



Personality is of central concern to understand health: towards a theoretical model for health psychology

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This paper sets out the case that personality traits are central to health psychology. To achieve this, three aims need to be addressed. First, it is necessary to show that personality influences a broad range of health outcomes and mechanisms. Second, the simple descriptive account of Aim 1 is not sufficient, and a theoretical specification needs to be developed to explain the personality-health link and allow for future hypothesis generation. Third, once Aims 1 and 2 are met, it is necessary to demonstrate the clinical utility of personality. In this review I make the case that all three Aims are met. I develop a theoretical framework to understand the links between personality and health drawing on current theorising in the biology, evolution, and neuroscience of personality. I identify traits (i.e., alexithymia, Type D, hypochondriasis, and empathy) that are of particular concern to health psychology and set these within evolutionary cost-benefit analysis. The literature is reviewed within a three-level hierarchical model (individual, group, and organisational) and it is argued that health psychology needs to move from its traditional focus on the individual level to engage group and organisational levels.

Keywords: personality; evolution; diagnosis

When exposed to the same health threat people respond differently: psychologically, behaviourally, physiologically, and ultimately in terms of prognosis and mortality. Explaining this variability is of central importance for medicine and health psychology, in terms of diagnosis, treatment, and prognosis. Personality theory offers a coherent conceptual framework to help explain some of this variability. The aim of this article is to highlight the central role personality has for health psychology. To achieve this, three broad, but interlinked, aims need to be addressed. First, it is necessary to establish that personality influences the health process (i.e., the behaviours, mechanisms, and processes associated with disease) as well as health outcomes (e.g., mortality). Second, to understand the link established with the first aim, it is necessary to move away from simple deterministic and descriptive uses of personality within health psychology and develop a more theoretical framework. It is not sufficient just to show associations between personality and health related variables, and without fulfilling this second aim the case for the central role of personality cannot be made. Therefore, current thinking in the biology, evolution, and neuroscience of personality are reviewed and an initial theoretical framework

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outlined. Third, having established links (Aim 1) and set these within a theoretical framework (Aim 2), it is important to establish the practical clinical relevance of personality traits. The current review adds to the existing literature as existing reviews of personality and health have focused primarily on the first aim and focused on specific aspects of the illness process such as (1) coping (Carver & Connor-Smith, 2010), (2) mortality (Roberts, Kuneel, Shiner, Caspi, & Goldberg, 2007), or (3) health behaviours with respect to a single trait (Bogg & Roberts, 2004), and have not explored the full illness process. In this review a more complete model of the illness process is explored and Aims 1 through 3 are addressed.

Section one: Personality a central construct for the illness process

The illness process may be understood as an individual's journey from health to illness (Figure 1). The individual journey may start with perceptual, behavioural, and cognitive processes that influence the identification of potential illness (via symptom reporting), or through routine checkups/screening in the absence of symptoms. Subsequently, there will be interactions with the health services, diagnosis, and ultimately disease progression, treatment choices, compliance, and mortality. Aspects of this process are influenced by health behaviours (e.g., smoking, exercise), social cognitions (e.g., theory of planned behaviour, TPB), and health beliefs, as well as stress and coping responses. Within this context personality traits are conceived as distal predictors of health outcomes (Chapman et al., 2009) that influence health outcomes either directly (Chapman et al., 2009) or via a number of *mechanisms*. Roberts et al. (2007) identified three mechanisms (1) pathogenesis (i.e., traits resulting in differential physiological reactions, susceptibility to illness, and reactions to external stimuli), (2) health behaviours, and (3) coping with illness. To this it is possible to add that traits will also influence (1) *social cognitions* (e.g., intentions to act; Conner & Abraham, 2001), which influence health behaviours; (2) *associative processes*, whereby environments become associated with symptoms and illness behaviours and act as triggers to illness presentation (Ader & Cohen, 1982); and (3) *communication* with health professionals. The first step in establishing the central role of personality within the health domain is to highlight that it influences all aspects of the illness process. Existing reviews to date have examined aspects of the illness process rather than all components (e.g., Carver & Connor-Smith, 2010; Roberts et al., 2007).

The illness process defined above should also be understood as embedded within a multi-level framework, where the health domain is divided into three hierarchical levels: individual (Level 1), group (Level 2), and organisational (Level 3; Ferguson & Kerrin, 2004; McManus, Winder, & Paice, 2002; see Table 1). The recent reviews by Carver and Connor-Smith (2010) and Roberts et al. (2007) focused on Level 1. This review extends these to look beyond Level 1 and to explore the role of personality across the wider spectrum of health outcomes across Levels 2 and 3.

The individual level reflects personal health outcomes (e.g., mortality, physical health), health processes (e.g., stress and coping), and health behaviours and all traits should influence aspects of this level. The vast majority of health psychology research takes place at this level. A central theme of this review is that health psychology research needs to expand beyond Level 1, if it is to engage with broader

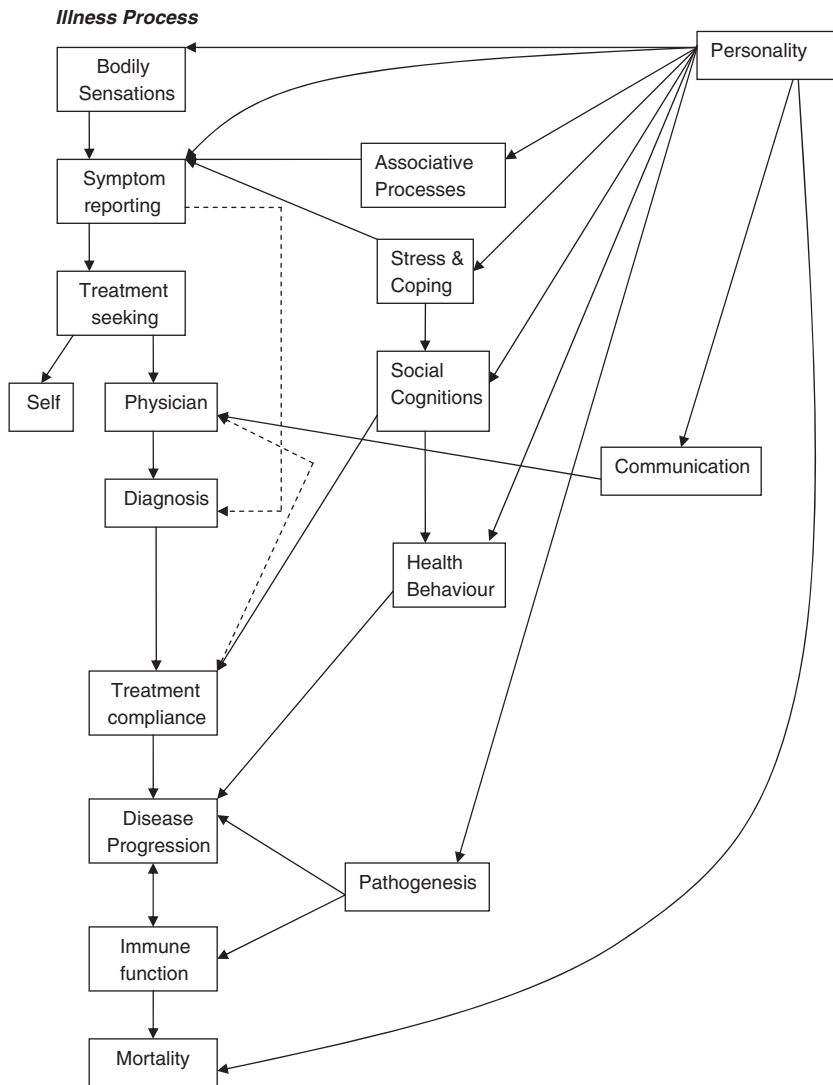


Figure 1. Illness process: personality and mechanisms.

policy issues. The group level focuses on the influence of small group interactions and requires data from all group members (e.g., doctor–patient interaction, family). The organisational level is concerned with the structure and functioning of the health care system and how it influences the delivery of health care (e.g., training and selection of health care staff, health care utilisation, or the health of health carers). A key feature of such a multi-level approach is that each of these levels can and do influence each other (Raudenbush & Bryk, 2002) and it is possible to identify which level of the hierarchy accounts for the most variability in an outcome, and this can be used to target interventions at the correct level (see McManus et al., 2002). Thus, it is possible to identify effects that cascade down the hierarchy and how intervening at a higher level can influence lower level outcomes.

Table 1. Summary of the evidence linking personality to health.

	Organizational	Group	Individual
Empathy	Selection of medical students ²⁹	Charitable giving & helping behaviour, ²⁷ doctor–patient interaction ²⁸	High: Pain, ²³ [longevity, well-being (via helping)], ²⁴ empathic distress, ²⁵ depression ²⁶ Low: Psychopathy, anti-social behaviour
Health anxiety	Doctor-shopping, ³⁴ health care utilization, ³³ medical student syndrome ³⁵	Doctor–patient interaction ³⁶	Symptom reporting, ³⁰ steeper diurnal cortisol, ³¹ SC, ³² S&C, ³³ SBS ³²
Type D	Health care utilisation ⁴⁴	Doctor–patient interaction ⁶¹	MI, ³⁷ immune function, ³⁸ HB, ³⁹ S&C, ⁴⁰ SC ⁴¹
Alexithymia	Negative attitudes to RCTs, ⁵⁸	Doctor–patient interaction, ^{36,55,56} not giving blood, ⁴⁸ not phase 1, ⁵⁰ partner’s personality is detrimental ⁵⁷	PI, ⁴² ACM, ⁴³ Symp, ⁴³ immune function, ⁴⁵ HB ⁴⁶
Neuroticism	Increased burnout ⁵⁹	blood, ⁴⁸ not phase 1, ⁵⁰ partner’s personality is detrimental ⁵⁷	PI, ¹ ACM, ²⁻⁴ HB, ⁶ SC, ^{9,10,12} S&C, ^{13,16,53} C&C, ¹⁴ Symp, ^{17,18} Assoc, ²⁰ SBS ⁵¹
Extraversion		Not giving blood, ⁴⁸ phase 1, ⁵⁰ doctor–patient interaction ⁵⁵	PI, ¹ ACM, ⁴ HB, ^{6,8} SC, ^{9,10} S&C, ^{13,16,53} C&C, ¹⁴ Cyt, ¹⁵ Infect, ¹⁹ DP ⁵⁴
Openness		Phase 1, ⁵⁰ doctor–patient interaction ^{55,56}	PI, ¹ ACM, ⁵ HB, ⁶ SC, ¹⁰ S&C, ^{16,53} Symp, ¹⁷ DP ⁵⁴
Agreeableness		Giving blood, ⁴⁷ helpline volunteer, ⁴⁹ not phase 1, ⁵⁰	PI, ¹ ACM, ²⁻⁴ HB, ⁶ SC, ^{9,10} S&C, ^{16,53}
Conscientiousness	Selection of medical students, ²¹ transition to work ⁶⁰	Compensatory health factors, ²² giving blood, ⁴⁸ doctor–patient interaction ^{55,56}	PI, ¹ ACM, ⁴ Longevity, ³ HB, ⁶⁻⁸ SC, ^{10,11} S&C, ^{16,53} DP ⁵⁴

Note: PI, physical illness; SC, social cognitions; S&C, stress and coping processes; MI, myocardial infarction; HB, health behaviours; CHD, coronary heart disease; FSS, functional somatic syndromes; ACM, all cause mortality; C&C, cortisol/cholesterol; Cyt, cytokines; Symp, symptom reporting; infect, susceptibility to infection; Assoc, associative mechanisms in health; SBS, sensitivity to bodily sensations; DP, disease progression; Phase 1, phase 1 studies.

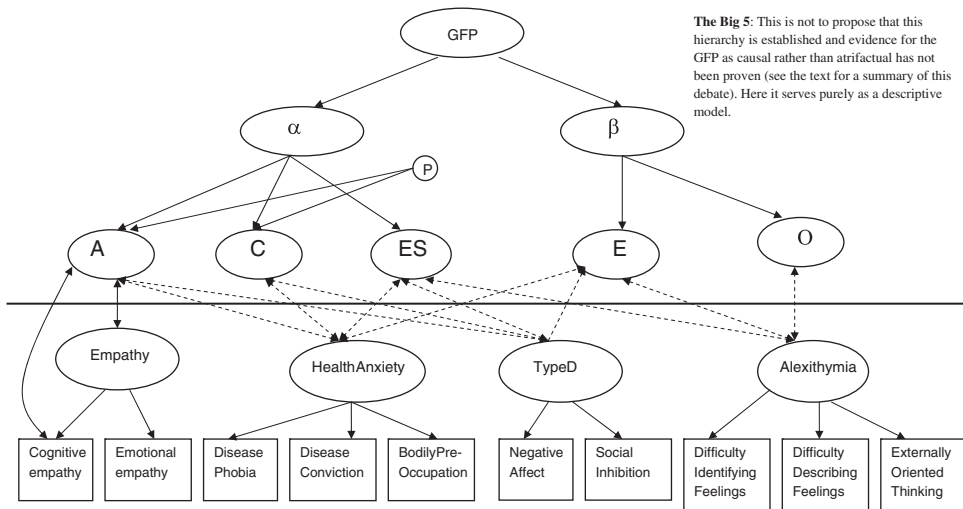
¹Goodwin and Friedman (2006); ²Chapman et al. (2010); ³Kern and Friedman (2008); ⁴Roberts et al. (2007); ⁵Taylor et al. (2009); ⁶Booth-Kewley and Vickers (1994); ⁷O’Connor et al. (2009); ⁸Raynor and Levine (2009); ⁹Gerend et al. (2004); ¹⁰Vollrath et al. (1999); ¹¹Chatzisarantis and Hagger (2008); and Conner and Abraham (2001); and Conner, Grogan, Fry, Gough, and Higgins (2009); ¹²de Bruijn et al. (2009); ¹³Ferguson (2001); ¹⁴LeBlanc and Ducharme (2005); ¹⁵Chapman et al. (2009); ¹⁶DeLongis and Holtzman (2005) and David and Suls (1999); ¹⁷Feldman et al. (1999); ¹⁸Watson and Pennebaker (1989); ¹⁹Totman et al. (1980); ²⁰Devriese et al. (2000); ²¹Ferguson et al. (2000a, 2002, 2003); ²²Roberts et al. 2009; ²³Schieman and van Gundy (2000); ²⁴Brown et al. (2003); ²⁵Schaller and Cialdini (1988); ²⁶Kim et al. (2007); ²⁷Ferguson et al. (2008); ²⁸Kim et al. (2004); ²⁹Ferguson (2000); ³⁰Noyes et al. (1993); ³¹Ferguson (2008); ³²Barsky et al. (1999); ³³Noyes et al. (1999); ³⁴Kasteler et al. (1976); ³⁵Moss-Morris and Petrie (2001); ³⁶Ferguson (2000); ³⁷Denollet et al. (1996); ³⁸Denollet et al. (2008); ³⁹Williams et al. (2008); ⁴⁰Williams et al. (2008); ⁴¹Mols and Denollet (2010); ⁴²Grabe et al. (2010); ⁴³Tolmunen et al. (2010); ⁴⁴Lumley et al. (2007); ⁴⁵Dewaraja et al. (1997); ⁴⁶Helmers and Mente (1996); ⁴⁷Bekkers (2006); ⁴⁸Ferguson (2004a); ⁴⁹Paterson et al. (2009); ⁵⁰Almeida et al. (2008); ⁵¹Paal et al. (2004); ⁵²Gramling et al. (1996); ⁵³Carver and Connor-Smith (2010) and Connor-Smith and Flachsbart (2007); ⁵⁴Ironson et al. (2008); ⁵⁵Eaton and Tinsley (1999); ⁵⁶Chapman et al. (2008); ⁵⁷Ruiz et al. (2006); ⁵⁸McCulloch et al. (2005); ⁵⁹Deary et al. (1996); ⁶⁰Cave et al. (2009); ⁶¹Rastling et al. (2005).

Whereas Level 1 is the traditional domain for the majority of health psychology research, there are key implications from Levels 2 and 3 for health psychology. Level 2 involves the influence of one person's behaviour on another, this not only concerns doctor–patient interactions and the role of the family but also the study of other-regarding preferences (i.e., acting with respect to the well-being of others). Such preferences play a major role with respect to health outcomes. Indeed, a large proportion of health service provision and research is only possible through charitable donations (e.g., hospital fund-raising), volunteering (e.g., blood and organ donation, clinical trials), and donations from medical charities. Other-regarding preferences also concern decisions made on behalf of others (e.g., 'Should I get my children vaccinated?'). Sometimes the personal (self-regarding) and societal (other-regarding) preferences may be in opposition, for example, vaccination. Getting vaccinated presents personal risks (e.g., side effects) and benefits (e.g., immunity) as well as benefits for others (reduces spread of infection, helps to attain herd immunity). Individuals could, however, choose to free-ride, waiting to let others get vaccinated, avoiding the risks of vaccination, and reaping the benefits (herd immunity: but only if the majority get vaccinated). Free-riding of this type is a major concern for vaccination programmes (Hershey, Asch, Thumasathit, Meszaros, & Waters, 1994; Meszaros et al., 1996). These examples of other-regarding preferences should be more fully explored and studied within health psychology and personality theory (empathic traits) can help to provide a starting point for this research. As governments increasingly emphasise the role of the third sector (e.g., charities) with respect to the provision of health care and mass public health interventions (e.g., recent Swine flu vaccination in the UK) require detailed planning, understanding Level 2 processes is crucial if health psychology is to influence policy in these areas.

Level 3 is also an important domain for health psychology research, reflecting the nature of health care provision and how this may impact on Level 1 as well as organisational factors (e.g., physician turnover). This includes the selection and training of health care workers. Selecting the right medical staff will have implications not only for patient compliance (this is an example of how Level 3 can affect Level 1) but also the health of medical staff.

The personality hierarchy and why it is important for health psychology

A key feature of all personality taxonomies is their hierarchical structure, with the higher-order traits causally responsible for the lower ones (Ashton, Lee, & Goldberg, 2004; Ashton, Lee, Goldberg, & de Vries, 2009). A central question within contemporary personality theory concerns whether one, two, three, or five factors mark the apex of this hierarchy (Ashton et al., 2009, see top section of Figure 2; Ferguson, Chamorro-Premuzic, Pickering, & Wiess, in press). From the perspective of health psychology, it is essential to know where the apex of this hierarchy resides. For example, if a general factor of personality (GFP) exists (see Rushton & Irwin, in press for a review), then the role of personality within health psychology would be concerned with how this *single* trait can influence a *wide* variety of health outcomes. If the apex is lower, and marked by five factors, the role of personality within health psychology would be concerned with whether specific personality traits influence specific health outcomes, or how traits interact with each other (both linearly and non-linearly) to influence health outcomes (Hoyle, 2000). For example, work on



The Big 5: This is not to propose that this hierarchy is established and evidence for the GFP as causal rather than artifactual has not been proven (see the text for a summary of this debate). Here it serves purely as a descriptive model.

Note for Figure 2: Solid arrowed lines represent positive associations and dashed arrowed lines represent negative associations. The above is a descriptive representation of how the health related traits are related to the Big 5. The arrows are two-headed to imply that these are associations and not lower traits of the FFM

Figure 2. The proposed personality hierarchy.

conscientiousness (C) has become a central focus for health psychology, for example, with respect to the prediction of longevity (Kern & Friedman, 2008) and its interaction with neuroticism to influence health outcomes (Roberts, Smith, Jackson, & Edmonds, 2009). The rationale for examining C is predicated on five orthogonal personality factors marking the apex of the personality hierarchy (Ashton et al., 2009). Therefore, prior to considering the role of personality and the health process it is necessary to address this issue.

Indeed, the most established apex for the personality hierarchy is defined by the domains of the five factor model (FFM): Neuroticism (N: high scorers are emotionally unstable, anxious, etc.), Extraversion (E: high scorers are outgoing and sociable, impulsive, etc.), Openness (O: high scorers are being creative, cultured, artistic, etc.), Agreeableness (A: high scorers are trustworthy, cooperative, etc.), and Conscientiousness (C: high scorers are methodical, hardworking, etc.). While theoretically derived as orthogonal, these domains are correlated leading some to argue *theoretically* that fewer dimensions are required to explain variation in normal personality (e.g., the ‘Giant 3’ of N, E, and Psychoticism; Eysenck, 1992), or empirically derived higher-order models such as α (reflecting socialisation processes) and β (reflecting agency and development; Digman, 1997). DeYoung (2006) developed these two factors theoretically in terms of their basic neuroscience referring to them as *stability* and *plasticity*, respectively. Recently, it has been suggested that a GFP resides at the top of this hierarchy (Rushton & Irwing, in press). The GFP is seen as a bipolar construct with the high end defined as ‘altruistic, emotionally stable, agreeable, conscientious, extraverted, and intellectually open, with high levels of well-being, satisfaction with life, self-esteem and emotional intelligence’ (Rushton & Irwing, 2009, p. 1091) and the low end defined by social challenge and personality disorder (Rushton & Irwing, 2009). From a health

psychology perspective this implies that low GFP scores will be associated with *all poor health outcomes across all contexts*.

A recent evaluation of the data and the theory mustered in support of the GFP casts serious doubts on the validity of the GFP, suggesting that the FFM domains mark the apex of the personality hierarchy (Ferguson et al., in press). Ferguson et al. (in press) offered their critique in terms of (1) the psychometric evidence for the GFP and (2) the GFPs theoretical underpinnings. Psychometrically there are concerns about (1) the wide use of modification indices in the confirmatory models to show the GFP, as these increase the probability of capitalising on chance (MacCallum, Roznowski, & Necowitz, 1992) and (2) using a mixture of exploratory and confirmatory analyses on the same samples. Furthermore, it is argued that the correlations between the FFM domains reflect a number of methodological artefacts, rather than substance, associated with trait measurement (e.g., Anusic, Schmmack, Oinkus, & Lockwood, 2009; Ashton et al., 2009). The five factors, for example, become orthogonal when method variance due to observer type (self, peer, etc.) is controlled in a multi-trait multi-method analysis (Biesanz & West, 2004). Further, the GFP disappears when social desirability is controlled (Backstrom, Bjorklund, & Larson, 2009). Theoretically there are also concerns. First, high scores on the FFM domains are associated with personality disorders, which is opposite to the GFP prediction (e.g., Samuel & Widiger, 2008). Second, GFP theory suggests that, like general IQ, the GFP was influenced by directional selection, however, the evidence that the GFP and general IQ are linked is weak. Third, GFP theory would suggest that high scores on the GFP predict fitness outcome such as reproduction. Eaves, Martin, Heath, Hewitt and Neale (1990) showed that reproduction for N varied along E, such that fitness was maximal for high N when E was low and maximal for high E when N was low. While the low N/high E finding is consistent with the GFP, the high N/low E finding is not. This is also an example of a non-linear association between traits with respect to fitness, whereas the GFP theory would predict linear associations with fitness. Fourth, in terms of mate preference, GFP would predict that high GFP would be positively selected for. However, consensually only O, A, and E are preferred by the majority as desirable in a partner (e.g., Wood & Brumbaugh, 2009) and there is little evidence for assortative mating on the basis of the FFM domains (Ozer & Benet-Martinez, 2006) except for O and to a lesser extent C (McCrae, 1996). As such, the FFM should be considered to be the highest level of description within the personality hierarchy and the rest of this article proceeds on this basis.

FFM and the health domain: the illness-process and three levels of analysis

Table 1 summarises the evidence concerning the impact of the FFM domains across all three levels of the health domain and this is used as the evidence for the links in Figure 1. The first thing to note is that there is much more evidence for Level 1 – this just reflects the focus of work in health psychology. The aim of Table 1, and this section, is not to provide a comprehensive meta-analysis, but to highlight the breadth of outcomes and mechanisms personality has been linked to, but where evidence is available from meta-analyses to provide effect size estimates. Thus evidence reported in Table 1 is based on studies that are either (1) meta-analyses (e.g., Roberts et al., 2007) or (2) contemporary studies offering replications of exiting findings. Indeed,

there is evidence that as research areas progress the effect sizes diminish due to improved methods and theory (Munafo, Matheson, & Flint, 2007). I will review the evidence for the central role of personality at Level 1 in terms of its effects on mortality and other key health outcomes (major chronic disease and symptom reporting) and in terms of the six broad mechanisms (associations, social cognitions, health behaviours, communication, pathogenesis, and stress, and coping) presented in Figure 1.

Personality and mortality

There is well-established, replicated, and robust evidence that the FFM domains influence the major illness outcomes of ‘all cause mortality’ (Chapman, Fiscella, Kawachi, & Duberstein, 2010; Jonassaint et al., 2007; Roberts et al., 2007), longevity (Kern & Friedman, 2008), physical illnesses (Goodwin & Friedman, 2006), and symptom reporting (Watson & Pennebaker, 1989).

Mortality reflects the ultimate outcome of the illness process and is a key objective marker used to make the case that a risk factor is important for policy (Deary, 2010). With respect to the FFM, Roberts et al. (2007) reported the following effect sizes with respect to mortality: (1) N = .05, (2) E/positive affect = $-.07$, (3) C = $-.09$, and (4) hostility/disagreeableness = .04. Thus, N and low A are related to increased mortality and E and C to reduced mortality. Roberts et al. (2007) did not report an effect for O. Indeed, Carver and Connor-Smith (2010) state that there is much less evidence linking O systematically to health outcomes. Ferguson (2010) has recently conducted a meta-analysis of 10 studies on O and mortality and reports a protective effect in terms of r of the same magnitude as N and hostility/disagreeableness reported by Roberts et al. (2007). One possibility suggested for the protective effect of O is its link to general IQ and adaptive coping (Ferguson, 2010).

The effect sizes for the personality–mortality link appear small. However, when interpreting these effect sizes, consideration should be given to guidelines set out by Prentice and Miller (1992). They argue that small effect sizes should be considered impressive under two conditions: (1) when the intervention is minimal or (2) when the outcome is difficult to influence. This is why within medicine a minimal intervention (e.g., aspirin) that has a small ($r = .034$), but significant, effect in reducing a difficult to influence outcome (e.g., risk of future cardiovascular events) has important public health implications (Steering, 1988). With respect to personality and mortality, the intervention is minimal (assessed traits) and the outcome (mortality) is difficult to influence. As such, the effect of personality on health and mortality should, as Chapman et al. (2009) states, be seen as a *key* distal predictor of mortality much as socio-economic status (SES) is. While work is advancing with respect to examining and summarising, both health behaviours and personality with respect to longevity and mortality, research on traditional social cognitions has tended not to pursue this type of work. It would be useful to know the effect sizes for social cognitive constructs with respect to mortality.

How does the effect of personality compare to other known predictors of mortality? Roberts et al. (2007) reported, after controlling for other risk factors, for mortality, the effect size for SES was .02 and for low IQ it was .06. Deriving correlations from Chapman et al. (2009), the effect size for smoking on mortality was .41, .07 for obesity, .20 for physical inactivity, with a negligible effect of heavy alcohol

consumption ($-.007$).¹ As such, personality is as good as, and in some cases better than, SES, obesity, and alcohol consumption in predicting mortality. Of course all of these constructs are interlinked and research needs to adopt structural modelling to tease effects apart. For example, Chapman et al. (2009) showed that the association between personality and mortality is not completely attributable to health behaviour and SES (also Mroczek, Spiro, & Turiano, 2009).

With respect to physical illness, Goodwin and Friedman (2006) explored 12 common illnesses (e.g., diabetes, stroke, asthma) and showed that for C there were significant effects for 10 and all were in the predicted direction (high C linked to the absence of the illness). For E there were 6 association and all 6 were in the predicted direction (high E linked to absence of illness). For O there were 8 associations and again all in the direction that high O is protective. For N there were 9 associations with all in the direction of high N being a risk factor. The picture for A was mixed. Thus, the effect of personality is consistent across a wide variety of major physical illnesses.

Finally *symptom reporting* is a key health outcome with respect to health care utilisation (Kroenke, 2001). This shows how Level 1 can influence Level 3. Influences on symptom reporting have been observed for N, C, and O (Feldman, Cohen, Doyle, Skoner, & Gwaltney, 1999; Watson & Pennebaker, 1989). Traits related to N have been linked to greater sensitivity to pain in healthy (Pud, Eisenberg, Sprecher, Rogowski, & Yarnitsky, 2004) and clinical samples (e.g., Granot, 2005), as well as less accurate reporting of bodily sensations (Bogaerts et al., 2005).

Thus with respect to mortality, disease diagnosis, and symptom reporting there are consistent and robust effects of personality. The next section explores the potential mechanisms for these personality–health outcomes.

Pathogenesis

Personality traits are linked to a wide variety of physiological and immune system responses (Chapman et al., 2009; LeBlanc & Ducharme, 2005) and susceptibility to infection (Totman, Kiff, Reed, & Craig, 1980). There is evidence that E is related to increased cortisol levels, reduced cytokine levels (implying reduced inflammation), and increased susceptibility to infection and, along with high levels of O and C, slower disease progression (see Ironson, O'Leirigh, Weiss, Schneiderman, & Costa, 2008; LeBlanc & Ducharme, 2003). In terms of meta-analytic findings, sub-analyses reported by Chida and Steptoe (2009) showed that positive psychological factors (e.g., positive affect, self-esteem) were related to reduced waking cortisol production. Further meta-analytic work shows that depressive affect – which is a good marker for neuroticism (see Kotov, Gamez, Schmidt, & Watson, 2010) – is associated with reduced cellular immune activity (Herbert & Cohen, 1993) and increased pro-inflammatory cytokine levels (Dowlati et al., 2010). Finally, it should be recognised that the effect of personality on pathogenesis may be moderated by demographics (see Gerritsen et al., 2009) and stress (Burke, Davis, Otte, & Mohr, 2005).

Health behaviours

The FFM domains have been consistently linked to health behaviours (e.g., Booth-Kewley & Vickers, 1994; O'Connor, Jones, Conner, McMillan, & Ferguson,

2009; Raynor & Levine, 2009). Meta-analytic evidence shows that C is positively linked to healthy behaviours (e.g., activity: $r = .05$) and negatively to unhealthy behaviours (e.g., tobacco use: $r = -.14$; Bogg & Roberts, 2004). Kotov et al.'s (2010) recent meta-analyses confirms that C is negatively linked to substance abuse ($r = -.48$: converted from their d of -1.10). They additionally showed that N was positively linked to substance abuse ($r = .43$: their $d = 0.97$). They showed no link between O and substance abuse and weaker links for A and E, with both negatively linked to drug use ($r_s = -.16$ and $-.25$, respectively). This confirms an earlier finding of Booth-Kewley and Vickers (1994) showing that N was linked to poorer health behaviours and E and A to healthier behaviours. However, risk taking for E may vary according to the type of behaviour, while this is negative for drug taking (Kotov et al., 2010), it is positive for sexual risk taking (Vollrath, Knoch, & Cassano, 1999). As N and low A are linked to increased mortality and O and E reduced mortality, C should no longer be seen as the only protective factor. Meta-analyses of the health behaviour links for N, A, O, and E are needed.

Coping

The FFM domains influence stress and coping processes (Carver & Connor-Smith, 2010; David & Suls, 1999; DeLongis & Holtzman, 2005; Ferguson, 2001) Meta-analytic evidence shows that E ($r = .15$), C ($r = .11$), and O ($r = .10$) are linked to overall engagement coping strategies (e.g., problem solving) that are generally beneficial to health, whereas N ($r = .27$) is associated with disengagement strategies (e.g., substance use; Kotov et al., 2010), which are not beneficial to health (see Connor-Smith & Flachsbart, 2007).

Social cognitions

There is also emerging evidence that FFM domains influence key components of social cognitive models. For example E, A, and C are linked to reduced perceived disease susceptibility and N to increased perceived susceptibility (e.g., Gerend, Aiken, & West, 2004; Vollrath et al., 1999). C moderates the effects of intentions, such that people are more likely to act in accordance with their intentions when they score high on C (e.g., Chatzisarantis & Hagger, 2008). Similarly, the C health behaviour link (Bogg & Roberts, 2004) is mediated by TPB variables such as attitudes (e.g., Conner & Abraham, 2001; de Bruijin, Brug, & van Lenthe, 2009).

Associative processes

There is ample evidence that people learn to associate places and odours with symptoms and illness (see Ferguson & Cassaday, 1999, 2002 for reviews). These processes are stronger for those high in N (Devriese et al., 2000).

Communication

There is evidence that those high in N are less likely to perceive doctor–patient interactions as reassuring (Ferguson, 2000).

Level 2

Exploration at this level extends previous reviews. The specific focus at this level is not only on the manner in which one person's behaviour affects the health of another, but also how the dynamic interaction between people affects each others health. Topics at this level include (1) dyadic interactions (doctor–patient, spouse/carer–patient), (2) other regarding preferences, and (3) small group effects. While the influence of one partner's behaviour, cognitions, and mood on the others is studied in psychiatry (e.g., Vaughn, Leff, & Sarner, 1999) and with respect to illness representations in health psychology (e.g., Hilbert, Martin, Zech, Rauh, & Rief, 2010), this level is generally under-researched in health psychology. Work on personality offers one avenue to explore the effects of dyadic interaction on health and there is a small but emerging literature here. Two types of dyad have been explored: spouses and doctor–patient interactions. For example, spouse's level of C has been shown to predict positive health outcomes in their partners: 'compensatory conscientiousness' (Roberts et al., 2009), whereas carer neuroticism predicts patient's depression and patient's neuroticism predicts carers' depression (Ruiz, Matthews, Scheier, & Schultz, 2006). Thus within dyads, one partner's personality can act as a protective or risk factor for the others health. Indeed, discrepancies between partners personality are known to influence relationship satisfaction (O'Rourke, Neufeld, Claxton, & Smith, 2010). Personality also influences doctor–patient communications. For physicians, C is related exploring the patient's psychosocial circumstances, however, both C and N were linked to involving patients less in their treatment (Chapman, Duberstein, Epstein, Fiscella, & Kravitz, 2008). Patient's personality (O, E, C, and N) predicts the content of their communication with their physician, the content of the physician's communication (O and N), and the physician's overall rating of the patient's communication success (O; Eaton & Tinsley, 1999). Personality effects on health care volunteering have also been observed. However, the exact nature of the relationship depends on the type of voluntary behaviour. For example, blood donation is related to high A (Bekkers, 2006), low N, low E, and high C (Ferguson, 2004a) and helpline volunteering to high A (Paterson, Reniers, & Vollm, 2009). However, those volunteering for phase 1 trials are low on N and A, but high on E and O (Almeida et al., 2008).

Level 3

Level 3 examines factors linked to health care provision (e.g., physician health, selection and training, staff turnover). In this context, C has been consistently linked to success in medical training (Ferguson, James, & Madelely, 2002; Ferguson, James, O'Hehir, & Sanders, 2003; Ferguson, Sanders, O'Hehir, & James, 2000a; Lievens, Coessier, De Fruyt, & Maeseneer, 2002; Lievens, Ones, & Dilchert, 2009) and preparedness for the transition from medical school to employment (Cave, Woolf, Jones, & Dacre, 2009). N has also been linked to increased physician burnout (Deary, Blenkin, Agius, Endler, & Zeally, 1996) and an increased tendency not to have a positive attitude towards randomised controlled trials (McCulloch, Kaul, Wagstaff, & Wheatcroft, 2005).

Synergistic trait effects and compensatory health behaviour

The above analysis has focused on the ‘main effects’ for the trait, however, traits can interact to modify each others influence. For example, Roberts et al. (2009) showed that a combination of high N and high C was related to reduced health limitations – yet N on its own was related to poorer health outcomes. Understanding the mechanisms that make trait interactions such as this beneficial can suggest new lines of cognitive-behavioural research for health psychology. For example, Roberts et al. (2009) argue that this combination of high C and N leads to greater vigilance and diligence about health. However, another possibility is that this combination of traits is associated with what may be termed *compensatory health behaviours* (CHBs). That is a combination of negative (e.g., smoking, binge drinking) and positive (e.g., exercising, eating healthily) health behaviours exhibited by an individual, because they erroneously believe that the effects of indulging in the positive behaviour offsets any deleterious effects of the negative behaviour. The positive health behaviours are more likely to be associated with C (Bogg & Roberts, 2004) and the negative ones with N (Booth-Kewley & Vickers, 1994). Thus, those high in both N and C should express good self-reported health as Roberts et al. (2007) showed, because people with this combination will believe they are healthy due to their erroneous compensatory health belief. A prediction would be that there should be a positive correlation between negative (e.g., smoking) and positive (e.g., exercise) health behaviours for people high in both N and C, while for other combinations of C and N the associations should be negative. Further, those high in both N and C, while expressing good self-reported health, should exhibit poorer objective health. Taking exercise will not negate the negative effect of smoking, ultimately this CHB belief will lead to maintained smoking and ultimately worse objective health. To date the concept of CHB has not been explored within health psychology. Thus considering synergistic trait effects can suggest new lines of research in health psychology.

Health specific traits: beyond the FFM

The above analysis has focused on the FFM. However, Carver and Connor-Smith (2010) have suggested that with respect to personality and health research it is useful to go beyond the FFM. Thus, while the FFM domains form a central framework – as detailed above – there are traits that lie beyond these five factors (see Saucier & Goldberg, 1988) that have particular relevance to health psychology. While not an exhaustive list, four traits are of particular relevance to health psychology: (1) empathy, (2) health anxiety, (3) type-D, and (4) alexithymia. While all are associated with the FFM domains (see Figure 2; Bagby, Taylor, & Parker, 1994; De Fruyt & Denollet, 2002; Ferguson, 2001; Luminet, Bagby, Wagner, Taylor, & Parker, 1999; Nettle & Liddle, 2008), they are not theoretically subsumed under the FFM domains.

These four traits are highlighted as essential for health psychology because of their focus on emotional processing. The role of emotional processing is becoming of theoretical importance for health psychology (Lawton, Conner, & McEachan, 2009; Lumley, Neely, & Burger, 2007), as is the use of emotion-based interventions (Lumley, 2004). Empathy is linked to understanding the emotions of others (Davis, 1983), health anxiety (HA) to the emotional tagging of health relevant information (Ferguson, Moghaddam, & Bibby, 2007), Type D to reduced emotional quality of life

(Pedersen, Herrmann-Lingen, de Jonge, & Scherer, 2010), and alexithymia to the inability to understand and utilise emotions. Assessment of each trait provides a means to explore the role of emotional processing in health behaviours (Lumley, 2004) and test theoretical models that focus on emotional processing. Furthermore, these four traits are highlighted because they are likely to have specific health-related policy implications (described below). Finally, (1) these traits are well developed and understood theoretically to enable health psychology to develop and test theory in a wide context, (2) relate to many key aspects of the illness process (Figure 1), and (3) have a clear biological basis. Other traits related to health such as optimism, self-esteem, and self-control load on a single factor ('optimistic control') and are strongly associated and subsumed within the five factor model, especially E and C (see Marshall, Wortman, Vickers, Kusulas, & Hervig, 1994). The importance of each trait for health psychology is detailed below.

The policy implications for empathy arise as it is seen as a desirable quality in health care providers (medical students may be selected on this trait; Ferguson et al., 2002) and political parties in the UK are arguing for an increased role for the third (e.g., charitable) sector in health care. Further it (1) is well understood in terms of its psychometric properties and neuroscience (Davis, 1983; Singer & Lamm, 2009), (2) has a key role in communication of health information (Kim, Kaplowitz, & Johnston, 2004), and (3) is heritable with an h^2 of .47 (Knafo, Zahn-Waxler, Hulle, Robinson, & Rhee, 2008). Health anxiety (HA) is likewise important for policy due to its association with medically unexplained syndromes (MUS), which account for up to 50% of admission in some hospital specialties (Nimnuan, Hotopf, & Wessely, 2001). Therefore, understanding the role of HA with respect to MUS has major policy implications. As with empathy it is (1) theoretically well specified (Kellner, 1986, Warwick & Salkovskis, 1990), (2) a key determinant of health care provision and unexplained symptom reporting (Noyes, Happel, & Yagla, 1999), and (3) heritable with a h^2 of .37 (Taylor, Thordarson, Jang, & Asmundson, 2006). Similarly, Type D has policy implications for the treatment of heart disease. Further it is also (1) theoretically grounded with respect to traits of negative affectivity and social inhibition, (2) has a number of identified mechanisms linking it to health outcomes (Denollet et al, 1996; Denollet, Pedersen, Vrints, & Conraads, 2006), and (3) is heritable with a h^2 of .52 (Kupper, Denollet, De Geus, Boomsma, & Willemsen, 2007). Finally, alexithymia, which is the inability to understand, verbalise, and interpret one's emotions affects up to 10% of the population, and has a higher prevalence in clinical populations such as those with (a) Type 1 diabetes (Chartzi et al., 2009), (b) cancer (Gritti et al., 2010), (c) Parkinson's Disease (Costa, Peppe, Carlesimo, Salamone, & Callagirono, 2010), and (d) Systemic Lupus Erythematosis (Barbosa et al., 2009). The high prevalence of alexithymia and its link to major health problems means it will have major policy implications for screening and treatment. Finally alexithymia is (1) well-understood psychometrically with a well-developed neuroscientific basis (Kooiman, Spinhoven, & Trijsburg, 2002; Lesser, 1981; Taylor, Bagby, & Parker, 1997), (2) is related to many aspects of the illness process (Lumley, Stettner, & Wehmer, 1996), and (3) is heritable with a h^2 of .33 (Jorgensen, Zachariae, Skytthe, & Kyvik, 2007).

A more detailed analysis of these traits and their implications for health psychology is given below.

Empathic traits

Empathy is multidimensional (Davis, 1983; Singer & Lamm, 2009), with four broad dimensions: cognitive, affective/emotional, distressed, and motor. Cognitive empathy (or perspective taking) is defined as understanding the emotional state of another (Blair, 2005; Jolliffe & Farrington, 2006; Smith, 2006), without necessarily feeling those emotions (Batson, Early, & Salvarani, 1997; Batson et al., 2007). Affective/emotional empathy (or empathic concern) represents the capacity to experience (Jolliffe & Farrington, 2006) and share (Smith, 2006) the emotions of others. Empathic distress relates to the negative emotions felt by the empathiser due to 'sharing' the emotions of the other person (Batson et al., 1997; Davis, 1983). This could be due to an inability to distinguish self from other as the source of emotional distress (as might be the case with mimicry or emotional contagion; Singer & Lamm, 2009) or feelings of personal distress may arise because of another's pain (Baumann, Cialdini, & Kenrick, 1981; Cialdini & Kenrick, 1976; Davis, 1983; Maner et al., 2002; Manucia, Baumann, & Cialdini, 1984). Finally, motor empathy refers to an individual's ability to mirror/mimic the emotional responses of others (Blair, 2005; de Wall, 2008; Preston & de Wall, 2002). Empathy is distinguished from emotional contagion as contagion occurs without awareness that the emotion the person is feeling is not their own, whereas empathy involves awareness that their emotion is elicited by another (Hein & Singer, 2008; Singer & Lamm, 2009). While the evidence that empathy is a precursor to helping behaviour (Batson et al., 1997) is not conclusive (Singer & Lamm, 2009), there is evidence that helping others is linked to longevity (Brown, Nesse, Vinokur, & Smith, 2003) and improved subjective well-being (Weinstein & Ryan, 2010). Finally, while there is some debate concerning whether empathy should be considered a trait or state phenomena (Singer & Lamm, 2009), trait empathy is correlated with many of the same brain areas involved in experimentally (state) induced empathy (Singer et al., 2004).

With respect to Level 1, the idea that high levels of empathy are beneficial for health needs to be reconsidered with respect to empathic distress (Hoffman, 1979). It is argued here that empathising may, in fact, lead to negative health outcomes (Lee, 2009). A number of lines of evidence support this. First, empathising with another can lead to physical symptoms in the empathiser. For example, high empathy is related to increased reported pain (Schieman & van Gundy, 2000) and empathy for another's pain leads to sensations of personal pain (Singer et al., 2004; Singer & Lamm, 2009). Second, as well as physical symptoms, empathy can also be linked to psychological symptoms. For example, becoming more empathic towards others is linked to increased depression (Kim, Schulz, & Carver, 2007). Third, psychometric evidence shows that emotions associated with empathic distress (e.g., sadness) load positively with emotions of compassion and empathy (e.g., compassionate) in helping contexts, suggesting that, in this context, helping is linked to distress (Maner et al., 2002). Fourth, experimental data shows that distress associated with empathy can be 'cancelled' by other mood enhancing events (anticipating listening to a comedy tape, receiving money or praise; Cialdini, Darby, & Vincent, 1973; Cialdini et al., 1987; Schaller & Cialdini, 1988). The later finding suggests an intriguing hypothesis for health psychology. If empathic distress can be cancelled by mood enhancing activities, then people may seek to alleviate any negative distress associated with helping others by seeking mood enhancement. This could be

achieved, not only by positive health behaviours (e.g., exercise), but also negative ones (e.g., smoking). However, there is evidence that negative mood is more likely to be linked to poorer health behaviours (Kendzor et al., 2008; but see Jones, O'Connor, Conner, McMillan, & Ferguson, 2007) and that the effects of positive affect are separate to those of negative affect (Steptoe, Dockray, & Wardle, 2009). Further the choice of mood enhancing behaviour may be influenced by current behaviour or biological sex. For example, Moghaddam and Ferguson (2007) have shown that smokers will have a cigarette to enhance their mood and Jones et al. (2007) demonstrated that the relationship between negative affect and health behaviour depends on sex, with negative affect linked to snacking in men and alcohol consumption in women. Thus feeling empathy towards others, in particular empathic concern, may not always be health enhancing and may have negative health effects.

At Level 2, physician empathy is seen as a desired quality in health care providers (Ferguson et al., 2002; Kim et al., 2004; Silvester, Patterson, Koczwara, & Ferguson, 2007). Physician empathy is important from a health psychology perspective as patient perceptions' of physician empathy is not only related to the physician's ratings of their own empathy (Glaser, Markham, Adler, McManus, & Hojat, 2007) but also to greater patient compliance and satisfaction (Kim et al., 2004). The concern, however, is that while beneficial to the patient, physician empathy may lead to empathic distress and ill health in the physician (through the mechanisms described above for Level 1). Indeed, studies of family carers show that as carer's become more empathic they are more likely to report depressive symptoms (Kim et al., 2007). With the increased focus of empathic skills in the caring professions and increases in family carer roles (Tonks, 2002), the hypothesis that empathising may have negative health outcomes for health care providers needs to be explored. This has policy implications. Physician selection and training currently emphasise empathy as a key component (Patterson & Ferguson, 2007; Patterson, Ferguson, Lane, & Norfolk, 2005). If empathy is to be selected for or trained in physicians, then contingency within training for the types of negative consequences described above needs to be recognised and skills for dealing with these incorporated into medical training.

A further implication for trait empathy at Level 2 is its role with respect to voluntary behaviour. Ferguson, Farrell, and Lawrence (2008) showed that with respect to blood donation both trait empathy and other regarding preferences did not predict blood donor behaviour or intentions. Rather empathy was important for low-cost helping (e.g., raising money for health-related charities). Theoretically, these findings help to identify the limit of the empathy–altruism hypothesis (low-cost helping behaviours). These findings also have policy implications, indicating that transfusion services – where the behavioural cost is high – are unlikely to increase recruitment by emphasising empathy or altruism. However, for low-cost health volunteering (e.g., fundraising) empathy may be more important (Ferguson et al., 2008; Ferguson, France, Abraham, Ditto, & Sheeran, 2007).

Health anxiety

Health anxiety (HA) represents fears and worries about illness, coupled with bodily preoccupations, in the *absence* of objective illness (American Psychiatric Association, 1994; Kellner, 1986; Warwick & Salkovskis, 1990). The HA has a population prevalence rate in the range of 4.8–7.05% (Creed & Barsky, 2004). At Level 1, HA is

linked to increased incidence of functional somatic syndromes (FSS) and non-specific symptom reporting (Ferguson, 1998, 2004a, 2004b; Gou, Kuroki, & Koizumi, 2001; Noyes et al., 1999, 2005). HA is also linked to 'poorer' stress and coping responses (Ferguson, 2001; Ferguson, Swairbrick, Clare, Bignell, & Anderson, 2000b; Katerngahl, 1999), unrealistic health beliefs (Barsky, Coeytaux, Sarnie, & Clearly, 1993; Barsky et al., 2001), a focus on health goals (Karoly & Lecci, 1993), altered physiological responses (e.g., steeper diurnal cortisol slope; Ferguson, 2008), and reduced pain thresholds (see Gramling, Clawson, & McDonald, 1996).

With respect to Levels 2 and 3, the strong link between HA and symptom reporting is particularly important as symptoms are the key to information people use when deciding to contact the health services (Kroenke, 2001). Those high in HA are more likely to consult physicians (Hiller & Fischter, 2004; Noyes et al., 1999) and exhibit doctor shopping behaviour (Kasteler, Kane, Olsen, & Thetford, 1976). Thus understanding how HA is linked to symptom reporting (Level 1), poor doctor-patient trust (Ferguson, 2000; Level 2), and increased doctor-shopping (Level 3) would offer a major step forward in helping those with HA and saving hospital resources. It should be noted that the association of symptoms with HA remains once N is controlled (Ferguson, 2000).

Type D

The Type D personality is defined as the 'tendency to experience negative emotions and to inhibit self-expression' (Denollet et al., 2006, p. 970), and is a key predictor of cardiovascular disease (CVD; Denollet et al., 1996). There is a wealth of evidence linking Type D to Level 1 outcomes including increased cytokine levels (Denollet et al., 2003), poorer health behaviours (Williams et al., 2008), poorer stress and coping behaviour (Williams, O'Carroll, & O'Connor, 2009), and more negative perceptions of health in patient groups (Mols, Holterhues, Nijsten, van de Poll-Franse, & Lonneke, 2010). However, there is no research on Type D in relation to social cognitive models such as TPB or at Levels 2 and 3, and this is a large gap in the Type D literature. For example, at Level 2, research could explore if there is a 'Detrimental D' effect. That is, does one partner's Type D have deleterious effects on the other partner's health? Further work should also examine the role of Type D with respect to health care utilisation and doctor patient consultation.

Alexithymia

Alexithymia is related to a wide variety of Level 1 outcomes including: (1) increased negative health behaviours (Helmets & Mente, 1996; Lumley, 2004), (2) poorer physical health outcomes (e.g., CVD; Grabe et al., 2010), (3) increased mortality (Kauhanen, Kaplan, Cohen, Julkunen, & Salonen, 1996; Tolmunen, Lehto, Heliste, Kurl, & Kauhanen, 2010), (4) increased psychosomatic illness (see Lumley et al., 1996, 2007; Taylor, 2000), (5) increased risk taking behaviours (Ferguson et al., 2009a; Taylor et al., 1997; Toneatto, Lecce, & Bagby, 2009); and (6) reduced immune system function (Dewaraja et al., 1997). At Level 2 it is associated with poor doctor-patient interactions (Rastling, Brosig, & Beutel, 2005) and at Level 3 to increased health care utilisation (Lumley et al., 2007). For alexithymics, the strength of any affective associations to health behaviours (Kiviniemi, Voss-Humke, & Seifert, 2007)

is likely to be weaker, and as such they are less likely to learn from negative emotional associations associated with poor health behaviours (Ferguson et al., 2009a). Similarly, alexithymia is associated with worse outcomes on emotional-based interventions such as emotional writing (Lumley, 2004; O'Connor & Ashley, 2008).

This section has set out the case that personality (the FFM and health specific traits), has important and clinically significant effects on health. The next section sets this within a theoretical framework.

Section two: towards a theoretical understanding of personality and health

Models of the biology, evolution, and neuroscience of personality

Section one showed that personality traits have a role across the illness process. However, personality traits should not be viewed as simple descriptive and deterministic entities. To extend previous reviews of personality and health this section draws on the biology, evolution, and neuroscience of personality, to set out an initial attempt to develop a theoretical perspective linking personality to health. Specifically the section aims to show how traits are both sensitive and reactive to environmental contingencies and associated with fundamental neurobiological processes. Further, this section sets out how traits can explain behavioural and cognitive (decision making), as well as cultural and physiological response to illness. It is hoped that this initial framework should allow health psychologists to make more precise predictions concerning the role of personality within the health process.

Biology of personality traits

Two theoretical concepts within biology are emerging as central to trait psychology – *sociogenomics and reaction norms*. Both offer a more fundamental understanding of traits, as biological entities, moving away from simple deterministic models (Denissen & Penke, 2008; Penke, Denissen, & Miller, 2007; Roberts & Jackson, 2008; van Oers, de Jong, van Noordwijk, Kempenaers, & Drent, 2005).

The sociogenomic approach is set out by Roberts and Jackson (2008) and based on the assumption that (1) genes and evolution influence all behaviour, (2) genes are conserved across species, and (3) there is a dynamic interaction between genes and the environment (see Robinson, Grozinger, & Whitfield, 2005). This latter point is crucial, with growing evidence indicating that gene expression (transcription and protein synthesis) is influenced by both variation in genes and the environment, rendering a simple nature–nurture dichotomy redundant (Robinson, 2004). Within this approach, environmental factors influence biology that in turn influences personality traits, which serve ‘as the conduit between biology and significant life outcomes’ (Roberts & Jackson, 2008; p. 1534). A major difference in the socio-genomic model is the central role ascribed to ‘state’ levels of beliefs, thoughts, and feelings. Prolonged changes in these states (which may be caused by environmental factors and influenced by traits) are seen as responsible for altering biological systems, which in turn lead to changes in the expression of the trait.

The concept of ‘reaction norms’, developed within behavioural ecology to understand animal personality, describes the relationship between genotype, phenotype, and the environment (see Penke et al., 2007). Reaction norms refer to a typical response function for an individual genotype to a specific context. Van Oers

et al., (2005) define them as the ‘function relating a phenotypic response of a genotype to a change in the environment’ (p. 1197). A classic example of this is Caspi et al.’s (2003) study showing that the association between life stress (environment) and depression (phenotype) was moderated by a polymorphism in the serotonin transporter gene (genotype) – the expression of the phenotype is dependent on the relationship between gene and environment. Recently Dingmanse, Kazem, Reale, and Wright (2010) have extended the idea of reaction norms, to behavioural reaction norms (BRN). BRN aim to explain the link between two key features of ‘traits’ that are often seen and studied as separate entities: (1) stability/difference between people across the average context and (2) plasticity (individual differences in variation across contexts). That is, stability does not imply individuals cannot vary their response and, indeed, stability and plasticity may be correlated, oblique, or indeed a trait may show plasticity but not stability or vice versa. This again highlights that the expression of a trait may vary as a function of context (plasticity), with the individual having a mean level of trait expression across context. This approach emphasises the need to take detailed assessment of the context (and indeed multiple contexts) in which the trait expression is to be observed and multiple indicators of the type of behaviour under study.

The concept of reaction norms avoids the ‘personality paradox’ – personality traits do not show strong cross-situational consistency (Mischel, 1968) – as the situation is part of the theoretical understanding of personality (see Mischel & Shoda, 1995, 1999; Penke et al., 2007). Within their *process-oriented approach* to personality, Mischel and Shoda (1999) drew similar conclusions. They stated that variation in individual behaviour across situations is not attributed to either all error variance or the situation, but is part of a stable dynamic personality system such that a ‘person’s behavior in a domain will necessarily change from one type of situation to another...even when the personality structure remains unchanged’ (Mischel & Shoda, 1999, p. 211). This conceptualisation of personality is set within Mischel and Shoda’s (1995, 1999) Cognitive-Affective-Personality System (CAPS) model. The CAPS framework is based on the assumptions that individuals differ (1) in the chronic accessibility (activation) of affective and cognitive representations and (2) in the interconnections of these chronic accessibilities. The situation is incorporated into the CAPS, as it activates the processing of the affective and cognitive representations. This results in a framework whereby personality is defined as a set of situationally contingent behavioural signatures based on *if...then* associations. Personality remains unchanged but behavioural responses are triggered contingent on the context. Mischel and Shoda (1999) see genetically heritable traits operating to influence the organisation and accessibility of cognitive, affective, and behavioural units.

Evolution of personality traits

Penke et al., (2007) reviewed three potential mechanisms for the evolution of personality concluding that *balancing-selection* based on a *frequency-dependent* model is the most plausible for the evolution of personality. This suggests that extremes of a trait are equally favoured by selection under different conditions, with frequency-dependency favouring a particular ratio of traits within a population.

Specifically a particular trait's fitness decreases as it increases in the population. This optimal ratio produces an evolutionary stable strategy (ESS; MacDonald, 1995).

The balancing selection mechanism implies that each trait has evolutionary costs and benefits, leading Nettle (2006) to develop a *cost-benefit trade-off model* for personality (see also Smith & Blumstein, 2008). This model suggests that all traits have costs and benefits associated with them, with the optimal balance between these depending on the context in which they are expressed. Nettle (2006), for example, identifies social allies as a benefit of extraversion, but a cost being low family stability. Buss (2009) further suggests that personality traits evolved as strategies to solve adaptive problems, with high scores on a trait adaptive for one problem but not another (e.g., high extraversion adaptive for negotiating status hierarchies and low extraversion for extracting resources).

Neuroscience of personality traits

Recently DeYoung et al., (2010) have hypothesised that the FFM domains are linked to fundamental underlying biological processes. Specifically they link E to sensitivity to rewards, N to sensitivity to punishment, A to altruism, C to impulse control, and O to working memory and attention (see also DeYoung, 2010). In support of this, they demonstrated that, using structural MRI, E was associated with variation in regional brain sizes for areas associated with reward (e.g., medial orbito-frontal cortex), N to regions sensitive to threat and punishment (e.g., dorso-medial pre-frontal cortex), A to areas associated with understanding others beliefs (e.g., posterior cingulate), and C to areas associated with planning (e.g., middle frontal gyrus). Evidence for associations with O were less clear with some indication that O was linked to areas associated with working memory and attention in the parietal lobe.

Summary

Drawing the above together, at a very broad level, it may be argued that personality traits evolved into normally distributed traits comprising constellations of behaviours (organised cognitions, affects, and behaviours), linked to fundamental biological processes that carry relative costs and benefits. However, while there may be a mean level of trait expression across contexts, there is also likely variation (plasticity) depending on context (e.g., a BRN) and indeed traits may reflect differential sensitivities to express a behaviour in a particular context. Thus while traits are heritable (Bouchard & Loehlin, 2001), the biology of heritable traits is not one of simple determinism and traits should be viewed as dynamic and environmentally contingent.

The biology/evolution of traits: the implications for health psychology

The model of personality detailed above moves away from description towards explanation and has three broad theoretical implications for health psychology: (1) contextual prediction of trait effects, (2) understanding cultural responses to disease, and (3) cost-benefit analyses of the health traits.

Contextual prediction of trait effects

The above approach allows for more a-priori predictions to be made concerning trait effects. I will illustrate this with respect to a recent paper by O'Connor et al. (2009) examining the role of C with respect to daily hassles and health behaviours within the varying contexts of daily work hassles and multiple health behaviours. This makes a BRN approach viable. Within the framework above, it would be noted that C has costs (obsessionality and rigidity) as well as benefits (planning, long-term fitness; Nettle, 2006) and these may result in different expressions of health behaviours dependent on context (e.g., BRN). O'Connor et al. (2009) observed the usual protective effects for C with respect to lower fat intake. However, they reported 'counter-intuitive' effects with high C linked to higher caffeine intake and increased smoking in smokers. Smoking reflects an additional context, to add to daily work stress. Importantly the facets of C, more likely to be associated with costs (order, self-discipline, and achievement striving), were the stronger predictors of daily smoking (in smokers), with self-discipline moderating the hassles-smoking link, such that those high in self-discipline smoked more when stressed. Thus there is variability (interaction with context: stress and smoking) in the expressed behaviour (smoking) as a function of variation on the trait across contexts. O'Connor et al. (2009) provided an explanation of these unexpected findings in terms of methodological differences to previous studies and smoking as a potential coping mechanism for driven and motivated individuals. However, it is argued here that based on the cost-benefit analysis of traits and the concepts from BRN, it should be possible to make a-priori predictions about pattern of behaviour and, as such, render the finding as not counter-intuitive. Thus contextual sensitivities (e.g., BRN) and cost-benefit analyses are key to understanding the role traits within the health domain.

Personality and the cultural responses to disease

There is evidence that personality can change as a consequence of direct exposure to a disease. For example, personality change (e.g., reduced novelty seeking) has been observed as a function of infection with *Toxoplasma gondii* (Skallová et al., 2005). It has further been argued that personality plays a wider role in the cultural response to illness (e.g., Lafferty, 2006; Schaller & Murray, 2008). Indeed, based on the arguments developed above, culture should be considered a wider environmental context in which personality operates.

Before examining the role of personality in cultural responses to disease, it is necessary to distinguish two aspects of culture: evoked (Tooby & Cosmides, 1992) and transmitted culture (see Gangestad et al., 2006; Nettle, 2009). Evoked culture refers to evolved mechanisms that are responsive to environmental features. These environmental features trigger an adaptive response from the organism's repertoire of evolved behaviours that is best calibrated to the local environmental contingencies (Gangestad et al., 2006; Nettle, 2009). By analogy, evoked culture is often referred to as a 'jukebox' (Tooby & Cosmides, 1992) with the environment acting as the trigger that selects which of the stored tracks is played (Nettle, 2009). Viewed in this way personality traits may be considered part of the psychological mechanisms that underlie evoked cultural responses. This is consistent with the model of personality

described above where the expressed behavioural response is environmentally contingent (e.g., Mischel & Shoda, 1999; Penke et al., 2007).

Evoked culture is seen as distinct from ‘transmitted culture’, which reflects the transmission of ideas, representations, norms, beliefs, and attitudes across individuals and groups via both trial-and-error and social learning (Nettle, 2009). For health psychology, ‘transmitted culture’ reflects the norms, attitudes, beliefs, and behaviours associated with health. It has been argued that these two forms of culture may not be that distinct, as similar evolved mechanisms may be involved in both (Gangestad et al., 2006). Below it is argued that personality may be part of both an evoked and transmitted cultural response to health.

At a cultural level, people are known to change their health behaviours (e.g., food preparation, mate preference) in response to pathogen prevalence (Sherman & Billing, 1999). Similarly, regional differences in personality also reflect pathogen prevalence (Lafferty, 2006; Schaller & Murray, 2008). Schaller and Murray (2008) showed that both E and O are lower in regions with historically high levels of pathogen exposure. They argue that low E and low O reflect traits associated with avoiding social contact and increased conformity to local rules. Both of these behaviours result in reduced pathogen exposure. These regional differences probably involve a mixture of evoked culture and natural selection. That is, personality traits may have evolved with relative costs and benefits (as described above). This evolved personality forms part of an evoked cultural system (see Schaller & Murray, 2008). When pathogen prevalence is high, all people will respond by changing their behaviour; however, those with certain traits (e.g., low E and O) will have an added advantage as aspects of their behavioural repertoire (which are more likely to be evoked in this context) will provide an extra advantage leading to increased survival. Personality traits also influence most aspects of health-related transmitted culture in terms of influences on attitudes, beliefs, and behaviours associated with health (see Table 1) as well as through the *processes* underling transmitted culture such as social learning (Hooker, Verosky, Miyakawac, Knight, & Esposito, 2008). For example, introversion is known to enhance social learning (Hooker et al., 2008) with introversion linked to more positive health behaviours (Raynor & Levine, 2009). Thus, in this wider cultural sense, personality traits can again be envisaged as central to the health process.

Health traits, evolution, and cost benefits

While Nettle (2006) has examined the differential costs and benefits for the FFM, the same has not been done with respect to the health-related traits identified here. For example, what is the benefit of having a Type D personality? The following provides an initial attempt at such an analysis for the four health traits with respect to health outcomes (Table 2).

Importantly each of the four traits has been shown to be heritable and, as such, they should have been open to selection pressures with identifiable costs and benefits (Nettle, 2006). Work on the evolution of empathy is the most established (de Wall, 2008; Preston & deWall, 2002). When considering the costs and benefits of empathy it is useful to consider the distinction between cognitive (CE) and emotional empathy (EE) (Smith, 2006). High EE has benefits associated with making strong emotional bonds, group cohesion, and kin altruism (Preston & de Wall, 2002). High CE is

Table 2. Evolutionary cost-benefit analysis for health-related traits.

	Costs	Benefits	Hypotheses
Emotional empathy	Empathic distress, exploitable	Helping others, reciprocity, kin selection, parental-child social bonds, group cohesion	<i>Mediation model for poorer health: Empathic distress mediates the link between EE and poor health behaviours</i>
Cognitive empathy	Empathic distress	Exploit, deceive, greater social understanding, reciprocity, cheater detection	<i>Alexithymia moderated model: Poorer health behaviour observed only for a combination of high CE and high alexithymia</i>
Health anxiety	Obsessive behaviours, lack of trust	Help seeking, health vigilance, 'reduced mortality', steeper diurnal cortisol slope	Reduced infection exposure and incidence of infection; reduced risk of cancer and other major illness
Type D	Poor health, negative health behaviours, poor social interaction	Vigilance	Reduced infection exposure and incidence of infection
Alexithymia	Poor social interactions, poor health behaviours, poor health, pathological gambling, autistic spectrum, reduced empathy	Risk taking, less exploitable	Increased status, reduced infection exposure

related to benefits arising through reciprocity, the ability to identify cheats, and reduced exploitability (Buss & Duntley, 2008; Smith, 2006). In terms of costs, however, those high on EE are open to exploitation (Buss & Duntley, 2008) and empathic distress. Indeed, Smith (2006) links autism to a combination of high EE and low CE and, conversely, antisocial personality disorder (ASPD) as reflecting low EE and high CE (see also Blair, 2005); indeed, Singer and Lamm (2009) have linked CE to exploiting others.

In terms of health outcomes it might be predicted that both EE (with low CE) and CE (with low EE) should be linked to poor health via different routes: EE should be mediated via empathic distress (see Table 2) and high CE (and low EE) moderated by alexithymia. Those who are able to understand another's point of view (high CE) but are unable to understand their own emotions (high alexithymia) have the potential to be exploitative, unlikely to learn from the emotional consequences of their actions (Ferguson et al., 2009a), and adopt unhealthy behaviours (see Table 1). As such, a combination of high CE and alexithymia should result with antisocial behaviour and poorer health. Whereas high CE with low alexithymia will relate to empathy associated with pro-social behaviour.

The costs of Type D, HA, and alexithymia in terms of poor health behaviours and inhibited social interactions have been articulated. However, are there any adaptive benefits associated with these traits? One potential benefit of Type D is that

it may be associated with reduced exposure to infection. That is, the facets of Type D should enhance reduced exposure to potential infections. Specifically, NA should be linked to increased vigilance for health threats and social inhibitions to avoiding social contact. Indeed, there is some evidence that the SI facet of Type D is related to reduced reporting of symptoms of infection (Smolderen, Vingerhoets, Croon, & Denollet, 2007). Similar benefits may be evident for alexithymia and HA as both are related to social isolation (low E). Thus it would be predicted that higher levels of Type D, HA, and alexithymia should be (1) associated with lower antibodies for infections, (2) associated with avoidance of individuals who are likely to have infections, (3) linked to avoidance of exposure to infectious environments, and (4) more prevalent in areas with high background of pathogen levels.

It has been suggested that anxiety associated with health threats may be beneficial with respect to avoiding them (Lee, Wadsworth, & Hotopf, 2006). HA is similarly related to increased vigilance and memory for health information, this may enhance health seeking and illness avoidance behaviour (Ferguson et al., 2007). HA may also have a protective role with respect to non-pathological illness, as evidence links HA to reduced mortality from non-pathological chest pain (Shekelle, Vernon, & Ostfeld, 1991). The exact mechanism for this is unclear, but one reasonable hypothesis would suggest it may be mediated by health care seeking. Finally there may be some biological benefits from HA. For example, a flattened diurnal cortisol slope is linked to increased risk of cancer (Abercrombie et al., 2004) whereas a higher level of HA is related to a steeper diurnal cortisol slope (Ferguson 2008). Whether or not this would infer some protective function is an empirical question worth pursuing.

There are two implications of the model of personality presented for methods: the use of animal models and assessment of traits.

Animal models of personality and health

The idea of gene conservation opens up the possibility of studying the personality–health link in non-human animals (Roberts & Jackson, 2008). Such a research agenda requires reliable and valid assessments of personality in non-human animals and appropriate animal models of the target disease to examine the relationship between the two. Reliable and valid measurements of personality traits across a wide variety of species are possible (Cavigelli, 2005; Gosling & John, 1999; Mehta & Gosling, 2008 for reviews). Animal personality has been assessed through behavioural responses (e.g., anxiety through defecation in an open field test), observer ratings, or manipulations of trait expression (e.g., anxiety) using selective breeding or genetic modification (Gosling & John, 1999; Mehta & Gosling, 2008 for reviews). Observer ratings are the most widely used and involve owners or keepers rating the animal using carefully chosen adjectives that are sensitive to the animals behaviour and niche (see Gosling, Kwan, & John, 2003). These are supplemented with two or three sentences to describe the interpretation of the adjective within the context of the animal's behaviour (see Weiss, King, & Perkins, 2006). Observational methods show psychometric properties equivalent to judgments of self and peer human personality (Gosling, 2001; Gosling et al., 2003). Anthropomorphising is a concern with such rating. Gosling (2001) and Gosling and John (1999) present the following arguments against this worry: (1) raters can reliably rank order animals on traits and such variability is unlikely with anthropomorphising, (2) factor structures

vary across species, and (3) similar structure emerge for ratings and behavioural coding. Crucially, ratings of animal personality predict future behaviour (Gosling et al., 2003). Thus reliable and valid assessments of animal personality can be achieved. These assessments can be related to a wide variety of animal models of major diseases such as asthma (e.g., Ricciardolo, Nijkamp, De Rose, & Folkerts, 2008) and MS (Tsunoda, Lane, Blackett, & Fujiinami, 2004).

Exploring the effects of personality on health within a specific animal model of disease is, therefore, feasible and the virtues of animal models for examining personality–health links have been set out by Mehta and Gosling (2008). These include greater experimental control, physiological assessment, and reduced time for longitudinal studies. Classic examples of the application of such an approach are provided by Capitano and colleagues, who have demonstrated the predictive roles of personality (Capitano, Mendoza, & Baroncelli, 1999), as well as personality and coping (Capitano et al., 2008), with respect to disease progression in monkeys with simian immunodeficiency virus (SIV) infection. While many of the animal models focus on major disease outcomes, the experience of general non-specific symptoms is of interest to health psychologists. This can be explored in terms of the ‘sickness response’ (Maier & Watkins, 1998). Finally, it is appreciated that a lot of research within health psychology is concerned with social cognition, for which animal models are less useful. However, animal models can be used to explore not only the behavioural *mechanisms* such as coping (see Capitano et al., 2008) and physiological responses (e.g., immune functions) linking personality to illness but also the role of *processes* such as associative processes associated with illness (Ader & Cohen, 1982, 1993). Also the roles of learning and cognition can be explored using procedures and designs from animal learning (MacKintosh, 1985; Walker, 1985). As such, health psychologists should consider exploring personality and health within the context of animal models.

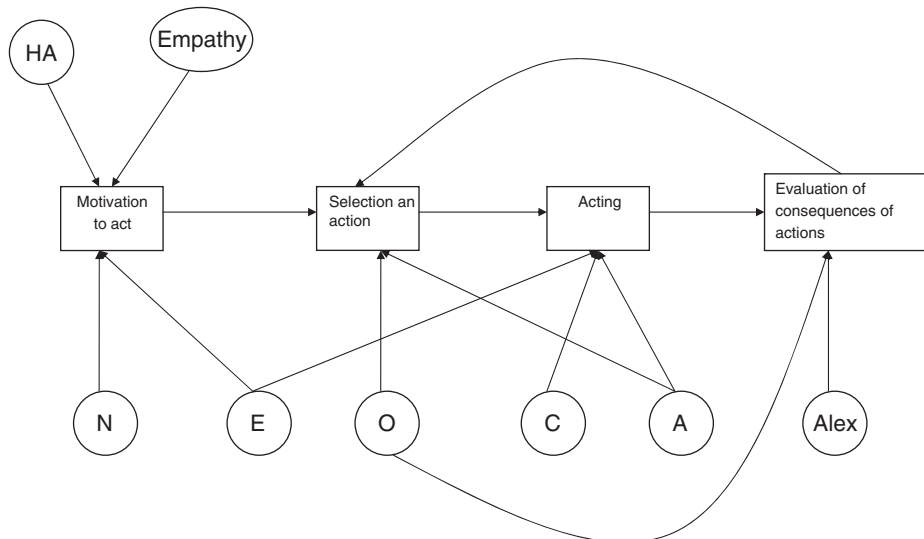
Assessment of traits

The model of personality described here suggests differential adaptiveness dependent on situational contingencies. This suggests that the measurement of personality traits may need some revision. Indeed, Denissen and Penke (2008) have developed an index of the FFM with the domains reflecting individual differences in motivation to react to environmental contingencies. Similarly, Lawrence (2006) developed an assessment of specific traits that show differential responding to environmental contingencies associated with provocation and frustration (Lawrence & Hodgkins, 2009).

Neuroscience of personality: implications for socio-cognitive models in health psychology

While broad theoretical implication arise from the biology/evolution of personality, more specific implications for socio-cognitive models arise from the neuroscience findings. The neuroscientific work of DeYoung et al. (2010) linking FFM domains to biological processes provides a theoretical basis for understanding how traits may be related in socio-cognitive decision making (see Figure 3). The model presented in Figure 3 draws on and develops theoretical models proposed by DeYoung (2010) and Van Egeren (2009). Both DeYoung (2010) and Van Egeren (2009) proposed that

personality traits influence different stages within goal-directed, cybernetic decision-making processes. DeYoung (2010) suggested three stages: (1) motivation to act, (2) selecting an action, and (3) carrying out that action. However, to this may be added evaluation of the action and feedback to earlier stages. These stages fit neatly with the two main stages that underlie most health behaviour models, namely motivation and action stages (Schwarzer & Fuchs, 1996), with motivation and selection constituting the motivational stage and action and evaluation/feedback the action stage. Based on DeYoung et al. (2010) and DeYoung (2010) it is possible to suggest how the FFM and health traits should be related to these stages of decision making. DeYoung (2010) suggests that E should influence motivation and energise action, specifically when the motivation is to approach a goal. Likewise N should influence motivation to avoid. N may also reduce the possibility of being derailed (Gollwitzer & Sheeran, 2006) from a goal by minor threats. DeYoung (2010) also argues that O will be important in the selection of actions, leading to more creative and complex solutions and aiding attention. Given its links to working memory, O may also be important in evaluating the outcomes of actions. Conscientiousness (C) should be important in evaluating between multiple plans of action, avoiding distraction, and ensuring that long-term actions and plans are organised and maintained. Agreeableness (A) was seen by both DeYoung (2010) and Van Egeren (2009) as important for planning and action when this involved group and not personal decision making. Specifically, A is more likely to be important when consideration involves the welfare of others. Similar arguments can be advanced for empathy. However, this motivation may only be translated into action if the costs of action are perceived to be low (see Ferguson et al., 2008; Neuberg et al., 1997). Alexithymia is hypothesised as central to the evaluation process.



Note: N = neuroticism, E = extraversion, O = Openness to Experience, C = Conscientiousness, A = agreeableness, Alex = Alexithymia, HA = Health Anxiety and Empathy = Empathy.

Figure 3. A theoretical model of personality and social cognition.

Specifically, alexithymics show an inability to learn or consolidate information about losses (Bibby & Ferguson, 2010; Ferguson et al., 2009a), with reduced activation in brain regions linked to loss aversion (Mantani, Okamoto, Shirao, Okada, & Yamawaki, 2005). As such, they should be less likely to learn from mistakes and losses and adapt their behaviour accordingly. Hence, alexithymics may get locked into a negative cycle of repeat negative behaviour. Finally, HA should have a salient role to play with respect to motivation to act with respect to all health-related goals. Thus, clear links between traits and health decision-making processes can be hypothesised and tested.

Section three: personality as risk factors for illness: dimension, taxons, and cut-offs

Aims 1 and 2 were addressed above and a case made that personality is central to health psychology. If this is taken on board, then a major clinical issue facing personality assessment within the context of health and medicine concerns its diagnostic and prognostic value. Can personality traits be used to identify those at risk of serious illness? Type D personality, for example, has become defined as a categorical risk factor for CVD, with predetermined cut-off scores (Denollet, 1998; Denollet et al., 2006). To adopt such a definitive diagnostic model it is essential to show that the trait is taxonic (i.e., individuals are differentiated into non-arbitrary groups or categories) rather than dimensional (i.e., distributed as a continuous variable, with individuals varying quantitatively from each other; Ruscio, Haslam, & Ruscio, 2006). The dangers of using arbitrary cut-off scores has been detailed elsewhere (see Ferguson et al., 2009b; Ruscio et al., 2006) and includes misdiagnosis of cases and non-cases and vice versa, reduced statistical power, unnecessary labelling, and applying incorrect theoretical models. If a trait is dimensional, then it should not be arbitrarily split into cases and non-cases and treated as a risk factor (Ruscio et al., 2006). However, it is appropriate to draw distinctions within a dimensional construct as long as these are systematic and empirically justifiable (Ruscio et al., 2006), via identifying inflection points (Kessler, 2002) or the cross-over points for sensitivity and specificity (Ferguson, 2008).

Simply inspecting distributions, applying cluster or factor analysis will not address this assumption of dimensionality (Beauchaine, 2007; Waller & Meehl, 1998). Rather taxometric procedures need to be applied. These represent a set of procedures to demonstrate if the latent trait is either dimensional or taxonic (see Meehl, 1995). Detailed overviews of these procedures have been published and the reader should refer to these for details (Beauchaine, 2007; Ruscio et al., 2006; Waller & Meehl, 2006). The overarching assumption is that personality traits are continuous and normally distributed in the population. There is taxometric evidence for three of the four health traits, showing that alexithymia (Parker, Keefer, Taylor, & Bagby, 2008), HA (Ferguson 2009), and Type D (Ferguson et al., 2009b) are all dimensional and, therefore, should be treated as continuous variables for research, prognosis, and treatment evaluations (Ferguson, 2008; Parker et al., 2008). If Type D personality is to remain a categorical risk factor for CVD, then it is essential that sensitivity and specificity analyses are conducted for varying cut-off scores and that this is combined with the identified inflection points.

Personality and clinical practice

The dimensional nature of traits notwithstanding, personality traits have a role to play in diagnosis and treatment. Specifically traits can be used (1) to identify those potentially at risk and for whom psychosocial interventions should be targeted and (2) as a marker for psychological change as a result of illness and intervention/treatment. With respect to identifying risk, personality traits could be used as an initial screening device (along with other known risk factors) to identify those in need of further exploration (Albus, Jordan, & Hermann-Lingen, 2004). Only traits that can be clearly shown to be related to the target clinical outcome and that have empirically defensible cut-offs should be used. The identified 'at risk' groups should be further examined with more extensive measures that identify potential risk behaviours and mechanisms linked to the specific clinical outcome (see Kupper & Denollet, 2007). These mechanisms, be they behavioural (e.g., exercise), physiological (e.g., cytokine activity), or psychological (e.g., depression) should be individually targeted in a tailored intervention programme (Noar, Benac, & Haris, 2007). In this way, a more complete picture of the patient's risk factors and clinical needs can be addressed.

The second key role for personality within the clinical context concerns the effect of illness on personality. For example, infection with specific pathogens (Skalova et al., 2005) as well as the experience of chronic pain (Lumley et al., 1996) can result in changes in personality. Personality traits should, therefore, be assessed with other outcome measures to gain a complete picture of changes in a patient as a consequence of illness as well as part of the evaluation of interventions. For example, alexithymia is more prevalent in many clinical populations (e.g., Chartzi et al., 2009) and linked to numerous negative health behaviours and consequences (see Table 1). Therefore, knowing about the patient's levels of alexithymia will help to identify appropriate interventions, helping to avoid the negative behaviours linked to alexithymia.

Thus, while personality traits are often viewed as too broad to be useful and not amenable to interventions, I hope that the above shows that when the mechanisms that link traits to illness are identified as well as using empirically robust cut-offs, personality traits can be used to develop individual interventions that should be beneficial to the patient.

General conclusions

It was argued that to establish the central role of personality for health it is not sufficient just to (1) show that traits are associated with the illness process, but (2) to move away from simple deterministic and descriptive models and develop a theoretical framework to explain this link, and (3) show how personality can be used clinically. This review has endeavoured to address these three aims and also extend existing reviews in three ways by (1) showing that personality has a potential influence across the whole illness process (Figure 1); (2) exploring the personality health link within a multi-level perspective, arguing that health psychology needs to move beyond the individual level perspective that dominates at present; and (3) specifying a broad theoretical framework to understand the personality health link and a specific theoretical model linking traits to socio-cognitive processes.

This review has aimed to present an initial attempt to develop a theoretical framework for exploring the role of personality within the health domain. There are

a number of key points that arise from this theoretical perspective. First, traits should *not* be viewed as deterministic entities but rather as an evolved mechanism, with associated cost and benefits, showing differential sensitivity to environmental contingencies in terms of behavioural expression. This is essential as a trait may lead to adaptive responses under some circumstances and not others. Second, the neuroscience evidence shows that the FFM traits are linked to fundamental biological processes that reflect key decision points in many socio-cognitive models used in health psychology, as such more precise predictions can be made concerning the relationship between traits and social cognitive processes. Third, is it necessary to apply evolutionary perspective to understand more conceptually specific traits that are of central importance to health psychology. For example, what is the selective advantage of Type D personality?

Note

1. These figures are after controlling for age, sex race, SES, and personality.

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