideal ages at first birth, their levels of future discounting and subjective life expectancies. Although study 1 revealed a possible effect of the manipulation on future discounting, the effect was not replicated in study 2. Further investigation revealed that this may have been because cues to age and health were confounded in the images used as stimuli. However, it was not possible to draw any firm conclusions based on our results.

Chapters 4 and 5 demonstrated the importance of making the distinction between extrinsic and intrinsic mortality risk. They reported studies that tested the hypothesis that the controllability of personal mortality risk should be an important determinant of behaviour. Chapter 4 showed that lower subjective socioeconomic status was associated with higher perceived extrinsic mortality risk, which in turn predicted lower reported health effort. The effect of subjective socioeconomic status on reported health effort was completely mediated by perceived extrinsic mortality risk. This indicates that extrinsic mortality risk, which is higher for those of lower socioeconomic status, may be a more important determinant of health behaviour than socioeconomic status per se.

The three experiments in Chapter 5 showed that manipulating the perceived controllability of prevailing mortality risks could alter a health-related decision: the choice between a healthy food reward (fruit) and an unhealthy alternative (chocolate). These results reinforce the correlational finding of Chapter 4 and also

show that the perceptions driving health behaviour may remain malleable later in life, updating rapidly in response to new information.

Chapter 6 tied together many of the ideas implicit in chapters 2 - 5. It made the case that there is a clustering of behaviours associated with socioeconomic status - a behavioural constellation of deprivation. It explains how the behavioural constellation of deprivation may be the result of socioeconomic inequalities in control, including control over mortality risk. It argues that the relatively limited control associated with lower socioeconomic status curtails the extent to which people can expect to receive future rewards, leading to more present oriented behaviour in a range of domains. Chapter 6 also used principles from evolutionary theoretical models to illustrate the likely dynamics of the effects of poverty. It explained how small initial disparities can lead to larger eventual inequalities, how feedback loops may embed early life circumstances, and how this effect may be perpetuated over generations.

## 7.2 The implications of my findings

There are various implications to my findings. One is that "impulsive" behaviours should be viewed, not as fixed traits, but as adaptive responses that reflect a person's environment and future prospects. If we start from the assumption that present oriented decisions are the result of pathology or poor judgement, we risk conflating cause and consequence. For example, it is often assumed that teenage pregnancy is the result of mistakes, driven by ignorance, and a cause of ill health and poverty (Geronimus 1996; Johns et al. 2011; Dickins et al. 2012). Yet, the perspective summarised in Chapter 6, and the findings in Chapter 2, suggest that factors associated with poverty, such as extrinsic mortality risk, cause a shortening of time horizons, including reproductive ones.

The results of chapters 4 and 5 suggest that interventions to adjust perceptions regarding extrinsic mortality risk might prove useful in improving health behaviour. We found that priming people to believe that prevailing mortality risks were controllable made them more likely to choose a healthy snack reward (Pepper & Nettle, 2014a, Chapter 5). More research is needed to determine whether longer term effects can be achieved and whether other health related behaviours can be influenced.

An important avenue for future investigation is the perception of control over mortality risk. What are the determinants of perceived control over mortality risk? Are perceptions accurate? It is possible that people's perceptions of their own mortality risk are accurate. Or, they may become skewed as a result of media scare stories (Sunstein 2003). It is also possible that exposure to violent movies and video games may alter perceptions (consciously, or unconsciously). Indeed, this may partly explain the association between violent video game play and impulsive and risky behaviours (Griffiths 1999; Hull et al. 2014).

An intriguing possibility, outlined in Chapter 6, is that high extrinsic mortality risk may reduce the optimum investment in physiological repair, leading to accelerated ageing. This hypothesis cannot be experimentally tested in humans, but, animal models may provide the opportunity. Experimental tests of this hypothesis could also make the distinction between internal and external predictive adaptive responses (see section 7.3.2).

Finally, one of the most important policy implications of the perspective presented in this thesis is that tackling sources of extrinsic mortality could pay off in more ways than one. It would reduce extrinsic mortality rates – a guaranteed win - and, as a side effect, it may improve health behaviours, educational outcomes, and even environmental behaviour.

## 7.3 Related questions

There are some interesting questions related to the theme of this thesis, which I have not discussed in the more focussed discussions contained within each chapter. Here, I shall briefly summarise these questions and how they tie in with my work so far.

## 7.3.1 The possibility that trade-offs vary with socioeconomic status

Chapter 4 and 5 of this thesis tested predictions arising from a theoretical behavioural ecological model (Nettle 2010b) about the effects of extrinsic mortality on health behaviour. However, a key assumption of this model was that resources invested in health could not be invested in other activities that would enhance Darwinian fitness (there is a trade-off). For example, time invested in healthy behaviour cannot also be spent accruing status or finding a mate. The

original model examined the effects of varying the strength of this trade-off. It showed that, as the strength of the trade-off increases, the optimum investment in health decreases. However, the levels of trade-off presented by Nettle (2010b) were constrained at low levels. I have reconstructed the model and explored the effects of the trade-off further. I found that, even at a low level of extrinsic mortality (2%), stronger trade-offs result in the optimum health behaviour being dramatically low (Figure 12). Thus, low extrinsic mortality risk may only incentivise healthier behaviour in individuals who do not experience strong conflicts between investing in health and investing in other things that would enhance their fitness. An implication of this is that the effects of extrinsic mortality risk may be most visible in populations experiencing minimal poverty. Extreme poverty might increase the trade-off to the point that decreasing extrinsic mortality rates would have little impact. Thus, this theoretical model may have more power to explain the persistence of health inequalities in welfare states (the paradox introduced in section 1.1) than to explain unhealthy behaviour in the context of extreme poverty.

The idea that the trade-off between healthy behaviour and other activities might vary with SES has been examined in the non-evolutionary literature. Some economists have argued that higher SES individuals face greater opportunity costs when investing time in health (e.g. exercising and cooking healthy meals) because their time is more highly valued in the workforce (Biddle & Hamermesh 1989). Indeed, a study examining trends in leisure time in the U.S. over recent decades shows that the least educated have gained the most leisure time (Aguiar & Hurst 2006). Thus, while those of lower SES may face stronger financial trade-offs, they may face lesser time constraints.


Figure 12. The decrease in optimum health behaviour for trade-offs varying from 0-1 with a fixed extrinsic mortality risk of 2% (Based on the model by Nettle, 2010b).

Future research should take into account the fact that some health behaviours will carry greater opportunity costs than others and that, under some circumstances, these opportunity costs will be more important than extrinsic mortality risk for determining health behaviour. It would also be of interest to investigate the extent to which SES determines the trade-off between healthy behaviour and other activities.

# 7.3.2 The question of internal versus external predictive adaptive responses

In my outline of how the effects of extrinsic mortality risk might become embedded and amplified over time (Chapter 6, section 6.4.6, Figure 11), I assumed that our hypothetical individuals make health investment decisions based on external cues to extrinsic mortality risk. This is known as an *external* predictive adaptive response. An organism making this type of response assumes that the early-life environment provides a good indicator of its later environment and adjusts its life history strategy accordingly (this is what occurs at the initial time point in Figure 11). For such a response to be adaptive, there must be a high level of environmental stability over time (Nettle et al. 2013).

An alternative account is the *internal* predictive adaptive response. This assumes that external forces cause damage to be accrued in early life, reducing longevity

so that it becomes adaptive to adopt a faster life history strategy. For the internal predictive adaptive response to evolve, damage accrued in early life must meaningfully impact upon adult mortality risk (Nettle et al. 2013). These two processes are not mutually exclusive and the model depicted in Figure 11 would have a similar outcome under an internal predictive adaptive response. Thus, the assumption of an external predictive adaptive response is not problematic.

However, the model depicted in Figure 11 is not a dynamic one. Dynamic programming models assume that only future decisions count for maximising fitness. They work backwards from the end of life, calculating each optimal decision based on the previous one (Cichoń 1997; McNamara & Houston 1996). This enables the models to account for the impact of current optimal decisions on future optimal decisions (something that should occur in a predictive adaptive response). By contrast, my model has only taken into account the impact of past decisions on future ones. It would be interesting to develop dynamic models to examine the impacts of changes in early environment on later phenotypes and this is something that I hope to acquire the requisite skills to do in future.

# 7.3.3 Sensitive periods, variability and the reversibility of the effects of early-life adversity

Related to the questions above (section 7.3.2), are questions about sensitive developmental periods. As discussed in Chapter 6 (section 6.4.6), a large volume of evidence shows that early life experiences are important for adult health and behaviour. Early life experiences may shape adult phenotypes by providing information about the world, or by altering the state of the organism. However, some questions remain open. Do sensitive periods occur under all conditions? What conditions determine the length of the sensitive period and the extent to which plasticity is retained?

Theoretical models suggest that phenotypes should be more flexible in individuals who have formed less confident estimates of their environments (Frankenhuis & Panchanathan 2011). This is more likely to occur in variable environments than in stable ones. Information availability might also explain why younger and older people tend to discount the future more steeply than middle aged people: Under uncertain hazard rates, younger people should have a higher

Bayesian estimate of extrinsic risk, whilst older people have aged more and thus have less time to live (Sozou & Seymour 2003).

There are practical reasons to consider the information gathered during early life and the impact it might have on adult behaviour. A substantial research effort goes into designing interventions to improve health behaviour. However, these interventions may have limited effects for people whose world views have become entrenched. If someone has consistently experienced cues suggesting that they live in a dangerous world, they are unlikely to alter their perceptions or behaviour on the basis of a brief intervention. It may take numerous cues suggesting less dangerous world to change their behaviour. Or, if the sensitive period has passed, behaviour change may never occur. Nonetheless, a greater understanding of sensitive periods and the effects of environmental variability might enable us to tailor interventions to past experience, making them more effective.

#### 7.3.4 Extrinsic mortality risk as a special case of lack of control

Although chapters 4, 5 and 6 of this thesis have focused primarily on the effects of extrinsic mortality risk, I have highlighted the fact that other extrinsic factors may be important (section 6.4.4). The idea that control over life in general may be important for health is supported by empirical evidence (Marmot et al. 1997; Bobak et al. 2000; Chandola et al. 2004). However, I believe that extrinsic mortality risk may be a special case. While other extrinsic factors may limit specific future outcomes, extrinsic mortality risk limits them all: A lack of control over the labour market may limit future job prospects. However, a lack of control over mortality risk limits the chance of being alive to have a job, to have children, to see the benefits of healthy living, or to see any other future outcome at all. As such, extrinsic mortality risk is the most all-encompassing case of lack of control. The importance of this distinction could be tested experimentally by comparing the effects of priming extrinsic mortality to the effects of priming lack of control over other, non-fatal, factors.

The only future payoffs that are not entirely limited by personal extrinsic mortality risk are those from investments in children. Investments made in children may enhance inclusive fitness, even if no direct rewards are received by the parent in

their lifetime. Thus, it is possible that parents might be less impulsive in the face of extrinsic mortality risk. However, this is unlikely. A parent who estimates their personal extrinsic mortality risk to be high is likely to apply a similarly high risk estimate to their children. Thus, they may respond by having more children and investing less in each of them (Lawson & Mace 2011).

#### 7.4 Reflections and future directions

As outlined in the introduction (1.2), my interest in my thesis topic began with an attempt to understand the cluster of health and social outcomes (*trust, mental illness, life expectancy, infant mortality, obesity, educational performance, teenage births, homicides, imprisonment and social mobility*) that are associated with economic inequality at the population level (Wilkinson & Pickett, 2009). I perceived that many of these health and social outcomes might be a result of the inequalities in mortality risk that accompany economic inequalities (as reviewed in Chapter 6). I have not tested the hypothesis that there are direct psychological effects of economic inequality. However, I now more clearly understand how inequalities in mortality risk might affect several of the behaviours that Wilkinson and Pickett (2009) reviewed. Within the framework I have used throughout this thesis, *life expectancy, infant mortality* and *homicide* can be seen as variables that might cause other outcomes such as *teen pregnancy, obesity* and poor *educational performance*, by shortening time horizons. Using a life history framework has helped to separate likely causes from likely correlates.

Nonetheless, it is also important to test the direct psychological effects of inequality, which may even contribute to inequalities in mortality risk. For example, inequality may have effects on male-male status competition, of which homicide can be an extreme result (Daly et al. 2001; Wilson & Daly 1985; Daly & Wilson 1988). Some studies have begun to test the effects of relative status on risk taking and competition. One found that men (but not women) who thought their decisions were being evaluated by potential status rivals were more likely to make risky decisions (Ermer et al. 2008); Other experiments have found that both individuals who experience relative disadvantage caused by uncontrollable elements of their environment (structural inequality) and individuals who perceive themselves to be at a competitive disadvantage were more risk-prone (Mishra et al. 2014a; Mishra et al. 2014b).

As an immediate extension of the work in this thesis, I hope to go on to examine the aetiology of perceptions about the controllability of mortality risk (questions discussed in section 7.2). I aim to investigate what sources of information and life experience lead people to their estimates of personal extrinsic mortality risk. I also hope to replicate the findings of Chapter 4 with a larger data set in which SES is well characterised and a range of health behaviours are recorded. I plan to extend the experiments summarized in Chapter 5, testing the effect of the priming method on new health behaviours, such as smoking and physical activity. This research will be valuable because it will test the feasibility of enhancing sense of control over mortality risk as a health behaviour intervention.

Alongside this work, I hope to develop more specific theory to make predictions regarding how the trade-off between investing in health and other things might vary with SES and type of health behaviour (the questions outlined in 7.3.1). I also hope to develop the requisite skills to use dynamic programming models to examine the expected effects of the timing of changes in environment (as discussed 7.3.2).

Finally, I am interested in quantifying the extent to which increases in wealth may "purchase" increases in control over risk. I have already begun discussing possible approaches to this question with others including statisticians, economists and risk specialists.

### **Chapter 8. References**

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### **Chapter 9. Appendices**

#### 9.1 Pepper & Nettle (2014c)

## Chapter 10 Socioeconomic Disparities in Health Behaviour: An Evolutionary Perspective

**Gillian V. Pepper and Daniel Nettle** 

Abstract Socioeconomic disparities in health behaviour are a reliable finding across many societies. Individuals of lower socioeconomic status (SES) more frequently undertake behaviours detrimental to health (e.g. smoking) than those of higher SES. Despite a large volume of research on the subject, there is still no consensus on the causes of these disparities. In this chapter, we discuss nine categories of explanation which have been put forward in the social science literature. We then outline a complementary behavioural-ecological approach based on the idea that as extrinsic mortality increases, the payoff to investment in preventative health behaviour declines. We discuss how this evolutionary approach alters the interpretation of existing explanations, allowing us to reorganise the nine categories of explanation into three; ultimate, proximate and constraint based. We then discuss how this perspective can help to guide future research in public health.

#### **10.1 Background and Aims**

Socioeconomic status (SES) refers to ranking in a social and economic hierarchy and is usually measured by education, occupation, income or wealth (Pampel et al. 2010). SES disparities in health outcomes are a reliable finding. There are SES inequalities in life expectancy, in physical health and in mental health (Feinstein 1993; Adler and Ostrove 1999). Indeed, SES is so consistently linked with health outcomes that it has been classified as a fundamental cause of SES disparities (Link et al. 1995). There has been a large volume of research on the subject of SES differences in health. The website of the MacArthur Research Network on Socioeconomic Status and Health (http://www.macses.ucsf.edu/) lists nearly 700 publications between 1998 and 2009. These only represent a portion of the relevant literature. In this chapter, we focus on the literature regarding socioeconomic gradients within and between developed societies. The majority of the literature focuses on developed societies, because of the paradox of the persistence of health inequalities in modern welfare states (Mackenbach 2012). Despite this large volume of research, there is still no consensus on the causes of the gradient. However, what is clear is that a large part of the gradient is attributable to the health behaviours associated with SES (Mokdad et al. 2004; Stringhini et al. 2010). Why the people in society who face the most challenging life circumstances respond to them with behaviours which seem to exaggerate their problems is also an unresolved issue. In this chapter, we will briefly review some common explanations of SES differences in health behaviour. These explanations are often treated as competing hypotheses that must be tested against one another. None of them currently make use of an evolutionary adaptive framework. We will then review Nettle's (2010a) behavioural-ecological model of adaptive allocation of energy to preventative health behaviour. We will show how this evolutionary approach aids interpretation of the evidence and helps to reconcile the existing, seemingly competing, explanations. Our discussion emphasizes that an evolutionary framework can guide a more cohesive approach to future investigations of, and intervention policies aimed at, socioeconomic gradients in health behaviour.

#### **10.2** SES Gradients in Health and Health Behaviour

Socioeconomic gradients in health outcomes within developed countries are well documented (e.g. Feinstein 1993; Adler and Ostrove 1999; Melchior et al. 2011). There are SES differences in life expectancy (Wilkinson 1992a, b; Phelan et al. 2010) and in healthy life expectancy (defined by the World Health Organization as "The average number of years that a person can expect to live in 'full health' by taking into account years lived in less than full health due to disease and/or injury"; Evans 2004; Liao et al. 1999; Crimmins and Saito 2001). Lower SES is associated with greater risk of a number of diseases. These include, but are not limited to: diabetes (Brennan et al. 2009), gastrointestinal diseases (Adler and Ostrove 1999; Levenstein and Kaplan 1998), tuberculosis (Cantwell et al. 1998), cardiovascular diseases (Laaksonen et al. 2008; Mobley et al. 2006) and arthritis (Sapolsky 2004; Kristenson et al. 2004). There are many mechanisms by which SES could influence health. However, this chapter will focus on socioeconomic disparities in health behaviour. We use the term health behaviour to encompass those activities which are beneficial for health. These could be either acts of omission (e.g. not smoking) or commission (e.g. getting health checks). Evidence suggests that SES differences in health behaviour account for a large portion of the gradient—up to half of it (e.g. Mokdad et al. 2004; Stringhini et al. 2010). People of lower SES more frequently exhibit risky health behaviours such as smoking and excessive drinking than those of higher SES (e.g. Pridemore et al. 2010; Harrell et al. 1998). Individuals of lower SES are also more likely to be obese, and less likely to take part in regular physical activity (e.g. McLaren 2007; Wardle et al. 2002). They are less likely to adhere to medication programmes and follow health-screening advice, even when these things are free (Barr et al. 2002; Goldman and Smith 2002; Qi et al. 2006). There is a higher incidence of teenage pregnancy (which is often conceptualised as a health issue) among lower-SES individuals and a higher rate of adverse birth outcomes (Johns 2010; Jewell et al. 2000; Adler and Ostrove 1999). There are even SES differences in the performance of basic self-protection behaviours, such as the use of seat belts (Colgan et al. 2004; Leigh 1990).

#### **10.3** Classifying Potential Causes

A wide variety of explanations have been put forward for SES differences in health behaviours. They come from a diverse range of fields including epidemiology, sociology, behavioural economics and health psychology. It is challenging to review these, as they are so diverse and numerous, but Pampel et al. (2010) helpfully grouped them into nine main types. Here, we provide an abridged summary of their categories, which we have edited slightly for clarity. For full details and references, see Pampel et al. (2010). From here onwards, we shall refer to these categories of explanation as explanation types (ET) 1–9:

*ET1. Deprivation and stress*: People of lower SES experience more stressful negative life events and use smoking, alcohol, drugs and junk food as buffers or self-medication against these.

*ET2. Fewer benefits of health behaviours*: The benefits of health behaviours are lower for people of low SES because they are less likely to live to see the result. This is known as the Blaxter hypothesis (Blaxter 1997). Pampel et al. (2010) also classify the idea that people of lower SES have a preference for more immediate over more deferred rewards under this heading, but we henceforth classify this as belonging to the following category.

*ET3. Latent traits*: Some third variable such as attraction to risk or to short-term gain explains individual differences in both SES attainment and health behaviours.

ET4. Class distinctions: High-SES individuals adopt healthy behaviours in order to set themselves apart from lower-SES individuals.

ET5. Lack of knowledge: People of lower SES lack knowledge that behaviours are bad for health.

*ET6. Efficacy and agency*: Increasing education is associated with a greater sense of control and ability to exert choices, which allows people of higher SES to adopt more healthy behaviours.

*ET7. Aids to health behaviour:* The resources needed to pursue a healthy lifestyle cost money that is less likely to be available to people of lower SES.

ET8. Community opportunities: Lower-SES neighbourhoods lack health-supporting options such as shops selling healthy produce.

ET9. Social support and influence: Lower-SES social networks are less likely to provide role models for healthy behaviours or sanctions against unhealthy ones.

Pampel et al. (2010) state that little has been done to systematically compare and contrast the categories of explanation which they reviewed. They say that, "this makes it difficult to offer an overarching framework that integrates or adjudicates between the various approaches". This situation is not uncommon in the social sciences where, in the memorable words of Davis (1994), "while each article/book/ course may be well

crafted, they have little or nothing to do with each other." We will argue that the use of an adaptive evolutionary framework helps clarify which of these diverse types of explanation are genuinely competing and which are different levels or components of the same overall story. To do so, we will now introduce Nettle's (2010a) behavioural-ecological model of optimal health behaviour. We will then show how this can be applied to SES gradients in health behaviour to provide a useful big picture, clarifying the relationships among ET1–9, showing where the fundamental issues lie and helping to make novel predictions about how health behaviours can be changed.

#### 10.4 Investment in Preventative Health Behaviour: A Behavioural-Ecological Model

Behavioural ecology is the study of behaviour from an adaptive evolutionary perspective; if many individuals living under some particular set of circumstances recurrently exhibit some suite of behaviours, then maybe those behaviours have an adaptive payoff under those circumstances (Davies et al. 2012; Nettle et al. 2013). Note that this does not mean assuming that the behaviours in question are under genetic control. Rather, natural selection on genes has endowed individuals with capacities for learning and plasticity that mean that they can find adaptive solutions to living in their local environments through non-genetic processes.

In the current case, it may seem counterintuitive to speak of adaptive payoffs. Survival is a central component of Darwinian fitness, and therefore, surely, investment in preventative health behaviours must always be adaptive. However, this ignores what is known in behavioural ecology as the principle of allocation. Individuals have finite energetic resources, and if they devote a unit of energy to one activity, they cannot be devoting that unit to something else. This leads us to the central behavioural-ecological idea of a trade-off. Investing a bit more in preventative health behaviour might always yield some improvement in survival chances, but there will come a point where the marginal benefit will not outweigh the cost, given that there are other things that could be done with the time and energy. Crucially, the terms of that trade-off might be different for people living under conditions of low versus high SES. They may have less time or money available to invest in health; or they may simply place a greater value on *other* things which they could invest time or money in. In addition to this, there may be short-term social benefits to unhealthy behaviours such as drinking, which outweigh the long-term repercussions for those living in lower-SES conditions, but not for those of higher SES. This will alter the terms of the trade off—especially if those of lower SES have less incentive to focus on the long term. The model, which we will go on to outline, demonstrates that this is likely to be the case.

Another central feature of behavioural ecology is the distinction between ultimate and proximate causes of behaviour (Mayr 1961; Tinbergen 1963). Ultimate explanations are about *why* a behaviour should occur in a given population and environment, in terms of the payoffs to that behaviour in that environment. Proximate explanations are about *how* that behaviour is generated, for example, the psychological or neural mechanisms involved. Importantly, these two different types of explanation are seen in behavioural ecology as complementary rather than competing. The distinction between ultimate and proximate explanations is not widely made in the social sciences, but it can be very useful. It will help us to make better sense of how ET1–9 relate to one another. We return to this below, but here we note that the model we outline is at the ultimate level. Ultimate explanations generally underdetermine the proximate mechanisms by which the adaptive behaviour is generated. This is true here; the model is compatible with several different hypotheses about the details of the psychology of investment in health behaviour.

Nettle's (2010a) model of optimal investment in health behaviour relies on three central axioms. (1) The first is that individuals experience some component of mortality which is *extrinsic*, meaning that it is not affected by decisions about health behaviour. The extrinsic mortality risk is the risk of mortality still faced by a person who has made all available investments in health behaviours. (2) The second is that investment in health behaviour is costly, in the sense that every unit of energy devoted to it is taken away from some other adaptively relevant activity or allocation. For example, time and energy devoted to health behaviour cannot be spent on activities such as gaining a mate, status or resources. (3) The third is that the effectiveness of health behaviour in reducing mortality risk is subject to diminishing returns. That is, the first unit of effort expended on preventative health behaviour has a slightly larger impact than the second unit, and so on. The model couples these axioms with the general principle of *optimization* (Parker and Maynard Smith

1990); that is, given these axioms, what would be the best thing for the individual to do if they were able to implement any behaviour?

The predictions in this case are very simple. As the risk of extrinsic mortality (the part people *cannot* do anything about) increases, the amount it is worth them investing in preventing the health risks they *can* do something about also decreases (Fig. 10.1a). This result is fairly intuitive. It seems quite pointless to make great effort to abstain from smoking if something you have no influence over is likely to kill you in the next few years anyway. Thus, people facing higher extrinsic mortality risks should reduce their allocated effort towards preventative health behaviour and reallocate their energy to other things. This in turn will increase their mortality risk, amplifying the initial difference in extrinsic mortality into a larger difference in total mortality (Fig. 10.1b). The model shows that even very small differences in extrinsic mortality can have quite large effects on optimal allocation to preventative health behaviour. This leads to a quite large final discrepancy in life expectancy. Note that although Nettle's model focuses on extrinsic mortality risk, the principle could also be extended to extrinsic morbidity risk. The payoff of health behaviour either in terms of healthy life expectancy or Darwinian fitness will be limited for individuals whose likelihood of suffering illness is beyond their control.



Fig. 10.1 Predictions from Nettle's (2010a) model. **a** As the risk of extrinsic mortality increases, the optimal investment in health behaviour (measured here on an arbitrary scale) decreases. **b** As the risk of extrinsic mortality goes up, the predicted total mortality rate goes up faster, through a combination of the primary effect of the extrinsic mortality and the secondary effect of people's response to it. (Reprinted from Nettle 2010a)

How can we apply this model to the SES gradient in health behaviour? Although we have pointed out that there are SES gradients in health behaviours, there is also evidence that people of lower SES are exposed to more risks of dying from things which are beyond their behavioural control. For example, there are strong SES gradients in mortality due to homicide (e.g. Cubbin et al. 2000; Shaw et al. 2005), assault and other violent crimes (e.g. Leyland and Dundas 2010; Markowitz 2003). There are SES gradients in death due to traffic accidents and other unintentional injuries (e.g. Belon et al. 2012; Singh et al. 2012). There is also evidence that low SES individuals are exposed to a greater number of environmental risk factors such as hazardous waste, toxins, air pollutants, ambient noise and crowded or unsafe residential and working environments (Evans and Kantrowitz 2002). Furthermore, evidence also suggests that people of lower SES do perceive that they are subject to a larger number of risks beyond their control (Wardle and Steptoe 2003). Although some of these risks might be reduced by avoidance behaviour (for example avoiding situations in which assault is likely), these sources of mortality are extrinsic with respect to the health behaviours which are typically examined in the literature (e.g. smoking, drinking or overeating). Furthermore, it may be that the best way to avoid these sources of mortality is simply to move away from deprived areas, a solution which is by definition unavailable to the poor. Lantz et al. (1998) demonstrated that, in a nationally representative US sample, mortality risk was greater for low-income groups than for middle-income groups, even after mortality due to all measured health behaviours was accounted for statistically. Thus, it seems reasonable to infer that people of low SES are indeed exposed to greater extrinsic mortality risk than their affluent peers (see also Lawlor et al. 2003). If we assume that the second and third axioms of the model hold, which is not unreasonable, then it actually makes adaptive sense for people of lower SES to be less concerned with preventing future health risks. The model predicts the most dramatic change in health behaviour with a small increment in extrinsic mortality where extrinsic mortality is low in absolute terms. This might help to explain why variation in health behaviour is more marked within developed nations that

have low overall levels of extrinsic mortality than in those that have higher extrinsic mortality levels (e.g. Singh and Siahpush 2006; Cristia 2009).

The idea that lower SES is associated with greater exposure to extrinsic mortality might explain other traits besides health behaviours which are associated with SES. For example, individuals facing higher extrinsic mortality could be expected to place a greater value on the present than on the future. This is because high mortality risk increases the likelihood that one will not survive to reap future rewards, or to experience future penalties. There is evidence to support SES differences in time perspective and also to suggest that these may mediate SES differences in smoking cessation and in body mass index (Adams 2009a; Adams and White 2009).

People facing higher extrinsic mortality might also be expected to have children at a relatively young age (Low et al. 2008; Nettle 2010b). This is because when there is high mortality, waiting to have children increases the chance that one may not survive to have children at all. In addition, if one does survive to have children, having them sooner will improve the odds of surviving long enough to provide adequate parental care. We would also expect to see interactions between mortality risk and resource availability, because for those of high SES, waiting offers an opportunity to gather resources which can buffer against a risky environment and can be invested in children. However, this may not be possible for those of lower SES, no matter how long they wait. Again, the evidence supports this. There is a sharp SES gradient in age at first childbearing (e.g. Nettle 2010b, 2011).

Thus, we would predict that people should have an evolved sensitivity to cues of what level of extrinsic mortality they currently face, and that these cues should shift their behavioural allocations between current and future benefits. This prediction has led to the development of relevant psychological experiments. Griskevicius et al. (2011a) found that in a county-level analysis of data from the USA, income and violent crime (a factor contributing to mortality) were significant unique predictors of age at first reproduction. By comparison, levels of property crime (which do not contribute to mortality) did not predict age at first reproduction. Based on this finding, Griskevicius et al. went on to perform an experiment. Their participants either read a fake news article about a rise in random violent crimes or a control article about a stressful afternoon spent searching for keys. They found that individuals who reported a less wealthy upbringing expressed a desire to have children sooner (and had a more positive attitude towards reproduction in general) when they had read the article about rises in violent crime. For participants who reported a wealthier upbringing, the same article produced a desire to further career and education at the cost of starting a family. Using a similar experimental method, Griskevicius et al. (2011b) found that individuals with low childhood SES who were exposed to the violent crime article subsequently chose smaller, sooner rewards over later, greater ones (they displayed greater future discounting) and were more likely to choose riskier options with larger rewards over smaller guaranteed rewards (they became more risk prone). Conversely, individuals of high childhood SES who read the violent crime article discounted future rewards less and became less risk prone.

These findings are a good example of how an evolutionary framework can guide investigations into the mechanisms underlying SES differences in behaviour.

# **10.5** How does the Adaptive Perspective alter our Understanding of Existing Explanations?

So far, we have summarized the nine categories of explanation for SES disparities in health behaviour put forward by Pampel et al. (2010), and outlined an adaptive explanation for SES disparities in health behaviour in terms of additional exposure to extrinsic mortality (Nettle 2010a). We have also made the distinction between proximate and ultimate levels of explanation. We will now go on to discuss how the adaptive approach and the proximate–ultimate distinction can change our perspective on ET1–9. There is in general no reason to think of evolutionary and non-evolutionary explanations as fundamentally at odds with one another. Indeed, formal evolutionary models may capture generalizations already made within social science (see Nettle et al. 2013 for discussion). The evolutionary perspective can provide a "big picture" overview on how the different parts of current knowledge interrelate.

In this light, we can divide ET1–9 into just three more inclusive groups (see Table 10.1). First, the contention that people of lower SES receive *fewer benefits of health behaviours for longevity* is an ultimate explanation, and is remarkably consilient with the extrinsic mortality model we have set out (see below). Second, several of the other explanations can be seen as different accounts of the proximate mechanisms

by which reduced investment in preventative health behaviours is delivered. For example, *self-medication and stress, latent traits* such as time preference and feelings of *efficacy* could all be aspects of the proximate psychology that delivers a disinvestment in taking preventative action for the future exactly when extrinsic mortality is perceived to be high. *Social support, class distinctions* and *community opportunities* are also explanations of proximate mechanisms, but these focus more on how patterns of health behaviours are maintained over time in particular social groups through social learning and norms. They do not explain why exactly those social groups initiate exactly those patterns of behaviour in the first place.

Third, the only types of explanation which do not relate at all to the adaptive approach are *lack of knowledge of health risks* and *aids for healthy behaviours*. These amount to claims that people of lower SES are simply ignorant in their health behaviour decisions, or do not have the option of behaving differently, and we can therefore label them non-adaptive, or constraint-based, explanations.

#### 10.6 Added Value of the Evolutionary Approach

We argued in Table 10.1 that many of the existing social science explanations (ET1–9) relate closely to the adaptive approach of Nettle (2010a) outlined above. So what, then, is the added value of taking an adaptive approach? Does it provide anything which was not already available? Here, we briefly discuss several ways in which we believe that developing an evolutionary model is useful:

- 1. For clarifying what explanations are competing and complementary
- 2. For deepening existing explanations
- 3. Because of the implications for public health interventions

 Table 10.1
 Pampel et al.'s (2010) nine types of explanation for SES disparities in health behaviour reclassified and reinterpreted from an adaptive perspective

Level of explanation	Pampel et al.'s explanation type (ET)	Interpretation in light of adaptive model
Ultimate	Fewer benefits of health behaviours (ET2)	Increasing extrinsic mortality reduces the adaptive benefits of healthy behaviour. This is an ultimate explanation because it explains <i>why</i> the behavioural response is adaptive
Proximate	Deprivation and stress (ET1)	Deprived environments may entail greater extrinsic mortality. This could act as a trigger for reduced investment in health. Features of low SES life may also trigger stress responses, which could lead to "self- medication" using food, tobacco or alcohol. These explanations are mechanistic because they explain <i>how</i> behaviours are triggered, but not <i>why</i> they exist. Our extrinsic mortality explanation explains <i>why</i> we should expect differences in health behaviour to correspond with deprivation
	Latent traits (ET3)	Latent traits arguments presume some third variable causes both SES and health behaviour. These are proximate explanations because they describe <i>how</i> latent traits might link SES and health behaviours, but not <i>why</i> there should be variation in that latent trait in the first place. Our extrinsic mortality explanation suggests that differences in factors such as time preference (triggered by cues associated with deprivation) could be a latent trait
	Class distinctions (ET4)	Once established, SES differences in health behaviour may be further perpetuated by class distinctions. This is a mechanistic explanation because it explains <i>how</i> behaviours are reinforced as class norms, but not <i>why</i> they become associated with class in the first place. Our extrinsic mortality model explains why we should expect class differences in health behaviour
	Efficacy and agency (ET6)	Explanations about efficacy and agency suggest that education enhances sense of control and thereby increases the tendency to seek out solutions to health problems. This is a mechanistic explanation, which describes <i>how</i> SES might influence health behaviour. The extrinsic mortality explanation suggests that SES differences in motivation towards health behaviour and feelings of control over health may produce an effect which looks a lot like SES differences in efficacy and agency

Level of explanation	Pampel et al.'s explanation type (ET)	Interpretation in light of adaptive model
	<i>Community opportunities</i> (ET8)	These arguments suggest that SES differences in health behaviour may be due to differences in community opportunities. This is a mechanistic explanation because it explains <i>how</i> behaviours are reinforced, but not <i>why</i> they become associated with SES in the first place. However, our extrinsic mortality explanation highlights the fact that community features such as safety may be particularly important in explaining SES differences in health behaviour
	Social support and influence (ET9)	Explanations about social support and influence suggest that social learning and peer support are important for the spread of health behaviours amongst low and high SES networks. These explanations are mechanistic, because they describe <i>how</i> behaviour spreads through social networks, but not <i>why</i> behaviours differ by SES in the first place. Our extrinsic mortality explanation explains why we should expect initial SES differences in health behaviour
Non-adaptive explanations (constraint based)	Lack of knowledge (ET5)	This is a non-adaptive explanation. A lack of knowledge about health risks could be a fundamental cause of SES differences in health behaviour. However, as we have discussed in the chapter, we have reason to believe that this cause contributes a limited amount to SES gradients in health behaviour. For example, it cannot explain the persistence of SES differences in smoking, when it is now compulsory to print messages such as "smoking kills" on cigarette packaging
	Aids for healthy behaviour (ET7)	This is also a non-adaptive explanation. A lack of ability to pay for health aids could be a fundamental cause of SES differences in health behaviour. However, this explanation cannot account for the fact that many of the poorest people have unhealthy habits, such as smoking and heavy drinking, which actually <i>cost</i> money
#### 1. Clarification of What is Competing and What is Complementary

The most useful merit of the adaptive approach is that it clarifies how the different parts of the story relate to one another. Pampel et al. discussed nine classes of explanation and stated that it was difficult to adjudicate or integrate between them. Our evolutionary approach suggests that there may not be nine, but only two, conflicting accounts to consider. The first is the non-adaptive or constraint account: People of lower SES make suboptimal choices about health behaviour through lack of information or options (ET5 and ET7). By suboptimal here, we mean choices that they would change if they had better information or options. The second are the adaptive accounts, both at the ultimate (ET2, our model) and proximate (ET1, 3, 4, 6, 8, 9) levels.

Evidence for the non-adaptive account is at best mixed, and it may depend which health behaviours are being considered. In the case of healthy eating, for example, it is possible to mount a convincing case that the healthiness of a diet is strongly influenced by how much money one can spend on it (Drewnowski and Specter 2004; Drewnowski et al. 2007). However, there are other cases where the evidence is in clear conflict with non-adaptive or constraint accounts. The simplest health protection behaviours (e.g. seat belt use) cost nothing and yet are less used by low-SES groups (Colgan et al. 2004; Leigh 1990). In addition, leading causes of the excess mortality in low-SES groups include tobacco and alcohol. Far from costing something to avoid, these habits are expensive to engage in. This suggests that SES gradients in health cannot be attributed solely to a lack of ability to purchase health.

Nor is lack of information likely to explain the gradient. Health warnings have been printed on cigarette packets for many years and in many countries. Since 2002, cigarettes sold in the EU countries have been obliged to display warnings such as "Smoking kills" and "Smoking seriously harms you and others around you" on at least 30 % of the front of the packaging and 40 % of the back (EU Directive 2001/37/EC concerning the manufacture, presentation and sale of tobacco products). Despite this, social gradients in smoking habits in the EU countries persist (Lader 2008; Buck and Frosini 2012). Indeed, evidence suggests that desire to quit and use of smoking cessation tools do not differ across social class, while quitting success does (Kotz and West 2009). This implies that the gradient may be created by differences in motivation toward healthy behaviour rather than by ignorance of the risks. This is supported by evidence regarding changes in health behaviours in the UK between 2003 and 2008. During this time period, there was extensive government investment in public health information campaigns. Buck and Frosini (2012) examined how four behaviours (smoking, excessive alcohol use, poor diet and low levels of physical activity) changed during this time. They found that high-SES individuals dramatically reduced their levels of unhealthy behaviour during the public health campaign period, while low-SES individuals did not. Receiving specific health information may have improved behaviour in individuals already motivated to invest in health, while failing to change behaviour in others. Thus, a key prediction of the lack of information hypothesis-that the gradient would disappear if everyone were given better information-is disconfirmed.

The major alternative to the non-adaptive account is something along the lines of the Blaxter hypothesis (ET2): People of lower SES invest less in their future health because the benefits of doing so are less for them than for people of higher SES. The behavioural-ecological model, by distinguishing analytically between extrinsic and intrinsic mortality and following through mathematics, provides a non-circular theoretical foundation for the Blaxter hypothesis. It also clarifies some anomalies with the existing economic models that have made similar arguments. For example, Cutler and Lleras-Muney (2006) suggest that individuals with better education may obtain greater incomes and therefore may expect to be happier in the future. They argue that this makes more educated individuals more likely to invest in protecting their future. But the same economic logic could be used to make exactly the opposite prediction (Pampel et al. 2010): High-earning individuals face greater opportunity costs in investing in time-consuming health behaviours (sleeping, exercising and preparing nutritious meals) than low-earning ones. Thus, without the fundamental distinction between extrinsic and intrinsic mortality as a foundation, it is hard to ground these intuitively plausible hypotheses in sound theory.

What of the remaining possibilities (ET1, 3, 4, 6, 8, 9)? We would argue that they constitute different claims about the proximate process by which an adaptively patterned disengagement from investment in the distant future under conditions suggestive of high extrinsic mortality might be delivered. As such, they do not conflict with ET2 at all, and they do not necessarily conflict with one another. Instead, we could think of them as different proximate pathways that might all contribute something, and to which studies might eventually be able to apportion different weights in terms of their centrality. Many or all of them could play a role, though, and indeed with most human behaviours, multiple mechanisms, both individual and social, are involved. Note that just because these are proximate mechanisms, they are not of lesser

importance than ET2. On the contrary, as we shall discuss below, mechanisms that were adaptive over evolutionary time may not optimize personal or societal welfare today, and those who design interventions need to understand the proximate mechanisms as much as if not more than the ultimate shaping forces.

#### 2. Deepening Explanations

The next major merit of the adaptive approach is that it deepens explanation. Many of the ET1–9 are likely to be correct, but provoke the immediate response, yes, but why should that be the case? For example, people of low SES may be more present oriented or motivated by immediate payoffs (ET3); yes, but why? People of low SES may feel that they have less control over their futures (ET6); yes, but why do they feel that way? Low-SES communities may have norms of smoking (ET9) and even use these as identity markers (ET4); yes, but why are SES gradients in norms consistently established in the same direction across different populations, so that they become available for identity marking? In each of these cases, the proximate factor is crying out for integration into a deeper explanatory framework. In this framework, preference for immediate payoff or subjective lack of control are responses delivered by an evolved psychology attuned to cues of extrinsic mortality, delivering adaptively patterned shifts in behaviour, which then become propagated through social transmission.

A related point is that several things which are often taken as exogenous traits may in fact be psychological reactions to living in environments containing cues suggestive of high extrinsic mortality. For example, time preferences (relative valuation of present and future benefits) are often invoked in the health behaviour literature, and they are generally assumed to be stable individual differences of endogenous origin (e.g. Fuchs 1982; Kirby 2009). They are not typically viewed as psychological responses to environmental cues. However, within the framework we have outlined above, it is possible to view SES differences in time preference as part of an adaptive response to differential exposure to extrinsic mortality risk. This insight has guided the experiments we reviewed above, whereby participants changed their future discounting behaviour in response to cues to extrinsic mortality (Griskevicius et al. 2011b). From this perspective, explanations about "attraction to short-term gain", which Pampel et al. classed as latent traits, may in fact be responses to an ecology in which there are fewer benefits of health behaviours for both longevity and Darwinian fitness. This might also apply to efficacy and agency and to risk preferences.

The strongest evidence for this contention comes from experiments showing that these "traits" can in fact be manipulated over short timescales (Mishra (under review); Ermer et al. 2008; Callan et al. 2009; Wilson and Daly 2004). For example, Callan et al. (2009) investigated the impact of "just world threat" on future discounting. They exposed participants to a video in which a woman talks about her experience of living with HIV. Half of their participants were told that the woman had contracted HIV after having unprotected sex with someone she met at a friend of a friend's party. The other participants were told that the woman contracted HIV after she was in a car accident and was given a blood transfusion with infected blood. The authors deemed the latter scenario a just world threat, because the woman could be perceived as an innocent victim, who contracted HIV without having done anything to deserve it. The participants that were exposed to this just world threat subsequently discounted future rewards more steeply than those who were told that the woman contracted HIV after unprotected sex. Callan et al. interpreted this finding as a link between the need to believe in a just world, and the ability to delay gratification. However, our evolutionary framework offers an alternative interpretation: the just world threat scenario acted as a cue, to extrinsic mortality risk. In the scenario where the woman contracts HIV through unprotected sex, the decision about whether to have unprotected sex with a relative stranger is under her control. In the scenario where she contracts HIV from a blood transfusion, the situation is beyond her control. The mortality risk is extrinsic.

Further evidence that future discounting may change in response to cues to extrinsic mortality risk comes from Li et al. (2012). They investigated discounting in Chinese earthquake survivors in comparison with controls, who lived in similar towns, but had not recently experienced earthquakes. They found that the earthquake survivors discounted future rewards more steeply than the controls. They also measured event-related brain potentials and found group differences in the neural responses to the discounting task. The results of such experiments suggest that the latent traits which have been treated (either implicitly or explicitly) as stable individual differences may in fact be flexible responses to cues from the environment. This may account for the inconsistencies in findings regarding time preference and health behaviours (Becker and Mulligan 1997). If variables, such as future discounting, are treated as fixed individual traits, our conclusions can be quite different from those drawn when considering that they may be flexible responses to ecological factors.

#### 3. Implications for Interventions

The adaptive perspective has potentially quite significant implications for the design of interventions. It argues that disinvestment in health behaviours represents a sensible response to living in certain types of environments, namely those rich in unavoidable danger. The corollary of this is that there is no reason to believe that giving people living in such environments more information about, say, the harms of smoking, is likely to make a dramatic difference to their behaviour. In fact, such information-giving can actually increase disparities in health behaviour when it is implemented across whole populations, exactly because the most affluent are most motivated to attend to the information and update their decisions using it, while the poorest have less incentive to do so (White et al. 2009; Capewell and Graham 2010).

An adaptive perspective naturally draws attention to broader structural-ecological parameters. Roughly speaking, it predicts that if the *extrinsic* dangers of deprived environments could be tackled, then the behaviours would more or less take care of themselves. That is, if societies reduce the relatively high rates of violence, the dangers of jobs and buildings, the differential exposure to accidents and toxins, etc. that beset deprived communities, then people in those communities would be more likely to be motivated to stop smoking. At the extreme, this kind of argument leads to an insupportably strong claim that no public health interventions aimed directly at health behaviour are worth carrying out, since all people are already assumed to be behaving adaptively anyway. The only action worth investing in is political action to improve socioeconomic conditions. Although we do recognize the force in this argument, we would not wish to go that far. We do however endorse the view that improving the socioeconomic environment is desirable and has a double yield; it is a good thing to do in its own right, and it will have a secondary benefit as people respond by looking after themselves better.

A more nuanced position would be the claim that the relatively low investment in preventative health behaviours seen in deprived communities is the result of adaptive *mechanisms*, while not always representing adaptive *behaviour*. That is, natural selection has sculpted psychological mechanisms which lead people to respond to conditions of high extrinsic mortality by becoming more present-oriented and investing less in their health. Although those mechanisms have on average been fitness enhancing over the millennia, it does not follow that every time they are engaged, particularly in modern environments, they improve the person's fitness, still less their wellbeing.

It is important to understand what determines people's perceptions of mortality risk, especially if perceptions of mortality risk are inaccurate. For example, evidence suggests that media coverage tends to skew people's perceptions of the risk of death due to given causes (e.g. Frost et al. 1997). It is possible that some portrayals of health scares in the news might worsen health behaviours, rather than improving them. Furthermore, reducing perceptions of extrinsic mortality may help to improve health behaviours, thereby reducing the inequalities that result from SES disparities in behaviour. There is much scope for applied evolutionary research in this area.

Finally, an understanding of the significance of psychological mechanisms attuned to cues of extrinsic mortality suggests some counterintuitive routes for intervention. Whereas intuition tells us that the most effective way to change health behaviour is to alert people to the risks of death that they face (as in the word "kills" on cigarette packets), it could be that such messages activate the mental schema of *extrinsic* mortality, making some people, perhaps especially those who live in harsh environments, feel that they are going to die anyway, and so there might be little point in trying hard to quit. If this were confirmed, then a health message pointing out that social conditions are improving and life expectancy has never been longer, and so there is all to try for, might actually have more effect than a negative message. This is a simple prediction that calls for further observation and experimental research.

So to conclude, the evolutionary perspective can bring a great deal of added value to much debated questions in public health. Rather than adding another seemingly competing explanation to the mix, taking an adaptive approach to understanding health behaviours can help to unite explanations from a diverse range of literature. It can help to clarify our understanding of what explanations are competing and what are complementary. It can deepen existing explanations and it can shed new light on the success and failures of health interventions. Yes, there is still much work to be done, but the evolutionary perspective undoubtedly has a good deal to offer.

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# Local norms of cheating and the cultural evolution of crime and punishment: a study of two urban neighborhoods

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# ABSTRACT

The prevalence of antisocial behavior varies across time and place. The likelihood of committing such behavior is affected by, and also affects, the local social environment. To further our understanding of this dynamic process, we conducted two studies of antisocial behavior, punishment, and social norms. These studies took place in two neighborhoods in Newcastle Upon Tyne, England. According to a previous study, Neighborhood A enjoys relatively low frequencies of antisocial behavior and crime and high levels of social capital. In contrast, Neighborhood B is characterized by relatively high frequencies of antisocial behavior and crime and low levels of social capital. In Study 1, we used an economic game to assess neighborhood differences in theft, third-party punishment (3PP) of theft, and expectation of 3PP. Participants also reported their perceived neighborhood frequency of cooperative norm violation ("cheating"). Participants in Neighborhood B thought that their neighbors commonly cheat but did not condone cheating. They stole more money from their neighbors in the game, and were less punitive of those who did, than the residents of Neighborhood A. Perceived cheating was positively associated with theft, negatively associated with the expectation of 3PP, and central to the neighborhood difference. Lower trust in one's neighbors and a greater subjective value of the monetary cost of punishment contributed to the reduced punishment observed in Neighborhood B. In Study 2, we examined the causality of cooperative norm violation on expectation of 3PP with a norms manipulation. Residents in Neighborhood B who were informed that cheating is locally uncommon were more expectant of 3PP. In sum, our results provide support for three potentially simultaneous positive feedback mechanisms by which the perception that others are behaving antisocially can lead to further antisocial behavior: (1) motivation to avoid being suckered, (2) decreased punishment of antisocial behavior, and (3) decreased expectation of punishment of antisocial behavior. Consideration of these mechanisms and of norm psychology will help us to understand how neighborhoods can descend into an antisocial culture and get stuck there.

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## **INTRODUCTION**

Why do humans behave antisocially? The converse of this question—why humans behave prosocially—has been studied extensively by experimental economists, and determinants of prosocial behavior may be mirror images of determinants of antisocial behavior. One proximate explanation for prosocial behavior is punishment; i.e., people will behave prosocially if not doing so results in punishment. Empirical evidence for this comes from economic games. Using a repeated public goods game, *Yamagishi (1986)* and *Fehr & Gachter*" (2000) showed that the opportunity for players to fine each other on the basis of contribution behavior can stabilize contributions to the public good at a high level. Following this, the cross-cultural covariation of prosocial behavior and punishment has received substantial interest (*Henrich et al., 2006; Herrmann, Thoni*" & *Gachter,*" 2008). Considerable local variation in prosociality has also been observed (*Wilson, O'Brien & Sesma, 2009; Nettle, Colleony & Cockerill, 2011; Lamba & Mace, 2011*), yet the question of whether prosocial behavior and punishment positively covary at the *local* level has spurred little research among experimental economists (but see *Kocher, Martinsson & Visser, 2012*).

However, the related question of whether *antisocial* behavior and a *lack of punishment* positively covary at the neighborhood level has generated substantial research within the field of sociology. Social disorganization theory posits that poverty, residential mobility, and family disruption can diminish the capacity a community has for creating relationships and establishing shared social norms. This low level of 'social capital' can lead to increased crime and delinquency via reduced collective action (*Shaw & McKay, 1942; Sampson & Groves, 1989; Bursik & Grasmick, 1993a; Bursik & Grasmick, 1993b; Sampson, Raudenbush & Earls, 1997*). Without trust and shared behavioral expectations, residents have decreased capacity to enforce desirable behavior through informal social control (i.e., informal surveillance and/or intervention by residents) (*Sampson, Morenoff & Gannon-Rowley, 2002*).

Of interest to researchers in both of these fields is how the local social environment can evolve over time to become more prosocial or more antisocial. This requires an understanding of the dynamic relationship between individual decisions (as typically studied by experimental economists) and the local social environment (as typically studied by sociologists). That is, individual decisions can be influenced by empirical expectations of the behavior of others in the local social environment (*Bichierri & Xiao, 2009*). These decisions, as manifest in observable behavior, then become part of the local social environment. Others will form expectations on the basis of their perception of the local environment and possibly alter their own behavior. That such a dynamic relationship exists is suggested by, for example, the interdependence of individual decisions to commit crimes (*Glaeser, Sacerdote & Scheinkman, 1996*).

In this paper, we attempt to bridge these two approaches of experimental economics and sociology and increase our understanding of the dynamic relationship between individual decisions and the social environment. We do so through consideration of the role of the individual's expectation of others' cooperative behavior—that is, the role of perceived local norms of cooperative behavior. *Cialdini, Reno & Kallgren (1990)* distinguish between *injunctive norms* and *descriptive norms*. Injunctive norms convey how people should behave. Descriptive norms, on the other hand, illustrate how most people actually do behave.

It is readily apparent that cooperative descriptive norms should be informative as to people's expectation of cooperation. However, cooperative descriptive norms may also be informative as to people's expectation of punishment for cooperative norm violation or antisocial behavior, particularly when there is a mismatch between injunctive and descriptive norms. A lack of alignment between injunctive and descriptive cooperative norms is implicit in broken windows theory—the idea that signs of social and physical disorder invite criminal behavior—in part because disorder is a cue that social control is lax (Kelling & Wilson, 1982). This mechanism for the 'spread of disorder' was elegantly tested by Keizer, Lindenberg & Steg (2008), who created public spaces in which an explicit injunctive norm was violated—e.g., a littered space (conveying a descriptive norm) next to a sign telling people not to litter (injunctive norm)—thereby communicating a lack of adherence to the injunctive norm and experimentally inducing further antisocial behavior. These results suggest that signs that others are flouting injunctive cooperative norms may serve as cues that antisocial behavior will not be punished. However, this remains a largely untested explanation of these results and of the broken windows effect in general (Traxler & Winter, 2012; but see Lochner, 2007).

Important to the studies we present in this paper, the work of *Keizer, Lindenberg & Steg (2008)* and *Keizer, Lindenberg & Steg (2013)* also demonstrated the possibility for 'cross-norm effects'—that is, the focus of the injunctive and descriptive norms was different from the behavioral outcome assessed by the researchers. Some of the observed cross-norm effects included public versus private goods. For example, graffiti and litter (destruction of a public good) each resulted in an increase in theft of an envelope with money in it (*Keizer, Lindenberg & Steg, 2008*). In another set of experiments, these same authors also demonstrated cross-norm effects for the restoration of a public good and prosocial behavior targeted at an individual; garbage bags on the street—in violation of city ordinance—resulted in a decrease in posting of a letter dropped next to a postbox (*Keizer, Lindenberg & Steg, 2013*).

Thus, studying injunctive and descriptive cooperative norms presents a way to assess individual perceptions of environmental variation in cooperative and, potentially, punitive behavior. It also offers a way to study how the social environment affects the behavior of the individual and individual's behavior in turn affects the social environment, by conveying information about descriptive norms. It is particularly appropriate when the focus is on local (rather than large-scale) variation in prosocial or antisocial behavior, as injunctive norms may be more similar in areas where people share common culture and history, while descriptive norms may still vary. Given a general consensus on injunctive norms, the emphasis can then be on perceived deviation from the injunctive norms.

#### The studies

Our studies were set in two nearby neighborhoods in Newcastle Upon Tyne, England, that we expected to have similar injunctive cooperative norms based on a shared cultural history. These two neighborhoods are similar in size, physical layout, and ethnic composition yet differ dramatically in rates of antisocial behavior and socioeconomic deprivation. While Neighborhood A is relatively affluent, Neighborhood B has experienced high rates of unemployment, physical decay, massive depopulation, and crime, following the collapse of mining and shipbuilding industries (see *Nettle, Colleony & Cockerill, 2011* and citations therein). In an earlier study, *Nettle, Colleony & Cockerill (2011)* used surveys, a Dictator Game, behavioral observation, and field experiments to reveal substantially less antisocial behavior, more social capital, and more prosocial behavior in Neighborhood A than B.

Here, we return to these neighborhoods to investigate whether individual decisions to engage in antisocial behavior and norm enforcement vary by neighborhood. To do so, we evaluated antisocial behavior, punishment, and expectation of punishment in an economic game. We used a questionnaire to investigate whether neighborhood differences in antisocial behavior, punishment, and expectation of punishment could be explained by neighborhood differences in trust and local descriptive cooperative norms. *Study 1* was observational and aimed to document and explain differences in perceptions and behaviors between the neighborhoods. *Study 2* introduced a novel experimental methodology to manipulate perceived injunctive norm adherence, allowing us to make causal inferences. We assessed whether information on injunctive cooperative norm adherence altered expectations of punishment for antisocial behavior.

## **STUDY 1**

*Camerer & Fehr (2004)* suggest that a real-world example of a third-party punishment game (3PP game) (*Fehr & Fischbacher, 2004*) is scolding of a neighbor for treating another person unacceptably. In this study, we administered a 3PP game along with a questionnaire (see Supplemental Information). Our variant of the game, which was played among residents within each neighborhood, enabled us to study differences between the neighborhoods in antisocial behavior and punishment for antisocial behavior. Player 1 was given the opportunity to steal from Player 2. Player 3 was given the opportunity to fine Player 1 if she took money from Player 2. Player 2 indicated whether she thought Player 3 would fine Player 1 if Player 1 took half of Player 2's money.

We used Player 1 and Player 2 decisions to assess whether residents of Neighborhood B were (1) more likely to behave antisocially and (2) less likely to expect someone in their neighborhood to intervene in antisocial behavior. In conjunction with the questionnaire, we also used Player 1 and 2 decisions to investigate (3) whether perceived local cooperative norm violation could explain the the hypothesized neighborhood differences in individual antisocial decisions and (4) punitive expectations.

We used Player 3 game decisions and the questionnaire to assess (1) whether residents of Neighborhood A were more willing than those of B to punish antisocial behavior in their neighborhood, and (2) whether, following social disorganization theory, neighborhood trust could explain the hypothesized relationship between neighborhood and punitive behavior.

# Study 1 methods

#### Sampling

The Ethics Committee of the Newcastle University Faculty of Medical Sciences approved the study protocol (Protocol #00503/2011 and Amendment #00503 \_1/2012). Written consent was not obtained because it would have been the only record of participation. We conducted the study from July 2012 to December 2012. A maximum of one participant per household was drawn from the electoral roll. Potential participants received a hand-delivered envelope with a cover letter describing the study, packet (questionnaire, explanation of the game, and game), and stamped return envelope. A minority of envelopes were delivered by subjects in another study (*Nettle et al., 2014*). We avoided sampling adjacent households and households sampled by *Nettle, Colleony & Cockerill (2011)*.

## Questionnaire

From the questionnaire, we recorded each participant's age and sex.

*Trust.* We asked individuals how much they trust people in their neighborhood, on a 10-point scale (10 = most trusting).

*Civic norms: condoned and perceived cheating.* We asked individuals about both injunctive and descriptive civic norms (Supplemental Information). For the injunctive norms, we described three behaviors and asked whether it is Never OK to do this behavior, Always OK, or somewhere in between. Answers were constrained to a 10-point scale (1 = 'Always OK' and 10 = 'Never OK'). The behaviors were (1) cheating the benefits system, (2) avoiding a fare on public transport, and (3) cheating on taxes. *Condoned cheating* is the average across behaviors. Larger values indicate that cheating on public goods is condoned. Note that *condoned cheating* is similar to the 'norms of civic cooperation' (*Knack & Keefer, 1997; Herrmann, Thoni & Gachter, 2008*) derived from the World Values Survey.

For the descriptive norms, we asked individuals whether they think many people in their neighborhood would do these behaviors (1 = 'No one would' and 10 = 'Everyone would'). We averaged across these responses to arrive at *perceived cheating*. Larger values indicate that neighborhood cheating on public goods is perceived as more common. We note that the cooperative norms used in the questionnaire pertain to public goods, while the possibility for antisocial behavior in the game is directed at a single person. However, as mentioned in the introduction, previous studies have experimentally demonstrated 'cross-norm effects' wherein destruction or restoration of a public good induced antisocial or prosocial behavior, respectively, directed at a single individual (*Keizer, Lindenberg & Steg, 2008; Keizer, Lindenberg & Steg, 2013*).

#### The 3PP game

Participants read instructions for the game, which followed the questionnaire, and then worked through examples (see Supplemental Information). (From this, we had responses to six *test questions*.) They were told that after receiving the packet in the post, we would determine the game outcome and then deliver their cash payoff along with a £5 payment for completing the survey.

The game worked as follows: all three players received an initial allocation of £10, to be paid after the decisions of all three players had been submitted. Player 1 had to decide how many pounds (integer from 0 to 10) to take from Player 2. If Player 1 took money from Player 2, Player 3 had to decide whether to fine Player 1. We used the strategy method for Player 3. Player 3 had to decide, for each amount greater than 0 that Player 1 could take, whether to pay to fine Player 1. Therefore, Player 3 had to make 10 choices, each corresponding to an amount that Player 1 might take from Player 2. The cost of the fine to Players 1 and 3 was constant (Player 3 paid £2 to make Player 1 lose £6). Player 2 could not make a choice in the game. We asked Player 2 to indicate whether she thought Player 3 would fine Player 1 if she took £5 from her (Supplemental Information).

Game behaviors are thus: *theft* (an integer from 0 to 10 representing the amount of money Player 1 took from Player 2), *expect 3PP* (whether Player 2 expected Player 3 to punish Player 1 if she took £5), and *punitiveness* (an integer from 0 to 10; this is the total number of potential thefts, from  $\pounds 1 - \pounds 10$ , that Player 3 would punish).

#### Subjective value of money

We expected the subjective value of money to differ between neighborhoods and impact game behavior. Therefore, following the game, we asked how much of a difference, on a scale of one to 10, an amount of money x would make to their weekly budget, where x was £1 for Player 1 (*value* £1) and £2 for Players 2 and 3 (*value* £2). After commencement of data collection, we revised the packets for Player 1 to include  $x = \pm 10$ . Thus, for some Player 1s we also have *value* £10.

### Statistical analyses

The majority of responses can be considered discrete ordered choices. Thus, to assess neighborhood differences in game behavior, trust, cooperative norms, and the value of money, we analyzed the data with ordered logistic regression. The exception to this is game behavior for Player 2, for which we used binary logistic regression. We compared the fit of different models with the Akaike information criterion (AIC) (*Akaike, 1974*). Ordered and binomial logistic regression analyses and plotted predictions (i.e., the predicted value based on the fitted model and the data used to fit the model) were produced in the R statistical and computing environment (*R Core Team, 2012*) with the following packages: MASS (*Venables & Ripley, 2002*), rethinking (*McElreath, 2012*), beeswarm (*Ecklund, 2012*), and ggplot2 (*Wickham, 2009*). Note that plotted predictions for *theft* and *punitiveness* are both (0, 8). For each of these game behaviors, two possible values were not observed (3 and 8 for *theft*, 2 and 9 for *punitiveness*); thus, for prediction we condensed the ranges. We report Odds Ratios (ORs) for a unit increase in the outcome for each unit increase of the predictor variable, accompanied by 95% confidence intervals.

**Table 1 Key variables from** *Study 1* **by neighborhood.** Columns one and two contain medians for Neighborhood A and Neighborhood B, respectively (median absolute deviation in parentheses). Column three contains the odds that a participant from Neighborhood B indicated a higher value (95% confidence interval in parentheses). Condoned cheating and perceived cheating are the mean of the three injunctive and descriptive norms, respectively.

Variable	Median A (scale 1 to 10)	Median B (scale 1 to 10)	Odds B higher
Trust neighbors	8 (1)	5 (2)	0.053 (0.031, 0.093)
Value £1	1 (0)	1 (0)	1.89 (0.77, 4.61)
Value £2	1 (0)	2.5 (1.5)	5.53 (2.51, 12.18)
Value £10	3 (1)	5 (3)	3.37 (1.04, 10.9)
Injunctive avoid fare	1 (0)	1 (0)	0.84 (0.52, 1.35)
Injunctive cheat benefits	1 (0)	1 (0)	1.43 (0.87, 2.34)
Injunctive cheat tax	1 (0)	1 (0)	1.38 (0.84, 2.23)
Descriptive avoid fare	3 (1)	6 (2)	11.02 (6.58, 18.46)
Descriptive cheat benefits	3 (1)	6 (2)	13.95 (8.16, 23.85)
Descriptive cheat tax	4 (1)	5 (2)	3.06 (1.95, 4.79)
Condoned cheating	1.33 (0.33)	1.50 (0.50)	1.25 (0.80, 1.95)
Perceived cheating	3.00 (0.67)	5.50 (1.83)	10.22 (6.18, 16.90)

# Study 1 results

#### Participants

We achieved sample sizes of 40 (16 male), 44 (22 male), and 49 (23 male) for Players 1, 2, and 3, respectively, in Neighborhood A and 34 (12 male), 43 (23 male), and 50 (23 male) in B (Table S1). Every week, new players from each neighborhood were combined into triads, and we determined game outcome from their decisions. For incomplete triads, players were drawn at random from all previous neighborhood players. We delivered to participants: the game outcome, debriefing sheet, money received from the game, and £5 for participating. The mean payoff from the game is £9.26 ( $\sigma$  =£3.49) in Neighborhood A and £9.16 in B ( $\sigma$  =£4.13). Descriptive statistics and neighborhood comparisons for key variables are in Table 1. We also report descriptive statistics in the text to assist the reader.

#### Trust

Participants in Neighborhood A indicated far higher *trust neighbors* (median 8 on a scale of 1:10, median absolute deviation (MAD) 1) than did participants in B (median 5, MAD 2) (Table 1) (OR 18.8, 95% CI [10.8–32.8]).

#### Punishment of antisocial behavior

As predicted, participants in Neighborhood A were more punitive than those in B (Fig. 1) (OR 3.3, 95% CI [1.6–7.0]). Median *punitiveness* is 6 (MAD 4) and 3 (MAD 3) for Neighborhoods A and B, respectively. Thus, more participants in Neighborhood A indicated that they would pay  $\pounds 2$  to fine Player 1 for a greater number of potential thefts.

The subjective cost of punishment in the game, *value £2*, had a negative effect upon *punitiveness* (OR 0.7, 95% CI [0.6–0.9]) and was larger for participants in Neighborhood B than A (Table 1). However, participants in Neighborhood A were still more punitive than those in B when we include *value £2* in the model (OR 2.1, 95% CI [0.9–4.6]). This result is robust to the inclusion of additional covariates *age, male,* and *test questions* (OR 2.9, 95% CI [1.2–7.2]).



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Figure 1 Player 3 behavior, punitiveness, by neighborhood. Each diamond represents one observation.
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Based on social disorganization theory, we hypothesized that greater trust among residents of Neighborhood A would partially explain the increased willingness of residents to engage in 3PP of antisocial behavior. Individuals who reported greater *trust neighbors* were slightly more punitive (OR 1.15, 95% CI [0.99–1.32]). The relationship between trust and punitiveness is less robust to the inclusion of *value £2* (OR 1.09, 95% CI [0.94–1.27]); however, including an interaction between *value £2* and *trust neighbors* improves model fit (AIC of 380.13 compared to 384.49).

Predictions from this model including the interaction are shown in Fig. 2. Value £2 still has a negative effect on *punitiveness*, but the slope is steeper for participants with high *trust neighbors*. Thus, participants with high *trust neighbors* are more punitive than those with low *trust neighbors* when *value* £2 is small, but less punitive when it is large. *Neighborhood* is no longer a reliable predictor of *punitiveness* when the interaction is included in the model (OR 1.8, 95% CI [0.7, 5.7]), nor does model fit improve with the addition of *neighborhood* (AIC = 380.67).

#### Civic norms: condoned and perceived cheating

In both neighborhoods, most participants indicated that it is not acceptable to cheat on public goods. We observed little variation in injunctive norms across cooperative behaviors (Table 1). Nor did we detect a clear difference between neighborhoods with respect to specific injunctive norms or *condoned cheating* (i.e., the within-participant mean of injunctive norms) (Fig. 3, Table 1).



**Figure 2** *Punitiveness* **modeled as an interaction between** *trust neighbors* **and** *value* £2. Blue is 'high trust' (8; median *trust neighbors* score for Neighborhood A). Orange is 'low trust' (5; median *trust neighbors* score for Neighborhood B). Dotted lines are 95% confidence intervals.



**Figure3 Neighborhood means and standard errors for** *condonedcheating* **and** *perceivedcheating*. For *condoned cheating*: 1, Never OK; 10, Always OK; and for *perceived cheating*, 1, No one would; 10, Everyone would.

However, there was a dramatic difference between neighborhoods with respect to *perceived cheating*. Participants in Neighborhood B indicated that more of their neighbors would cheat on a public good than those in A (median 3.00, MAD 0.67 for A; median 5.50, MAD 1.83 for B) (Fig. 3, Table 1). Participants who thought more of their neighbors cheat on public goods were also less trusting of their neighbors (OR 0.54, 95% CI [0.48–0.62]).

Juxtaposition of *condoned cheating* and *perceived cheating* reveals that although participants in Neighborhood B tended to state that many of their neighbors cheat on public goods, we lack strong evidence that they view this behavior as more acceptable than those in A. This fits with our prior expectation that injunctive cooperative norms would be similar in Neighborhoods A and B. We therefore use *perceived cheating* as a within-participant measure of perceived local cooperative norm violation, or deviation from the injunctive cooperative norm.

#### Antisocial behavior

Participants in Neighborhood B took more from their neighbors in the game. *Theft* is also more variable in Neighborhood B than A. The median value of *theft* is 5 in Neighborhood B (MAD 5), compared to 0 in A (MAD 0) (odds that *theft* is greater in Neighborhood B: OR 2.9, 95% CI [1.2–7.1]). The neighborhood difference in *theft* is robust to the inclusion of *age*, *male*, and *value* £1 (OR 2.8, 95% [2.5–6.9]). For the reduced dataset for which we had data on *value* £10 (40 participants, 23 from Neighborhood A), substituting this variable in the model increases the odds that a participant in B stole more in the game (OR 4.1, 95% CI [0.9–17.5]). Inclusion of *test questions* in the model reduces confidence in the neighborhood difference in *theft* (OR 2.1, 95% [0.8–5.8]). However, incomplete test questions are heavily patterned for Player 1; only participants in Neighborhood B for whom *theft* > 0 did not complete the questions. Irrespective of the participant's comprehension of the entire game, the opportunity for Player 1 to behave antisocially (the outcome of interest to us) should be very clear from the packet (i.e., "How many pounds do you choose to take from Player B?") (Supplemental Information).

As expected, *perceived cheating* is a robust predictor of *theft*, even controlling for *value £1* (Fig. 4; OR 1.3, 95% CI [1.0–1.6]). When both *neighborhood* and *perceived cheating* are considered in the same model, neither is a reliable predictor of *theft*. Nor does AIC offer strong support for a single model (235.40 for the model with *perceived cheating*, 234.67 for *neighborhood*, and 234.60 for *perceived cheating* + *neighborhood*). This suggests that to understand the greater *theft* in Neighborhood B, we need to consider *perceived cheating*.

## **Expectation of 3PP**

We asked Player 2 whether she thought Player 3 would fine Player 1 if Player 1 took £5 from her (*expect 3PP*). Contrary to our expectations, *neighborhood* was not a reliable predictor of *expect 3PP*. Of participants in Neighborhood A, 36.36% expected 3PP,

compared to 30.23% of participants from Neighborhood B (OR 1.2, 95% CI [0.5–3.2]). However, as predicted, we did observe a negative relationship between *perceived cheating* and *expect 3PP* (Fig. 5; OR 0.8, 95% CI [0.6–1]). This relationship does not change with inclusion of *value* £2 as a proxy for the local subjective value of £2 (OR 0.8, 95% CI [0.6–1]).



perceived cheating





perceived cheating

Figure 5 Probability of *expect 3PP* dependent on *perceived cheating*. Dotted lines are 95% confidence intervals.

#### Study 1 summary and discussion

Study 1 reveals that individual perceptions of local cooperative descriptive norms (i.e., *perceived cheating*) vary dramatically by neighborhood, in concordance with previous observations of neighborhood discrepancies in antisocial behavior (including crime), prosocial behavior, and social capital (*Nettle, Colleony & Cockerill, 2011*). Participants in Neighborhood B were far more likely than those in A to think that more of their neighbors behave uncooperatively. We could not, however, attribute this to a neighborhood difference in injunctive cooperative norms. Thus, a perceived lack of adherence to injunctive cooperative norms was pervasive in Neighborhood B.

This general perception in Neighborhood B that others are behaving antisocially appears justifiable: participants in Neighborhood B stole more money in the game. However, the results of our analyses suggest that this neighborhood difference in theft in the game can be explained by neighborhood differences in descriptive cooperative norms. That is, individuals who perceived cheating to be common were more likely to steal, and stole more in the game. These individuals tended to reside in Neighborhood B. Thus, the perception that others in the community are cheating may have induced further antisocial behavior in the game. While this observation is purely correlational, it is in accordance with the experimental results of *Falk & Fischbacher (2002)*, who demonstrated a positive effect of observed theft on a participant's subsequent choice to steal in the lab. It is also in agreement with those of *Cialdini, Reno & Kallgren (1990)* and

*Keizer, Lindenberg & Steg (2008)*, who showed that observed norm violation can result in an increase in norm violation.

Correspondingly, participants in Neighborhood B indicated far less trust in their neighbors than did those in A. This result fits with the far lower self-reported social capital in Neighborhood B previously observed. Our measure of trust in the current study, *trust neighbors*, approximates one of six items in the social capital index of *Nettle*, *Coll'eony & Cockerill (2011)*, which was highly positively correlated with the overall index (0.77, *p*-value < 0.05).

As expected, and in concordance with social disorganization theory, *trust neighbors* was a positive predictor of punitiveness. *Kocher, Martinsson & Visser (2012)* similarly found that trust in members of a participant pool was positively correlated with punitiveness in a public goods game. Although they interpreted this outcome as stemming from greater disappointment in free-riding behavior, they suggest it merits further investigation of the role of social capital in norm enforcement.

One possible interpretation of the unpredicted interaction we observed between *trust neighbors* and *value*  $\pm 2$  lies in consideration of the multiple ways in which the cost of punishing can vary for the punisher. We showed that participants were more punitive when value £2 was smaller. Punitiveness is also less costly when there are fewer defectors and/or more punishers (Boyd et al., 2003, Gurerk, Irlenbusch & Rockenbach, 2006; Boyd, Gintis & Bowles, 2010). Trust neighbors may be informative as to whether Player 3 thinks there are many punishers and defectors in her neighborhood and thus construed as a measurement of the cost of intervening in antisocial behavior. From this perspective, our results are consistent with the idea that people are more punitive when punishment is cheap—with respect to both material resources and the behavior of others. This also highlights a limitation of this study, which is that Player 3 was able to punish anonymously and therefore 'cheaply' with respect to possible retribution. In the real world, third-party punishment may be associated with risk of retribution or other costs that are not captured by the £2 Player 3 paid to exact punishment. Decreased resiliency to retribution could also vary by neighborhood, perhaps partly as a result of differing material resources.

We are unable to determine whether participants in Neighborhood B stole more money than those in Neighborhood A because they thought punishment was less likely. This is because a participant's motivation to steal a particular amount of money can be ascribed to inequity aversion as well as the expected probability of punishment. However, our data from Player 2 address expectation of punishment. While we did not observe a robust neighborhood difference in *expect 3PP*, we did observe a strong negative relationship between *perceived cheating* and *expect 3PP*. That is, a participant who thought many of her neighbors cheat on public goods was less likely to expect a neighbor to pay  $\pounds 2$  to fine Player 1 if Player 1 took half her money.

This result supports the idea that descriptive cooperative norms are indeed informative as to expectation of punishment (*Traxler & Winter, 2012*). It also suggests that expectation of punishment is one of the mechanisms by which signs of norm violation can lead to further violation (*Traxler & Winter, 2012*; *Kelling & Wilson, 1982*). However, the causality of the observed relationship between *perceived cheating* and *expect 3PP* remains unknown. Surveys of the kind in *Study 1* can only establish correlation; examining the causal significance of one variable for another requires experimental manipulation of the first variable. With this in mind, we undertook *Study 2*, in which we used selective feedback from *Study 1* to experimentally alter perceptions of perceived cheating in the two neighborhoods.

## **STUDY 2**

Feedback on or manipulation of descriptive norms has been used to alter people's behavior—in diverse domains from littering (*Cialdini, Reno & Kallgren, 1990*) to energy use (*Nolan et al., 2008*). In *Study 2*, we used a novel method for manipulation of descriptive norms to investigate the causality of the relationship between *perceived cheating* and *expect 3PP*. In each neighborhood, we provided novice Player 2s with information on what their neighbors thought about the descriptive cooperative norms of the neighborhood ('Norms treatment'). We manipulated this information so as to present *Study 2* participants from Neighborhood A with a less positive picture of descriptive norms than was really the case, and participants from Neighborhood A who received the Norms treatment would be less likely to expect Player 3 to 3PP on their behalf, compared to those participants in the same neighborhood B.

## Study 2 methods

### Sampling

We collected data for *Study 2* from October to December 2012, while *Study 1* was ongoing (Supplemental Information), following the same protocol as in *Study 1*.

#### Norms questionnaire

We refer to the questionnaire used in *Study 1* as 'Baseline treatment'. The questionnaire for the Norms treatment differed as follows.

*Civic norms manipulation: perceived cheating.* The Norms questionnaire did not include questions about injunctive and descriptive norms. We presented participants with information on the responses of a subset of *Study 1* participants in their neighborhood to the questions about descriptive civic norms (Supplemental Information).

The following backstory was used: as a part of the Tyneside Neighbourhoods Project, we had asked 10 people in their neighborhood how common they think avoiding a public transport fare, cheating the benefits system, and cheating on taxes, are in that neighborhood. We averaged these answers to get an idea of how common people think certain behaviors are. We wanted to know what other people in the neighborhood thought of these answers, and thus were asking them (Supplemental Information).

We presented one scale for each of the behaviors. The information in each scale was manipulated: in Neighborhood A, we took the mean of the 10 responses that gave the least favorable impression of cheating (i.e., high *perceived cheating*), and in Neighborhood B, we took the mean of the 10 responses that gave the most favorable impression of cheating (i.e., low *perceived cheating*). The information presented for Neighborhood A was: 5.7 for avoid a fare on public transport, 5.5 for cheat the benefits system, and 6.7 for cheat taxes (where 1 = 'No one would' and 10 = 'Everyone would'). In Neighborhood B the information presented was: 2.2 for avoid a fare, 2.3 for cheat benefits, and 1.7 for cheat taxes. Beneath each scale, *Study 2* participants were asked to circle 'Fewer people would do this', 'This is about right', or 'More people would do this' (Supplemental Information).

*Contamination.* To assess whether participants knew *Study 1* participants, we included a contamination question: 'Do you know of other people in your neighborhood who got a questionnaire and plan to post it or already have posted it?' ('Yes', 'Not sure', or 'No').

#### 3PP game

For *Study 2*, we measured the following behavior: *expect 3PP* (yes or no; representing whether Player 2 expected Player 3 to punish Player 1 if Player 1 took *£*5 from her).

#### Statistical analyses

We used binary logistic regression to assess the effect of the Norms treatment on *expect 3PP* within each neighborhood.



**Figure 6** Proportion of Player 2s, by neighborhood and treatment, who indicated that they expect Player 3 to 3PP on their behalf.

## **Study 2 results**

## Participants

For *Study 2*, we sampled 41 participants from Neighborhood A (21 male) and 39 participants from B (16 male) (Table S2).

## Reaction to normative information

Participants in Neighborhood B were far more likely than those in A to indicate 'This is about right' when presented with the manipulated norms scales for *cheat benefits* and *avoid fare* (OR 3.63, 95% CI [1.23–10.70] and OR 3.74, 95% CI [1.34–10.49], respectively). In Neighborhood B, 38.46%, 43.59%, and 46.15% of participants indicated 'This is about right' for *cheat benefits, avoid fare*, and *cheat taxes*, respectively. In contrast, the majority of participants in Neighborhood A indicated 'Fewer people would do this' when presented with the manipulated scales for *cheat benefits* and *avoid fare* (78.05% of participants for each behavior). Only 51.28% of participants in Neighborhood A indicated 'Fewer people would do this' when presented with the manipulated scales for *cheat benefits* and *avoid fare* (78.05% of participants for each behavior).

## Expectation of 3PP: norms treatment

Participants in Neighborhood B who received the Norms treatment—i.e., who received information that their neighbors perceive cheating to be uncommon— were more likely to expect Player 3 to 3PP on their behalf, compared to those in B who received the Baseline treatment. The proportion of participants who expected 3PP is 58.97% for the Norms treatment, compared to 30.23% for Baseline (OR 3.32, 95% CI [1.33–8.25]; Fig. 6). Exclusion of participants for whom *contamination* was 'Not sure' (five) or 'Yes' (two) does not qualitatively change the results. (One participant circled both.)

We did not observe a robust effect of the Norms treatment on *expect 3PP* in Neighborhood A. Contrary to our prediction, the proportion of participants in A who expected 3PP is 41.46% for Norms treatment, compared to 36.36% for Baseline treatment (OR 1.24, 95% CI [0.52–3.00]; Fig. 6).

However, the Norms treatment generated an unanticipated response in Neighborhood A. Some participants attempted to redirect their money by asking us to: donate it to charity (three participants), keep it for research/university funds (two participants), or not pay them (one participant). The rate of 'opting out of payment' is 14.63% for Norms treatment participants in Neighborhood A, compared to 1.15% of Baseline participants in A (OR 11.25, 95% CI [2.18–57.97]). This spontaneous change in game play was not observed in Neighborhood B.

## Study 2 summary and discussion

In *Study 2*, participants in Neighborhood B received information that their neighbors think there is little cheating on public goods in their neighborhood, relative to what we actually observed in *Study 1*. They were far more likely to expect a neighbor to punish antisocial behavior compared to those in Neighborhood B who did not receive the manipulation. Whether disorder can play a causal role in an increase in crime rates (*Kelling & Wilson, 1982*) has been debated (*Sampson, Morenoff & Gannon-Rowley, 2002; Markowitz et al., 2001*). Our results provide empirical evidence of a mechanism by which norm violation can lead to the further violation of a different norm—through change in the expectation of punishment.

There are at least three plausible routes by which this effect is achieved. One possibility is that people expect cooperators to be more likely than non-cooperators to punish. The second is that people perceive other's behavior to reflect other's expectation of punishment. That is, people think that others are not behaving antisocially because of their expectations of punishment for behaving antisocially. The third possibility, closely related to the second, is that if antisocial behavior is very common, people may intuit that it persists because antisocial behavior is going unpunished and thus have a decreased expectation of punishment.

We did not observe a reliable negative effect of the norms manipulation on expectation of 3PP in Neighborhood A. It is not clear why we observed the expected result in Neighborhood B and not Neighborhood A. In *Study 1*, we found greater variation in trust and norms in Neighborhood B than in Neighborhood A (Table 1). One interpretation of this is that the environment is more heterogeneous and unpredictable in Neighborhood B. If so, perhaps residents of Neighborhood B are less certain than residents of Neighborhood A of the behavior of their neighbors and therefore were more accepting of the manipulation. Indeed, far more Neighborhood B participants circled 'This is about right' when presented with the manipulated descriptive norms. Another possibility is that participants in Neighborhood B were more accepting of the information provided by an authority figure (university personnel/scientist).

# **GENERAL DISCUSSION**

The aim of this paper was to consider how the local social environment affects individual decisions to engage in and sanction antisocial behavior, and how an individual's antisocial behavior can in turn affect the local social environment, by conveying information about descriptive norms. In *Study 1*, we observed that

subjects in Neighborhood B took more money from their neighbors and were less punitive in an economic game of crime and punishment. The perception that others are cheating on public goods varied dramatically by neighborhood, was fundamental to the neighborhood difference in theft in the game, and was negatively associated with the expectation of third party punishment for antisocial behavior. Subjects in Neighborhood B were also less punitive of antisocial behavior, and punitiveness was negatively associated with trust in one's neighbors.

In *Study 2*, we showed that providing participants in Neighborhood B with information that cheating is perceived as uncommon within their neighborhood led to a sharp increase in the expectation of third-party punishment for theft. An increase in the perceived likelihood of punishment would presumably lead to greater cooperation, given the close relationship between these two variables. Thus, these results provide novel empirical support for a mechanism by which cues of norm violation can lead to further norm violation (*Cialdini, Reno & Kallgren, 1990; Keizer, Lindenberg & Steg, 2008*): altered expectation of punishment (*Kelling & Wilson, 1982; Traxler & Winter, 2012*).

We consider these results within a framework where culture is dynamic, subject to evolutionary processes that can lead to more or less cooperative outcomes (Boyd & Richerson, 1985). Unlike in recent cross-cultural studies of cooperation and punishment (Henrich et al., 2006; Herrmann, Thoni" & Gachter," 2008), our two study populations share many cultural components, including the institutions that formally sanction their civic violations (although how those institutions are experienced may vary) and injunctive cooperative norms. The apparently large discrepancy between desired and achieved cooperative outcomes in Neighborhood B, as assessed with injunctive and descriptive cooperative norms, adds a new perspective on the cultural evolution of variable cooperative outcomes. Our results provide evidence for three potential routes by which perceived cooperative norm violation can lead to further violation of cooperative norms.<sup>1</sup>All of these processes have been postulated or investigated by others; however, to our knowledge, they have not been considered simultaneously as processes that may, in concert, lead to substantial cultural change. These positive feedback processes are: (1) To avoid being 'suckered', conditional cooperators are motivated to defect if they perceive that defection is common (Fischbacher, Gachter" & Fehr, 2001; Falk & Fischbacher, 2002; Bichierri & Xiao, 2009; Raihani & Hart, 2010; Irwin & Simpson, 2013). (2) Perceived cheating leads to lower trust. Low trust results in reduced informal punishment of norm violation (Kocher, Martinsson & Visser, 2012). In this vein, Traxler & Winter (2012) observe a direct effect of the perceived frequency of norm violations on expressed willingness to sanction violations. Similarly, extensions of social disorganization theory include feedback processes between crime/disorder and social cohesion/control, via fear or residential instability (Sampson & Raudenbush, 1999; Markowitz et al., 2001; Steenbeck & Hipp, 2011). (3) When the perceived frequency of cooperative norm

<sup>&</sup>lt;sup>1</sup> Our studies focused on cooperative norm violation ("perceived cheating") and a very specific type of antisocial behavior (theft). However, based on work on cross-norm effects referenced in the introduction, we think that we can draw inferences here not just about theft but cooperative norm violation in general.

violation is high, expectation of punishment for violation is lower (*Sah, 1991*; *Traxler & Winter, 2012*).

We hypothesize that these three positive feedback mechanisms, wherein perceived cooperative norm violation leads to further cooperative norm violation, could act simultaneously to result in a rapid downward spiral, leading to low levels of cooperation. As Cialdini, Reno & Kallgren (1990) note, descriptive norms are informative as to adaptive behavior. In a community with low levels of cooperation and minimal punishment of cooperative norm violation, non-cooperative strategies may outperform others (Wilson & Csikszentmihalyi, 2007). Other processesprestige-biased (Henrich & Gil-White, 2001) or conformist (Henrich & Boyd, 1998) transmission and self-selection of people with preferences for an antisocial community—could further reinforce uncooperative or overtly antisocial strategies. While cooperative norms are considered a component of social capital (Knack & Keefer, 1997; Bowles & Gintis, 2002), our results demonstrate the need for explicit integration of cultural transmission and norm psychology—i.e., psychological adaptations for determining and adopting local norms and punishing violators (Chudek & Henrich, 2011)—with social disorganization theory. Scholars of criminology will note some similarities between the social learning theory of deviance (Akers, 2009) and theories of cultural transmission. However, we extend this bridge between the social environment and individual behavior by emphasizing the feedback from the individual to the social group. That is, we have outlined three routes by which an individual's defection can lead other individuals to adopt similar behavioral strategies, thus altering the local cultural ecology (Camerer & Fehr, 2006).

Missing from this hypothesized downward spiral is an initial perturbation that could result in an increase in cooperative norm violation (or perceived violation) in the neighborhood. Poverty and economic uncertainty are also striking differences between Neighborhoods A and B. Without middle class buffers of savings and credit, institutional safety nets, or strong reciprocal networks, crises such as illness create the potential for dire outcomes, thus altering the costs and benefits of defecting. For people already living at the margin, material crises might result in a higher probability of defection. Especially for crises that hit broad swaths of a community simultaneously, such as the widespread job loss in Neighborhood B resulting from the collapse of the shipbuilding and coal mining industries, one can imagine an increase in the frequency of defection that alters the descriptive cooperative norms enough to start a downward spiral in defection. Importantly, although we hypothesize that poverty and economic uncertainty were linked to an initial perturbation of cooperative norm violation in the current study, the positive feedback of norm violation could continue in the absence of poverty. There has been debate as to whether there are direct, as well as indirect, effects of poverty and/or income inequality on crime (Patterson, 1991; Bursik & Grasmick, 1993a; Bursik & Grasmick, 1993b). The story we have sketched is compatible with both possibilities, as an historical direct effect of poverty on norm violation may lead to cultural dynamics that persist beyond the duration of the poverty itself. (For a similar example of such cultural inertia, see Sah (1991), who argues that a transient change in the economics of crime can lead to persistently high crime rates, due to a postulated relationship between higher crime rates and decreased expectation of punishment.)

However, we can only speculate as to whether these dynamics are at play in Neighborhood B (outside of the 3PP game) and to what extent they can explain the observed high rates of crime and antisocial behavior.

This paper also makes contributions to empirical gaps in two fields. In Study 1, we demonstrated that the covariation of cooperation and punishment of noncooperation, which has been observed cross-culturally with economic games (Henrich et al., 2006), can extend to the local level. Participants in Neighborhood A stole less money and were more punitive in the game than those in B. Also in Study 1, we demonstrated an association—albeit small—between third-party punishment of antisocial behavior and trust in one's neighbors, as well as a neighborhood-level association between antisocial behavior in the game and decreased third-party punishment of antisocial behavior. These results provides additional, novel empirical support for the relationship between (1) low social control and low social capital, and (2) low social control and high rates of antisocial behavior. Data on actual social control (rather than the potential that residents will engage in social control, as measured by survey data) are difficult to come by, limiting the strength of the inference that low social capital and high rates of antisocial behavior are correlated due to lack of social control (Bursik & Grasmick, 1993a; Bursik & Grasmick, 1993b; Steenbeck & Hipp, 2011).

We acknowledge that there are a number of limitations to our studies. We could not control the order at which participants looked at or filled out packet components. It is possible that participants 'justified' their behavior in the game with their questionnaire answers. However, we might then expect a robust positive effect of value £1 on theft. Presenting Player 1s with the threat of punishment for theft could have decreased intrinsic motivation to behave cooperatively (Frey & Jegen, 2001), although it is unclear how this would produce a spurious correlation between *perceived cheating* and *theft* in the game. We cannot account for the neighborhood residents who chose not to respond, although in both neighborhoods we likely reached a segment of the community biased towards prosocial preferences (registered voters and research participants). Additionally, although participants were anonymous to each other in the game, they were not anonymous to us. The neighborhood differences in game behavior we observed could be partly attributed to participants in Neighborhood A, but not Neighborhood B, regarding a university professor as someone in their social milieu and thus being concerned about reputational repercussions.

Finally, we have two related suggestions for future study that may increase our understanding of why some communities appear to be stuck at uncooperative equilibria, despite concerted efforts by city planners to chart a different course (*Robinson, 2005*), or even substantial temporal changes in the demographic makeup (*Shaw & McKay, 1942*). The first is further investigation of the potential for multiple, simultaneous paths of positive feedback on cooperative norm violation, including not just conditional cooperation but also punitiveness and expectation of punishment. The second is consideration of how psychological adaptations for

recognizing and adopting local norms, as well as biased inand out-migration (*Chudek & Henrich, 2011*), can reinforce an antisocial culture.

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# **Competing Interests**

The authors declare there are no competing interests.

# **Author Contributions**

- Kari Britt Schroeder conceived and designed the experiments, performed the experiments, analyzed the data, wrote the paper, prepared figures and/or tables, reviewed drafts of the paper.
- Gillian V. Pepper and Daniel Nettle conceived and designed the experiments, performed the experiments, reviewed drafts of the paper.

# **Human Ethics**

The following information was supplied relating to ethical approvals (i.e., approving body and any reference numbers):

The Ethics Committee of the Newcastle University Faculty of Medical Sciences approved the study protocol: Protocol #00503/2011 and Amendment #00503  $_{-}$  1/2012.

# **Supplemental Information**

Supplemental information for this article can be found online at http://dx.doi.org/ 10.7717/peerj.450.

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# 9.3 Nettle, Pepper, Jobling & Schroeder (2014)

# Being there: a brief visit to a neighbourhood induces the social attitudes of that neighbourhood

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# ABSTRACT

There are differences between human groups in social behaviours and the attitudes that underlie them, such as trust. However, the psychological mechanisms that produce and reproduce this variation are not well understood. In particular, it is not clear whether assimilation to the social culture of a group requires lengthy socialization within that group, or can be more rapidly and reversibly evoked by exposure to the group's environment and the behaviour of its members. Here, we report the results of a two-part study in two neighbourhoods of a British city, one economically deprived with relatively high crime, and the other affluent and lower in crime. In the first part of the study, we surveyed residents and found that the residents of the deprived neighbourhood had lower levels of social trust and higher levels of paranoia than the residents of the affluent neighbourhood. In the second part, we experimentally transported student volunteers who resided in neither neighbourhood to one or the other, and had them walk around delivering questionnaires to houses. We surveyed their trust and paranoia, and found significant differences according to which neighbourhood they had been sent to. The differences in the visitors mirrored the differences seen in the residents, with visitors to the deprived neighbourhood reporting lower social trust and higher paranoia than visitors to the affluent one. The magnitudes of the neighbourhood differences in the visitors, who only spent up to 45 min in the locations, were nearly as great as the magnitudes of those amongst the residents. We discuss the relevance of our findings to differential psychology, neighbourhood effects on social outcomes, and models of cultural evolution.

Subjects Anthropology, Epidemiology, Psychiatry and Psychology, Public Health Keywords Neighbourhood effects, Paranoia, Trust, Cultural evolution, Social disorder, Mental health, Social capital

## **INTRODUCTION**

There are substantial differences between human groups in social behaviours and the attitudes that underlie them. Much of the literature demonstrating these differences has compared different ethnic or national groups (e.g., *Gachter & Herrmann, 2009; Henrichet al., 2005; Henrich et al., 2010; Herrmann, Thoni & Gachter, 2008*). However, differences at a much smaller scale, such as villages within one ethnic population or neighbourhoods within one city, can be equally marked (*Falk & Zehnder, 2007; Gurven, Zanolini & Schniter 2008; Lamba & Mace, 2011; Nettle, Colleony & Cockerill, 2011; Wilson, O'Brien & Sesma, 2009*). Whilst these observations are relatively novel, they are conceptually related to what can broadly be termed neighbourhood effects, which have been intensely studied in social science for several decades. The literature on neighbourhood effects is concerned with the consequences of the features of the immediately surrounding ecology for outcomes such as criminality, violent conduct, antisocial behaviour, trust, paranoia, and depression, which are clearly related to social behaviour (see *Aneshensel & Sucoff, 1996; Leventhal & Brooks-Gunn, 2000; Sampson, Morenoff & Gannon-Rowley, 2002; Sampson, Raudenbush & Earls, 1997*).

Previous research has ably described between-group differences, and established some of the ecological and economic correlates of different levels of pro- and anti-sociality. However, much less progress has been made in understanding the proximate mechanisms that produce (or reproduce) the behavioural and attitudinal differences within the individual. Prevalent proximate explanations for between-group differences invoke cultural transmission and social norms (*Henrich et al., 2010*). Such explanations are compelling, but merely invoking culture and norms is not in itself an explanation of how individuals acquire them. The psychological mechanisms involved need to be identified (Chudek & Henrich, 2011). Acquisition of local attitudinal patterns might involve lengthy socialization through childhood, followed by relative intra-individual stability, or attitudes could be updated dynamically throughout life according to current context. Explicit verbal instruction might be required. Alternatively or additionally, psychological mechanisms might respond to particular classes of subtle behavioural or physical cues that have, over evolutionary time, been reliably associated with social environments in which particular social behaviours are adaptive. Correlational studies are in general limited in their potential to be able to address these kinds of issues (see Henrich et al., 2012b; van Hoorn, 2012, for recent discussion).

Recent experimental work suggests that mechanisms for calibrating pro- and anti-social behaviours to the local socio-ecology remain highly plastic in adulthood, and are continuously updated using input from the current environment (*O'Brien & Wilson, 2011*). *Peysakhovich & Rand (2013)* showed that high- or low-cooperation behaviour could be readily induced amongst experimental volunteers by pre-exposing them to experience of cooperation or defection by others. The authors suggest that people develop heuristics of social cooperation based on experiences of social interaction from

their daily lives. These heuristics can be readily and continuously updated by new experience.

Direct personal interaction with others in an environment may not even be necessary to change social behaviour. In a series of field studies inspired by the 'broken windows' theory from criminology, Keizer, Lindenberg & Steg (2008) showed that experimentally introducing signs of social disorder, such as graffiti or littering, into the urban environment had remarkably large effects on the propensity of passers-by to litter, violate local rules, and even steal money. These effects were seen immediately, and crossed domains of behaviour; for example, observing that others had littered a public space increased the probability of stealing. Keizer, Lindenberg & Steg (2008, see also *Keizer, Lindenberg & Steq, 2013*) suggested that individuals have a psychological goal to behave well in the local social context (that is, to uphold norms that are generally agreed to be desirable for all parties). However, the strength of activation of this goal relative to their other goals depends on factors to do with the context and their state. In particular, they are motivated to uphold prosocial norms at cost to themselves only to the extent that others in the social environment are also motivated to do so. The environment provides cues of the motivation of others locally to uphold prosocial norms, in the form of their behaviour and its crystallized consequences in the landscape. These cues can include both disorder (perceptible consequences of others' not being motivated to uphold prosocial norms), and also order restoration (perceptible consequences of others expending effort in the service of upholding or restoring a prosocial norm). The results of the experimental interventions imply that people are very sensitive to these cues, and use them to continuously calibrate the strength of their own prosocial goals relative to other motivations.

Fessler and colleagues, using psychological priming paradigms, have suggested more specific mechanisms by which such continuous calibration may operate (Fessler & Holbrook, 2013; Schnall, Roper & Fessler, 2010). In particular, witnessing others upholding prosocial goals produces a specific emotion of elevation, which increases the subject's own prosocial motivation, whilst witnessing the opposite produces declination, a pessimism about others in general that decreases prosocial motivation. We can speculate that, in real-world environments, the continuous calibration via a diet of cues triggering elevation or declination results in a locally distinctive attitudinal stance towards other people in the environment. In social science, this stance is usually operationalized as trust, measured with a question such as 'To what extent do you think people in general can be trusted?' Trust measured in this way varies markedly between populations (Bond et al., 2004; Delhey & Newton, 2005; Knack & Keefer, 1997), is predictive of prosocial behaviours (Balliet & Van Lange, 2013; Gachter, Herrmann & Thoni, 2004), and relates to crime rates rates (Kennedy et al., 1998; Roh & Lee, 2013), and the functioning of social institutions (Knack, 2002). Low trust has several consequences. It can produce paranoia, a related and more extreme attitude involving the appraisal that others are trying to cause personal harm (*Mirowsky & Ross, 1983*). It directly reduces prosocial behaviour, thus leading to the creation of further environmental cues to which others will respond to by reducing *their* trust. It also reduces motivation to engage in acts of prosocial punishment or social control (*Schroeder, Pepper & Nettle, 2013*). Communities in which trust is low lack collective efficacy; that is, the capacity of their members to sanction those whose behaviour is antisocial (*Sampson, Raudenbush & Earls, 1997*), further exacerbating antisociality. Thus, a culture of low trust and low prosociality can become socially entrenched from small beginnings.

If, as suggested by the work described above, the mechanisms calibrating social attitudes remain highly plastic in adulthood, update rapidly, and respond to specific cues in the immediate environment, then people should assimilate to the culture of a population (in the sense of its locally distinctive social attitudes) very rapidly upon encountering it. We hypothesized that putting people temporarily into the environment inhabited by a population, thereby exposing them to the cues that result from the social behaviours of that population, would have a measurable effect on their social attitudes. This paper reports an experiment in which we attempted to test this hypothesis. The setting for our study was two different neighbourhoods within the city of Newcastle upon Tyne. These neighbourhoods have been the focus of ongoing fieldwork for several years (Nettle, 2012; Nettle, Colleony & Cockerill, 2011; Nettle, Coyne & Colleony, 2012; Schroeder, Pepper & Nettle, 2013). They are within a few kilometres of one another and are similar in many regards (size, population, population density, architectural layout, distance from city centre, approximate ethnic composition), but radically different in terms of socioeconomic fortunes. Whereas one neighbourhood (neighbourhood A) is economically thriving and has largely professional homeowner residents, the other (neighbourhood B) has suffered loss of economic activity, blight and continued uncertainty following the deindustrialisation of Newcastle beginning in the 1970s. Neighbourhood B is now classified by the UK government as within the 1% most deprived areas in England. It sustains a rate of crime that is twice that of neighbourhood A, and a rate of violent crime that is 6 times as high (see Nettle, Colleony & Cockerill, 2011, for more detail). We have previously found marked differences between the two neighbourhoods in terms of residents' play in Dictator, Theft and Third-Party Punishment economic games, and their likelihood of volunteering for a study or returning a lost letter on the pavement (Nettle, Colleony & Cockerill, 2011; Schroeder, Pepper & Nettle, 2013). There is, effectively, a large cultural difference between the two neighbourhoods in terms of pro- and anti-social behaviours and the attitudes that underlie them.

Our experiment had two parts. In the first part, the resident sample, we used our ongoing survey fieldwork amongst the residents to characterize the social attitudes of the residents of the two neighbourhoods. We did this by asking them questions about trust and paranoia. Trust, as previously mentioned, is widely studied in social research. It is generally held to be a central attitudinal variable relevant to the propensity towards prosociality and away from anti-sociality, both at the individual and community level (*Balliet*)
& Van Lange, 2013). In particular, it is trust in people in general (henceforth social trust), rather than trust in those one knows well (personal trust) that varies most amongst populations and best predicts prosocial outcomes (Uslaner, 2002). Paranoia is the belief that other people are actively trying to harm the subject. It is closely related, conceptually and empirically, to low trust, and has been previously found to be elevated in deprived socioeconomic groups (*Mirowsky & Ross, 1983; Ross, Mirowsky & Pribesh, 2001*). Paranoia is also related to persecutory symptoms of psychosis that are elevated in dense urban environments (*van Os et al., 2001*), and amongst psychotic patients, paranoia can be experimentally exacerbated by a short walk in such an environment (*Ellett, Freeman & Garety, 2008*). We predicted that social trust would be lower, and paranoia higher, amongst residents of neighbourhood B than neighbourhood A.

The second part of our experiment (the visitor sample) tested our main hypothesis regarding assimilation to the social attitudes of a neighbourhood by brief exposure to it. As described below, we randomly assigned a sample of student volunteers to be transported to one or other of the two neighbourhoods, where they completed an urban walk, under the guise of delivering surveys to the houses of the residents. They too completed measures of social and personal trust, and paranoia. We predicted (1) that there would be an effect of which neighbourhood the volunteer had been sent to on their trust and paranoia scores; and (2) that these differences would mirror the pattern of differences between the residents of the two neighbourhoods. If these predictions were met, we would have effectively induced a temporary version of the difference in social attitudes between the residents of the two neighbourhoods by exposure to the cues to which the residents are exposed.

#### **METHODS**

#### **Ethics statement**

All work reported in this paper was approved by the Faculty of Medical Sciences Research Ethics Committee, Newcastle University.

#### Data availability

The raw data from residents and visitors are downloadable as Supporting Information.

#### **Study sites**

Our research was based in the two neighbourhoods, A and B, within the city of Newcastle upon Tyne, Northeast England, that have been described fully in previous papers (*Nettle, 2012; Nettle, Colleony & Cockerill, 2011*). For this study, the boundaries of neighbourhood B were enlarged slightly compared to our previous work, due to a desire to avoid repeatedly sampling the same residents in surveys. The area into which the expansion occurred is socially similar to the core of neighbourhood B.

#### **Resident sample**

Between July 2012 and June 2013, we used the city's electoral roll to address questionnaires and accompanying letters to randomly chosen residents of each neighbourhood. These were longer questionnaires that formed part of our ongoing fieldwork and which contained measures that are reported elsewhere (*Schroeder, Pepper & Nettle, 2013*), as well as the two trust measures used in the current study (see *Measures* below). Residents returned the questionnaires by post, and received £5 in cash as a participation incentive, which was hand-delivered to their houses. From April to June 2013, we modified the resident questionnaire to contain, as well as the trust measures, a measure of paranoia (see *Measures* below). Response rates were approximately 24% in neighbourhood A and 17% in neighbourhood B. Respondents' geographical origin was established by asking for the post-code or city in which they had resided at age 10. The total resident sample reported here consisted of 259 responses for trust only, and a further 65 for paranoia and trust.

#### Visitor sample

In October and November 2012 and April and May 2013, we recruited 52 student volunteers from Newcastle University to visit the two neighbourhoods and post questionnaires through letterboxes of designated resident addresses. They received £5 or course credit for participation, and were aware that they were taking part in an experiment, though not aware of its exact hypothesis. Volunteers did not reside in either neighbourhood and neither neighbourhood was referred to by name to at any point in the session. Their geographical origin was established by asking for the postcode or city in which they had resided at age 10. On arrival at a rendezvous point on the university campus, participants were randomly assigned to be sent to one neighbourhood or the other. They were then taken in groups of 1-4 in a minibus or taxi, with at least one experimenter, to a drop-off point in the neighbourhood, where they were deposited with a packet of questionnaires, a list of resident addresses and a personalised map. They were instructed to find the addresses on foot and deliver the questionnaires, and then return to the waiting vehicle. Participants in the same vehicle set off from the drop-off separately, and were instructed to return after 45 min even if they had not successfully found all target addresses. The time away from the vehicle was 10–48 min (mean  $\pm$  sd 30.39  $\pm$  11.47; precise times were not recorded for the first 14 participants but were not more than 45 min). On return to the waiting vehicle, participants were asked to write down two open-ended comments about the neighbourhood they had just visited. Their answers were prompted as follows. "We would like to know what you thought of the neighbourhood you have been delivering questionnaires in. Please write about two things that seemed important about the neighbourhood. Please tell us why you chose these things". They were then handed a questionnaire to fill in, ostensibly as part of a separate study. This questionnaire included the measures of trust and paranoia (see Measures below), and a general measure of mood. After completing the questionnaire, they were debriefed and the vehicle returned them to the rendezvous point.

#### Measures

Our main outcome measures were identical for the resident and visitor samples. In accordance with much previous trust research, we measured each kind of trust with a single item. For social trust, the question was 'How much do you trust people you meet for the first time?', whilst for personal trust it was 'How much do you trust people you know personally?' The response scale varied from 1 to 10 in each case. For paranoia, we used the conviction subscale of the paranoia checklist from *Freeman et al. (2005)*. This consists of 18 items and is designed to measure paranoid symptoms in non-clinical samples. Cronbach's  $\alpha$  for the paranoia measure was 0.88 in the resident sample and 0.87 in the visitor sample. Visitors additionally rated their current mood on a 10-point scale. The trust and paranoia measures referred to how participants were in their life in general, and for the visitors, made no reference at all to their immediate acute experience, the neighbourhood they just visited, or how they would hypothetically feel if they lived there. The experience they had just had was not alluded to in the questionnaire.

#### Analysis strategy

All analysis was carried out in SPSS version 19 with a uniform  $\alpha$ -value of 0.05 for statistical significance. We had three outcome variables, personal trust, social trust and paranoia. Where there are multiple dependent variables within the same experiment, it is desirable to use a single MANOVA for statistical inference, rather than several ANOVAs, in order to minimize multiple testing. For the resident data, it was unfortunately not possible to use

a single MANOVA, since we had social and personal trust scores for 323 and 324 residents respectively, but paranoia scores for only a subset of 65. We therefore conducted separate ANOVA analyses for each outcome variable. In each case, we first performed an ANOVA with neighbourhood as the sole independent variable (henceforth, the simple model). Subsequently we ran a model containing neighbourhood plus sex, age, and – since being in a local minority is associated with paranoid symptoms (*Halpern*, *1993*) – local origin and the neighbourhood by local origin interaction. In the results section, we refer to this as the adjusted model.

For the visitor data, all three outcome measures were taken from the same set of 52 people, so we were able to use a MANOVA to test for an effect of neighbourhood on the set of three measures. Again, a first simple model contained neighbourhood as the sole predictor, whilst a second model adjusted for age and sex. We could not adjust for local origin, since all but one of our visitor participants grew up outside the Newcastle area.

We coded each of the open-ended comments made by the visitors before completing the questionnaire as a basically positive (+), basically negative (-) or unclassifiable (0)

reaction to the neighbourhood environment. We thence gave each participant a reaction score, which varied from -2 (two negative comments) to +2 (two positive comments). To establish whether it was the participant's reaction to the environment they had walked through that was driving any neighbourhood effects on trust and paranoia, we ran additional MANOVA analyses using reaction score as a dependent variable. Finally, for each variable in each neighbourhood, we tested whether the visitor means differed significantly from the estimated marginal means for the residents from the adjusted model. This was done using one-sample *t*-tests.

#### RESULTS

#### Trust and paranoia amongst residents

In the resident sample, social trust and personal trust were moderately positively correlated ( $r_{323} = 0.43$ , p < 0.01). The correlations of the two trust measures with paranoia, though negative, were not significant (social trust:  $r_{65} = -0.06$ , p = 0.62; personal trust:  $r_{64} = -0.22$ , p = 0.09).

For social trust, there was a significant neighbourhood difference in the simple model ( $F_{1,322}$  = 45.48, p < 0.01; means±se: Neighbourhood A 5.00±0.15, Neighbourhood

B 3.53±0.16), with trust approximately 0.7 pooled standard deviations higher in Neighbourhood A than B. The neighbourhood difference remained significant in the adjusted model ( $F_{1,308}$  = 29.41, p < 0.01; estimated marginal means±se: Neighbourhood A 4.95±0.16, Neighbourhood B 3.58±0.20). No other effects approached statistical significance in the adjusted model.

For personal trust, there was a significant neighbourhood effect in the simple model ( $F_{1,321} = 13.18$ , p < 0.01; means±se: Neighbourhood A 8.61±0.09, Neighbourhood B 7.97±0.15). This represents a difference of approximately 0.4 pooled standard deviations, with personal trust higher in neighbourhood A. Again, the neighbourhood difference remained significant in the adjusted model ( $F_{1,307} = 9.29$ , p < 0.01; estimated marginal means±se: Neighbourhood A 8.60±0.13, Neighbourhood B 7.98±0.16). No other effects approached significance in the adjusted model.

For paranoia, there was no significant neighbourhood difference in the simple model ( $F_{1,63} = 0.001$ , p = 0.97; means±se: Neighbourhood A 25.14±1.21, Neighbourhood B 25.21±1.58). However, in the adjusted model, the effect of neighbourhood was significant, with neighbourhood B having higher paranoia once age, sex and local origin are controlled for ( $F_{1,56} = 4.46$ , p = 0.04; estimated marginal means±se: Neighbourhood A 24.77±1.31, Neighbourhood B 30.57±2.38). The neighbourhood difference in marginal means in the adjusted model represents approximately 0.7 pooled standard deviations. None of the other effects in the adjusted model was statistically significant, although there were marginally non-significant trends for effects of sex ( $F_{1,56} = 3.81$ , p = 0.06, males higher, estimated marginal means±se: M 29.68±1.79, F 25.66±1.59) and local origin ( $F_{1,56} = 3.64$ , p = 0.06, non-locals higher, estimated marginal means±se: local 25.12±1.22, non-

local 30.22±2.38). Figure 1A summarises the resident neighbourhood differences in the three outcome variables.





#### Trust and paranoia amongst visitors

In the visitor data, social trust and personal trust were moderately positively correlated with each other ( $r_{51} = 0.58$ , p < 0.01), and showed significant or marginal negative correlations with paranoia (social trust:  $r_{51} = -0.30$ , p = 0.03; personal trust:  $r_{51} = -0.27$ , p = 0.06). Time away from the vehicle was not significantly correlated with any of the trust and paranoia measures (social trust:  $r_{37} = -0.02$ , p = 0.91; personal trust:  $r_{37} = 0.29$ , p = 0.09, paranoia:  $r_{38} = -0.10$ , p = 0.57).

In the simple MANOVA, there was a significant effect of neighbourhood visited ( $F_{3,47}$  = 3.68, p = 0.02, Wilk's  $\lambda$  = 0.81). The neighbourhood effect was driven by a substantial neighbourhood-visited difference in social trust (means±se: Neighbourhood A 4.73±0.46, Neighbourhood B 3.68±0.37; difference equates to 0.5 pooled standard deviations), with visitors to neighbourhood A having the higher social trust. There was a small neighbourhood difference in personal trust, with the higher mean actually found in visitors to neighbourhood B (means±se: Neighbourhood A 7.62±0.40, Neighbourhood B 7.96±0.27; 0.2 pooled standard deviations). We found a substantial difference in paranoia, with paranoia scores being higher in visitors to Neighbourhood B than in visitors to Neighbourhood A (means±se: Neighbourhood A 26.11±1.04, Neighbourhood B 29.64±1.76; 0.5 pooled standard deviations). It should be noted that none of the outcome variables considered in isolation shows a significant neighbourhood difference on an

ANOVA (respectively,  $F_{1,49}$ = 3.16, p = 0.08;  $F_{1,49}$ = 0.50, p = 0.48;  $F_{1,50}$ = 3.08, p = 0.09). Nonetheless, the significance of the MANOVA confirms that the effect of neighbourhood

visited on the set of outcomes taken together is statistically significant by conventional criteria.

The adjusted model did not change the significance or magnitude of the neighbourhoodvisited effect ( $F_{3,45}$  = 3.55, p = 0.02, Wilk's  $\lambda$  = 0.81; adjusted marginal means very similar to unadjusted means), and the effects of sex and age were not significant. However, in the visitor sample the age range was limited (18–24) and the sex ratio highly unbalanced (10 male, 42 female), so power to detect age and sex effects was low. Means for social and personal trust were similar between the two sexes (means±se: social trust, M 4.10±0.55, F 4.24±0.35; personal trust, M 8.20±0.47, F 7.68±0.28). Mean paranoia was somewhat higher for the male than female visitor participants, in line with the trend for the residents (means±se: M 30.20±1.50, F 27.24±1.21).

The visitor neighbourhood differences are summarised in Fig. 1B. Visitors to neighbourhoods A and B did not differ in self-rated mood after completing their deliveries (means±se: Neighbourhood A 7.12±0.38, Neighbourhood B 7.16±0.39;  $t_{49}$  = 0.08, p = 0.93).

#### Visitor reaction scores

The open-ended comments given by the visitors to neighbourhood A were uniformly positive (all participants' scores 2). The comments of visitors to neighbourhood B were much more variable (mean 0.24, s.d. 1.67, range –2 to 2). The reaction score difference between the neighbourhoods was significant ( $t_{24}$  = 5.29, p < 0.01). In a MANOVA with the trust and paranoia measures as dependent variables and reaction score as the independent, the effect of reaction score was significant ( $F_{3,47}$  = 3.43, p = 0.02, Wilk's  $\lambda$  = 0.82). When both reaction score and neighbourhood visited were entered in the same MANOVA, the effect of neighbourhood visited was no longer significant ( $F_{3,46}$  = 2.33, p = 0.09, Wilk's  $\lambda$  = 0.87), though reaction score also missed statistical significance ( $F_{3,46}$  = 2.56, p = 0.07, Wilk's  $\lambda$  = 0.86).

#### Relationship of visitor responses to the responses of the local residents

To facilitate the direct comparison of residents and visitors for each of the outcome variables, Fig. 2 replots the data from Fig. 1, but with data from residents of and visitors to each neighbourhood shown directly adjacent. To formally compare residents and visitors, we conducted a series of one-sample *t*-tests comparing the trust and paranoia levels of visitors to each neighbourhood with the trust and paranoia levels of the residents of that neighbourhood. The results of these are given in Table 1. For social trust and paranoia, the pattern is extremely clear: the visitors to a neighbourhood were not significantly different from the residents of the other neighbourhood (the one they did not visit). For personal trust, the pattern was different. Visitors to either neighbourhood had significantly lower personal trust than the residents of neighbourhood A, and did not differ significantly from the residents of neighbourhood B.





#### DISCUSSION

In the first part of our study, we characterized the social attitudes of our two study neighbourhoods using a survey of residents that included measures of trust and paranoia. In accordance with our expectations from previous literature and known facts concerning the socioeconomic context and crime rates, we found that people living in neighbourhood B trusted significantly less, and were significantly more paranoid, compared to people living in neighbourhood A. The neighbourhood effect was larger for social trust than personal trust, and for paranoia it was only detectable once sex, age and local origin had been adjusted for. For none of the outcome variables were sex, age or local origin themselves significant predictors, though, suggesting that we might be detecting consequences of living in the neighbourhood environment, rather than compositional differences – for example of age or ethnic background – between the two populations.

In the second part of the study, we randomly assigned student volunteers to be transported to one or the other neighbourhood and walk around distributing questionnaires to houses. Our prediction (1) was that there would be significant differences in trust and paranoia according to which neighbourhood the participant had been sent to. This prediction was met, with a significant neighbourhood effect on the set of three outcome variables, albeit that none significantly differed between the neighbourhoods when considered in isolation. Our prediction (2) was that the neighbourhood differences amongst the visitors would mirror those seen amongst the residents. This prediction was supported for social trust and paranoia, where the visitor differences were of the same direction and approximately the same magnitude as the differences found amongst the residents. For these two variables, visitors to a neighbourhood did not differ significantly from the residents of that neighbourhood, but did differ significantly from the residents of the other neighbourhood. Thus, for social trust and paranoia, we had effectively induced the attitudinal difference between people in neighbourhood A and those in neighbourhood B through an urban walk lasting 45 min or less. The prediction was not met for personal trust, which was the variable showing the smallest difference amongst the residents. This is comprehensible in retrospect; we had not manipulated participants' experience with people they knew well, and so there is no reason that the experimental treatment should have any effect on their trust in those people.

There were no significant differences in general mood between visitors who had been to one neighbourhood and those who had been to the other. However, there were marked differences in their gualitative comments about the neighbourhoods, with the comments uniformly positive in neighbourhood A and more mixed in neighbourhood B. There was some evidence that people's qualitative appraisal of the environment was a mediator of the neighbourhood difference in trust and paranoia, but the strong multicollinearity between neighbourhood and reaction score made this difficult to demonstrate statistically. These findings thus suggest, in accordance with the findings of other recent studies (Fessler & Holbrook, 2013; Keizer, Lindenberg & Steg, 2008; Keizer, Lindenberg & Steq, 2013; O'Brien & Wilson, 2011; Peysakhovich & Rand, 2013; Schnall, Roper & Fessler, 2010), that the mechanisms regulating social attitudes (and thence behaviours) are highly plastic in adulthood, and can be influenced by cues from the surrounding environment in real time. We believe these findings to have important implications for three areas of research in particular, research in differential psychology, research on neighbourhood effects, and research on cultural evolution.

Table 1 Results of one-sample *t*-tests comparing the trust and paranoia of the visitors to each neighbourhood to those of the residents of the two neighbourhoods. Statistically significant differences are underlined. The resident means are marginal means from the model adjusting for age, sex and local origin.

v	isitors to	Neighbourhood A		Neighbourhood B
		Social trust		
		Neighbourhood A	<i>t</i> <sub>25</sub> = 0.48, <i>p</i> = 0.64	t <sub>25</sub> = 2.53, <i>p</i> = 0.02
		Neighbourhood B	t24= 3.41, <i>p</i> < 0.01	t <sub>24</sub> = 0.27, p = 0.79
		Personal trust		
		Neighbourhood A	<i>t</i> <sub>25</sub> = 2.46, <i>p</i> = 0.02	<i>t</i> <sub>25</sub> = 0.91, <i>p</i> = 0.37
		Neighbourhood B	t24= 2.34, p = 0.03	t <sub>24</sub> = 0.07, p = 0.94
		Paranoia		
		Neighbourhood A	t <sub>26</sub> = 1.29, <i>p</i> = 0.21	t <sub>26</sub> = 4.27, <i>p</i> < 0.01
		Neighbourhood B	t <sub>24</sub> = 2.77, p = 0.01	t <sub>24</sub> = 0.53, <i>p</i> = 0.60

Compared to residents' mean of...

#### Implications for differential psychology

Within differential psychology, there is a long-standing debate about the extent to which psychological characteristics should be seen as trait-like rather than immediately situation-driven (Fleeson, 2004). When social factors are shown to be associated with psychological characteristics, the causal nexus is often assumed to be an irreversible developmental effect (e.g., *McCullough et al., 2013*). The results of this study suggest, however, that trust and paranoia are subject to immediate contextual influence in adulthood, supporting the general importance of current situational variables in driving social behaviours (Zimbardo, 2007). Thus, to explain associations between social deprivation or environmental harshness and behaviour, we may need to consider not just irreversible developmental effects, but also people's ongoing 'diet' of exposure to particular current contextual cues (Nettle, Coyne & Colleony, 2012). This is the process that Buss & Greiling (1999) refer to as *enduring situational evocation*. Individuals might be quite stable in their trust and paranoia if measured repeatedly over time, but this could simply mean that their exposure to the triggering cues occurs continually. It does not mean that their trust and paranoia would not change if their environment changed.

A number of other recent studies have reached similar conclusions about plasticity in psychological characteristics related to environmental adversity or unpredictability. *Mani et al. (2013)* investigated the hypothesis that poverty causes poorer cognitive performance. In an experimental study, they showed that people with lower incomes showed poorer cognitive performance than people with higher incomes only when their financial problems were made salient. When financial problems were not salient, there was no difference between the groups. In a related observational study of poor farmers, Mani et al. showed within individuals that cognitive performance declined when money was scarce, and improved again with the harvest when money became available. *Kidd, Palmeri & Aslin (2013)* studied a classic 'delay of gratification' task where children choose between one marshmallow immediately or two after a delay. Variation in performance on this task has been attributed to trait-like differences in self-control. Kidd et al. showed experimentally that giving children an immediate cue that the experimenter was unreliable caused a large reduction in the time the child was able to wait for gratification. Thus, if children from certain social groups show reduced delay of gratification, this may be because they are chronically exposed to cues of unreliability, rather than because their delay of gratification is fixed.

These studies mean that demonstrating differences between groups of people on some characteristic does not mean that those differences are not plastic within each individual, even if they are shown to be stable over time. Cross-sectional studies that purport to show, for example, that a particular social group has low social trust, only really show that people currently in that environment report low social trust. They do not in themselves justify any inference about what those participants would be like if they migrated elsewhere, their state changed, or their public environment was altered. To be clear, we are not claiming that a person's long-term developmental and cultural history leave no stably internalized influences on social attitudes. It is likely that they do, and indeed, some of the variability in the responses of our samples may well be explained by such influences. We merely wish to draw attention to the relatively strong effects of current situation, and make the methodological point that cross-sectional surveys cannot be used as evidence about how labile social attitudes are within the individual, or what the psychological mechanisms maintaining those attitudes are.

#### Implications for neighbourhood effects

Neighbourhood effects – associations between neighbourhood characteristics and individual-level outcomes such as health, wellbeing and prosociality – are widely studied in social science, and there are a vast number of correlational studies suggesting their importance (*Aneshensel & Sucoff, 1996; Leventhal & Brooks-Gunn, 2000; Pickett & Pearl, 2001; Sampson, Morenoff & Gannon-Rowley, 2002; Sampson, Raudenbush & Earls, 1997*).

However, the principal challenge with these studies is demonstrating causality (*Sampson, Morenoff & Gannon-Rowley, 2002*). That is, it is hard to exclude the possibility that people who at the outset have poor health or antisocial tendencies are differentially likely to end up in certain neighbourhoods, rather than the neighbourhood environment causing poor health or antisocial tendencies. Researchers have appreciated that the experimental method is what is required to demonstrate causality (*Sampson, Morenoff & Gannon-Rowley, 2002*). The (quasi-) experimental designs typically used involve permanent mobility from one type of environment to another (*Katz, Kling & Liebman, 2001; Kling, Liebman & Katz, 2007*). There has been much less consideration of the fact that the changes induced by living in a neighbourhood might become manifest in real time, and so, much easier and briefer experiments can also be of interest. Spending 45 min or less in a neighbourhood knowing that there is a vehicle waiting that will take one away is not of course the same as living there. Nonetheless, the fact that social trust and

paranoia were so similar for residents of and visitors to a neighbourhood is striking. If a short visit is sufficient to induce detectably lowered trust and heightened paranoia, then how much more powerful must be the effects of living in the place every day? Trust is related to physical and mental health, crime rates, and other social indicators (De Silva et al., 2005; Kawachi, Kennedy & Glass, 1999; Kawachi et al., 1997; Kennedy et al., 1998), whilst paranoia is a clinical psychiatric construct (Freeman et al., 2005), so the outcomes that were affected by our experiment are important for long-term social and health outcomes. Thus, our results tend to support the view that neighbourhood effects are not only causal, but powerful and very rapidly acting. This means that disorder can spread very fast (Keizer, Lindenberg & Steg, 2008), but it does also imply, hopefully, that some of the negative impacts of an environment might be relatively rapid to reverse if environments can be improved (see Keizer, Lindenberg & Steg, 2013). Thus, apparently stable negative consequences of living in a particular environment might actually be labile, adaptively-patterned responses that could quickly change with appropriate social intervention.

#### Implications for models of cultural evolution

The social attitudes found in particular populations are generally thought of as culturally transmitted (Henrich et al., 2012a; Henrich et al., 2012b; Henrich et al., 2010; Uslaner, 2002). Cultural transmission has been conceptualized as a Darwinian evolutionary process, with the most important change arising through processes analogous to mutation and natural selection (Mesoudi, Whiten & Laland, 2006, though see Claidi'ere & Andr'e, 2012). In simple models of cultural evolution, cultural transmission is modelled as occurring once in each lifetime, presumably through socialization in childhood (Boyd & Richerson, 1985). Thereafter, the individual's cultural traits are fixed and serve as input to the next generation. This maximizes the analogy with genetic evolution. However, our data and that in the other studies reviewed above suggests greater plasticity and lability than such models allow for: social attitudes are continuously updated in adulthood in response to very recent experience. This means that the dynamics of cultural change will be quite different from those of genetic evolution, with cultural patterns able to bloom and fade rapidly in periods much shorter than a generation (Strimling, Enguist & Eriksson, 2009). Darwinian processes of inheritance and selection are not such an appropriate framework for examining this kind of process. Instead, we need bespoke models of cultural dynamics that are built around the actual psychological processes involved in transmission of social attitudes from one person to another, including their intra-individual plasticity. What is needed is to understand the cultural transmission of social behaviours is an empiricallyinformed 'epidemiology of representations' (Sperber, 1985).

#### Limitations and future directions

Our study had a number of important limitations that should be noted, and future work should seek to overcome these. Our key comparisons in the visitor sample were between subjects. Because of this, we were not able to determine whether individual visitors to neighbourhood A became more trusting as a result of their visit, visitors to neighbourhood B became less trusting, or both. Our methodology also provides no information about which cues are important in explaining the observed effect. We see it is as a proof of principle that being in an environment induces the social attitudes of that environment. Future work using different methodologies will be needed to isolate which cues or interactions are causally important in producing the effect. For example, *Hill, Pollet & Nettle (2013)* showed experimental volunteers slideshows of street scenes from neighbourhoods A and B, with police presence either prominent or absent in the slideshows. They found that perceptions of safety and social support were lower for neighbourhood B than A, and police visibility had no effect at all. This implies that the high-visibility policing that is a feature of life in neighbourhood B (*Nettle, Colleony & Cockerill, 2011*) is not one of the main cues people use to calibrate their social perceptions.

Another limitation of our methodology is that it provides a one-off snapshot of the consequences of being in a neighbourhood. We were not able in this experiment to determine the time course of the effects, or establish what would happen with repeated exposure. Although social trust and paranoia were very similar in residents of and visitors to a neighbourhood, the mechanisms producing the differences in the residents may not be exactly the same ones producing the differences in the visitors (though they could be). For example, cues of disorder are very powerful in driving short-term responses (*Keizer, Lindenberg & Steg, 2008*; O'Brien & Wilson, 2011), but it has been suggested that in the longer term, personal social relationships become more important (O'Brien & Kauffman, 2013). In our data, residents of neighbourhood B showed relatively lowered personal trust, whereas the personal trust of visitors to neighbourhood B was not lowered by their visit. This suggests long-term consequences of living in a neighbourhood that are more than just the immediate visitor reaction. Thus, future work will need to tease out the ways different influences may become more or less important with repeated exposure.

Our resident samples were not representative of the two communities, since only small minorities responded to our surveys. This is hard to avoid in this kind of research, and its consequences are difficult to infer; we may for example have underestimated the true effect size of the neighbourhood differences, if the least trusting and most paranoid residents of neighbourhood B were least likely to respond. There are also important covariate variables that we lacked. We did not know for example how many participants were substance users or had a diagnosed mental illness, and this could have been relevant to understanding variation in paranoia. As for our visitor sample, here we also lacked the sample size and range of measures to assess factors that might have accounted for variation in the response to the neighbourhood, such as cultural and socioeconomic background, and initial level of trust. The visitor sample also had few males, hampering inference about sex differences in attitudes and responsiveness. However, amongst the residents, the only sex difference of note was a near-significant trend for males to have higher paranoia. This is an expected finding (Lewis, 1985), and the means amongst the visitors suggested the same pattern.

#### **CONCLUSIONS**

Our resident data revealed striking differences in trust and paranoia between people living in two different neighbourhoods. Had we stopped there, we would have assumed that these differences were stable within the individual, and, to the extent they were caused by the neighbourhood, arose from lengthy residence and socialization in those groups. The fact that groups of visitors who spent less than one hour in the neighbourhoods produced very similar patterns of trust and paranoia suggests that immediate contextual experience is relatively important in modulating social attitudes. This may mean that differences in social attitudes between individuals and between populations might be more labile and more context-dependent than previously thought.

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#### **Competing Interests**

The authors declare they have no competing interests.

#### **Author Contributions**

- Daniel Nettle conceived and designed the experiments, performed the experiments, analyzed the data, contributed reagents/materials/analysis tools, wrote the paper.
- Gillian V. Pepper and Kari Britt Schroeder conceived and designed the experiments, performed the experiments, contributed reagents/materials/analysis tools, revised the manuscript.
- Ruth Jobling performed the experiments, revised the manuscript.

#### **Human Ethics**

The following information was supplied relating to ethical approvals (i.e., approving body and any reference numbers):

The study was approved by the Faculty of Medical Sciences Ethics Committee, Newcastle University, under approvals 00325 and 00503.

#### **Supplemental Information**

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## 9.4 Questionnaire for Chapter 2, Pepper & Nettle (2013)

**Note:** Questions were presented online using the SocialSci platform. They are represented in plain text below.

#### Are you male or female?

Drop-down menu: Male Female Other

How old are you? Open text box

#### What is your annual income before tax?

Please enter your gross annual income (\$USD) in the box below. Open text box

### Do you have children?

Drop-down menu: Yes No

#### How old were you when your first child was born?

Please enter the age you were when you first started having children. Drop-down menu: 16:45

#### What would be your ideal age to start having children?

Please enter your ideal age to start having children. Drop-down menu: 16:45

#### Making choices about money

For each of the following choices, please indicate which of the two options you would take if you were offered the choice right now. This is a hypothetical question, but please answer as though it were a real situation.

Forced-choice check-box options:

\$100 in one year's time	\$1 today
\$100 in one year's time	\$2 today
\$100 in one year's time	\$3 today
\$100 in one year's time	\$5 today
\$100 in one year's time	\$10 today
\$100 in one year's time	\$20 today
\$100 in one year's time	\$30 today
\$100 in one year's time	\$40 today

\$100 in one year's time	\$45 today
\$100 in one year's time	\$50 today
\$100 in one year's time	\$55 today
\$100 in one year's time	\$60 today
\$100 in one year's time	\$65 today
\$100 in one year's time	\$70 today
\$100 in one year's time	\$75 today
\$100 in one year's time	\$80 today
\$100 in one year's time	\$85 today
\$100 in one year's time	\$90 today
\$100 in one year's time	\$95 today
\$100 in one year's time	\$99 today

#### You may consider the following questions to be of a personal nature.

If so, remember that you are free to continue without answering them.

#### Has anyone you know died in the past 5 years?

Check-box options: Yes No

How many people that you know have died in the past 5 years? Open text box

How many of those people did you feel you were very close to? Open text box

#### Money and you

Click to tell us how much you agree with each of the statements below, where 1 means "strongly disagree" and 7 means "strongly agree".

Forced-choice check-box options:

- 1: strongly disagree
- 2: disagree
- 3: slightly disagree
- 4: neutral
- 5: slightly agree
- 6: agree
- 7: strongly agree
  - I don't worry too much about paying my bills
  - I have enough money to buy things I want
  - I don't think I'll have to worry about money too much in the future

## 9.5 Questionnaire for faces studies (Chapter 3)

#### Welcome

"Welcome to the personality and perception study. Thank you for agreeing to take part. The aim of the study is to investigate whether people with different personalities perceive faces differently. There are two parts to the study. First, you will be asked to categorise 50 faces, which will appear one at a time on the screen. Then you will complete a short personality questionnaire, which will take around 5 minutes. The whole session should take no longer than 10 minutes to complete. Your participation is voluntary and you are free to withdraw from the study at any time without giving a reason. The electronic data captured during the study are anonymous and will be kept on password-protected computers. Participants will not be personally identifiable in any research papers arising from this study.

Please click ">>" to continue."

### Consent

"Please click next to each of the following statements to indicate that you understand them and wish to take part in the study. Then click ">>" to continue.

I understand that participation in this study is entirely voluntary and I can withdraw at any time without giving any reason and without any of my rights being affected. (1)

I understand that all electronic data created during the study will be stored in anonymised form on password-protected computers. (2)

I understand that all the information will be treated as confidential, and that I will not be personally identified in any way. (3)"

### **Practice item**

"In the first part of this study, you will be asked to drag and drop images into the appropriate boxes. This is a practice item. Click on the image with the left mouse button. Drag the image into the correct box using the mouse and then release the left mouse button. When the image is in the correct box, click ">>" to continue."

Animal Vegetable

Hedgehog image Hedgehog image

## Practice achieved

"Well done. You have mastered the drag and drop method. Please click ">>" to continue to the real task."

## Images

"Is this person male or female? Drag the image into the correct box."

Male Female

Image X Image X

Qualtrics continues to present another 49 faces in the same manner as outlined above. The faces will be from either the "younger faces" treatment or the "older faces" treatment. The treatment is randomly allocated and the order of image presentation is also randomized.

## Personality questionnaire

"Welcome to the personality questionnaire. Remember that any information you enter into this questionnaire is confidential and will be treated as anonymous. However, if you do not wish to answer a question, you are free to continue without doing so. Thank you.

Please click ">>" to continue."

The following questions are presented in a randomized order.

## Having children

"What would be your ideal age to start having children, if you were to do so?

\_\_\_\_\_ Age"

## Making choices about money

"For each of the following choices, please indicate which of the two options you would take if you were offered the choice right now."

Choice 1 (1)	£100 in one year's time, or (1)	£1 today (2)
Choice 2 (2)	£100 in one year's time, or (1)	£2 today (2)
Choice 3 (3)	£100 in one year's time, or (1)	£3 today (2)
Choice 4 (4)	£100 in one year's time, or (1)	£5 today (2)
Choice 5 (5)	£100 in one year's time, or (1)	£10 today (2)
Choice 6 (6)	£100 in one year's time, or (1)	£20 today (2)
Choice 7 (7)	£100 in one year's time, or (1)	£30 today (2)
Choice 8 (8)	£100 in one year's time, or (1)	£40 today (2)
Choice 9 (9)	£100 in one year's time, or (1)	£45 today (2)
Choice 10 (10)	£100 in one year's time, or (1)	£50 today (2)
Choice 11 (11)	£100 in one year's time, or (1)	£55 today (2)
Choice 12 (12)	£100 in one year's time, or (1)	£60 today (2)
Choice 13 (13)	£100 in one year's time, or (1)	£65 today (2)
Choice 14 (14)	£100 in one year's time, or (1)	£70 today (2)
Choice 15 (15)	£100 in one year's time, or (1)	£75 today (2)
Choice 16 (16)	£100 in one year's time, or (1)	£80 today (2)
Choice 17 (17)	£100 in one year's time, or (1)	£85 today (2)
Choice 18 (18)	£100 in one year's time, or (1)	£90 today (2)
Choice 19 (19)	£100 in one year's time, or (1)	£95 today (2)
Choice 20 (20)	£100 in one year's time, or (1)	£99 today (2)

## How long you might live

"What do you think the chances are that you will live to be 75 or more? (Answer on a 100-point scale where 0 is 'No chance' and 100 is 'Definitely')

\_\_\_\_\_ Move the slider with your mouse"

## Gender

"Are you male or female?

Male (1)

Female (2)"

Age

"How old are you?" \_\_\_\_\_

## Childhood postcode

"What was the postcode of the house you lived in when you were 15 years old? If you can't remember the postcode, please enter the name of the street you lived on and the town or city it was in." \_\_\_\_\_

## Demand characteristic check

"You have now finished the experiment. Thank you for taking part. Before you go, we would like to know if you guessed what this study was about. If you think you know what we were trying to measure, please write your idea in the box below. Then click '>>' to complete the study." \_\_\_\_\_

## Debriefing

"Thank you for participating in this study. Your answers have been recorded.

This study was about how people respond to cues to life expectancy. We know that people who live in areas where life expectancies are shorter have different attitudes to people who live in areas where life expectancy is long: For example, they differ in long they prefer to wait for rewards and when they choose to have children. It is possible that these differences in behaviour reflect a rational response to different environments. The images which you categorised as "male" or "female" will have come from one of two sets, chosen randomly by the computer programme. Your set of images will have contained either a higher proportion of older faces, or a lower proportion of older faces. We expect the number of older faces in the set you were given to have acted as a temporary "prime", which might have slightly altered your answers to the questionnaire.

If you want to know more about the rationale for this study you might wish to read the following paper:

Nettle, D. (2009). Social class through the evolutionary lens. *The Psychologist*, 22(11), 133-136.

If you have any questions about the study please email g.pepper@ncl.ac.uk"

Choice #	Future £ (A)	Now £ (V)	Delay in days (D)	k-parameter
1	100	1	365	0.271232877
2	100	2	365	0.134246575
3	100	3	365	0.088584475
4	100	5	365	0.052054795
5	100	10	365	0.024657534
6	100	20	365	0.010958904
7	100	30	365	0.006392694
8	100	40	365	0.004109589
9	100	45	365	0.003348554
10	100	50	365	0.002739726
11	100	55	365	0.002241594
12	100	60	365	0.001826484
13	100	65	365	0.001475237
14	100	70	365	0.001174168
15	100	75	365	0.000913242
16	100	80	365	0.000684932
17	100	85	365	0.000483481
18	100	90	365	0.000304414
19	100	95	365	0.000144196
20	100	99	365	0.000027674

9.6 Table of values for *k*, the indifference point for each choice in the set used as a measure of future discounting (Chapter 3).

		D	р	df
Ideal age at first birth	Older faces	0.172**	0.005	39
	Younger	0.168**	0.006	40
Future discounting	Older faces	0.150*	0.027	39
score	Younger	0.153*	0.020	40
Subjective life	Older faces	0.130	0.094	39
expectancy	Younger	0.171**	0.005	40

# 9.7 Table of Kolmogorov-Smirnov test values for outcome variables in (Chapter 3, study 1)

\*= p<.05. \*\*=p<.01

	F	df1	df2	р
Ideal age at first birth	0.216	3	54	0.885
Future discounting	2.281	3	54	0.090
Subjective life	0.206	3	54	0.892

9.8 Levene's Test of Equality of Error Variances for outcome variables (Chapter 3, study 1).

		D	р	df
Ideal age at first birth	Older faces	0.155**	0.006	47
	Younger faces	0.146	0.065	34
Future discounting	Older faces	0.172**	0.001	47
score	Younger faces	0.150	0.052	34
Subjective life	Older faces	0.158**	0.005	47
expectancy	Younger faces	0.192**	0.003	34

# 9.9 Table of Kolmogorov-Smirnov test values for outcome variables (Chapter 3, study 2).

\*= p<.05. \*\*=p<.01

	F	df1	df2	р
Ideal age at first birth	0.863	3	48	0.467
Future discounting	1.373	3	48	0.262
Subjective life	4.460*	3	48	0.008

# 9.10 Levene's Test of Equality of Error Variances for outcome variables (Chapter 3, study 2).

\*\*=p<.01

## 9.11 Experiments 1 & 2 – participant information, consent, questionnaire and debrief (Chapter 5)

## **Participant Information**

## The UK health behaviours study

Welcome to the UK health behaviours study. We want to understand why people in some parts of the UK live longer than others do. (For recent information about differences in life expectancies across the UK, please see this BBC news article.)

## What is involved?

We will ask you for your age, gender and location (postcode). Then we will ask you some questions about your health habits. You must be over 18 and living in the UK to take part. The whole survey should take no more than 5 minutes. Participation is voluntary and you are free to withdraw from the study at any time without giving a reason. The study has ethical approval from the Newcastle University Faculty of Medical Sciences (ref: 00554). If you have any concerns about the way the study was conducted please contact either Prof Daniel Nettle (daniel.nettle@ncl.ac.uk) or the ethics committee (fmsethics@newcastle.ac.uk).

## What happens to the information gathered?

The information recorded for the study is anonymous and will be kept on password-protected computers, or in a locked filing cabinet at the University. Participants will not be personally identifiable in any research papers arising from this study. We will not share your information with any third parties.

### **Questions?**

If you have any questions about the study please contact the lead researcher (below) by email or post.

Gillian Pepper, Henry Wellcome Building, Newcastle University, NE2 4HH

Email: g.pepper@ncl.ac.uk

Thank you!

## **Timing recorded**

First Click (1)

Last Click (2)

Page Submit (3)

Click Count (4)

## Consent

Please click next to each of the following statements to indicate that you understand them and wish to take part in the study. Then click ">>" to continue.

I have read and understood the information provided for the study and have seen the email address I can contact to ask questions about it. (1) I understand that participation in this study is entirely voluntary and I can withdraw at any time without giving any reason and without any of my rights being affected. (2) I understand that all electronic data created during the study will be stored in anonymised form on password-protected computers. (3) I understand that all the information will be treated as confidential, and that I will not be personally identified in any way. (4) I confirm that I wish to take part in the study. (5)

## Location information

Your IP address indicates that you are currently in Newcastle Upon Tyne. We only need people who live in the UK to complete our survey. However, we understand that you may be taking the survey whilst away from home. If you are not from the UK we can still generate a code so you get credit for trying to take part, but we will not be able to use your answers as part of our study. Do you currently live in the UK? Select your answer then click ">>" to continue.

Yes (1) No (2)

[If No Is Selected, Then Skip To End of Survey]

## **Demographic questions**

## How old are you?

[Drop-down menu, age options 18-100]

## Are you male or female? male (1) female (2)

## What is your current postcode?

[Text box]

## **Processing screen**

Thanks for submitting your information. It may take a while to match it to health data for people of your age and gender in your postcode area. Please wait a few moments. Thank you.

## **Timing recorded**

First Click (1)

Last Click (2)

Page Submit (3)

Click Count (4)

## [Random allocation to primes – see manuscript for prime text]

## **Timing recorded**

First Click (1)

Last Click (2)

Page Submit (3)

Click Count (4)

## Health intention questions

How much effort will you put into looking after your health and safety over the coming week? 0 means no effort at all, 100 means the maximum effort you could make.

[Sliding scale 0-100]

How likely is it that you will eat 5 portions of fruit or vegetables per day over the coming week? 0 means you definitely won't eat 5 portions of fruit and veg per day, 100 means you definitely will eat 5 portions of fruits or veg per day.

[Sliding scale 0-100]

How likely is it that you will do 30 minutes or more of physical exercise three times over the coming week? 0 means you definitely won't do three 30-minute blocks of exercise, 100 means you definitely will.

[Sliding scale 0-100]

How many units of alcohol are you likely to drink over the coming week. (One standard glass of wine or pint of lager is about 2.3 units.) If you are unsure about how many units of alcohol are in different drinks, you can calculate them here: NHS Units Calculator

[Text box]

## Prize draw question

Thanks for taking part in our study. To say thank you, we would like to enter you into a prize draw. There are two choices of prize available. Please select the prize you would prefer to win, then enter your email address or telephone number in the box below, so that we can contact you to organize delivery if you win.

Privacy: We will not use your contact details for any other purpose, or pass them to any third parties. Your contact details will be deleted from our records once the prize draw has taken place.

What would you prefer to win? A Riverford Farm Organic Fruit box worth £11. (1) A Thorntons Summer Chocolate Collection box worth £11. (2)

Contact If you win the prize draw, what is your preferred contact email address or phone number?

[Text box]

## End of survey debrief message

## The health behaviours study

Thanks for taking part in this study. If you are not living in the UK we could not include you in the study, but thanks for trying to take part. You will still be awarded credit.

Your validation code for Crowdflower is [insert code]

The statistics we presented to you about people in your area were false. We did not tell you the true purpose of the study because we wanted you to answer our questions as honestly as possible.

The experiment was actually about whether changing people's ideas about how long they might live alters their motivation to look after their health. You will have been told that people of your age, living in your area, are living longer than others in the UK, or that they are dying younger than others in the UK are. This was not real information.

We expect the experiment to have had a short-term influence on your motivation to look after your own health. However, this effect should not be long lasting. If you feel that you have been unduly affected by your experience of this study, please contact Prof Daniel Nettle on +44 (0)191 222 8993 or daniel.nettle@ncl.ac.uk

If you want to know more about our reasons for running study you might wish to read the following paper:

Nettle, D. (2010). Why are there social gradients in preventative health behavior? A perspective from behavioral ecology *PLoS ONE* 5(10): e13371: Link to paper PDF

If you have any questions about the study please email g.pepper@ncl.ac.uk

## 9.12 Participant location checks for experiments 1 and 2 (Chapter 5)

We needed to ensure that our participants were from the UK, because the primes were based on UK postcode statistics. Thus, when we requested participants through Crowdflower, we made UK residency a criterion. However, the Crowdflower system relied on participant honesty to ensure that they fulfilled this condition. Therefore, we took additional measures to ensure that the data that we used came from participants who had been honest about their location. After completing the consent form, participants were moved onto a location-check screen, which read "Your IP address indicates that you are currently in [LOCATION]." Qualtrics was programmed to present the location from which the participant was accessing the web page, based on their Internet Protocol address (IP address). The rest of the screen read, "We only need people who live in the UK to complete our survey. However, we understand that you may be taking the survey whilst away from home. If you are not from the UK we can still generate a code so you get credit for trying to take part, but we will not be able to use your answers as part of our study. Do you currently live in the UK?" Participants then had the option to click "Yes" or "No." Participants who clicked "no" were filtered straight to the debriefing screen and were given Crowdflower credit for their time. Participants who declared themselves to be from the UK were moved on to the rest of the experiment.

### Experiment 1 - exclusions based on location

We requested 100 responses through Crowdflower. However, we were compelled to exclude some of the data. A key element of the prime was that the mortality risk information was tailored to participants' postcodes. Therefore, we only used data for which we could be reasonably certain that the participants had entered their real postcode. We excluded data from our analysis if: 1) The participant's postcode was missing or was not a valid UK postcode, 2) the participant's IP address was not UK based, or 3) the participant IP address recorded by Crowdflower did not match that recorded by Qualtrics (indicating possible use of a proxy server or an attempt to take the same survey multiple times from different machines in order to get extra Crowdflower credit.) After these exclusions we were left with 72 responses.
## Experiment 2 - exclusions based on location

The exclusion criteria were the same as those used in experiment 1 (above). Since both experiments 1 and 2 used Crowdflower as a recruitment platform, we used both IP addresses and postcode, age and gender combinations to check that participants in experiment 2 had not previously taken part in experiment 1. No repeat participants were identified. After exclusions, we were left with a sample of 195 participants.

## 9.13 Prize draw cards used in experiment 3 (Chapter 5)

Personal details box – used in both treatments:



Controllable long life priming card:



## Newcastle Iniversity

Our question: Recent statistics show that people in Tyne and Wear are living longer now than they were in the year 2000. Why do you think this is?

- Because people have more control over the kind of healthcare they receive.
- Because people are looking after themselves better.
- Both: people have more control over their care and are looking after themselves better.

This study has received ethical approval from the Newcastle University Faculty of Medical Sciences ethics committee (reference: 00653/2013). By completing this card you are giving your consent for your answers to be used in our study. We will not share your contact details with anyone else. We will only use them to deliver your prize to you if you win.

Uncontrollable long life priming card:



The end 🙂