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

G. V. Pepper (✉) · D. Nettle
Centre for Behaviour and Evolution, Newcastle University,
Newcastle upon Tyne, Tyne and Wear, UK
e-mail: g.pepper@newcastle.ac.uk

M. A. Gibson, D. W. Lawson (eds.), *Applied Evolutionary Anthropology,* Advances in the Evolutionary Analysis of Human Behaviour, DOI 10.1007/978-1-4939-0280-4_10,

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 sci-

ences 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.

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

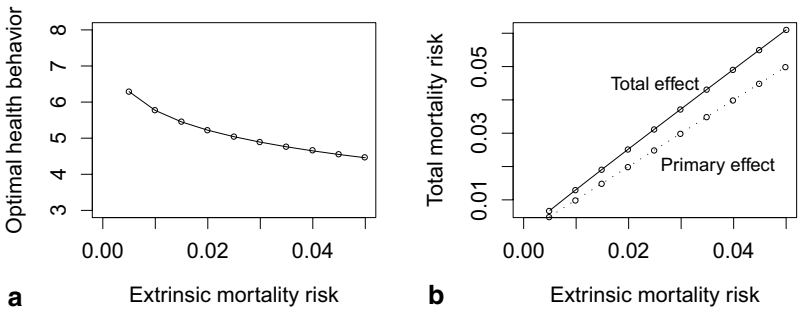

Fig. 10.1 Predictions from Nettle's (2010a) model. **a** As the risk of extrinsic mortality increases, the optimal investment in health behaviour (measured here on an arbitrary scale) decreases. **b** As the risk of extrinsic mortality goes up, the predicted total mortality rate goes up faster, through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

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; Adams and Nettle 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 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

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 *why* 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 *how* behaviours are triggered, but not *why* they exist. Our extrinsic mortality explanation explains why 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 *how* latent traits might link SES and health behaviours, but not *why* 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 *how* behaviours are reinforced as class norms, but not *why* 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 *how* 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

Table 10.1 (continued)

Level of explanation	Pampel et al.'s explanation type (ET)	Interpretation in light of adaptive model
	Community opportunities (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 *how* behaviours are reinforced, but not *why* 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 *how* behaviour spreads through social networks, but not *why* 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 *cost* money

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

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 exogenous 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.

References

Adams, J. (2009a). The mediating role of time perspective in socio-economic inequalities in smoking and physical activity in older english adults. *Journal of Health Psychology, 14*(6), 794–799.

Adams, J. (2009b). The role of time perspective in smoking cessation amongst older English adults. *Health psychology: Official Journal of the Division of Health Psychology, American Psychological Association, 28*(5), 529–534.

Adams, J., & Nettle, D. (2009). Time perspective, personality and smoking, body mass, and physical activity: an empirical study. *British Journal of Health Psychology, 14*(Pt 1), 83–105.

Adams, J., & White, M. (2009a). Socio-economic and gender differences in nutritional content of foods advertised in popular UK weekly magazines. *European Journal of Public Health, 19*(2), 144–149.

Adams, J., & White, M. (2009b). Time perspective in socioeconomic inequalities in smoking and body mass index. *Health Psychology: Official Journal of the Division of Health Psychology, American Psychological Association, 28*(1), 83–90.

Adler, N. E., & Ostrove, J. M. (1999). Socioeconomic status and health: What we know and what we don't. *Annals of the New York Academy of Sciences, 896,* 3–15.

Barr, R. G., Somers, S. C., Speizer, F. E., & Camargo, C. A. (2002). Patient factors and medication guideline adherence among older women with asthma. *Archives of Internal Medicine, 162,* 1761–1768.

Becker, G. S., & Mulligan, C. B. (1997). The endogenous determination of time preference. *The Quarterly Journal of Economics, 112*(3), 729–758.

Belon, A. P., Barros, M. B., & Marin-Leon, L. (2012). Mortality among adults: Gender and socioeconomic differences in a Brazilian city. *BMC public health, 12*(1), 39.

Blaxter, M. (1997). Whose fault is it? People's own conceptions of the reasons for health inequalities. *Social Science and Medicine, 44*(6), 747–756.

Brennan, S. L., Henry, M. J., Nicholson, G. C., Kotowicz, M. a, & Pasco, J. a (2009). Socioeconomic status and risk factors for obesity and metabolic disorders in a population-based sample of adult females. *Preventive Medicine, 49*(2–3), 165–171.

Buck, D., & Frosini, F. (2012). *Implications for policy and practice. Clustering of unhealthy behaviours over time* (pp. 1–24). http://www.kingsfund.org.uk/publications/clustering-unhealthy-behaviours-over-time

Callan, M., Willshead, N., & Olson, J. (2009). Foregoing the labor for the fruits: The effect of just world threat on the desire for immediate monetary rewards. *Journal of Experimental Social Psychology, 45*(1), 246–249.

Cantwell, M. F., McKenna, M. T., McCray, E., & Onorato, I. M. (1998). Tuberculosis and race/ethnicity in the United States: impact of socioeconomic status. *American Journal of Respiratory and Critical Care Medicine, 157*(4 Pt 1), 1016–1020.

Capewell, S., & Graham, H. (2010). Will cardiovascular disease prevention widen health inequalities? *PLoS Medicine, 7*(8), e1000320.

Colgan, F., Gospel, A., Petrie, J., Adams, J., Heywood, P., & White, M. (2004). Does rear seat belt use vary according to socioeconomic status? *Journal of Epidemiology and Community Health, 58*(11), 929–930.

Crimmins, E. M., & Saito, Y. (2001). Trends in healthy life expectancy in the United States, 1970-1990: gender, racial, and educational differences. *Social Science & Medicine (1982), 52*(11), 1629–1641.

Cristia, J. (2009). Rising mortality and life expectancy differentials by lifetime earnings in the United States. *Journal of Health Economics, 28,* 984–995.

Cubbin, C., LeClere, F. B., & Smith, G. S. (2000). Socioeconomic status and injury mortality: Individual and neighbourhood determinants. *Journal of Epidemiology and Community Health, 54*(7), 517–524.

Cutler, D., & Lleras-Muney, A. (2006). Education and health: evaluating theories and evidence. NBER Working Paper Series. No 12352, 1–37.

Cutler, D., & Lleras-Muney, A. (2008). Education and health: evaluating theories and evidence. In G. K. and H. P. Robert F. Schoeni, James S. House (Ed.), Making Americans Healthier: Social and Economic Policy as Health Policy. New York: Russell Sage Foundation.

Davies, N. B., Krebs, J. R., & West, S. A. (2012). *An introduction to behavioural ecology* (4th ed.). Oxford: Blackwell.

Davis, J. A. (1994). What's wrong with sociology? *Sociological Forum, 9,* 179–197.

Drewnowski, A., & Specter, S. E. (2004). Poverty and obesity: the role of energy density and energy costs. *American Journal of Clinical Nutrition, 79*(1), 6–16.

Drewnowski, A., Monsivais, P., Maillot, M., & Darmon, N. (2007). Low-energy-density diets are associated with higher diet quality and higher diet costs in French adults. *Journal of the American Dietetic Association, 107*(6), 1028–1032.

Ermer, E., Cosmides, L., & Tooby, J. (2008). Relative status regulates risky decision-making about resources in men: Evidence for the co-evolution of motivation and cognition. *Evolution and Human Behavior: Official Journal of the Human Behavior and Evolution Society, 29*(2), 106–118.

Evans, G. W., & Kantrowitz, E. (2002). Socioeconomic status and health: the potential role of environmental risk exposure. *Annual Review of Public Health, 23*(1), 303–331.

Feinstein, J. S. (1993). The relationship between socioeconomic status and health: A review of the literature. *The Milbank Quarterly, 71*(2), 279–322.

Frost, K., Frank, E., & Maibach, E. (1997). Relative risk in the news media: A quantification of misrepresentation. *American Journal of Public Heath, 87*(5), 842–845.

Fuchs, V. (1982). Time preference and health: an exploratory study. In E. V. R. Fuchs (Ed.), *Economic aspects of health* (Vol. I, pp. 93–120). Chicago: University of Chicago Press.

Goldman, D. P., & Smith, J. P. (2002) Can patient self-management help explain the SES health gradient? *Proceedings of the National Academy of Sciences of the United States of America, 99,* 10929–10934.

Griskevicius, V., Delton, A. W., Robertson, T. E., & Tybur, J. M. (2011a). Environmental contingency in life history strategies: The influence of mortality and socioeconomic status on reproductive timing. *Journal of Personality and Social Psychology, 100*(2), 241–254.

Griskevicius, V., Tybur, J. M., Delton, A. W., & Robertson, T. E. (2011b). The influence of mortality and socioeconomic status on risk and delayed rewards: A life history theory approach. *Journal of Personality and Social Psychology, 100*(6), 1015–1026.

Harrell, J., Bangdiwala, S., Deng, S., Webb, J., & Bradley, C. (1998). Smoking initiation in youth: The roles of gender, race, socioeconomics, and developmental status. *Journal of Adolescent Health, 23,* 271–279.

Jewell, D., Tacchi, J., & Donovan, J. (2000). Teenage pregnancy: Whose problem is it? *Family Practice, 17*(6), 522–528.

Johns, S. E. (2010). Perceived environmental risk as a predictor of teenage motherhood in a British population. *Health & Place, 17*(1), 122–131.

Kirby, K. N. (2009). One-year temporal stability of delay-discount rates. *Psychonomic Bulletin & Review, 16*(3), 457–462.

Kotz, D., & West, R. (2009). Explaining the social gradient in smoking cessation: It's not in the trying, but in the succeeding. *Tobacco Control, 18*(1), 43–46.

Kristenson, M., Eriksen, H. R., Sluiter, J. K., Starke, D., & Ursin, H. (2004). Psychobiological mechanisms of socioeconomic differences in health. *Social Science & Medicine (1982), 58*(8), 1511–1522.

Laaksonen, M., Talala, K., Martelin, T., Rahkonen, O., Roos, E., Helakorpi, S., Laatikainen, T., et al. (2008). Health behaviours as explanations for educational level differences in cardiovascular and all-cause mortality: A follow-up of 60 000 men and women over 23 years. *European Journal of Public Health, 18*(1), 38–43.

Lader, D. (2008). *Smoking-related behaviour and attitudes. Office for National Statistics Opinions Survey Report No. 40*. 1–115

Lantz, P. M., House, J. S., Lepkowski, J. M., Williams, D. R., Mero, R. P., & Chen, J. (1998). Socioeconomic Factors, Health Behaviors, and Mortality. *The Journal of the American Medical Association, 279*(21), 1703–1708.

Lantz, P. M., House, J. S., Mero, R. P., & Williams, D. R. (2005). Stress, life events, and socioeconomic disparities in health: Results from the Americans' Changing Lives Study. *Journal of Health and Social Behavior, 46*(3), 274–288.

Lawlor, D. a, Frankel, S., Shaw, M., Ebrahim, S., & Smith, G. D. (2003). Smoking and ill health: Does lay epidemiology explain the failure of smoking cessation programs among deprived populations? *American Journal of Public Health, 93*(2), 266–270.

Leigh, J. (1990). Schooling and seat belt use. *Southern Economic Journal, 57*(1), 195–207.

Levenstein, S., & Kaplan, G. (1998). Socioeconomic status and ulcer: A prospective study of contributory risk factors. *Journal of Clinical Gastroenterology, 26*(1), 14–17.

Leyland, A. H., & Dundas, R. (2010). The social patterning of deaths due to assault in Scotland, 1980-2005: Population-based study. *Journal of Epidemiology and Community Health, 64*(5), 432–439.

Li, J.-Z., Gui, D.-Y., Feng, C.-L., Wang, W.-Z., Du, B.-Q., Gan, T., & Luo, Y.-J. (2012). Victims' time discounting 2.5 years after the Wenchuan earthquake: An ERP study. *PLoS ONE, 7*(7), e40316.

Liao, Y., McGee, D. L., Kaufman, J. S., Cao, G., & Cooper, R. S. (1999). Socioeconomic status and morbidity in the last years of life. *American Journal of Public Health, 89*(4), 569–572.

Link, B. G., & Phelan, J. (1995). *Social conditions as fundamental causes of disease. Journal of Health and Social Behavior, 35*, 80–94.

Low, B. S., Hazel, A., Parker, N., & Welch, K. B. (2008). Influences on Women's Reproductive Lives: Unexpected Ecological Underpinnings. *Cross-Cultural Research, 42*(3), 201–219.

Markowitz, F. (2003). Socioeconomic disadvantage and violence: Recent research on culture and neighborhood control as explanatory mechanisms. *Aggression and Violent Behavior, 8*(2), 145–154.

Mayr, E. (1961). Cause and effect in biology. *Science, 134,* 1501–1506.

McLaren, L. (2007). Socioeconomic status and obesity. *Epidemiologic Reviews, 29,* 29–48.

Melchior, M., Choquet, M., Le Strat, Y., Hassler, C., & Gorwood, P. (2011). Parental alcohol dependence, socioeconomic disadvantage and alcohol and cannabis dependence among young adults in the community. *European Psychiatry: the Journal of the Association of European Psychiatrists, 26*(1), 13–17.

Mackenbach, J. P. (2012). The persistence of health inequalities in modern welfare states: the explanation of a paradox. *Social Science and Medicine, 75*(4), 761–9.

Mishra, S., Son Hing, L. S., & Lalumière, M. L. (under review). Mind the gap: An examination of the effect of inequality on risk-taking.

Mobley, L. R., Root, E. D., Finkelstein, E. a, Khavjou, O., Farris, R. P., & Will, J. C. (2006). Environment, obesity, and cardiovascular disease risk in low-income women. *American Journal of Preventive Medicine, 30*(4), 327–332.

Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *JAMA: The Journal of the American Medical Association, 291*(10), 1238–1245.

Nettle, D. (2010a). Why are there social gradients in preventative health behavior? A perspective from behavioral ecology. (E. Von Elm, Ed.). *PLoS ONE, 5*(10), 6.

Nettle, D. (2010b). Dying young and living fast: Variation in life history across English neighborhoods. *Behavioral Ecology, 21*(2), 387–395.

Nettle, D. (2011). Flexibility in reproductive timing in human females: Integrating ultimate and proximate explanations. *Philosophical Transactions of the Royal Society of London - Series B: Biological Sciences, 366*(1563), 357–365.

Nettle, D., Gibson, M. A., Lawson, D. W., & Sear, R. (2013). Human behavioral ecology: current research and future prospects. *Behavioral Ecology, 24*(5), 1031–1040.

Pampel, F. C., Krueger, P., & Denney, J. (2010). Socioeconomic disparities in health behaviors. *Annual Review of Sociology, 36,* 349–370.

Parker, G. A., & Maynard Smith, J. (1990). Optimality theory in evolutionary biology. *Nature, 348,* 27–33.

Phelan, J. C., Link, B. G., & Tehranifar, P. (2010). Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. *Journal of Health and Social Behavior, 51*(Suppl), S28–S40.

Pridemore, W. A., Tomkins, S., Eckhardt, K., Kiryanov, N., & Saburova, L. (2010). A case-control analysis of socio-economic and marital status differentials in alcohol- and non-alcohol-related mortality among working-age Russian males. *European Journal of Public Health, 20*(5), 569–575.

Qi, V., Phillips, S. P., & Hopman, W. M. (2006). Determinants of a healthy lifestyle and use of preventive screening in Canada. *BMC Public Health, 6,* 275.

Sapolsky, R. M. (2004). Social status and health in humans and other animals. *Annual Review of Anthropology, 33*(1), 393–418.

Shaw, M., Tunstall, H., & Dorling, D. (2005). Increasing inequalities in risk of murder in Britain: Trends in the demographic and spatial distribution of murder, 1981-2000. *Health & Place, 11,* 45–54.

Singh, G. K., & Siahpush, M. (2006). Widening socioeconomic inequalities in US life expectancy, 1980-2000. *International Journal of Epidemiology, 35*(4), 969–979.

Singh, G. K., Azuine, R. E., Siahpush, M., & Kogan, M. D. (2012). All-cause and cause-specific mortality among US youth: Socioeconomic and rural-urban disparities and international patterns. *Journal of Urban Health: Bulletin of the New York Academy of Medicine, 90*(3), 388–405.

Stringhini, S., Sabia, S., Shipley, M., Brunner, E., Nabi, H., Kivimaki, M., & Singh-Manoux, A. (2010). Association of socioeconomic position with health behaviors and mortality. *JAMA: The Journal of the American Medical Association, 303*(12), 1159–1166.

Tinbergen, N. (1963). On aims and methods of ethology. *Zeitschrift für Tierpsychologie, 20,* 410–433.

Wardle, J., Waller, J., & Jarvis, M. J. (2002). Sex differences in the association of socioeconomic status with obesity. *American Journal of Public Health, 92*(8), 1299–1304.

Wardle, J., & Steptoe, A. (2003). Socioeconomic differences in attitudes and beliefs about healthy lifestyles. *Journal of Epidemiology and Community Health, 57*(6), 440–3.

White, M., Adams, J., & Heywood, P. (2009). How and why do interventions that increase health overall widen inequalities within populations. In S. J. Babones (Ed.), *Social inequality and public health* (pp. 65-81). Bristol: Policy Press.

Wilkinson, R. G. (1992a). Income distribution and life expectancy. *British Medical Journal, 304,* 165–168.

Wilkinson, R. G. (1992b). National mortality rates: The impact of inequality? *American Journal of Public Health, 82*(8), 9–11.

Wilson, M., & Daly, M. (2004). Do pretty women inspire men to discount the future? *Proceedings of the Royal Society B Biological Sciences, 271*(Suppl 4), S177–S179.