GENES, BEATS AND TRAITS:

A MULTI-MARKER EXPLORATION OF PERSONALITY

By

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Abstract

This research explores associations between genetic polymorphisms in dopamine and serotonin systems (DAT1, DRD4 and 5HTTLPR polymorphisms), physiological and environmental variables and multiple personality traits. 113 participants were genotyped, participated in a stressful cross-cultural negotiation exercise and completed personality scales while wearing heart-rate monitors. Heart-rate variability and stressful life events were associated with conscientiousness and neuroticism traits. Contradicting previous research, no reliable gene x stressful life event interactions were found. Gender and ethnicity masked genetic effects on neurotic and sensation-seeking traits, particularly for DAT1 and 5HTTLPR. The DRD4-7R allele was associated with higher agreeableness and lower neuroticism, and contrary to prediction, with lower sensation-seeking. Gene-trait relations are complex, interactionist and multiply-determined, suggesting that personality variation is influenced by – but not reducible to – genetic variation.

Keywords: DAT1, DRD4, 5HTTLPR, personality genetics, physiology, heart-rate variability, stressful life events, gene-trait associations

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Genes, Beats and Traits

Introduction

Personality is a key area of psychological inquiry, as humans try to understand and explain individual and group differences in thought, feeling and behaviour. The Ancient Greeks believed that personality was determined by the four 'humors', namely the bodily fluids black bile, yellow bile, blood and phlegm. The relative balance of these fluids was considered important for healthy functioning, with extreme levels of any humor believed to be dysfunctional. Although 'humorism' was eventually discredited as a medical system, modern developments in biology and molecular genetics suggest that the structure, function and interactions of bodily genes, enzymes, hormones and neurotransmitters have a large influence on personality. The current research aims to explore personality using a multi-method approach including potential genetic and physiological influences alongside an environmental measure that may modulate gene-trait associations. By combining micro (biological) and macro (trait) approaches alongside environmental and physiological measures of stress exposure and reactivity, an interactionist account of personality is proposed and examined.

Last century, psychoanalytic (Freudian) and social learning models of personality were gradually superceded by psychobiological and trait models. In mainstream social psychology, the most influential personality model is the five-factor model ('FFM' or 'Big Five') popularised by Costa and McCrae (1992) with their widely-used 240-item NEO-PI-R instrument. The 'Big Five' is a lexical model based on lists of adjectives that describe personality traits and sub-traits. Five high-level traits emerged via factor analysis: Neuroticism, Extraversion, Conscientiousness, Agreeableness and Openness to Experience/Intellect; each with six lower-level 'facets' (Costa & McCrae, 1992). Many replication and validation studies have supported the universality of this model, with impressive factor congruence and stability across cultures, developmental stages and settings

(Allik, 2005; McCrae et al., 2000; McCrae & Terracciano, 2005). Some gender differences have been identified; females are often higher in Neuroticism and males higher in Extraversion (McCrae et al., 2000; Terracciano et al., 2010). More recently, researchers have begun to investigate the genetic underpinnings of the FFM (Loehlin, 2011; Terracciano et al., 2010; Yamagata et al., 2006)

Despite its popularity, the Big Five has been criticised as a purely descriptive model, which fails to address the *causes* of personality content and structure. In contrast, psychobiological models, guided by animal research, attempt to find the biological bases of personality (Zuckerman, 1991). Most of these models are based around two or three 'super-factors' centred on approach and avoidance stimulus-response tendencies (Elliot & Thrash, 2002). These factors have been variously identified as extraversion and neuroticism (Costa & McCrae, 1992; Eysenck, 1952); behavioural activation, behavioural inhibition and fight-flight-freeze systems (Gray, 1970); positive and negative emotionality and constraint (Tellegen, 1985); and as novelty-seeking, harm avoidance and reward dependence (Cloninger, 1987). Attempts to co-ordinate these different structural models into a comprehensive personality construct are ongoing, but there is strong evidence for a large degree of genetic and phenotypic overlap between models (Savitz & Ramesar, 2004).

The Zuckerman-Kuhlman-Aluja Personality Questionnaire (ZKA-PQ) (Aluja, Kuhlman & Zuckerman, 2010) is a relatively new psychobiological instrument created as an alternative to the Big Five. The five factors in the ZKA-PQ are Neuroticism (NE), Sensation-seeking (SS), Extraversion (E), Aggressiveness (AG), and Activity (AC), which are closely centred on stimulus-response tendencies (Stelmack, 2004; Zuckerman, 1991). This instrument has 200 items in total, with 40 items per factor. In structural analyses of the ZKA-PQ and NEO-PI-R scales (Aluja et al., 2010; Garcia, Escorial, Garcia, Blanch & Aluja, 2012a), ZKA-SS correlated positively with NEO-Extraversion and Openness, and negatively

with NEO-Conscientiousness and Agreeableness. ZKA-NE correlated positively with NEO-Neuroticism. Another study (Garcia, Aluja, Garcia, Escorial & Blanch, 2012b) comparing the ZKA with Cloninger and colleagues' (1993) Temperament and Character Inventory (TCI) indicated that ZKA-SS correlated positively with TCI-Novelty-Seeking and negatively with Harm Avoidance, while ZKA-NE correlated positively with Harm Avoidance.

This pattern of correlations points to two universal approach/avoidance traits; one centred on openness, exploration and extraversion; and another centred on sensitivity to potential threat/harm. Low Conscientiousness is associated with both high approach (SS) and high avoidance (NE) traits, so Conscientiousness may function to 'reign in' maladaptive extremes of release and restraint. Sensation-seeking may be differentiated from Extraversion by its focus on agentic rather than affiliative approach behaviours. Sensation-seeking involves seeking out new/exciting/risky/distracting activities, rather than engaging in social interaction. This distinction is clear in the ZKA facet scales for Extraversion (Positive Emotions, Social Warmth, Exhibitionism, and Sociability) and Sensation-seeking (Thrill and Adventure-seeking, Experience-seeking, Disinhibition, and Boredom Susceptibility-Impulsivity). However, it could be argued that there is much potential overlap between extraversion and sensation-seeking constructs, as well as across other traits. Multivariate genetic analyses of personality have indeed shown that facets used to define one subscale may share a common genetic basis with facets defining other subscales (Ando et al., 2004; Yamagata et al., 2006).

The influence of dopamine and serotonin on personality

Serotonin (5HT) and dopamine (DA) are both monoamine neurotransmitters with varied roles in the brain and elsewhere in the body. Serotonin is especially important in mood, appetite and sleep regulation, while dopamine is important in motivation, reward,

pleasure and movement regulation. Along with other molecular systems, DA and 5HT systems interact to influence and regulate affect, cognition and behaviour. Elliot and Thrash (2008) stress that approach and avoidance processes are essential for successful adaptation to the environment, as they are linked to agentic and affiliative (dopamine-influenced) and anxiety-reduction (serotonin-influenced) motivations. Individuals must learn how to balance and regulate these sometimes competing motivations in their daily lives and interactions.

Hirsch, DeYoung & Peterson (2009) have proposed a personality 'meta-trait' theory whereby serotonin-regulated 'stability' reflects the shared variance of Big Five Conscientiousness, Agreeableness and Neuroticism, while dopamine-regulated 'plasticity' reflects the shared variance of Extraversion and Openness to Experience/Intellect. In this conception, a well-functioning personality is regulated through serotonergic control of negative affect and aggression-impulsivity restraint, and dopaminergic control of approach behaviour and cognition-impulsivity release. This model aligns with the super-factor models, and studies on the genetic basis of personality which associate dopamine with approach/behavioural activation and serotonin with avoidance/behavioural inhibition (Ando et al., 2004; Yamagata et al., 2006). Note that in the meta-trait model impulsivity is influenced by both dopamine and serotonin, while lexical and psychobiological personality models have impulsivity as part of a specific Sensation-seeking (Aluja et al., 2010) or Neuroticism (Costa & McCrae, 1992) facet scale.

Personality Genetics

Recent reviews (Ebstein, 2006; Munafo & Flint, 2011; Reif & Lesch, 2003) suggest that the 'genetic architecture' of personality, like that of psychiatric disorders, is polygenic and moderated by multiple systems inside and outside the body. However, detailed investigation into the genetic basis of personality has proved challenging. Heritability research using twin and adoption studies indicates 30-60% heritability rates for personality

traits, with the bulk of the remaining variance attributed to non-shared environment (reviewed in Turkheimer, 2000) – although this type of research does not examine *which* genes are responsible. Personality molecular genetics was 'born' in 1996 with the publication of three seminal papers (Benjamin et al., 1996; Ebstein et al., 1996; Lesch et al., 1996) which indicated connections between specific personality traits and specific polymorphisms ('common' gene variants present in 1% or more of the population) in serotonin and dopamine genes. The current research considers the effect of three polymorphisms on personality.

The serotonin-transporter-linked polymorphic region (5HTTLPR) polymorphism

The 5HTTLPR polymorphism is a 44 base-pair insertion/deletion in the regulatory region of the serotonin transporter gene in chromosome 17, creating short (S), long (L) and (more rare) extra-long (XL) variants (Delbruck et al., 1997; Gelernter, Cubells, Kidd, Pakstis & Kidd, 1999). The 5HTTLPR-S (short) variant is most often linked to higher Harm Avoidance, anxiety, angry hostility, depression and impulsiveness (Lesch et al., 1996), higher Neuroticism (Benjamin, Ebstein & Belmaker, 2002; Munafo et al., 2009), negative emotionality (Burt, 2008), affective disorders (D'Souza & Craig, 2006; Szekely et al., 2004), lower Openness, Agreeableness and Conscientiousness (Harro et al., 2009); and lower Novelty-Seeking (Serretti et al., 2006). The 5HTTLPR-S allele is thus associated with a range of avoidance traits and heightened emotional reactivity/sensitivity.

The dopamine receptor 4 (DRD4) polymorphism

The DRD4 polymorphism is a 48 base-pair VNTR (variable number tandem repeat) in exon 3 of chromosome 4, found in 2-8 repeat units (Lerman et al., 1998). As there are multiple repeat alleles at the DRD4 VNTR locus there are many options for DRD4 allele and genotype groupings. The 2, 4 and 7 repeats are the most common (Kidd, 2012), but depending on the study, the DRD4 polymorphism is measured in 'long' versus 'short' alleles, or with the 7R allele the lone 'long' variant versus all other alleles. Long alleles have been

variously grouped as 5-8R, 6-8R, 7-8R, or just 7R (with rare variants excluded). The long allele is frequently linked to higher Novelty-Seeking (Benjamin et al., 1996; Benjamin et al., 2000; Ebstein et al., 1996); higher impulsivity and Extraversion, and lower Conscientiousness (Benjamin et al., 1996), lower Harm Avoidance (Serretti et al., 2006), higher thrill-seeking (Campbell et al., 2010); and less negative emotionality and more free play (versus structured) in infants (Laucht, Becker & Schmidt, 2006). The DRD4 long allele has also been linked to Attention Deficit Hyperactivity Disorder (ADHD), pathological gambling and drug abuse, suggesting that it may be a "non-specific vulnerability gene for a range of impulsive, disinhibited and reward-motivated behaviors" (Stelmack & Rammsayer, 2008, p.45).

The dopamine transporter (DAT1) polymorphism

The DAT1 (dopamine transporter) VNTR is a 40 base-pair repeat sequence in the 3' untranslated region of chromosome 5, found in 7-11 repeat units but with the 9R and 10R alleles being by far the most prevalent (Bidwell et al., 2011). This polymorphism has not been investigated as extensively as 5HTTLPR and DRD4, and research is still quite divided as to specific allelic/genotypic trait associations (D'Souza & Craig, 2008; Reif & Lesch, 2003). For example, both the 9R (Das & Mukhopadhyay, 2007) and the 10R allele (Bidwell et al., 2011; D'Souza & Craig, 2008) have been associated with ADHD. The DAT1-9R variant has been linked with both approach and avoidance behaviours (Enter, Colzato, & Roelofs, 2012), lower agentic extraversion (Osinsky et al., 2010), and the 10R with increased novelty-seeking (Kazantseva, Gaysina & Khusnutdinova, 2008; Van Gestel et al., 2002).

The Complexity of Gene-Trait Associations

Some studies have reported conflicting gene-trait associations and many studies have failed to replicate significant findings (reviewed in Ebstein, 2006; Munafo et al. 2003; 2009). For example, 5HTTLPR-S has been linked with lower Harm Avoidance (Samochowiec et al., 2001; Van Gestel et al., 2002), and lower anxiety (reviewed in Savitz & Ramesar, 2004).

DRD4-7R has been associated with higher Harm Avoidance (Szekely et al., 2004), and DRD4-2R and 5R associated with Novelty-Seeking, rather than 7R (Keltikangas-Jarvinen & Salo, 2009). Also, the DRD4 and 5HTTLPR variants are polymorphic in sequence as well as length (Benjamin et al., 1996; Delbruck et al., 1997; Whisman, Richardson & Smolen, 2011), so observed personality associations could conceivably be due to a particular sequence variant, rather than the actual length/size of the gene.

Informed by the idea that the environment moderates the effect of genes – and genes may moderate the effects of the environment (Caspi, Hariri, Holmes, Uher & Moffitt, 2010) – increasing numbers of studies now evaluate gene-gene and gene-environment interactions for personality (reviewed in Keltikangas-Jarvinen & Salo, 2009). Gene-gene interactions have been found even in the absence of main effects (Ebstein, Benjamin & Belmaker, 2000). Savitz and Ramesar (2004) note that DRD4 and 5HTTLPR variants appear to function antagonistically to each other, in that long (6-8R) DRD4 alleles increase Novelty-Seeking and short DRD4 alleles (2-5R) increase avoidant behaviours – but only in the presence of the 5HTTLPR-S allele. However, Benjamin and colleagues (2000) found higher Novelty-Seeking in individuals with the DRD4-7R allele and the 5HTTLPR-L allele, while Kim, Kim, Lee, Kim and Kim (2006) found individuals with DAT1-10R and 5HTTLPR-L reported higher Harm Avoidance.

Polymorphisms associated with specific phenotypes in one gender may not be associated with these phenotypes in others (Reif & Lesch, 2003). For example, Pelka-Wysiecka and colleagues (2012) found a gender dissociation in the DAT1 influence on Temperament & Character Inventory (TCI) co-operativeness, where 10R+ females had lower scores while 10R+ males had higher scores. Laucht, Becker and Schmidt (2006) found DRD4-7R was associated with more exploratory behaviour and higher adolescent Novelty-Seeking in males only, with no effect for females. This gender dissociation extends to

interactive as well as main effects; Van Gestel and colleagues (2002) found DAT1-10R females had higher Novelty-Seeking in the *absence* of DRD4-7R, while DAT1xDRD4 interactions were not significant for males.

These mixed findings are difficult to interpret but suggest that heterogeneous genetic and phenotypic measures, as well as sample characteristics, have a strong effect on personality genetics results. Other environmental and physiological variables may also moderate gene-trait relationships, potentially explaining some of the contradictory results in the literature.

Environmental measures – Stressful Life Events

It is becoming more accepted that environmental experiences can both enhance and suppress genetic influences on personality traits (Burt, 2008). Psychiatric research indicates that behavioural traits such as impulsivity and negative emotionality are risk factors for psychopathology – but these genetically-influenced predispositions may only be activated in certain environmental contexts, for example experiencing parental neglect or exposure to specific stressors (Kazantseva, Gaysina & Khusnutdinova, 2008). Kandler (2012a) reasoned that genetic influences are more important in childhood, whereas environmental factors like social/work roles and normative life transitions are more important in early adulthood, but then reduce in influence as these factors stabilise in mid-life. Kandler et al. (2012b) also stress the importance of gene-environment correlations, where genetically-influenced personality traits influence *which* life events people experience, and *how* they experience them. There is more evidence that personality influences life events rather than vice versa (Kandler et al., 2012b), such that only very extreme life events have a direct influence on personality (e.g. severe abuse/injury/illness, death of a parent at a young age).

In now-classic gene-environment interaction research, Caspi et al. (2003; 2010) identified the 5HTTLPR-S allele as a moderator between stressful life events (SLEs) and depression, providing evidence for genetically-driven differences in stress-sensitivity. The S allele acted as an exacerbator, so that the more stressful life events one had experienced, the more depressive symptoms experienced – but only for individuals with the S/S genotype (Caspi et al., 2003; Karg, Burmeister, Shedden & Sen, 2011). Harro and colleagues (2009) suggest that the S allele produces a general 'negative affect' trait that may translate to clinical disorder in adverse circumstances, while others (Lazary et al., 2008) argue that the S allele only has an effect in combination with other polymorphisms. Although the S/S genotype was associated with higher depression in the Lazary study, some S+ (S/S and S/L) haplotypes were associated with *lower* depression depending on the presence/absence of other 5HT polymorphisms.

Physiological measures – heart-rate variability

Exposure to stressors leads to adaptive responses via the autonomic nervous system (ANS) which interrelates with subjectively-experienced emotions and personality traits (Hellhammer & Schubert, 2012). It seems clear that physiological states influence psychological states, and vice versa (Martens, Greenberg & Allen, 2009). De Geus and Neumann (2008, p.313) note that psycho-physiological testing may advance personality research beyond the limitations of "potentially flawed subjective linguistic self-report", because voluntary control over biological signals is extremely limited. Using physiological measures is therefore a chance to illuminate the biological underpinnings of personality and to eliminate some of the potential biases involved with self-reported personality, mediated as it is by language.

Recent research suggests that high Sensation-seeking individuals have a lower base level of physiological arousal and sensory under-stimulation, leading to increased exploratory behaviours and social engagement compared to low Sensation-Seeking individuals (Tyrka et al., 2007). Low Sensation-seeking is associated with stronger responses to social stressors due to sensory over-stimulation, leading to withdrawal from social situations and increased avoidance of risk and uncertainty (Stemmler & Wacker, 2010). The current research uses a physiological measure of stress reactivity to examine whether individuals who differ in Sensation-seeking and Neuroticism traits also differ in their physiological responsivity both during and after a stressful negotiation task.

Heart-rate variability (HRV) is a non-invasive technique used to examine autonomic nervous system function, and it is often used as an indicator of regulated emotional responding and stress reactivity (Thayer, Ahs, Frederikson, Sollers and Wager, 2012). Low HRV indicates response rigidity and resistance to change, while high HRV indicates response plasticity and is associated with a greater ability to self-regulate, greater behavioural flexibility, and greater adaptability to change (Thayer & Ruiz-Padial, 2006). Low HRV has been associated with low Novelty-Seeking (Di Simplico et al., 2012) and high Harm Avoidance (Puttonen et al., 2008), while high HRV has been associated with high Extraversion (Stemmler & Wacker, 2010) and high Novelty-Seeking (Puttonen et al., 2008). Cardiac tone has also been associated with neurotic traits including anxiety, depression and hostility (reviewed in Martens, Greenberg & Allen, 2009). The ZKA-PQ has not been subject to physiological investigation as yet, so the current research has a unique contribution to make in examining potential HRV associations with this psychobiological personality scale.

Research Questions and Rationale

In the 21st century, new sub-disciplines like personality genetics and personality neuroscience are attempting to study personality beneath the skin – in search of the new ‘humors’. At the

same time, reliance on self-report scales in personality psychology is being bolstered by the use of more direct measures of brain and body activity to index personality. Increasingly complex explanatory models of personality (Cramer et al., 2012; Mischel & Schoda, 1995; Read et al., 2010) are now challenging lexical trait and psychobiological personality models with the inclusion of situational variables, cognitive-affective mediating units, neural networks and trait clusters. The current research uses multiple self-report personality scales and multiple categorisations of genetic variants alongside other potential sources of influence (heart-rate variability, Stressful Life Events, gender, ethnicity) in order to try and clarify the mixed gene-trait findings of previous studies.

Lexical and psychobiological personality models are compared in terms of their trait associations with genes, physiology and environment. I focus on Neuroticism and Sensation-seeking traits, which appear to have the strongest biological foundation in the dopaminergic and serotonergic systems, and are thus expected to have the strongest associations with the analysed DA and 5HT polymorphisms (Savitz & Ramesar, 2004). The IPIP-50 scale (International Personality Item Pool: Goldberg, 1992) will be used alongside the ZKA scales as a (short) measure of the Big Five in the lexical trait approach. The use of a Big Five scale means that potential HRV and SLE associations with personality traits other than Sensation-seeking and Neuroticism can also be investigated.

The use of expanded genotype categories and gene-gene interactions has the potential to reveal associations that may have been masked by simple single allele +/– categorisation. Conflicting assumptions of allelic dominance using this two-group genotyping have resulted in differential (mis)coding and analysis of heterozygotes, clouding associations with other variables. In the absence of clear molecular evidence for the dominance of a particular allele in the three polymorphisms under consideration (Munafo & Flint, 2011; Reif & Lesch, 2003), running alternative models is ideal. For example, for the 5HTTLPR polymorphism this means

S+/S–, L+/L–, and S/S, S/L, L/L groupings. If a specific genotype is strongly associated with facet scales from different personality traits, this might help to explain the plethora of contradictory and null findings in the gene-trait literature – real and significant variance has been hidden by composite measures of genes and traits. Even within the same personality trait, individuals may score very high for one facet and very low for another. This results in an intermediate mean score and a probable null genetic association when using the higher-level factor score and allele present/absent categories only. For example, agentic and affiliative extraversion may oppose each other: an individual might be highly pro-social but low Sensation-seeking, or highly anti-social and high Sensation-seeking; a distinction lost if these characteristics are subsumed in one 'Extraversion' factor (Elliot & Thrash, 2002).

The current research will use a convenience sample of university students from Victoria University of Wellington, New Zealand. In order to maximize the stressfulness of the negotiation task, and to identify potential ethno-cultural group differences in personality trait/genetic/physiological profiles, a 'domestic' New Zealand European/Caucasian student and an 'international' East Asian student will be paired for each lab study. Associations of specific personality traits with specific DAT1, DRD4 and 5HTTLPR genotypes and haplotypes will provide support for previous research that has identified these connections, and should encourage further study in this area. Using multiple gene variants and multiple personality measures should increase the power of the study to find significant associations, particularly interactions. However, with a relatively small expected sample size of 100-200, it may be difficult to examine all the predicted gene-physiology-environment-trait associations, given likely genotype distributions and gene-trait effect sizes (Kidd, 2012; Munafo & Flint, 2011).

Hypotheses

The current research explores associations between three gene variants, heart-rate

variability, Stressful Life Events and personality traits. Multiple personality measures are used to cover a broad spectrum of behavioural phenotypes and lower-level ZKA facet scales will also be examined in an exploratory (non-hypothesis-driven) fashion in order to address the lack of consistent, detailed measurement of facet-level genetic/environmental/ physiological associations in previous personality research. It is hypothesised that the ZKA-PQ and IPIP-50 personality measures will be correlated, with ZKA Sensation-seeking positively and moderately correlated with IPIP Extraversion and Openness/Intellect, and ZKA Neuroticism negatively and strongly correlated with IPIP Emotional Stability (the opposite 'pole' of Big Five Neuroticism). It is expected that Sensation-seeking–Extraversion correlations will be weaker than Neuroticism–Emotional Stability correlations, due to the greater heterogeneity of the Extraversion construct (i.e. the combination of agentic and affiliative behaviours). No specific hypotheses are made for facet-level correlations, but these will be reported and interpreted.

Informed by the previously-cited literature and using multiple allele/genotype categories in alternative models, it is predicted that participants in the DRD4 long/7R+ group will have higher Sensation-seeking scores than those in the short/7R– group, and the 5HTTLPR-S+ group will have higher Neuroticism scores than the S– group. The DAT1-9R+ group will have higher Neuroticism and lower Sensation-seeking scores than the 9R– group. DRD4-7+ with 5HTTLPR-S+ will have high Sensation-seeking and high Neuroticism scores, while DRD4-7R– with 5HTTLPR-S– will have low Sensation-seeking and low Neuroticism scores. DRD4-7R+ with DAT1-9R+ will have low Sensation-seeking scores, and 5HTTLPR-S+ with DAT1-9R+ will have high Neuroticism scores. Due to the complexity of three-way interactions, no specific hypotheses are proposed concerning DAT1xDRD4x5HTTLPR interactions.

It is hypothesised that females will have higher ZKA Neuroticism and lower IPIP Emotional Stability scores, while males will have higher ZKA Sensation-seeking scores. No gender differences are expected for heart-rate variability or Stressful Life Event scores. Post-negotiation HRV will be higher than during-negotiation HRV, and higher Neuroticism will be associated with lower HRV during both periods. The 5HTTLPR-S+ group will have lower HRV, and the 5HTTLPR-S– higher HRV, during both periods. No specific hypotheses are made for the association of DRD4 or DAT1 genotypes with HRV.

A higher number of reported SLEs will be associated with higher ZKA Neuroticism and lower IPIP Emotional Stability, but this association will be moderated by 5HTTLPR genotype. Fewer reported SLEs will be associated with higher ZKA Sensation-seeking and IPIP Extraversion, with the association moderated by DRD4 genotype. No specific hypotheses are made for the association of DAT1 genotype with SLEs.

Summary

The current research examines the main and interactive effects of three gene variants, heart-rate variability, and the experience of stressful life events, on personality traits. Few if any previous studies into personality have combined self-reports, physiological, environmental and genetic measures in a single multi-method research project. If the hypotheses are borne out by the data, this would indicate that genes, physiology and environmental experiences are important influences on personality. The pattern of associations may prove informative for future research using more complex network and cluster models of personality. This study may also provide support for the more optimistic view of personality genetics, i.e. that specific gene variants have a measurable effect on personality traits – in contrast with the pessimistic view of no reliable associations, or an inability to measure them (Munafo & Flint, 2011).

Method

Participants

120 students from Victoria University of Wellington, New Zealand, completed the study, with full data available from 59 gender-matched pairs. Participants were either first year Psychology students from the Introduction to Psychology Research Programme (IPRP) who were invited to participate, or international students approached in person on campus during International Orientation Week. Two students (one international, one domestic) completed the survey and genetic measures but did not engage in the negotiation exercise. The final sample was 71.6% female, 48.3% NZ European/Caucasian, 38.3% Asian/Pacific and 13.3% Other European/Caucasian, and the age range was 17-43 years old (M = 21.19, $S.D.$ = 5.41).

Instruments

Personality: Zuckerman-Kuhlman-Aluja Personality Questionnaire

Sensation-seeking (SS) and Neuroticism (NE) traits were measured via 60 items from the SS and NE sub-scales of the Zuckerman-Kuhlman-Aluja Personality Questionnaire (ZKA-PQ) (Aluja, Kuhlman & Zuckerman, 2010) - see Appendix A for the specific items used. Thought/belief-focused statements were rated on a 4-point Likert scale, from 1 = strongly disagree to 4 = strongly agree, for example "I sometimes feel depressed". Three of four 10-item facet scales from each sub-scale were used: the Sensation-seeking 'Thrill-seeking', 'Experience-seeking' and 'Disinhibition' scales, and the Neuroticism 'Anxiety', 'Depression', and 'Low Self-Esteem' scales[1].

Personality: International Personality Inventory Pool

Big 5 personality traits (Conscientiousness, Agreeableness, Neuroticism, Openness to Experience/Intellect, and Extraversion) were measured via the 50-item International Personality Inventory Pool (IPIP-50) (Goldberg, 1992) - see Appendix B for the specific

[1] The SS 'Boredom-Susceptibility' and NE 'Dependency' facets had sub-standard reliability in previous testing (Aluja et al., 2010) and were not used in the current research.

items used. Behaviour-focused statements were rated on a 5-point Likert scale, from 1 = very inaccurate to 5 = very accurate, for example "Get chores done right away".

Negotiation Measure: Towers Market Task

Participants engaged in a verbal negotiation exercise which functioned as a mild stressor. The Towers Market task (Weingart, Bennett & Brett, 1993) is a standardised multi-issue exercise used to study negotiation tactics and performance in dyads and larger groups. This measure was chosen to expose participants to a realistic social stressor, in order to monitor the size and direction of changes in heart-rate and heart-rate variability during and after a stressor.

Environmental Measure: Stressful Life Events (SLE) Scale

A 10-item Stressful Life Events scale measured how many (if any) of ten stressful life events had occurred during the previous six months, for example "Have you suffered from a serious illness, injury or an assault?" This measure uses a 'yes/no' item format and was adapted from a published scale (Brugha, Bebbington, Tennant & Hurry, 1985) to be more relevant to a student sample - please see Appendix C for a full list of items.

Physiological Measure: Heart-Rate Variability

Heart-rate variability (HRV) was measured using POLAR2 mobile heart-rate monitors, straps, base station and associated software (POLAR Electro, Finland). The monitors provided a continuous measure of each participant's heart-rate over the course of the laboratory session, which ranged from 40 to 75 minutes. The researcher explained how to fit the monitors around the torso/breastplate using an adjustable elastic strap, and each participant fitted their own monitor under their clothing in a nearby bathroom. After wetting the strap to ensure appropriate signal conductivity, the plastic heart-rate monitor is clipped to the strap in the centre of the breastplate, with a beeping sound and green flashing light indicating successful operation (see Figure 1).

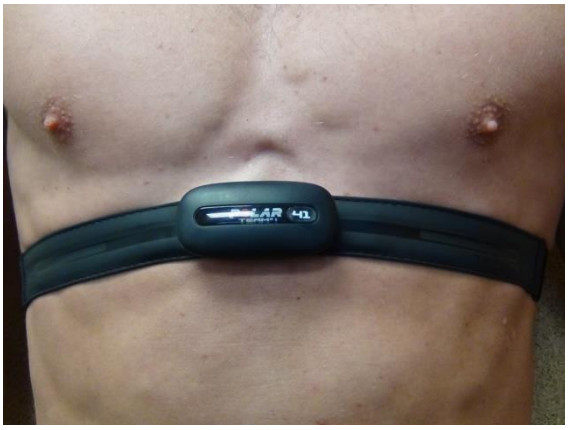

Figure 1. POLAR2 mobile heart-rate monitor and strap fitted on a male model

The Kubios programme (Kubios Software, University of Eastern Finland) was later used to analyse the heart-rate data in order to produce specific measures of heart-rate variability. Software-defined moderate artifact correction was applied to account for ectopic beats and other sources of error. Time-domain series parameters were used to calculate beat-to-beat (BTB) intervals and the root mean square of successive [BTB] differences (RMSSD) for each participant during two 3-minute periods during the lab session. These two periods were the first 3 minutes of the negotiation activity (negotiations ranged from 3-15 minutes), and the first 3 minutes immediately after the negotiation activity, when participants were filling out the final online survey items.

Genetic Measures

Genomic DNA was isolated from buccal cells, collected and analysed for variation at the specific polymorphic regions using protocols described for DAT1 (Enter, Colzato & Roelofs, 2012), DRD4 (Keltikangas-Jarvinen et al., 2003), and 5HTTLPR (Whisman, Richardson & Smolen, 2011). All genetic measure procedures required optimisation to ensure successful collection, extraction and amplification of DNA products and clear discrimination of genetic polymorphisms. Please see Appendix G for a description of genotyping protocol development.

DNA Collection

113 participants provided a genetic sample for DNA analysis. Participants used the 'brush' end of an interdental brushpick (The Doctor's Brushpicks, USA) to gently scrape the inside of their cheek to collect genetic material (DNA). The brush was placed in a 1.2ml hinge-capped plastic collection tube containing 200µl Chelex100 buffer (Bio-Rad, USA), which keeps the DNA stable during storage. The handle of the pick was cut off with scissors and discarded, and the sealed tube was kept frozen at -20°C until required for analysis. The scissors were cleaned with 70% ethanol between participants to prevent cross-contamination of samples. Participants provided two samples each, in case of technical/biochemical failure, and each sample was labeled with participant ID and collection date.

Primer Description and Stock Preparation

Six ABI fluorescent primers (Applied Biosystem Instruments, USA) were made from custom oligonucleotide sequences provided by Dr. Andrew Smolen of the Institute for Behavioral Genetics (IBG) at the University of Colarado, USA, as listed below. Each forward/reverse primer pair 'cuts' out the specific sequence of interest from the DNA sample, before the sequence is amplified via polymerase chain reaction (PCR). Each forward primer is tagged with a sequence-specific fluorescent dye from the ABI DS-33 dye set (6FAM=blue, VIC=green and NED=yellow), in order to discriminate each gene polymorphism when they are pooled together in a multiplex PCR (i.e. one participant = three reactions in one tube).

- DAT1 Forward (+) primer: **6FAM**-TGT-GGT-GTA-GGG-AAC-GGC-CTG-AG
- DAT1 Reverse (–) primer: CTT-CCT-GGA-GGT-CAC-GGC-TCA-AGG
- DRD4 Forward (+) primer: **VIC**-GCT-CAT-GCT-GCT-GCT-CTA-CTG-GGC
- DRD4 Reverse (–) primer: CTG-CGG-GTC-TGC-GGT-GGA-GTC-TGG
- 5HTTLPR Forward (+) primer: **NED**-ATG-CCA-GCA-CCT-AAC-CCC-TAA-TGT
- 5HTTLPR Reverse (–) primer: GGA-CCG-CAA-GGT-GGG-CGG-GA

Concentrated stock solutions of each primer (supplied at 10nm) were made by adding 50µL of ph8.0 Tris-EDTA (TE) buffer (Sigma-Aldrich, USA) to each primer vial, giving 200µM concentrations. Much weaker (10µM) working primer solutions were prepared to use directly in the PCR reactions, by 20x dilution with double-distilled water (ddH20); 10µL of concentrated stock primer was added to 190µL ddH2O to make 200µL volumes of each working primer. All primers were kept frozen at -20°C until required for use.

DNA Extraction

Samples were thawed in a 95°C heat-block for 10 minutes, then centrifuged at 6000xg (relative centrifugal force) for 2 minutes. Using a pipette, 4µL of DNA was carefully extracted from each tube, avoiding the Chelex beads now spun to the bottom of the tube. Leftover materials (original collection tubes with Chelex and brushpick heads remaining) will remain in storage for up to three years, after which time they will be securely destroyed according to Victoria University School of Biological Sciences protocols.

PCR Amplification

After discussion and thorough testing as described in Appendix G, Dr. Ryan Steel (Victoria University School of Biological Sciences) adapted the published IBG protocols (http://ibgwww.colorado.edu/genotyping_lab/protocols.html) for use with the Kapa2G Robust PCR kit (Kapa Biosystems, USA).

Multiplex Recipe

All recipe measurements are in µL. The recipe listed is for a single 20µL multiplex reaction (i.e. one participant) and can be scaled up appropriately for multiple reactions (increasing/reducing the ddH20 as required).

- Primers: DAT1 + (1.2) – (1.2), DRD4 + (1.2) – (1.2), 5HTTLPR + (1.2) – (1.2)

- Reagents: Kapa2G Robust polymerase (0.16), dNTPs (0.4), GC Buffer (4), Enhancer (4), DMSO (1)
- DNA Template: Sample DNA (4)
- ddH20 (to make up to desired volume of 20µL): (5.24)

PCR settings

The PCR amplification was conducted on a Bio-Rad iCycler (Bio-Rad, USA) using a 35-cycle programme with denaturation at 95°C, annealing at 60°C, and a final 10-minute 72°C extension. Please see Appendix G for more detail on the exact steps in the programme. After amplification, 10µL of each multiplex sample was run for 45 minutes in a 1.2% agarose gel at 90mv to electrophorese the samples, allowing visualisation of amplicons (allelic repeat sequences) under UV light. All sample lanes and the DNA ladder (for sizing accurate to 50 base-pairs) visualised and lanes running negative controls (Chelex only, no DNA) were empty.

Genotyping via Capillary Separation

1.5µL volume multiplex samples at 1:1 (i.e. no) dilution were sent to New Zealand Genomics Ltd. (NZG) for capillary separation in order to make precise allele size calls accurate to 1 base-pair. All 113 participant samples were successfully allele-called for each of the three genetic polymorphisms under study. This allowed participants to be coded and analysed by allele, genotype (e.g. DRD4: 4/7) and haplotype (e.g. DAT1-DRD4-5HTTLPR: 9/10-4/7-S/L). Alleles in this study were called at the following base-pair sizes in GeneMarker (Applied Biosystems, USA) software:

- DAT1 amplicons: 8R=390, 9R=435, 10R=474, 11R=513
- DRD4 amplicons: 2R=271, 3R=319, 4R=367, 5R=415, 6R=463, 7R=521
- 5HTTLPR amplicons: S=369, L=411, XL=493

Procedure

Participants were invited for a one-hour laboratory session where they undertook a negotiation exercise with another student, had their heart-rate monitored, completed survey scales and provided a DNA sample. On arrival at the lab, participants were given a briefing sheet outlining the nature and purpose of the research, and given an opportunity to ask questions before providing informed consent to participate. Participants were advised that they could withdraw from the study up to a week after their participation, with no penalty of any kind. Participants were then given verbal instructions for fitting their heart-rate monitors, and their participant ID number and heart-rate monitor number was noted down. Once fitted, the two participants sat opposite each other at a table, each facing a laptop computer, with the survey measures pre-loaded onscreen. On completion of the personality scales, they engaged in a short negotiation exercise which lasted from 3 to 15 minutes. After the negotiation exercise they answered additional survey measures[2] that do not form part of the current thesis. Once finished, participants were taken to a nearby kitchen area where a buccal cell sample was taken for DNA analysis. Participants were finally thanked, debriefed, and given NZ$20 worth of supermarket vouchers as an appreciation for completing the study. Participants were advised that a summary of the study findings would be available to them via email after collation and analysis of the results. Please see Appendices D, E and F for copies of the briefing sheet, consent form and debriefing sheet used.

Ethical approval

Ethical approval for the research was obtained from the Human Ethics Committee and School of Psychology Ethics Committee at Victoria University of Wellington, New Zealand.

[2] Including a post-negotiation survey, and cultural and biological essentialism scales.

Results

Personality, Heart-Rate Variability and Stressful Life Events: Descriptives

The IPIP-50 and ZKA-PQ are established personality instruments, with acceptable validity and reliability as tested by previous authors (Aluja et al., 2013; Garcia et al., 2012a, 2012b; Goldberg, 1992). In this sample, two of the ZKA facet scales had rather poor reliability (SS_Experience-seeking with .67 and SS_Disinhibition with .42), as shown in Table 1 below.

Table 1. Descriptive Statistics and Reliabilities

Variable	N	Scale	Mean	SD	α
NE_ANX	117	1-4	2.32	0.57	.82
NE_DEP	113	1-4	2.35	0.58	.78
NE_LSE	115	1-4	2.26	0.67	.81
NE_Total	109	1-4	2.31	0.56	.92
SS_THR	114	1-4	2.64	0.63	.81
SS_EXP	112	1-4	2.86	0.53	.67
SS_DIS	113	1-4	2.68	0.55	.42
SS_Total	107	1-4	2.73	0.46	.79
IPIP-Extra	119	1-5	3.23	0.76	.86
IPIP-Agree	119	1-5	3.98	0.58	.81
IPIP-Cons	119	1-5	3.40	0.68	.82
IPIP-EmoStab	119	1-5	2.97	0.81	.87
IPIP-Intel	119	1-5	3.62	0.57	.78
N_MeanHR	109	N/A	89.13	15.06	N/A
PN_MeanHR	108	N/A	83.00	15.25	N/A
N_RMSSD	110	N/A	35.16	19.55	N/A
PN_RMSSD	110	N/A	38.33	20.76	N/A
SLE_Total	119	0-20	2.57	1.77	N/A

Participants showed significant differences between negotiation (N) and post-negotiation (PN) mean heart-rate (HR): $t(107) = 10.79$, $p < .001$, and between negotiation and post-negotiation heart-rate variability (RMSSD): $t(109) = 3.80$, $p < .001$. Mean heart-rate was higher and HRV was lower during the negotiation, indicating that participants did find the negotiation task (moderately) stressful.

Descriptives by Gender and Ethnicity

When mean scores were compared by gender and ethnic group using ANOVA, a number of differences were apparent, as shown in Table 2. Gender showed a significant between-subject

effect for NE_Total: $F(1, 117) = 3.95$, $p < .05$, with females higher in Neuroticism than males. For IPIP Intellect, males had a higher mean score than females: $F(1, 117) = 4.46$, $p < .05$. When comparing the participants by ethnicity, IPIP Extraversion was higher for Caucasians than Asian/Pacific participants: $F(1, 117) = 8.62$, $p < .01$. The Asian/Pacific group had a higher mean heart-rate than Caucasians, during both the negotiation period: $F(1, 107) = 4.96$, $p < .05$, and the post-negotiation period: $F(1, 106) = 4.42$, $p < .05$. No other variables displayed significant differences by gender or ethnicity, and there were no gender by ethnicity interaction effects.

Table 2. Descriptive Statistics by Gender and Ethnicity

	Male (*n*=29 to 34)		Female (*n*=81 to 86)		Caucasian (*n*=69 to 74)		Asian/Pacific (*n*=41 to 45)	
Variable	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
NE_ANX	2.16	0.67	2.38	0.52	2.36	0.54	2.24	0.62
NE_DEP	2.21	0.66	2.41	0.55	2.37	0.55	2.33	0.65
NE_LSE	2.07	0.85	2.33	0.57	2.29	0.71	2.20	0.59
NE_Total	**2.15***	**0.70**	**2.37***	**0.49**	2.34	0.55	2.25	0.58
SS_THR	2.62	0.82	2.64	0.54	2.71	0.58	2.52	0.69
SS_EXP	2.77	0.68	2.90	0.45	2.89	0.49	2.82	0.59
SS_DIS	2.65	0.81	2.69	0.42	2.75	0.54	2.57	0.58
SS_Total	2.68	0.66	2.74	0.36	2.78	0.40	2.63	0.55
IPIP-Extra	3.15	0.80	3.27	0.75	**3.39***	**0.76**	**2.98***	**0.70**
IPIP-Agree	3.93	0.59	4.00	0.58	4.00	0.63	3.95	0.49
IPIP-Cons	3.58	0.60	3.33	0.70	3.45	0.69	3.33	0.67
IPIP-EmoStab	3.17	0.86	2.90	0.79	2.96	0.84	2.98	0.77
IPIP-Intel	**3.80***	**0.61**	**3.56***	**0.54**	3.70	0.56	3.50	0.57
N_MeanHR	93.38	17.04	87.60	14.08	**86.68***	**13.68**	**93.20***	**16.48**
PN_MeanHR	87.66	20.09	81.37	12.90	**80.67***	**13.80**	**89.96***	**16.89**
N_RMSSD	31.48	18.18	36.48	19.96	37.17	20.32	31.79	17.92
PN_RMSSD	33.94	18.95	39.90	21.26	40.59	21.13	34.54	19.79
SLE_Total	2.82	1.96	2.48	1.70	2.47	1.78	2.73	1.76

*Variables exhibit a significant ($p < .05$) mean difference between groups.

Personality, Heart-Rate Variability and Stressful Life Events: Correlations

Table 3 details the two-tailed Pearson correlations between personality, heart-rate variability and stressful life events scores. The ZKA Sensation-seeking and Neuroticism facets had low-moderate positive correlations ($r(112) = .18$ to $.32$), indicating that there is some overlap between these constructs. Within the IPIP scale, moderate positive intra-correlations were

found for Extraversion, Agreeableness and Intellect (r(118) = .23 to .35). Extraversion correlated moderately with SS_Thrill-seeking, SS_Disinhibition and SS_Total (r(118) = .29 to .31), but not SS_Experience-seeking. Experience-seeking had moderate correlations with IPIP Conscientiousness (negative) and Intellect (positive). In line with expectations, IPIP Emotional Stability had strong negative correlations with all ZKA Neuroticism facets and total Neuroticism (r(118) = -.56 to -.64). Interestingly, Conscientiousness had moderate negative correlations with two ZKA NE facets (Depression and Low Self-Esteem) and total Neuroticism (r(118) = -.27 to -.38), suggesting that high Conscientiousness may be a protective factor against negative emotions and beliefs about the self and the world. Conscientiousness also had moderate negative correlations with SS_Experience-seeking, SS_Disinhibition, and SS_Total (r(118) = -.22 to -.25), indicating that high Conscientiousness also protects against impulsive, hedonic actions. Meanwhile, IPIP Intellect had low to moderate positive correlations with all ZKA SS facets and total Sensation-seeking (r(118) = .19 to .27), suggesting that openness and curiosity are likely to co-occur with exploratory and sensation-seeking activity.

In line with the literature associating high heart-rate variability with low neuroticism, both HRV measures correlated moderately and negatively with all four of the ZKA Neuroticism measures (r(109) = -.21 to -.31). However, there was no HRV correlation with IPIP Emotional Stability, suggesting that the ZKA and IPIP measures of neuroticism are not interchangeable. However, heart-rate variability was positively correlated with Conscientiousness: N_RMSSD r(109) = .31, and PN_RMSSD r(109) = .29; suggesting that this trait may be linked to physiological reactivity. The Stressful Life Events measure had low negative correlations with Conscientiousness: r(119) = -.22, Emotional Stability: r(119) = -.20, and post-negotiation HRV: r(109) = -.19; an intriguing pattern which is explored in the discussion.

Table 3. Personality, Heart-Rate Variability and Stressful Life Events Pearson Correlations

Variable	NE DEP	NE LSE	NE Total	SS THR	SS EXP	SS DIS	SS Total	IPIP Extra	IPIP Agree	IPIP Cons	IPIP Emo Stab	IPIP Intel	N RMSSD	PN RMSSD	SLE Total
NE_ANX	**.80****	**.75****	**.91****	.04	.16	**.27****	**.19***	-.11	.00	-.14	**-.61****	.09	**-.26****	**-.21***	.01
NE_DEP		**.79****	**.93****	.14	**.29****	**.32****	**.30****	-.04	.02	**-.27****	**-.62****	.11	**-.28****	**-.23***	.09
NE_LSE			**.92****	.02	**.18***	**.31****	**.20***	-.07	.05	**-.38****	**-.56****	-.02	**-.31****	**-.26****	.11
NE_Total				.07	**.23***	**.32****	**.25****	-.08	.03	**-.29****	**-.64****	.06	**-.31****	**-.26****	.08
SS_THR					**.48****	**.43****	**.80****	**.31****	.08	-.08	-.06	**.19***	.11	.09	.13
SS_EXP						**.59****	**.83****	.07	.17	**-.25****	-.12	**.23***	.02	.04	-.07
SS_DIS							**.82****	**.31****	.10	**-.22***	-.15	**.25****	.01	.02	-.03
SS_Total								**.29****	.14	**-.22***	-.14	**.27****	.06	.06	.03
IPIP-Extra									**.35****	-.04	.06	**.23***	.09	.04	.03
IPIP-Agree										-.14	.10	**.28****	-.02	.03	-.17
IPIP-Cons											.15	.00	**.31****	**.29****	**-.22***
IPIP-EmoStab												-.03	.16	.11	**-.20***
IPIP-Intel													-.04	-.05	-.06
N_RMSSD														**.91****	-.15
PN_RMSSD															**-.19***

**Correlation is significant at the *p*<.01 level (2-tailed). *Correlation is significant at the *p*<.05 level (2-tailed). Cell *N*s = 109 to 119.

Genetic Data: Descriptives by Total Sample and by Gender and Ethnicity

Tables 4-6 display allele and genotype distributions for participants with genetic data available (N=113). Allele and genotype frequencies varied by both gender (unexpected) and ethnicity (expected). Long DRD4 alleles (5-7 repeats) were more common in Caucasians (15.7%) than Asian/Pacific participants (8.2%), while short 5HTTLPR alleles (S) were more common in Asian/Pacific participants (54.7%) than Caucasians (47.1%). Mirroring these results, short DRD4 genotypes (DRD4 2/2 and 2/4) were more prevalent in Asian/Pacific participants (23.2%) than Caucasians (12.8%), and the short 5HTTLPR genotype (S/S) was more common in Asian/Pacific participants (28.0%) than Caucasians (21.4%). DAT1 allele and genotype distributions did not differ significantly by gender or ethnicity, however the rare 8R and 11R alleles were only found in the Caucasian group. Females had more 5HTTLPR S alleles (53.0%) and S/S genotypes (27.7%) than males (38.3% and 13.3% respectively). Females also had more short DRD4 alleles (18.1%) and genotypes (21.7%) than males (6.7% and 3.3% respectively).

Table 4. Allele (N=226) and genotype (N=113) distributions for all participants

DAT1 n (%)		**DRD4 n (%)**		**5HTTLPR n (%)**	
Allele	**Genotype**	**Allele**	**Genotype**	**Allele**	**Genotype**
8R = 1 (0.4)	8/9 = 1 (0.9)	2R = 23 (10.2)	2/2 = 2 (1.8)	S = 113 (50.0)	S/S = 27 (23.9)
9R = 53 (23.5)	9/9 = 8 (7.1)	3R = 11 (4.9)	2/4 = 17 (15.0)	L = 111 (49.1)	S/L = 55 (48.7)
10R = 170 (75.2)	9/10 = 36 (31.9)	4R = 161 (71.2)	4/4 = 58 (51.3)	XL = 2 (0.9)	L/L = 29 (25.7)
11R = 2 (0.9)	10/10 = 66 (58.4)	5R = 3 (1.3)	4/7 = 17 (15.0)		S/XL = 2 (1.8)
	10/11 = 2 (1.8)	6R = 2 (0.9)	3/4 = 7 (6.2)		
		7R = 26 (11.5)	3/7 = 4 (3.3)		
			4/5 = 3 (2.7)		
			7/7 = 2 (1.8)		
			2/7 = 1 (0.9)		
			2/6 = 1 (0.9)		
			4/6 = 1 (0.9)		

Table 5a. Allele distribution by ethnicity

DAT1 n (%)		DRD4 n (%)		5HTTLPR n (%)	
Caucasian n=140	**Asian/Pacific n=86**	**Caucasian n=140**	**Asian/Pacific n=86**	**Caucasian n=140**	**Asian/Pacific n=86**
8R=1 (0.7)		2R=11 (7.9)	2R=12 (14.0)	S=66 (47.1)	S=47 (54.7)
9R=33 (23.6)	9R=20 (23.3)	3R=6 (4.3)	3R=5 (5.8)	L=73 (52.1)	L=38 (44.2)
10R=104 (74.3)	10R=66 (76.7)	4R=101 (72.1)	4R=60 (69.8)	XL=1 (0.7)	XL=1 (1.2)
11R=2 (1.4)		5R=3 (2.1)			
		6R=1 (0.7)	6R=1 (1.2)		
		7R=18 (12.9)	7R=6 (7.0)		

Table 5b. Genotype distribution by ethnicity

DAT1 n (%)		DRD4 n (%)		5HTTLPR n (%)	
Caucasian n=70	**Asian/Pacific n=43**	**Caucasian n=70**	**Asian/Pacific n=43**	**Caucasian n=70**	**Asian/Pacific n=43**
8/9=1 (1.4)		2/2=1 (1.4)	2/2=1 (2.3)	S/S=15 (21.4)	S/S=12 (28.0)
9/9=5 (7.1)	9/9=3 (7.0)	2/4=8 (11.4)	2/4=9 (20.9)	S/L=35 (50.0)	S/L=20 (46.5)
9/10=22 (31.4)	9/10=14 (32.6)	4/4=37 (52.9)	4/4=21 (48.8)	L/L=19 (27.1)	L/L=10 (23.3)
10/10=40 (57.1)	10/10=26 (60.5)	4/7=12 (17.1)	4/7=5 (11.6)	S/XL=1 (1.4)	S/XL=1 (2.3)
10/11=2 (2.9)		7/7=1 (1.4)	7/7=1 (2.3)		
		2/7=1 (1.4)			
		3/4=3 (4.3)	3/4=4 (9.3)		
		4/5=3 (4.3)			
		3/7=3 (4.3)	3/7=1 (2.3)		
		4/6=1 (1.4)	2/6=1 (2.3)		

Table 6a. Allele distribution by gender

DAT1 n (%)		DRD4 n (%)		5HTTLPR n (%)	
Female n=166	**Male n=60**	**Female n=166**	**Male n=60**	**Female n=166**	**Male n=60**
8R=1 (0.6)		2R=22 (13.3)	2R=1 (1.7)	S=88 (53.0)	S=23 (38.3)
9R=40 (24.1)	9R=13 (21.7)	3R=8 (4.8)	3R=3 (5.0)	L=76 (45.8)	L=37 (61.7)
10R=124 (74.7)	10R=46 (76.7)	4R=116 (69.9)	4R=45 (75.0)	XL=2 (1.2)	
11R=1 (0.6)	11R=1 (1.7)	5R=2 (1.2)	5R=1 (1.7)		
		6R=2 (1.2)			
		7R=16 (9.6)	7R=10 (16.7)		

Table 6b. Genotype distribution by gender

DAT1 n (%)		DRD4 n (%)		5HTTLPR n (%)	
Female n=83	**Male n=30**	**Female n=83**	**Male n=30**	**Female n=83**	**Male n=30**
8/9=1 (1.2)		2/2=2 (2.4)		S/S=23 (27.7)	S/S=4 (13.3)
9/9=6 (7.2)	9/9=2 (6.7)	2/4=16 (19.3)	2/4=1 (3.3)	S/L=40 (48.2)	S/L=15 (50.0)
9/10=27 (32.5)	9/10=9 (30.0)	4/4=42 (50.6)	4/4=16 (53.3)	L/L=18 (21.7)	L/L=11 (36.7)
10/10=48 (57.8)	10/10=18 (60.0)	4/7=8 (9.6)	4/7=9 (30.0)	S/XL=2 (2.4)	
10/11=1 (1.2)	10/11=1 (3.3)	7/7=2 (2.4)			
		2/7=1 (1.2)			
		3/4=5 (6.0)	3/4=2 (6.7)		
		4/5=2 (2.4)	4/5=1 (3.3)		
		3/7=3 (3.6)	3/7=1 (3.3)		
		2/6=1 (1.2)			
		4/6 = 1 (1.2)			

Note that columns may not add to exactly 100% due to rounding to two decimal places.

Exploratory Analyses: Genotype Main Effects on Personality

The small final sample size of 113 provided limited options for multivariate analysis, so a number of exploratory comparisons were undertaken using the simplest genetic groupings as informed by previous literature (see Tables 7-9 for personality descriptives by +/– allelic genotypes[3]). With a larger sample size, it would be possible to explore multiple gene-by-gene interactions and the effect of other variables (such as gender and ethnicity) in the same model. Instead, I ran a number of separate mean comparisons via ANOVA to gain a richer understanding of the trends and patterns in the data. Informed by the literature identifying ethnic and gender differences in mean personality traits (McCrae et al., 2000; Terracciano et al., 2010), additional ANCOVA analyses were also run to control for the effect of these variables. No adjustments for multiple comparisons were made due to the small cell sizes and the results should be interpreted with care in light of this limitation. Replication studies using larger samples (with a more balanced gender and ethnicity mix) should investigate these preliminary findings in more depth.

DAT1 analyses

Table 7. Personality by DAT1 genotype

	9R+ (n=44)		**9R– (n=68)**	
Variable	**M**	**SD**	**M**	**SD**
NE_ANX	2.37	0.46	2.31	0.65
NE_DEP	2.31	0.53	2.41	0.63
NE_LSE	2.25	0.62	2.26	0.72
NE_Total	2.31	0.47	2.32	0.63
SS_THR	2.65	0.65	2.59	0.64
SS_EXP	2.80	0.46	2.88	0.58
SS_DIS	2.71	0.48	2.66	0.62
SS_Total	2.72	0.44	2.71	0.50
IPIP-Extra	3.32	0.71	3.18	0.81
IPIP-Agree	3.93	0.68	3.99	0.54
IPIP-Cons	3.43	0.66	3.39	0.71
IPIP-EmoStab	3.02	0.72	2.90	0.88
IPIP-Intel	3.57	0.46	3.65	0.64

[3] Please see Appendix H for personality trait scores by specific bi-allelic genotypes.

DAT1 Main Effects on Personality

As discussed in the literature review, the 10R allele of DAT1 has been associated with difficulties with social interaction, and with psychological disorders including ADHD. When comparing the 9R+ (*n*=44) and 9R– (*n*=68) groups, no mean differences in personality were found. This did not change when rare genotypes (*n*=4; 8/9 and 10/11) were excluded.

DAT1 Main Effects on Personality (Controlling for Gender and Ethnicity)

When controlling for gender and ethnicity, ANCOVA indicated a significant main effect of DAT1 on NE_Anxiety, SS_Thrill-seeking, SS_Disinhibition, and SS_Total, with the 9R+ group indicating higher neuroticism and higher sensation-seeking, compared to the 9R– group (see Table 8 and Figure 2[4]).

Table 8. DAT1 Main Effects, controlling for Gender and Ethnicity (df=1,10

	Group	N	M_{adj}	SE	F	η^2_p
NE_ANX	9R+	44	2.38	.10	6.15	.06
	9R-	68	2.02	.11		
SS_THR	9R+	44	2.76	.10	10.55	.09
	9R-	68	2.27	.11		
SS_DIS	9R+	44	2.77	.10	3.79	.04
	9R-	68	2.49	.10		
SS_Total	9R+	44	2.79	.08	7.75	.07
	9R-	68	2.47	.08		

All comparisons are significant at the $p<.05$ level.

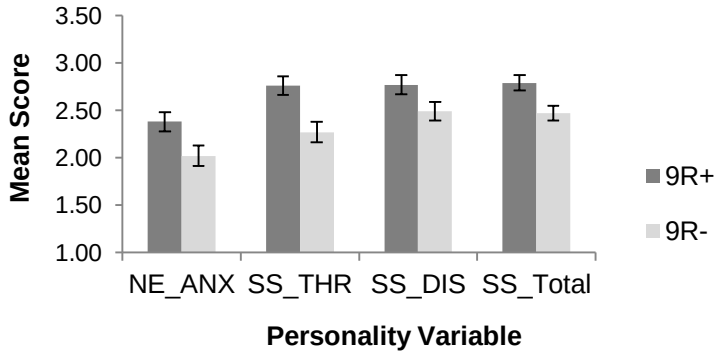

Figure 2. Mean Differences in Personality by DAT1 status, controlling for Gender and Ethnicity

[4] Error bars show standard errors of the estimated marginal means.

DAT1 Alternative Models

No mean differences were found when comparing 9/9 (*n*=8) and other (*n*=102), or when comparing 9/9 (*n*=8), 9/10 (*n*=35) and 10/10 (*n*=66), and this did not change when rare genotypes were excluded.

DRD4 analyses

Table 9. Personality by DRD4 genotype

	7R+ (n=24)		**7R– (n=89)**	
Variable	**M**	**SD**	**M**	**SD**
NE_ANX	2.11	0.76	2.39	0.52
NE_DEP	2.16	0.78	2.42	0.53
NE_LSE	2.15	0.99	2.28	0.57
NE_Total	2.14	0.81	2.36	0.49
SS_THR	2.43	0.84	2.66	0.57
SS_EXP	2.70	0.73	2.89	0.47
SS_DIS	2.65	0.94	2.69	0.43
SS_Total	2.60	0.69	2.74	0.40
IPIP-Extra	3.49	0.66	3.16	0.79
IPIP-Agree	4.21	0.42	3.90	0.62
IPIP-Cons	3.46	0.65	3.38	0.70
IPIP-EmoStab	3.17	0.93	2.89	0.79
IPIP-Intel	3.61	0.56	3.62	0.58

DRD4 Main Effects on Personality

As discussed in the literature review, the DRD4 7R allele has been associated with increased Novelty-seeking, Sensation-seeking and Extraversion – as well as risk-taking and impulsive behaviours like substance abuse and gambling, and disorders such as ADHD. There is debate, however, about the phenotypal traits associated with non-7R alleles including the most common 4R allele and the 2R allele, while still less is known about the 3R, 5R, 6R and 8R alleles. For this reason, a number of analyses were run with different genotype grouping combinations in order to try to identify functional personality phenotypes as measured in this study. DRD4 model comparisons are summarised below and significant mean differences are displayed in Table 10.

Table 10. DRD4 ANOVA Model Comparisons

Variables	Group	N	Mean	SD	F	η^2_p
Model 1: 7R+ vs 7R- (*df* = 1, 110)						
NE_ANX	7R+	23	2.11	.76	4.26	.04
	7R-	89	2.39	.52		
NE_DEP	7R+	23	2.16	.78	3.82	.03
	7R-	89	2.43	.53		
IPIP-Agree	7R+	24	4.21	.42	5.18	.05
	7R-	88	3.90	.62		
Model 2: 7R+ vs 4/4 vs Other 7R- (*df* = 2, 109)						
IPIP-Agree	7R+	24	4.21	.42	4.66	.08
	4/4	57	3.99	.64		
	Other 7R-	31	3.74	.55		
Model 3: 7R+ vs 4/4 vs 2R+ (*df* = 2, 97)						
IPIP-Agree	7R+	23	4.22	.43	3.83	.07
	4/4	57	3.99	.64		
	2R+	20	3.72	.62		
Model 4: 2R+&5R+ vs Other (*df* = 1, 110)						
IPIP-Agree	2R+&5R+	24	3.72	.57	5.48	.06
	Other	88	4.04	.59		

All comparisons are significant at the $p<.05$ level.

When comparing the DRD4 7R+ (n=24) and 7R– (n=89) groups (Model 1), mean differences were found in NE_Anxiety, NE_Depression, and IPIP Agreeableness. The 7R+ group were lower in Anxiety and Depression, and higher in Agreeableness (as shown in Figure 3[5]).

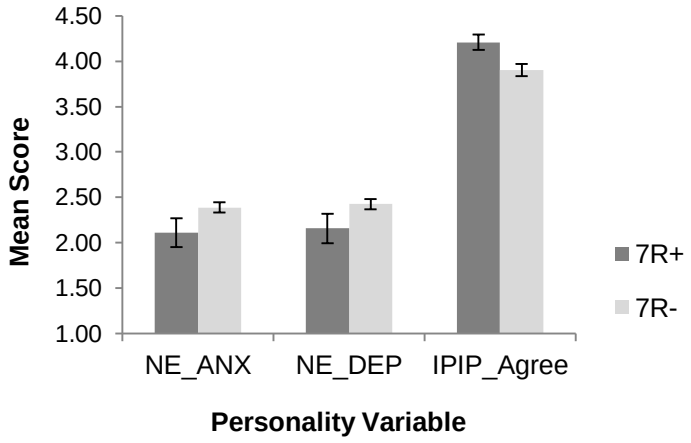

Figure 3. Mean Differences in Personality by DRD4 status

[5] Error bars display standard errors of the means.

DRD4 Alternative Models

When 7R+ (*n*=24) was compared to 4/4 (*n*=58) and other 7R– (*n*=31) in Model 2, a mean difference in Agreeableness was found, with a significant pairwise comparison between the 7R+ (highest) and the other 7R– (lowest) groups. When 7R+ (*n*=23), 4/4 (*n*=57), and 2R+ (*n*=20) groups[6] were compared in Model 3, there was a mean difference in Agreeableness, with a significant pairwise comparison between the 7R+ (highest) and 2R+ (lowest) groups. Using the genotype grouping of Keltikangas-Jarvinen and Salo (2009), when comparing the groups containing 2R and/or 5R alleles (*n*=24) and others (*n*=88), there was a mean difference in Agreeableness, with the 2R5R group lower.

DRD4 Main Effects on Personality (Controlling for Gender and Ethnicity)

ANCOVA indicated a significant main effect of DRD4 on NE_Anxiety, NE_Depression, NE_Total, SS_Thrill-seeking, SS_Experience-seeking, and SS_Total, indicating that the 7R+ group exhibit lower Neuroticism and lower Sensation-seeking than the 7R– group (see Table 11 and Figure 4[7]). However, the main effect of DRD4 on Agreeableness was not maintained.

Table 11. DRD4 Main Effects, controlling for Gender and Ethnicity

(df=1,102)	**Group**	**N**	**M_{adj}**	**SE**	**F**	**η^2_p**
NE_ANX	7R+	23	2.02	.13	5.98	.06
	7R-	89	2.38	.07		
NE_DEP	7R+	23	2.12	.13	4.13	.04
	7R-	89	2.43	.08		
NE_Total	7R+	23	2.08	.13	4.06	.04
	7R-	89	2.38	.07		
SS_THR	7R+	23	2.34	.13	5.01	.05
	7R-	89	2.69	.08		
SS_EXP	7R+	23	2.61	.12	3.96	.04
	7R-	89	2.88	.07		
SS_Total	7R+	23	2.49	.01	6.08	.06
	7R-	89	2.77	.06		

All comparisons are significant at the $p<.05$ level.

[6] One participant in the sample who had the 2/7 genotype was excluded from this analysis.
[7] Error bars show standard errors of the estimated marginal means.

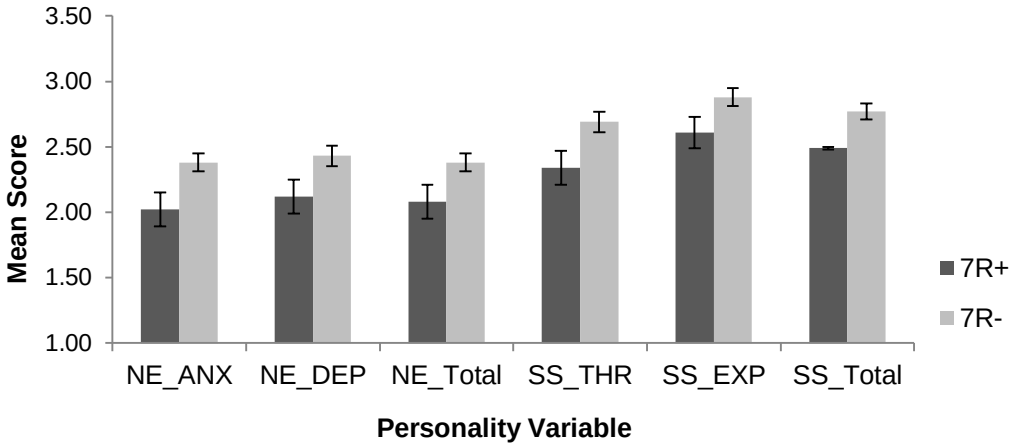

Figure 4. Mean Differences in Personality by DRD4 status, controlling for Gender and Ethnicity

5HTTLPR analyses

Table 12. Personality by 5HTTLPR genotype

	S+ (n=84)		S– (n=29)	
Variable	**M**	**SD**	**M**	**SD**
NE_Anx	2.36	0.54	2.25	0.71
NE_Dep	2.38	0.52	2.35	0.78
NE_LSE	2.24	0.61	2.31	0.86
NE_Total	2.32	0.51	2.30	0.74
SS_THR	2.62	0.58	2.59	0.80
SS_EXP	2.87	0.48	2.81	0.66
SS_DIS	2.68	0.54	2.67	0.65
SS_Total	2.72	0.42	2.69	0.62
IPIP_Extra	3.18	0.77	3.38	0.77
IPIP_Agree	3.92	0.61	4.09	0.55
IPIP_Cons	3.46	0.69	3.24	0.67
IPIP_EmoStab	3.00	0.85	2.79	0.72
IPIP_Intel	3.64	0.57	3.54	0.60

5HTTLPR Main Effects on Personality

As discussed in the literature review, the S allele of 5HTTLPR has been associated with increased neuroticism, anxiety, depression and stress reactivity. When comparing the S+ (*n*=82) and S– (*n*=28) groups, no mean differences in personality were found. This did not change when rare genotypes (*n*=2; S/XL) were excluded.

5HTTLPR Main Effects on Personality (Controlling for Gender and Ethnicity)

No main effects of 5HTTLPR were found using ANCOVA.

5HTTLPR Alternative Models

No mean differences were found when comparing S/S (*n*=27) and other (*n*=83) groups, or when comparing S/S (*n*=27), S/L (*n*=52) and L/L (*n*=29). This did not change when rare genotypes (*n*=2; S/XL) were excluded.

Moderation: Heart-Rate Variability and Stressful Life Events

Moderated regression analyses were used to measure the relative effects of Stressful Life Events and heart-rate variability on personality traits, as well as the moderating effects of SLEs and HRV on gene-trait relationships. Two sets of regression analyses were run for SLE x during-negotiation HRV and SLE x post-negotiation HRV. Stressful Life Events and heart-rate variability scores were centred and entered in the first step, followed by the SLE x HRV interaction term in the second step. As no significant gender or ethnicity differences were found in SLE or HRV scores, gender and ethnicity were not controlled for. There were no significant main effects ($p < .05$) of heart-rate variability, or of Stressful Life Events, on personality traits. Neither measure of heart-rate variability moderated any of the 39 (3x13) gene-trait relationships examined. However, Stressful Life Events did moderate the effect of DRD4 on two Sensation-seeking traits: 15% of the variance in Disinhibition, and 7% of the variance in total Sensation-seeking was explained by the SLExDRD4 interaction (see Table 13). To investigate the nature of these moderations, the data file was split by DRD4 genotype (7R+/7R–) and SLE–Sensation-seeking correlations examined (see Table 14).

Table 13. SLExDRD4 on Sensation-seeking

Variable	**B**	**∆r2**	**∆F (3,108)**	**t**
SS_Dis	-.58	.15	18.46	-4.30
SS_Total	-.34	.07	8.48	-2.91

All moderations are significant at *p*<.05.

Table 14. SLE–SS correlations by DRD4 status

Variable	**SS_DIS**		**SS_Total**	
Genotype	**7R+**	**7R-**	**7R+**	**7R-**
SLE_Total	-.39^	.17	**-.50***	.17

^ *p*=.07. * *p*<.05. 7R+ *n*=24, 7R- *n*=89.

Results indicated that the experience of more Stressful Life Events was associated with lower Sensation-seeking for the 7R+ genotype group, while there was no SLE moderation of Sensation-seeking in the 7R– group. However,

the low reliability of the Disinhibition facet in this study, and the large number of gene-trait relationships tested, means this result may be neither stable nor replicable.

Exploratory Analyses: Gene-Gene Interactions on Personality

Comparisons were conducted using three different two-genotype interaction groups (DAT1xDRD4, DAT1x5HTTLPR, and DRD4x5HTTLPR) based on the +/– genotype divisions used to compare main effects (see Table 13). Three-way interactions were not examined due to the small cell sizes in our data. Significant gene interactions were followed up with post-hoc tests to identify which combination of genetic polymorphisms significantly differed from each other. Concerning the previously reported main effects, DRD4 remained marginally significant ($.05 < p < .07$) for NE_Anxiety, NE_Depression and Agreeableness in the DRD4x5HTTLPR and DAT1xDRD4 interactions. The main effect for DRD4 on Sensation-seeking traits was no longer significant when examining interactions with 5HTTLPR or DAT1. Subsequent ANCOVAs revealed that all gene-gene interactions remained significant after controlling for gender and ethnicity (data not shown).

Table 15. Genotype Interaction Groups (Total N=112)

DATxDRD4 (n)	DATx5HT (n)	DRD4x5HT (n)
9+7+ (12)	9+S+ (30)	7+S+ (14)
9+7- (32)	9+S- (54)	7+S- (9)
9-7+ (14)	9-S+ (14)	7-S+ (70)
9-7- (14)	9-S- (14)	7-S- (19)

DAT1xDRD4 Interactions on Personality

Significant ($p < .05$) DAT1xDRD4 interactions were found for SS_Thrill-seeking: $F(1, 108) = 11.59$, $\eta^2_p = .10$ and SS_Total: $F(1, 108) = 5.34$, $\eta^2_p = .05$, as well as a trend for SS_Experience-seeking: $F(1, 108) = 3.29$, $\eta^2_p = .03$, $p = .07$. Follow-up tests showed that the 9–7+ group was lower in Sensation-seeking than other groups (see Table 14 and Figure 5).

Table 16. DAT1xDRD4 Significant Interactions

Variable	SS_THR	SS_EXP	SS_Total

Pairwise Comparison	t	df	t	df	t	df
9+7+ vs 9+7-	1.20	42	0.32	42	0.72	42
9+7+ vs 9-7+	**2.78***	**21**	0.97	21	1.53	21
9-7- vs 9-7+	**3.72***	**66**	**2.17***	**66**	**2.54***	**66**
9-7- vs 9+7-	0.99	87	1.51	87	0.91	87

*Significant (p<.05) comparisons.

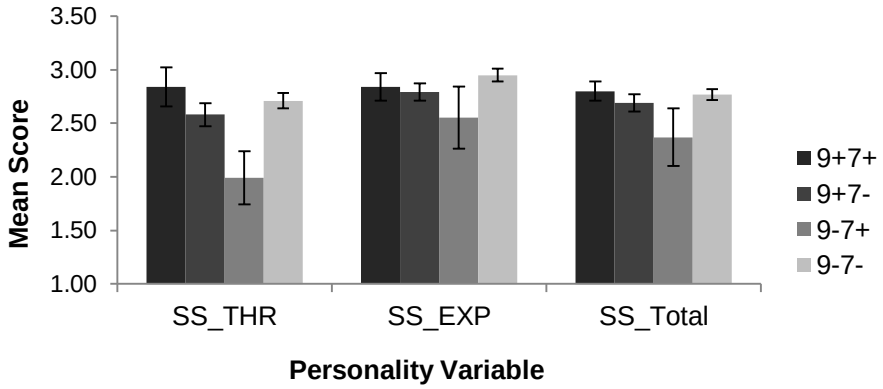

Figure 5. DAT1xDRD4 Significant Interactions on Personality

DAT1x5HTTLPR Interactions on Personality

Significant (p < .05) DAT1x5HTTLPR interactions were found for NE_Anxiety: $F(1, 108) = 8.41$, $\eta^2_p = .07$, NE_Depression: $F(1, 108) = 4.67$, $\eta^2_p = .04$, NE_Total: $F(1, 108) = 5.97$, $\eta^2_p = .05$ and IPIP Agreeableness: $F(1, 108) = 4.32$, $\eta^2_p = .04$. Follow-up tests showed that the 9+S– group was higher for Anxiety, Depression, and total Neuroticism; 9–S– lower for Anxiety and total Neuroticism, and 9+S+ lower for Agreeableness (see Table 15 and Figure 6).

Table 17. DAT1x5HTTLPR Significant Interactions

Variable	NE_ANX		NE_DEP		NE_Total		IPIP-Agree	
Pairwise Comparison	**t**	**df**	**t**	**df**	**t**	**df**	**t**	**df**
9+S+ vs 9+S-	**1.91***	**42**	1.72	42	**2.09***	**42**	**2.24***	**42**
9+S+ vs 9-S+	1.00	82	**2.10***	**82**	1.52	82	1.65	81
9-S- vs 9-S+	**2.37***	**66**	1.44	66	**2.37***	**66**	0.42	66
9-S- vs 9+S-	**2.47***	**26**	1.07	26	1.59	26	1.52	27

*Significant (p<.05) comparisons.

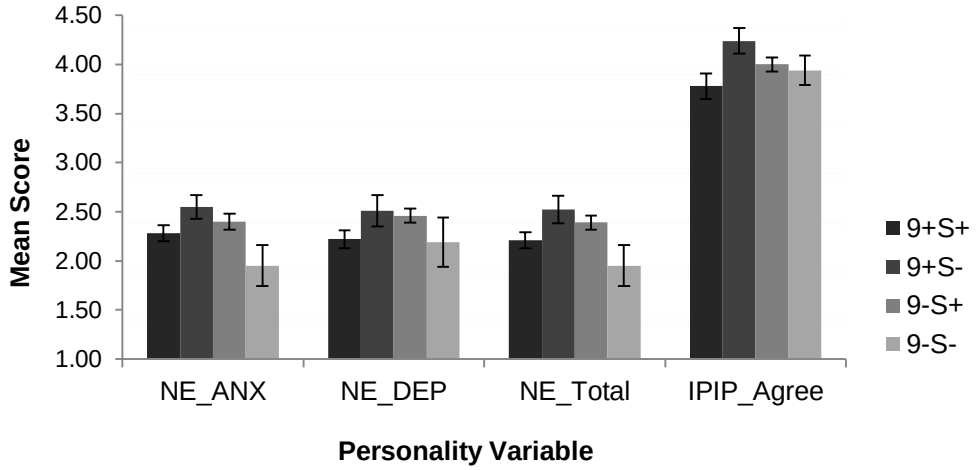

Figure 6. DAT1x5HTTLPR Significant Interactions on Personality

DRD4x5HTTLPR Interactions on Personality

Significant ($p < .05$) DRD4x5HTTLPR interactions were found for SS_Disinhibition: $F(1, 108) = 8.64$, $\eta^2_p = .07$ and SS_Total: $F(1, 108) = 4.84$, $\eta^2_p = .04$. Follow-up tests showed that the 7+S– group was significantly lower in Sensation-seeking than the other groups, and the 7–S– group was significantly higher (see Table 16 and Figure 7).

Table 18. DRD4x5HTTLPR Interactions

Variable	**SS_DIS**		**SS_Total**	
Pairwise Comparison	**t**	**df**	**t**	**df**
7+S+ vs 7+S-	1.54	21	1.33	21
7+S+ vs 7-S+	1.57	82	0.23	82
7-S- vs 7-S+	**1.96***	**87**	1.24	87
7-S- vs 7+S-	**2.31***	**26**	**2.04***	**26**

*Significant (p<.05) comparisons.

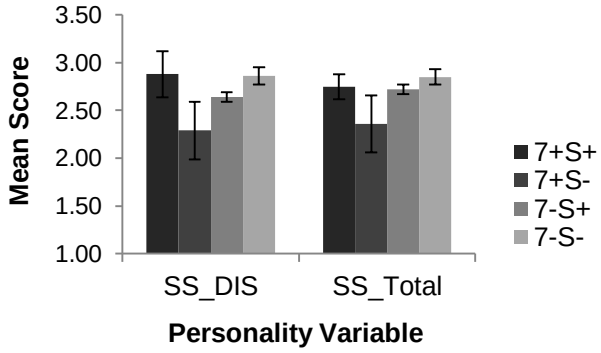

Figure 7. DRD4x5HTTLPR Significant Interactions on Personality

Summary of Genetic Analyses

Genetic main effects on personality were apparent for DRD4, where the 7+ group had lower (anxious and depressive) Neuroticism and higher Agreeableness than the 7– group. When controlling for gender and ethnicity the 7+ group also had lower (thrill and experience-seeking, and total) Sensation-seeking, while the DAT1-9+ group had higher (anxious) Neuroticism and higher (thrill-seeking, disinhibitory, and total) Sensation-seeking, compared to the 9– group. Gene-gene interactions on personality were found for DAT1xDRD4, with lower (thrill and experience-seeking, and total) Sensation-seeking in the 9–7+ group. For DAT1x5HTTLPR, the 9–S– group was lower for (anxious and total) neuroticism, 9+S– higher for (anxious, depressive, and total) Neuroticism, and the 9+S+ group lower for Agreeableness. For DRD4x5HTTLPR, the 7+S– group was lower in (disinhibitory and total) Sensation-seeking, while the 7–S– group were higher in both those traits. Heart-rate variability and stressful life events did not directly affect personality traits, and nor did they consistently moderate gene-trait relationships.

Discussion

The current research adds to the personality genetics literature in a number of ways. Little research has been conducted in the Pacific/Oceania region, so the testing of a sample population from this area increases the diversity of participants tested for gene-trait associations. The development of a genotyping protocol which can be used for both singleplex and multiplex DAT1, DRD4 and 5HTTLPR polymorphic analysis builds research capability for future gene-trait studies at Victoria University of Wellington. This paves the way for future students to engage in multi-disciplinary research that crosses the 'knowledge silos' often found in academia. The melding of Social Psychology theory and Biological Science techniques opens up new possibilities for testing multi-level (micro, macro and moderating) variables and multi-method hypotheses.

Strengths of the current study include the measurement of genetic, physiological, environmental and phenotypic variables in the same study. Using multiple gene variants and multiple personality measures has given this exploratory study a broad reach to grasp significant associations, particularly interactions. There appear to be different genetic influences for facets within the same trait, which has been obscured by studies looking only at higher-level factors (Munafo & Flint, 2011). The inclusion of facet scores in the personality measures, rather than relying on overall trait scores, means that higher-level factors like Neuroticism and Sensation-Seeking have been drilled down into lower-level component parts. Measurement and control of ethnicity means that the potential confound of genetic population stratification is avoided. Measurement and control of gender means that the relative strengths of different genetic effects on traits can be considered.

There is rather scant research presently available on the effect of heart-rate variability on personality traits. The current research used a life-like negotiation exercise to model a stressful cross-cultural interaction. Rather than ask participants how they might behave/feel in

such a situation, a direct measure of physiological stress reactivity was indexed via heart-rate monitoring. The advantage of a physiological measure which is not normally under conscious voluntary control is an increase in external validity for this lab-based study. The reliability of this physiological measure across participants (as indicated by higher mean heart-rate and lower HRV during the stressor) means that the portable and easy-to-use POLAR2 equipment may be effectively utilised in other studies examining the regulation of behavioural and emotional responses.

Little research has been conducted on the effect of Stressful Life Events on personality traits (rather than on negative outcomes/disorders like depression), or on the moderating effect of SLEs on dopaminergic (rather than serotonergic) gene-trait associations. The current research shows that both physiology and environment have measurable associations with personality, though not always in ways predicted by previous literature. More gene-gene interactions were apparent that gene main effects (when not controlling for gender and ethnicity), emphasising the importance of testing multiple gene variants in the same study to discover a fuller array of gene-trait associations.

Gene-Trait Associations

The hypothesised gene-trait associations were mostly unsupported by the results, and in some cases associations were in the opposite direction to expectations. DRD4-7R+ was associated with lower rather than the predicted higher Sensation-Seeking scores, while 5HTTLPR-S+ had no direct effect on Neuroticism. DRD4-7R+ was actually more strongly associated with (lower) Neuroticism than with Sensation-Seeking. DAT1-9R+ was associated with higher Neuroticism (as predicted) but with higher rather than lower Sensation-Seeking, contrary to prediction. The 9R+ effect was broader for Sensation-Seeking (including two facets and SS_Total) than for Neuroticism, where only one facet (NE_Anxiety) was involved.

Contrary to expectations, the 7R allele lowered Sensation-seeking scores for Thrill-seeking, Experience-seeking and SS_Total, and had no effect on Disinhibition. The same pattern was evident for the DAT1xDRD4 interactions, indicating that the DRD4-7R influence on Sensation-seeking is strong and persistent in this sample. The Disinhibition facet is perhaps less similar to the 'seeking' facets of the Sensation-seeking scale, with items that seem more socially-influenced such as liking 'wild' parties and 'intense rock music', and disliking 'melodic popular music' (see Appendix A). Also recall that the Disinhibition facet has sub-standard reliability in this study and therefore may not exhibit stable, replicable associations.

There were no simple main effects of DAT1-9R+ or 5HTTLPR-S+, perhaps due to a lack of power and the low prevalence of particular 'high-association' genotypes like 5HTTLPR S/S. However, once gender and ethnicity were controlled, more associations were revealed. The DAT1-9R+ group was now higher in both Sensation-seeking (Thrill-seeking, Disinhibition and total) and NE_Anxiety. This pattern could reflect heightened sensitivity to the environment such that 9R+ individuals seek out highly-arousing/rewarding positive stimuli, but are also strongly affected by negative/punishing stimuli. Research into sensory-processing sensitivity (Aron & Aron, 1997) identifies a personality type with a similar combination of increased temperamental and physiological susceptibility to environmental stimuli.

Some of the earliest personality genetics studies reported that genetic variants were associated with some, but not all, facets of a personality trait. For instance, Lesch et al. (1996) found that the 5HTTLPR-S+ group had higher NEO Neuroticism for 4/6 facet scales (anxiety, angry hostility, depression, impulsiveness); but with no effect for NE self-consciousness nor vulnerability. It is noteworthy that impulsiveness is a neurotic trait in the Big Five, but part of Sensation-seeking in the ZKA-PQ. DeYoung and Gray (2009) suggest

that the 5HTTLPR-S allele and the DRD4-7R allele both function to increase impulsivity. If we consider Sensation-seeking a form of impulsivity, the current research does not support this view.

Alone, DRD4-7R was associated with lower Neuroticism and higher Agreeableness, with no effect on Sensation-seeking until gender and ethnicity covariates were included, while 5HTTLPR-S was not directly associated with any personality trait. The DRD4x5HTTLPR interaction also did not support DeYoung and Gray's (2009) theory, as the 7+S– haplotype was associated with lower Sensation-seeking than the 7–S– group. Rather than 5HTTLPR-S and DRD4-7R alleles, in the current study it was DRD4-7R and DAT1-9R alleles which opposed each other's effects. DRD4-7R was associated with lower Neuroticism and lower Sensation-seeking, while DAT1-9R was associated with higher Neuroticism and higher Sensation-seeking. This shows that Sensation-seeking and Neuroticism are not simply the ends of a see-saw but have a more complex relationship.

In the current research, significant DAT1x5HTTLPR interactions indicated that the 9+S+ group had lower Agreeableness, the 9+S– group higher Neuroticism and the 9–S– group lower Neuroticism. This suggests that the DAT1-10/10 genotype may facilitate emotional stability and interpersonal warmth. This association of 5HTTLPR S– with higher Neuroticism is contrary to prediction but the pattern suggests that DAT1 is more influential than 5HTTLPR on Neuroticism. The current study thus partially supports the Lesch et al. (1996) findings but indicates that other genes may enable or disable the effect of 5HTTLPR on traits.

One of the strongest gene-trait effects was unanticipated – the DRD4-7R allele was associated with higher Agreeableness across multiple genotype models. If we consider the DRD4-4/4 genotype the 'norm' (at least in terms of genotype frequency), then the 2R allele appears to decrease Agreeableness and the 7R allele increase it. This is a clear example of the

variation hidden when comparing only 7R+ vs 7R–, or of comparing the common 4R+ with a general 'other' group. This result suggests that the 7R allele may aid social interaction and co-operation, and limit aggression. Could this reflect a 7R connection to a broader sociality factor which includes items from multiple traits? Depue and Collins (1999) propose a reward-processing theory of extraversion, which differentiates social closeness (associated with consumptive enjoyment or 'liking'), and social potency (associated with reward-seeking and appetitive desire or 'wanting'). However, the 'social closeness' factor may be something more akin to Big Five Agreeableness rather than a part of Extraversion. Cramer and colleagues (2012) for example, constructed a network architecture model of personality, and found that Big Five Extraversion and Agreeableness were largely intertwined. Future research could use additional measures of sociality to explore this connection.

The change in results when covariates were included shows that effects of gender and ethnicity on personality traits may suppress genetic effects. Interestingly, it seems that gender and ethnicity are more important for Sensation-seeking than for Neurotic traits. No Sensation-seeking traits were associated with single genes in the ANOVA analyses, but six effects became significant in the ANCOVA analyses for DRD4 and DAT1. This supports a relatively weak dopamine–Sensation-seeking connection, which is masked by stronger gender-trait associations. Gender may influence Sensation-seeking environmentally, through sexually-differentiated socio-cultural norms for exploratory behaviours. Gender may also influence Sensation-seeking through sexually-differentiated biological processes via the influence of sex hormones (Keltikangas-Jarvinen & Salo, 2009).

Frequencies of genetic polymorphisms in this sample were in line with previous research (Kidd et al., 2012), whereby Asian/Pacific participants had more 5HTTLPR-S and DRD4-2R alleles than Caucasians. Interestingly, the rare XL allele was found in two participants in this sample: one Malaysian Chinese and one NZ European. Previous research

has identified this allele in subjects of African origin (Delbruck et al., 1997), and in Japanese but not Caucasian subjects (Nakamura, Ueno, Sano & Tanabe, 2000). The current research provides evidence that this rare genotype is also found in European/Caucasians. The gender differences in genotype frequency are likely due to the small, self-selected sample comprised mainly of female psychology students, rather than any population genetic variation.

Heart-rate Variability and Stressful Life Events

As hypothesised, post-negotiation HRV was higher than during-negotiation HRV, showing that the negotiation task functioned well as an induced stressor, with a direct and measurable impact on a physiological index of stress reactivity. Higher Neuroticism was negatively correlated with HRV during both periods, with slightly stronger correlations during the negotiation (r = -.26 to -.31) than after it (r = -.21 to -.26). That HRV was associated with Neuroticism but not Sensation-seeking traits suggests that physiological reactivity is connected more closely to inhibition and avoidance rather than exploratory traits. The negative direction of the correlation, and the fact that both measures of HRV were involved, indicates that those with higher Neuroticism may be chronically rigid in both physiological and behavioural responses. This sits well with the literature indicating that cardiac tone indexes the ability to control negative cognitive and emotional states (Martens, Greenberg & Allen, 2009; Thayer et al., 2012). Heart-rate variability was not associated with the (reversed) neuroticism measure of IPIP Emotional Stability, which adds weight to claims that the ZKA model explains more biological variance than the Big Five. Both measures of heart-rate variability were also moderately positively correlated with IPIP Conscientiousness, suggesting that behavioural control via discipline and delay of gratification is associated with more flexible response regulation as indicated by higher HRV.

The pattern of correlations between physiological, environmental and personality variables sets the scene for gene-trait associations and HRV and SLE moderations. The

Thrill-seeking subscale was the only Sensation-Seeking facet that failed to produce both a low/moderate positive correlation with Neuroticism subscales and a moderate negative correlation with IPIP Conscientiousness. This suggests that thrill-seeking may be a 'cleaner' reward-centred hedonic trait without strong emotive content – thrills and ills are conceptually distinct. This pattern also supports the meta-trait theory connecting Big Five Conscientiousness and Neuroticism as 'stability' traits in opposition to 'plastic' Extraversion and Openness/Intellect; although in the current study Agreeableness was more strongly correlated with the plastic traits. Another way to look at the meta-traits, informed by physiology, might be as inhibitory versus exploratory traits.

The low negative correlation of Stressful Life Events with Conscientiousness, Emotional Stability and post-negotiation heart-rate variability is interesting. More Stressful Life Events were associated with lower Conscientiousness and lower Emotional Stability. This suggests that exposure to more stressors may lower the ability to regulate one's actions and emotions, perhaps via the perception that one is not able to avoid, or control the effects of, (negative) life events. The experience of loss of control in the environmental realm thus produces a loss of control in the behavioural realm. The more stressful life events one has experienced, the lower emotional, cognitive and physiological resilience displayed. Stress exposure may lead to affective (emotional stability) and effective (conscientiousness) dysfunction as well as response rigidity (lower HRV). An alternative interpretation is that the chain of causation lays in the opposite direction, such that pre-existing personality and physiology traits lead to exposure to more or less stressful life events. Longitudinal studies that measure variables at multiple time-points would allow this question to be tested empirically.

When considering stress exposure as an environmental variable, it would be useful to look at a wider range of stressors, both chronic and acute. There is evidence that stress as a

moderator of negative outcomes like depression is associated with specific medical conditions or maltreatment rather than stressful life events per se; these effects were also stronger when tested with objective measures or interview assessments as opposed to self-reports (Karg et al., 2009). That Emotional Stability was associated with the SLE measure suggests that the Big Five neuroticism may be more sensitive to environmental variation than ZKA Neuroticism facets. This divergence of association across genetic, physiological and environmental measures shows that using multiple (lexical and psychobiological) personality instruments can reveal different trait associations in different realms.

Higher heart-rate variability was associated with higher Conscientiousness and lower Neuroticism. This is interesting in that high conscientiousness could be characterised as adaptive behavioural regulation, and low neuroticism as adaptive emotional regulation, of stimuli responsivity. It then makes sense that a physiological measure associated with flexible responding would predict this adaptive trait pattern. Rather than the classic diathesis-stress model, where unidirectional 'risk' alleles/genes lay latent until activated by specific (negative) environments or experiences, Belsky and colleagues (2009) put forward a plasticity hypothesis where particular gene variants make their carriers more susceptible to the influence of the environment, for better AND worse. For example, in Belsky et al.'s study 5HTTLPR S/S carriers exhibited the highest depression and anxiety in a negative/stressful environment, but the lowest depression and anxiety in an enriched/stress-diminished environment. Similarly, children with the DRD4-7R+ genotype had the highest externalising problems if they experienced insensitive parenting, but the lowest problems in a context of sensitive parenting (Belsky et al., 2009). Future studies of gene-environment interactions on personality and other outcomes should therefore be careful to consider not just negative environments and experiences, but also positive/enriched ones; so that a wider range of genetic moderation may be revealed.

Limitations

Genotyping accuracy is a potential source of error in the current research. The PCR process favours amplification of smaller alleles over larger ones (Serretti et al., 2006), so what looks like a 5HTTLPR S/S genotype in an electrophoresed gel could actually be S/L (i.e. the L band is not visible, so the visible S band is interpreted as a homozygote). GC-rich sites – as found in the DRD4 and 5HTTLPR sequence variants – can also result in the formation of multiple (spurious) bands, making accurate allele calling even more difficult. However, these problems were addressed by running offsite capillary analysis as well as in-house gel electrophoresis. Although some samples had low peaks (outside the recommended 500-15,000 fluorescent unit height range), these peaks were still clearly discernible from background noise and the LIZ1200 size standards (see Appendix G for a visual representation). Full gene sequencing is an even more accurate genotyping technique which should be used in larger well-funded personality genetic studies.

The serotonin and dopamine systems consist of numerous different genes, including transporters and receptors. Each gene also has multiple polymorphisms, and even the functional subset (that code for proteins) may have different impacts on different individuals (Lazary et al., 2008). For example, the DAT1 polymorphism is in a non-coding area (the 3' untranslated region), but at present it is unknown exactly how non-coding polymorphisms affect molecular, physiological or behavioural outcomes. Studying only one polymorphic site in each of three genes, alongside one measure of the environment (Stressful Life Events) is therefore only examining a very small proportion of the potential gene x environment influences on personality. Researchers have pointed out that gene-trait relationships may be rendered latent through the influence of multi-system epistasis (gene-gene interactions), epigenetics (changes to gene expression and function over the lifespan), gene-environment

correlations and interactions, and the presence of other confounding variables (Savitz & Ramesar, 2004). Unravelling the effects of these variables is extremely difficult.

Keltikangas-Jarvinen and Salo (2009) suggest that dopamine and serotonin systems moderate the effect of environmental conditions on a range of psychological outcomes, including personality traits. The authors propose that gene x environment interactions are likely to be of more influence than direct gene-trait associations, and that failure to measure environmental variables has resulted in the highly-divergent results of previous studies. The effects of genes may only become evident when studied in the context of environmental factors, especially because people may be differentially sensitive to environmental conditions (Belsky et al., 2009). Certain genetic variants may be risk factors/exacerbators in some environments (or for some populations), but protective factors/buffers in others. This aligns with evolutionary psychology niche construction theories (Penke, Denissen, & Miller, 2007). Different populations adapt to different ecological niches so that some personality traits are adaptive in one niche (e.g. high sensation-seeking in a low-risk, low-predator environment), but maladaptive in another (e.g. high sensation-seeking in a high-risk, high-predator environment).

Other genes strongly linked to personality include brain-derived neurotropic factor (BDNF), the norepinephrine transporter (NET), catechol-O-methyltransferase (COMT) and monoamine oxidase A (MAOA), with genome-wide association studies (GWAS) identifying many more – each with a tiny individual effect (Terraciano et al., 2010). Other molecules involved to some degree in personality include endogenous opioids, sociosexual neuropeptides like oxytocin and vasopressin, and hormones such as estrogen, testosterone, cortisol and glucocorticoids (DeYoung, 2010). Other environmental features such as parenting style, early attachment style, sexual/physical abuse, substance use and allostatic load have also been identified as environmental factors contributing to personality (Caspi et

al., 2010; Kandler, 2012b). Careful study planning and design is needed to identify key personality influencers in multiple realms.

The short 10-item Stressful Life Event measure used in the current research is limited – other stressors not included in the list may have a significant impact on the variables under consideration. As the measure only covers the last six months, it also excludes any events which happened prior to this time period. There is no indication of the severity of impact of the measured events. For example one respondent may be strongly negatively affected by a particular event that another respondent barely registers, or reacts to positively (e.g. a new job or moving house), depending on the reasons for and meanings attributed to the event. There is evidence that the perception of the controllability of stressful life events is a particularly important consideration (Kandler et al., 2012b). Future research should include relevant environmental measures to provide a fuller picture of personality correlates and determinants over time and situation. A longitudinal design could identify the level of stability and change in personality, physiology and environmental measures and their inter-relations. It is likely that meta-analyses considering a broad range of these variables will be useful in determining the relative importance of particular environmental moderators of personality traits.

The heart-rate variability measures used were relatively brief (three minutes each), and no true baseline was used (where participants are sitting or laying down still, and not performing any kind of task). The use of a single metric (RMSSD) also limits the physiological measure to an index of high-frequency, parasympathetic influences on heart-rate variability rather than high frequency/low frequency balance which captures both parasympathetic and sympathetic influences (Task Force of The European Society of Cardiology, 1996; Zohar, Cloninger & McCraty, 2013). The use of a more physiologically-attuned personality instrument such as the SSP (Swedish universities Scales of Personality: Gustavsson et al., 2000) which includes items covering autonomic and somatic responses,

might more readily reveal physiological moderation of gene-trait relationships. Future personality genetics research should therefore include both more nuanced and more comprehensive measures of individual differences in physiology/life experience and environment.

Using a convenience sample of New Zealand-based university students has its pros and cons. One benefit is relative homogeneity in terms of age and education level, and one cost is that the results may not generalise to other populations. However, using a non-clinical sample means that the results should be comparable to other 'normal' populations of the same age, and the use of ANCOVA means that the potentially confounding/masking effects of gender and ethnicity have been addressed. Future research could use more diverse samples, alongside alternative tests to investigate convergent and discriminant validity of the study variables (e.g. skin conductance, cortisol and neuroimaging physiology measures, inclusion of other genetic polymorphisms, and other cognitive and/or behavioural measures of personality).

The current research tested a variety of genotype groups to attempt to more fully explore potential gene-trait associations. However, the analysis has been restricted by the small sample size (N=113) and the unequal group sizes created via the genotyping measures. Some genotypes are present in very small numbers (e.g. the DRD4 7/7 genotype: $n = 2$), which renders extensive comparative analysis problematic and reduces the study's power. Haplotype analysis beyond +/– two-gene interactions was not possible due to the extremely small cell sizes this would have necessitated. Future alternative designs might genotype a large initial sample and then select equal numbers of each genotype category to complete the study measures. This would be very expensive as it would require genotyping a very large sample, and this technique was not feasible within the constraints of an MSc thesis research project. Still, sample sizes of more than 2000 may be necessary to address concerns that the

majority of gene-trait associations in the literature have come from small sample size (N<200) studies, raising the possibility of spurious results (Savitz & Ramesar, 2004).

In the current study, personality is measured only by self-report scales, which may be subject to systematic biases including social comparison, social desirability and response style effects. For example, when answering questions about oneself, people may use different individual 'yardsticks' for comparison. I may compare my level of Sensation-seeking to that of my friends, while others may compare themselves to a family member, colleague, or a more general representation of a 'normal' person. Members of particular socio-cultural groups may consistently rate themselves higher or lower on specific desired or non-desired traits, according to their personality norms/ideals. Research into response styles indicates that some people tend towards extreme scores (avoiding middle/neutral categories), while others show the reverse pattern. Using peer/observer reports alongside more direct methods like experience-sampling and video diaries is one way to enhance the external validity of personality research. This is of course more time and labour-intensive than using survey scales.

Future directions

Many psychological disorders are associated with 'extremes' of personality, particularly Neuroticism (anxiety, depression) and Sensation-seeking (antisocial personality, substance abuse). Studying variation in 'normal' (i.e. non-clinical) populations may provide insight into 'pathological' personality and mood variation. If different gene variants have different biochemical effects in different populations (as suggested by Kim et al., 2006 for 5HTTLPR), serotonergic and dopaminergic drugs to treat depression, anxiety and impulse control disorders could be tested and targeted more effectively. Future clinical research could usefully include genetic and physiological testing alongside self-reports and clinical interviews. Specific bio-physio-psychological profiles could be developed to identify

dysfunctional mood and personality phenotypes in different populations. In non-drug-based therapy, individuals and groups with particular stress reactivity/personality profiles may also benefit from multi-modal treatments tailored to their specific needs.

Larger, multi-national personality genetic studies might indicate that different personality factors are mal/adaptive in different cultures and/or contexts (e.g. in intra- or inter-cultural situations). Appreciating these different genetic/cultural profiles of personality could aid intercultural understanding and increase tolerance of divergence from acknowledged personality norms or ideals. Educational/workplace training and development programmes could be customised to suit participants with different personality profiles. Perhaps the psychometrics industry might add physiology tests to the battery of measures that job applicants increasingly face, in an effort to distinguish those who respond well and poorly to stress. It might even be possible for socially-desirable traits to be 'learned', and undesirable traits 'unlearned'. The continued vigour of the self-help and therapy industries is some indication that people are interested in these possibilities.

New research paradigms involving behavioural and physiological measures that correlate with personality variation (such as prepulse inhibition, economic games, responses to emotional pictures, cognitive tests) should be used to examine more context-dependent trait associations. As the field of bio-informatics develops alongside increases in computing power and the development of increasingly sophisticated statistical techniques, complex gene-environment-physiology-trait analyses will become more common. Advanced techniques like biometrical moderation (South & Krueger, 2008), computational neural networks (Read et al., 2010), and hierarchical clustering (Suranyi, Hitchcock, Hittner, Vargha & Urban, 2013) will extend the possibilities for association and prediction of personality beyond correlation, moderation and analysis of variance. Rather than simply producing high/low mean scores on single traits, these tools may be able to predict actual context-

dependent behaviour based on trait clusters and multi-trait personality profiles. However, these methods also require carefully-constructed hypotheses, research designs and variable measurement.

Cramer and colleagues (2012) offer a network architecture perspective where personality traits are not causes of behaviour but emergent properties produced by connections and feedback between affective, behavioural and cognitive components. These components may be central or peripheral, and tightly or loosely bound. Applying a network model to personality genetics may be the way forward if we want to predict or shape behaviour rather than just describe it. Straightforward dopamine-approach and serotonin-avoidance theories are simplistic but offer a good starting point for testing psychobiological personality theories. Just as plants display varying 'reaction norms' in different environments, personality traits could be thought of as context-dependent, physiologically and environmentally-influenced 'reactivity norms' (Penke, Denissen & Miller, 2007), which provide a probabilistic rather than deterministic portrait of behaviour.

Studies based on a single instrument or single experimental paradigm are unlikely to improve our understanding of personality (Ebstein, 2006). The current research has attempted to rise to this challenge through the development and execution of a multi-marker exploration of personality. Gene-trait relations are complex, interactionist and multiply-determined, suggesting that personality variation is influenced by – but not reducible to – genetic variation.

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Appendix A. Survey Measures: ZKA-PQ

A number of statements are shown below that describe some ways in which people act and think. Please, indicate for each statement how much you agree or disagree. If you have not experienced that situation, please try to describe how you would act or what you would think about that situation. If you **strongly disagree** circle **1**, if you **somewhat disagree** circle **2**, if you **somewhat agree** circle **3**, and if you **strongly agree** circle **4**. Be sure to indicate your agreement or disagreement for every statement.

1	**2**	**3**	**4**
Strongly disagree	**Somewhat disagree**	**Somewhat agree**	**Strongly agree**

I often feel restless for no apparent reason.	1	2	3	4
I often feel like crying.	1	2	3	4
I sometimes fear I am not up to life's challenges.	1	2	3	4
Often I feel uneasy.	1	2	3	4
I sometimes feel depressed.	1	2	3	4
I am not very confident about myself or my abilities.	1	2	3	4
I am a very nervous person.	1	2	3	4
Negative thoughts sometimes obsess me.	1	2	3	4
I often think people I meet are better than I am.	1	2	3	4
I often worry about things that other people think are unimportant.	1	2	3	4
I sometimes seem to be lacking any energy.	1	2	3	4
I often feel unsure of myself.	1	2	3	4
I find it hard to keep my mind on a task or job.	1	2	3	4
I sometimes find it difficult to concentrate.	1	2	3	4
I am somewhat disappointed when I look back on my efforts.	1	2	3	4
I do not worry about unimportant things.	1	2	3	4
I do not feel guilty about anything in particular.	1	2	3	4
I am generally rather proud of myself.	1	2	3	4

I do not worry too much about temporary failures.	1	2	3	4
I have a positive attitude towards myself.	1	2	3	4
I am generally relaxed.	1	2	3	4
I have never wanted to die.	1	2	3	4
I am content with what I am.	1	2	3	4
I am not a worrier.	1	2	3	4
I wish I could be as happy as others seem to be.	1	2	3	4
I have little confidence in myself.	1	2	3	4
I am often bothered by unimportant thoughts that come into my mind.	1	2	3	4
On occasion I feel irritated and it bothers me to be with others.	1	2	3	4
I would like to have more self-respect.	1	2	3	4
I enjoy the sensations of speeding in a car.	1	2	3	4
I would like to take off on a trip with no pre-planned or definite routes or timetables.	1	2	3	4
I'll try anything once.	1	2	3	4
I like some physical activities that are somewhat risky.	1	2	3	4
I enjoy getting into new situations where you can't predict how things will turn out.	1	2	3	4
I like "wild" uninhibited parties.	1	2	3	4
I prefer fast-moving physical activities or sports.	1	2	3	4
I would like the kind of life where one is on the move and travelling a lot, with lots of change and excitement.	1	2	3	4
I like to let myself go and do impulsive things just for fun.	1	2	3	4
I would like to learn to fly an airplane.	1	2	3	4
I would like to travel to foreign lands where the people are quite different from the people in my own country.	1	2	3	4
I go to parties to meet exciting and stimulating people.	1	2	3	4
I think I would enjoy being a fire-fighter.	1	2	3	4
I like people who are unusual or different from most other people.	1	2	3	4
I do not try to restrain my urges to have exciting experiences.	1	2	3	4

If I were in the Army I might volunteer for exciting but dangerous duties.	1	2	3	4
I enjoy many types of loud, intense rock music.	1	2	3	4
I prefer quiet parties where one can have good conversations.	1	2	3	4
I do not like to engage in sports or activities in which there is a significant risk of getting hurt.	1	2	3	4
I would not like a job involving a lot of travel.	1	2	3	4
I am not interested in having new experiences just for the sake of experiencing new sensations.	1	2	3	4
I don't think I would like flying in a small airplane.	1	2	3	4
I do not like people who behave in uncontrolled and unconventional ways.	1	2	3	4
I enjoy quiet, melodic popular or classical music.	1	2	3	4
Given a choice I would never volunteer for any activity that is physically risky.	1	2	3	4
I am comfortable with the familiarity of a fixed daily routine.	1	2	3	4
One should not go too far in physical intimacy until one gets to know the other person.	1	2	3	4
I would never travel to countries where there is unrest and the threat of violence.	1	2	3	4
I would prefer to travel to places where people speak my language and have the same customs.	1	2	3	4
One of my main goals in life is to experience intense and pleasurable sensations.	1	2	3	4

Appendix B. Survey Measures: IPIP-50

On the following pages, there are phrases describing people's behaviours. Please use the rating scale below to describe how accurately each statement describes ***you***. Describe yourself as you generally are now, not as you wish to be in the future. Describe yourself as you honestly see yourself, in relation to other people you know of the same sex as you are, and roughly your same age. So that you can describe yourself in an honest manner, your responses will be kept in absolute confidence. Please read each statement carefully, and then tick the box that applies best to you.

1 **Very Inaccurate**	**2** **Moderately Inaccurate**	**3** **Neither Inaccurate nor Accurate**	**4** **Moderately Accurate**		**5** **Very Accurate**	
Am the life of the party.	1	2	3	4	5	
Feel little concern for others.	1	2	3	4	5	
Am always prepared.	1	2	3	4	5	
Get stressed out easily.	1	2	3	4	5	
Have a rich vocabulary.	1	2	3	4	5	
Don't talk a lot.	1	2	3	4	5	
Am interested in people.	1	2	3	4	5	
Leave my belongings around.	1	2	3	4	5	
Am relaxed most of the time.	1	2	3	4	5	
Have difficulty understanding abstract ideas.	1	2	3	4	5	
Feel comfortable around people.	1	2	3	4	5	
Insult people.	1	2	3	4	5	
Pay attention to details.	1	2	3	4	5	
Worry about things.	1	2	3	4	5	
Have a vivid imagination.	1	2	3	4	5	

Keep in the background.	1	2	3	4	5
Sympathise with others' feelings.	1	2	3	4	5
Make a mess of things.	1	2	3	4	5
Seldom feel sad.	1	2	3	4	5
Am not interested in abstract ideas.	1	2	3	4	5
Start conversations.	1	2	3	4	5
Am not interested in other people's problems.	1	2	3	4	5
Get chores done right away.	1	2	3	4	5
Am easily disturbed.	1	2	3	4	5
Have excellent ideas.	1	2	3	4	5
Have little to say.	1	2	3	4	5
Have a soft heart.	1	2	3	4	5
Often forget to put things back in their proper place.	1	2	3	4	5
Get upset easily.	1	2	3	4	5
Do not have a good imagination.	1	2	3	4	5
Talk to a lot of different people at parties.	1	2	3	4	5
Am not really interested in others.	1	2	3	4	5
Like order.	1	2	3	4	5
Change my mood a lot.	1	2	3	4	5
Am quick to understand things.	1	2	3	4	5
Don't like to draw attention to myself.	1	2	3	4	5
Take time out for others.	1	2	3	4	5
Shirk my duties.	1	2	3	4	5
Have frequent mood swings.	1	2	3	4	5
Use difficult words.	1	2	3	4	5
Don't mind being the centre of attention.	1	2	3	4	5
Feel others' emotions.	1	2	3	4	5

Follow a schedule.	1	2	3	4	5
Get irritated easily.	1	2	3	4	5
Spend time reflecting on things.	1	2	3	4	5
Am quiet around strangers.	1	2	3	4	5
Make people feel at ease.	1	2	3	4	5
Am exacting in my work.	1	2	3	4	5
Often feel sad.	1	2	3	4	5
Am full of ideas.	1	2	3	4	5

Appendix C. Survey Measures: SLE-10 Stressful Life Events Scale

Please circle Y for 'yes' or N for 'no'. In the last 6 months:

1. Have you suffered from a serious illness, injury or an assault? Y/N
2. Has a serious illness, injury or assault happened to a close relative? Y/N
3. Has a close friend or relative of yours died? Y/N
4. Have you had a separation due to marital difficulties or a relationship breakup? Y/N
5. Have you had a serious problem with a close friend, neighbour or relative? Y/N
6. Have you moved from your usual place of residence? Y/N
7. Have you had a major financial crisis? Y/N
8. Have you had problems with the police or other government agency? Y/N
9. Has something you valued been lost or stolen? Y/N
10. Have you lost or had to give up a job? Y/N

Appendix D. Information Sheet

TE WHARE WĀNANGA O TE ŪPOKO O TE IKA A MĀUI
VICTORIA
UNIVERSITY OF WELLINGTON

What is the purpose of this research?
This study examines the connections between personality characteristics in terms of traits, physiology and genes. We hope that this research will provide new information about the relationships between these different measures of personality.

Who is conducting the research?
This research is conducted by Anna Lee and Yee-Wei Ooi, MSc students in the School of Psychology at Victoria University of Wellington, supervised by Dr. Ronald Fischer. This research has been approved by Victoria University of Wellington's Human Ethics Committee.

What is involved if you agree to participate?
If you agree to participate in this study you will be asked to complete surveys which measure various aspects of your personality and ask questions about significant events in your life. You will undertake a video-taped negotiation exercise with another participant while you have your heart rate monitored. We will examine your negotiation behaviour and tactics. You will provide saliva for genetic analysis of specific regions of your genes that have been related to differences in personality. The genetic material that we collect will be destroyed during the analysis process. By signing overleaf, you indicate that you have provided informed consent to participate in the study. During the research you are free to withdraw at any point up to two weeks after your data have been collected.

Privacy and Confidentiality
We will keep your consent forms and data for at least five years after publication. You will never be identified in this research project or in any other presentation or publication. The information you provide will be coded by number only, and your email address and name will be stored in a separate file. In accordance with the requirements of some scientific journals and organisations, your data - without identifying details - may be shared with other competent researchers. Data without identifying names may also be used in other, related studies. A copy of data without identifying names will remain in the custody of Anna Lee, Yee-Wei Ooi and Dr Ronald Fischer.

What happens to the information that you provide?
The overall findings will be part of Master's theses that will be submitted for assessment. The overall findings may be submitted for publication in a scientific journal, presented at scientific conferences, or published in the media. If you would like to know the results of this study, they can be emailed to you in about 10 weeks when we have completed initial analyses. Please contact us via email to request the results.

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Appendix E: Consent Form

TE WHARE WĀNANGA O TE ŪPOKO O TE IKA A MĀUI
VICTORIA
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Personality: Genes, Physiology and Traits Research Study Statement of Consent

- I have read the information about this research and any questions I wanted to ask have been answered to my satisfaction.
- I agree to participate in this research. I understand that I can withdraw my consent up to two weeks after my data have been collected.
- I agree to participate in a paired negotiation exercise with another student.
- I agree to be video-taped during this research.
- I agree to have my heart rate monitored during this research.
- I agree to have my DNA collected via a saliva sample. I understand that this sample of my genetic material will be coded by number and not by my name. I understand that my genetic material will be destroyed during the analysis process.

Name: _____

Signature: _____

Student ID: _____

Date: _____

Copy to:

[a] participant,

[b] researcher (initial both copies below)

Appendix F: Debriefing Sheet

TE WHARE WĀNANGA O TE ŪPOKO O TE IKA A MĀUI
VICTORIA
UNIVERSITY OF WELLINGTON

Personality: Genes, Physiology and Traits Research Study Debriefing Information

- Thank you for participating in this research into personality. This research looks at the relationships between personality traits (how you think, feel and behave), physiology (your heart rate variability), and genes that have been associated with differences in personality (dopamine and serotonin genes in your DNA). We will try to find connections between your survey responses, your heart rate variability, and specific genetic variants (called polymorphisms) in your DNA.
- Genetic research has shown that polymorphisms in specific genes (in the dopamine and serotonin systems) may influence personality traits like neuroticism and sensation-seeking. Neuroticism includes things like anxiety, depression and low self-esteem. Sensation-seeking includes things like thrill-seeking, experience-seeking and disinhibition.
- Physiological research suggests that people with high levels of neuroticism have decreased heart rate variability, while people with high levels of sensation-seeking have increased heart rate variability.
- This research is conducted by Anna Lee and Yee-Wei Ooi, MSc students in the School of Psychology at Victoria University of Wellington, supervised by Dr. Ronald Fischer. This research has been approved by Victoria University of Wellington's Human Ethics Committee.
- Thank you again for participating in this research. If you have any questions regarding your involvement in the research, or issues regarding the research in general, please do not hesitate to contact us via email. If you would like to know the results of this study, they can be emailed to you once the results are available. Please contact us via email to request the results.

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Appendix G: Genotyping Protocol Development

DNA Collection

DNA samples collected from the researchers were used for early tests of DNA collection and amplification procedures. Cotton-buds, wooden toothpicks and plastic brushpicks were all tested as collection tools. After repeated testing, the brushpicks were the best option, in terms of ease of use and maximum DNA yield. The wooden toothpicks could be painful and draw blood when scraped across the inside of the cheek, and they also collected a very small amount of DNA. Meanwhile, the glue and bleach used in the cotton-buds appeared to interfere with the PCR process.

DNA Extraction

Chelex-100 chelating resin (6% solution) was chosen as the best DNA buffer/extraction medium, as it is comparatively cheap, non-toxic and does not require pre-PCR purification/desalting as some alternative media do (Walsh, Metzger & Higuchi, 1991).

PCR Amplification

To plan and run the PCR, we began with the published 5HTTLPR/DAT1/DRD4 protocols of the University of Colorado (Boulder) Institute for Behavioral Genetics (http://ibgwww.colorado.edu/genotyping_lab/protocols.html). Personal correspondence with IBG lab director Andrew Smolen suggested that setting up and optimising the protocols would be a significant amount of work. With this in mind, a further step involving another fluorescent primer pair to identify an A/G SNP (via restriction digest) to further classify 5HTTLPR long alleles as L(A) or L(G) was not undertaken. L(G) is believed to function more like the S allele (Hu, et al., 2005; Whisman, Richardson, & Smolen, 2011), but Dr. Smolen noted that few L(G)s are found in Caucasian populations. As our study sample was over 60% Caucasian, we decided to rely on bi-allelic classification (S/L).

The IBG protocols list very specific primer volumes for the Primer Mastermix: 9µL of DAT1 + and - primers, 18µL of DRD4 + and - primers, 19µL of 5HTTLPR + and - primers, and ddH20 to make up a total volume of 1342µL. This level of precision is rather difficult to maintain when making multiple batches of the recipe rather than a one-off 'feast', so a simplified Mastermix using 3µL of DAT1 +/- and 6µL for both DRD4+/- and 5HTTLPR+/- was prepared. The IBG PCR Supermix Recipe is made from scratch, with DMSO (dimethyl sulfoxide), ABI buffer, MgCl (magnesium chloride), dNTPs (deoxynucleotide triphosphates), deazaGTPs (7-deaza-2´-deoxyguanosine 5´-triphosphate), and AmpliTaq polymerase all added to the Mastermix.

After consultation with Dr. Darren Day and Dr. Ryan Steel in the Biology lab, we tested a number of pre-prepared Kapa polymerase Supermix kits (Kapa Biosystems, USA). The first kit was the Kapa2G Fast Multiplex PCR Kit, which contained Kapa2G Fast HotStart DNA polymerase, Kapa2G Buffer A, dNTPs, MgCl and stabilisers. To make up 20µL PCR volumes, 2µL of DNA template is added to 18µL of Supermix. As the DNA samples had not been processed immediately, and cheek cell/mouth swab samples may provide less DNA than other methods (saliva, blood etc.), a higher template proportion of 4µL template to 16µL Supermix was used in order to mitigate the effects of potential DNA degradation/low yield. IBG protocols used the following 40-cycle Touchdown PCR on an ABI3130 PCR machine:

- Denaturation: 1x [95°C for 10:00]
- Annealing: 10x [95°C for 0:30, 65-55°C for 0:30 (Touchdown: drop temperature by 2°C every 2nd cycle), 72°C for 1:30]
- Extension: 30x [90°C for 0:30, 55°C for 0:30, 72°C for 1:30]
- Final Extension: 1x [72°C for 30:00, then 4°C hold until removed from machine]

This very long 40-cycle programme was adapted to more closely follow the recommended cycling parameters for the Kapa2G Fast Multiplex Kit. The first PCR test programme 'PCQx30' reduced both the number of cycles and the length of steps within the cycles:

Samples were run for each primer pair separately (singleplex DAT1, DRD4 and 5HTTLPR), as well as the 3-in-1 multiplex.

PCQx30

- Denaturation: 1x [95°C for 3:00]
- Annealing: 10x [95°C for 0:15, 65°C for 0:15, 72°C for 0:30]
- Extension: 20x [90°C for 0:15, 55°C for 0:30, 72°C for 1:30]
- Final Extension: 1x [72°C for 10:00, then 4°C hold until removed from machine]

To visualise the genetic polymorphisms, the full 20µL volume for each test sample (plus 1µL blue loading dye) was electrophoresed at 150mv along lanes in a 2% agarose gel stained with ethidium bromide (EthBr), using a 50-base-pair DNA ladder for product size comparison. The gel was visually examined under UV illumination and photographed on a black & white Kodak GL100 camera using Kodak 1D software (Eastman Kodak Company, USA). Test one results indicated under-amplification: the primers had run through to the end of each lane (with plenty of primer remaining) but no PCR product bands were visible.

The second PCR test programme 'PCQx40' returned the extension cycles to the IBG 40x and reduced the annealing temperature from 65 to 60°C, after melting temperatures (T_M) were calculated for each of the primer pairs, suggesting that a lower annealing temperature might be justified[8]. The same agarose gel and photography process was used, and this time over-amplification was indicated: there was little primer left at the end of each lane, and there were multiple (10+) PCR product bands visible. Note that the expected number of bands is 1 (homozygote) to 2 (heterozygote) per singleplex lane, and 1 (homozygote for all genes, and the product sizes overlap) to 6 (heterozygote for all genes, and the product sizes do not overlap) per multiplex lane.

[8]DAT1+ 60.6°C, - 62.5°C; DRD4 + 62.5°C, - 65.9°C; 5HTTLPR + 57.4°C, - 62°C.

PCR test three 'PCQx35gradient' reduced the number of cycles to 35x, and experimented with running different annealing temperatures in the same batch. The 'gradient' feature on the iCycler allows researchers to set different areas of the machine at different temperatures. Multiple samples from the same participant were run at 70°C, 64°C and 60°C to try to determine the optimum annealing temperature. Singleplexes only were run in order to try to visualise which, if any, of the primer pairs were working. Clear single bands were visible for 5HTTLPR and DAT1, suggesting the test participant was a homozygote for both polymorphic loci (namely 5HTTLPR S/S and DAT1 10/10). However, no bands were present in any of the DRD4 lanes. There was not much visible difference between the annealing temperatures, though the 70°C lanes seemed slightly clearer/stronger. Careful analysis of the DRD4 48bp VNTR amplicon revealed extremely high GC content (>90%). As G-C molecular bonds are twice as strong as A-T bonds, products with a high GC content can be very difficult to amplify successfully as they may fold in on themselves and/or form secondary structures that make it harder for the primers to cut the amplicon in the right place.

The fourth PCR test 'PCQx35' was designed specifically to try to get the DRD4 primers working at 35 cycles with a longer annealing step of 70°C for 0:30, and without the final extension of 72°C for 10:00. The results were disappointing, with no bands visible in any lane – not even DAT1 or 5HTTLPR. After consideration, a new Kapa2G Robust kit designed for 'difficult samples' was trialed. The fifth PCR test ran the 'PCQx35new' with the 'PCQx35gradient' settings but with a uniform 70°C for 0:15 annealing step, using Kapa2G Robust instead of Multiplex. Again, results were disappointing with no bands visible in any lanes. PCR test six returned to the Kapa2G Fast Multiplex and the 'PCQx35gradient' programme, but due to a technical malfunction, the machine failed to progress from the initial 95°C denaturation step – leaving the samples at 95°C for over two hours. Surprisingly, products were clearly visible for DAT1 and 5HTTLPR, and DRD4 showed a large 'smudge' of overlapping bands.

More research uncovered the importance of using DMSO to inhibit the formation of DNA secondary structures (Lerman et al., 1998), as in the IBG protocols. PCR test seven ran three sets of samples from the same participant, using the Kapa2G Fast Multiplex and adding 0, 5 and 10% DMSO to the Supermix recipe, with other conditions unchanged from test six. Results were encouraging – and best for the 10% DMSO singleplexes - but with some evidence of over-amplification (more bands per lane than expected). The eighth PCR test 'Smolen' went back to the IBG protocols, using 40 cycles, the Touchdown 65 to 55°C annealing step and the 10:00 72°C extension. Three different participant samples were run to try to depict a range of different genotypes. Results were not as promising as hoped, with very faint bands visible in most lanes but strong evidence of under-amplification shown by very bright wells of unused primer at the end of every lane.

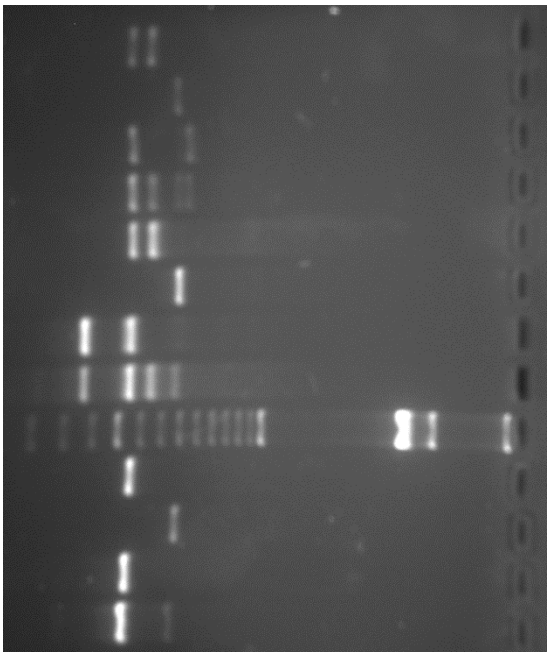
Alongside the Fast Multiplex polymerase, PCR test nine trialed a new Kapa2G Robust + Kapa dNTP mix kit, which included a number of buffers, enhancers and dNTPs as well as the polymerase. After some experimentation, Dr. Ryan Steel developed the followingmultiplex recipe for a single 20µL reaction (i.e. one participant sample). For multiple reactions, the recipe is scaled up as appropriate.

- Primers: DAT1 + (0.6µL) – (0.6µL), DRD4 + (1.2µL) – (1.2µL), 5HTTLPR + (1.2µL) – (1.2µL)
- Reagents: Kapa2G Robust polymerase (0.16µL), dNTPs (0.4µL), GC Buffer (4µL), Enhancer (4µL), DMSO (1µL)
- DNA Template: Sample DNA (4µL), ddH20 (to make up to desired volume of 20µL): (5.24µL).

New PCR settings were created for the '35x60w72' programme with 35 cycles, annealing at 60°C and a 10:00 72°C extension. Singleplex DAT1 and 5HTTLPR lanes were run for four participants, comparing the different polymerases. In order to increase fidelity, only 10µL of each PCR reaction was run in the agarose gel. The electrophoresis voltage was also lowered

from 150 to 90mv, making for a longer run time and greater detail visible in the gel. Results looked good for the Robust kit, and lanes running negative controls (Chelex only, no DNA) were reassuringly empty.

PCR test ten used the Kapa2G Robust recipe with the '35x60w72' programme for three different participants, running all three singleplex reactions as well as the multiplex. Results were good, with all lanes showing bands of appropriate sizes and no bands in the negative control lanes (see Figure G1).



DOM608:	5HT – 376,419bp (S/L)
DOM608:	DAT1 – 480,480bp (10/10)
DOM608:	DRD4 – 375,375bp (4/4)
DOM608:	ALL
DOM609:	5HT – 376,419bp (S/L)
DOM609:	DAT1 – 480,480bp (10/10)
DOM609:	DRD4 – 279,375bp (2/4)
DOM609:	ALL
50 base pair SIZE STANDARD	
INT729:	5HT – 376,376bp (S/S)
INT729:	DAT1 – 480,480bp (10/10)
INT729:	DRD4 – 375,375bp (4/4)
INT729:	ALL

Figure G1: Agarose gel image of three participant samples, showing selected DAT1, 5HT and DRD4 singleplexed and multiplexed polymorphisms.

At this point, 1µL volume multiplex samples from two participants were sent to New Zealand Genomics Ltd. (NZG) for capillary separation in order to make precise allele size calls accurate to 1bp. Following NZG recommendations suggesting an optimal sample dilution of between 1:3 and 1:100, each sample was sent at 1:25 and 1:50 dilution with ddH2O. Using GeneMarker software (Applied Biosystems, USA), the results from NZG showed that less dilution was required, as allele peaks were low and barely registering on the peak height scale of 500-20,000 units. The same two samples were sent off at 1:5 and 1:1 (i.e. no) dilution,

with better results – although the DAT1 peaks were still very weak (but still discernible from background noise and LIZ1200[9] size standard peaks).

A 1:1 sample dilution was used for all following samples, in order to streamline the process and avoid further opportunity for contamination and processing errors. The PCR recipe was also revised in order to increase DAT1 primer concentrations – now using the same amounts as the DRD4 and 5HTTLPR primers (i.e. 1.2µL + and 1.2µL -). Three multiplexed samples were sent to NZG using the new recipe, and using 1.5µL volume samples to mitigate any evaporation/spillage during processing, with encouraging results (see figure A2). The rest of the samples were batch processed, with every sample run on a gel to provide a backup/comparison for the NZG capillary separation. Ten samples did not initially visualise in a gel, and required reprocessing and rerunning before being sent on to NZG. All 113 participant samples were successfully allele-called for each of the three genetic polymorphisms under study.

[9] LIZ1200 (Applied Biosystems, USA) is an orange fluorescent dye used with the ABI DS-33 set, to size DNA fragments in the 20-1200bp range.

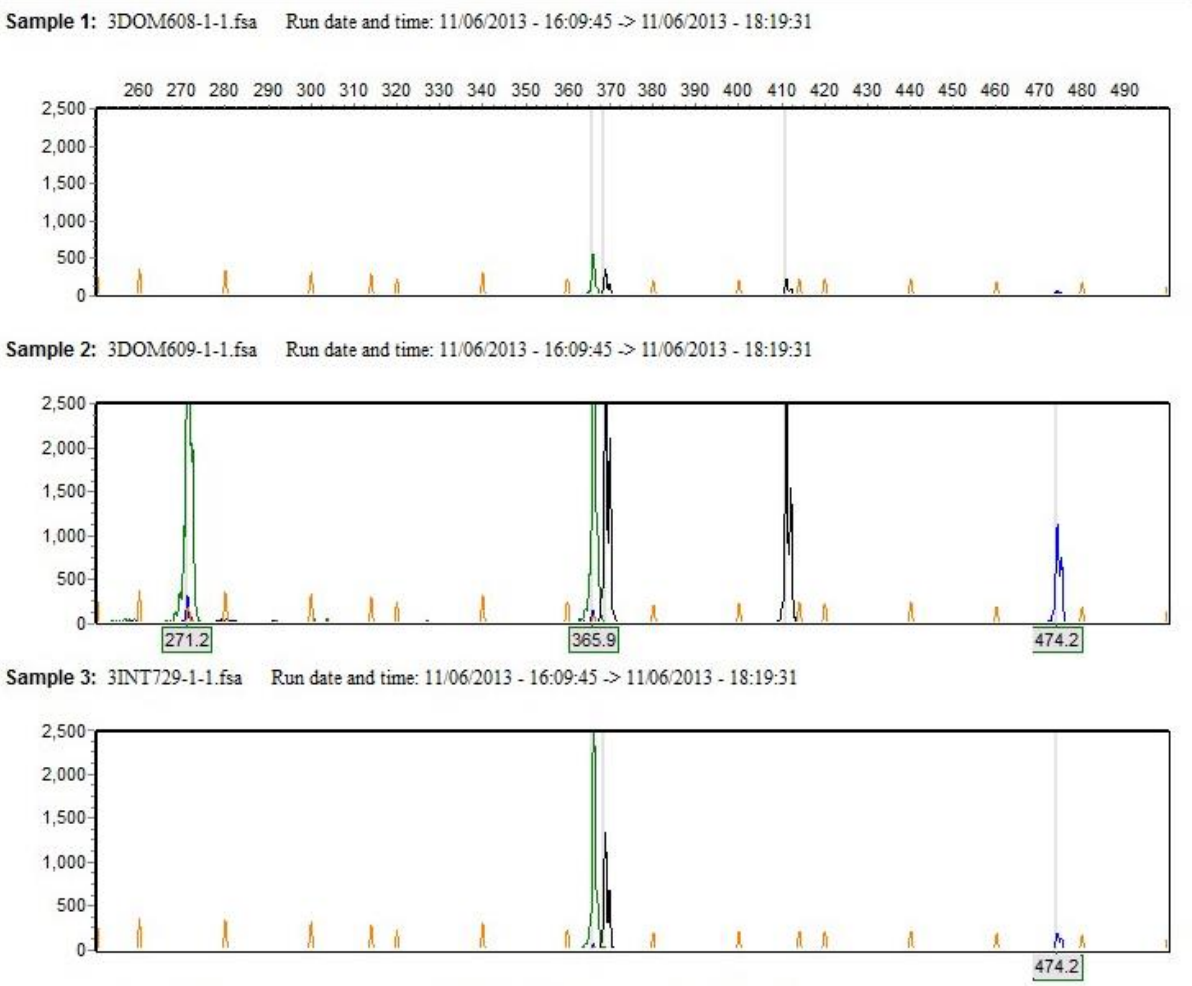


Figure G2: Electropherogram of three participant samples, showing DAT1 (blue), 5HT (black) and DRD4 (green) polymorphisms. Alleles are within 10bp of expected sizes. Note DOM608 (top panel) DAT1 peaks are outside accepted height range but are at expected size.

Appendix H: Personality Traits by DAT1, DRD4 and 5HTTLPR Genotype

DAT1 genotype		9/9 (*n*=8)		9/10 (*n*=36)		10/10 (*n*=66)		Other (*n*=4)	
Personality Variable	**Scale**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
IPIP_Extraversion	1 to 5	3.64	0.40	3.28	0.74	3.14	0.79	3.70	1.21
IPIP_Agreeableness	1 to 5	4.04	0.42	3.96	0.69	3.99	0.53	3.43	1.27
IPIP_Conscientiousness	1 to 5	3.26	0.89	3.46	0.61	3.38	0.72	3.43	0.35
IPIP_Emotional Stability	1 to 5	2.90	0.58	3.07	0.75	2.91	0.89	2.43	0.78
IPIP_Intellect	1 to 5	3.69	0.27	3.55	0.49	3.63	0.63	3.97	0.81
NE_Anxiety	1 to 4	2.56	0.32	2.31	0.48	2.30	0.66	2.60	0.26
NE_Depression	1 to 4	2.46	0.32	2.24	0.54	2.41	0.64	2.80	0.56
NE_Low Self Esteem	1 to 4	2.51	0.42	2.18	0.65	2.26	0.73	2.33	0.31
SS_Thrill-Seeking	1 to 4	2.46	0.51	2.69	0.68	2.57	0.63	3.17	0.42
SS_Experience-Seeking	1 to 4	2.88	0.23	2.78	0.50	2.89	0.58	2.80	0.26
SS_Disinhibition	1 to 4	2.59	0.43	2.73	0.50	2.65	0.63	2.90	0.10
NE_total	1 to 4	2.51	0.29	2.25	0.49	2.32	0.64	2.58	0.37
SS_total	1 to 4	2.64	0.22	2.73	0.48	2.70	0.50	2.96	0.10
DRD4 genotype		2R (*n*=20)		4/4 (*n*=58)		7R (*n*=23)		Other* (*n*=12)	
Personality Variable	**Scale**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
IPIP_Extraversion	1 to 5	3.30	.82	3.14	0.79	3.53	.65	3.03	.77
IPIP_Agreeableness	1 to 5	3.72	.62	3.99	0.64	4.22	.43	3.79	.42
IPIP_Conscientiousness	1 to 5	3.18	.67	3.37	0.67	3.47	.66	3.80	.75
IPIP_Emotional Stability	1 to 5	2.87	.72	2.91	0.84	3.20	.94	2.79	.62
IPIP_Intellect	1 to 5	3.50	.53	3.66	0.62	3.63	.56	3.60	.50
NE_Anxiety	1 to 4	2.43	.57	2.34	0.52	2.14	.77	2.45	.49
NE_Depression	1 to 4	2.51	.58	2.40	0.53	2.19	.78	2.33	.49
NE_Low Self Esteem	1 to 4	2.29	.44	2.25	0.60	2.19	1.00	2.34	.69
SS_Thrill-Seeking	1 to 4	2.74	.49	2.64	0.60	2.43	.86	2.64	.56
SS_Experience-Seeking	1 to 4	2.96	.46	2.86	0.48	2.70	.75	2.90	.41
SS_Disinhibition	1 to 4	2.72	.42	2.69	0.43	2.69	.94	2.54	.49
NE_total	1 to 4	2.41	.48	2.33	0.50	2.17	.82	2.37	.52
SS_total	1 to 4	2.80	.35	2.73	0.41	2.60	.70	2.70	.42
5HTTLPR genotype		S/S (*n*=27)		S/L (*n*=55)		L/L (*n*=29)		S/XL (*n*=2)	
Personality Variable	**Scale**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
IPIP_Extraversion	1 to 5	3.10	.80	3.25	.76	3.38	.77	2.55	.49
IPIP_Agreeableness	1 to 5	3.92	.61	3.94	.62	4.09	.55	3.55	.07
IPIP_Conscientiousness	1 to 5	3.34	.68	3.54	.70	3.24	.67	3.00	.14
IPIP_Emotional Stability	1 to 5	3.12	.86	2.94	.87	2.79	.72	3.05	.64
IPIP_Intellect	1 to 5	3.52	.52	3.72	.58	3.54	.60	3.20	.28
NE_Anxiety	1 to 4	2.27	.50	2.38	.56	2.25	.71	2.85	.21
NE_Depression	1 to 4	2.25	.46	2.43	.55	2.35	.78	2.60	.57
NE_Low Self Esteem	1 to 4	2.19	.59	2.26	.63	2.31	.86	2.45	.49
SS_Thrill-Seeking	1 to 4	2.52	.70	2.66	.52	2.59	.80	2.80	.28
SS_Experience-Seeking	1 to 4	2.89	.53	2.84	.46	2.81	.66	3.45	.07
SS_Disinhibition	1 to 4	2.58	.50	2.72	.56	2.67	.65	2.95	.35
NE_total	1 to 4	2.23	.46	2.36	.54	2.30	.74	2.63	.42
SS_total	1 to 4	2.66	.49	2.74	.38	2.69	.62	3.07	.19

* One participant with the 2/7 genotype was excluded.