

The Etiology of Personality Function: The University of British Columbia Twin Project

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The UBC Twin Project is an on-going study of personality and personality disorder that has collected personality data on approximately 1000 adult volunteer twin pairs. The primary purpose of the study is to develop and validate measures of the major forms of personality disorder as a quantitative alternative to the classificatory system first proposed by the DSM-III using the standard methods of personality and behavioural genetics research. To date, the study has explored the univariate and multivariate relationships between the major personality and personality disorder inventories, and their relationships to other psychopathologies including the mood and anxiety disorders and key psychological variables, such as cognitive ability, attachment and attitudes. The results have several implications for personality and personality disorders, most notably a rationalized diagnostic/nosological system to enhanced measurement instruments that index and reflect the influence of specific genetic and environmental influences.

The UBC Twin Project began recruiting twins from the general population in 1991. The study grew out of previous studies of the phenotypic structure of personality disorder that led to the development of a self-report questionnaire that provides a comprehensive assessment of the different components of personality pathology. The specific research questions and issues that the study was designed to address, and which continue to be the focus of our endeavors, are: 1) to establish the genetic and environmental etiology of personality disorder traits, 2) to investigate the genetic architecture of normal and disordered personality, 3) to explore the relationship between normal and disordered personality, 4) to investigate etiological relationships among personality, personality disorder, and psychopathology.

The sample consists of a register of about 1000 adult pairs drawn from the major urban centers of southwestern British Columbia. Twin pairs were recruited via media appeals. This included classified and box advertisements in the major daily newspapers and weekly community and special interest newspapers that reach most homes free-of-charge. Appearances by the authors on local and national radio and television shows and interviews in popular women's and family magazines were also used to recruit participants. Invitations from local twin organizations and family groups to speak about the research program at community centers and some public schools have also yielded participants. This

has been particularly helpful for establishing a register of child and adolescent twins for possible future studies.

The study adopted some of the suggestions outlined in Lykken et al. (1987) to overcome some of the problems that plague purely volunteer based samples (e.g., low participation and completion rates). Following this advice, the UBC Twin Project provided substantial cash honorariums (\$50.00 – \$100.00 per member of a pair), mementos (e.g., customized pens, pencils and refrigerator magnets with the “UBC Twin Project” logo and toll-free telephone contact numbers), and copies of the *Twin Times*, an annual newsletter that summarizes recent research findings and facts about twins. As a result, the study has enjoyed excellent completion rates (over 90%) and equal number of MZ to DZ twin pairs from quite diverse socioeconomic levels. However, the cash honorarium did encourage more male pairs to participate, but relatively few in comparison to the number of female participants.

Several of our projects are collaborative in nature, and because we have collected data on a variety of measures, our datasets are easily merged with other sets to permit a wide range of analyses that is the consequence of larger sample sizes. We presently collaborate and wish to collaborate with other twin and genetic groups as well as clinical service units.

The Development of the Phenotype

The twin study research was built upon investigations of the phenotypic structure of personality disorder that began in the late 1970s. The publication of the DSM-III in 1980 created interest in phenotypic structure of personality disorder. The initial focus of this work was to validate the major categories of personality disorder proposed by the DSM-III using the standard methods of personality research. The first step was to develop systematic descriptions of personality disorders based on extensive reviews of the clinical literature (Livesley, 1985a, 1985b; 1986). This produced descriptions of the traits comprising the

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various categories of personality disorder. Clinical judgments were used to organize these descriptions into a smaller number of traits (Livesley, 1987). This resulted in personality disorders as a whole being described in terms of approximately 100 traits.

The next step was to develop self-report scales to assess each trait. Factor analysis of these scales based on data from general population subjects identified 15 factors (Livesley et al., 1989). Higher-order analyses revealed four broad factors, currently labeled Emotional Dysregulation, Dissocial Behavior, Inhibitedness and Conscientiousness, that resemble four of the five major factors described in the five-factor model of personality (Livesley, 1991). The 15-factor structure was subsequently shown to be stable across clinical and non-clinical samples (Livesley et al., 1992), supporting a dimensional model of personality function: personality disorders are extreme variants of normal personality traits.

These findings were used to construct a new self-report scale — the Dimensional Assessment of Personality Pathology (DAPP; Livesley & Jackson, in press). This scale taps 18 traits derived from multivariate studies: Anxiousness, Affective Lability, Callousness, Cognitive Distortion, Compulsivity, Conduct problems, Identity problems, Insecure Attachment, Intimacy problems, Narcissism, Oppositionality, Rejection, Restricted Expression, Self-Harm, Social Avoidance, Stimulus Seeking, Submissiveness, and Suspiciousness. As noted above, several factorial analyses show that these basic dimensions are organized into four higher order factors. Because the 18 scales were derived from a longer list of more specific traits, each of the basic traits subdivides into two or more specific traits. For example, the Anxiousness scale can be divided into four specific traits, trait anxiety, rumination, guilt proneness, and indecisiveness, each assessed by an eight-item scale. The most extensive version of this instrument that we have used in twin study research is therefore organized into three levels: four higher-order factors, 18 basic trait scales, and 69 specific traits. This has provided opportunities to explore the genetic architecture of personality and personality disorder in greater detail than is possible with measures that only assess broad dimensions, such as the Eysenck Personality Questionnaire (EPQ-R; Eysenck & Eysenck, 1992), or measures such as the NEO-PI-R (Costa & McCrae, 1992) that only have two levels of construct: higher-order domains and facet scales. Moreover, scales such as the NEO-PI-R typically assess personality function in the normal range and suffer from possible ceiling effects in clinical research, thus limiting its use in studies of extreme behaviour (see Jang & Vernon, 2001; Jang et al., 2001 for a discussion).

The Heritability of Personality Dysfunction

The initial focus of our twin research was to establish the heritability of the various components of personality disorder measured by our instrument. When this work was originally planned, the prevailing opinion within psychiatry was that personality disorder was largely the result of psychosocial adversity. At that time, the extensive literature on the heritability of normal personality had not influenced clinical thinking about the nature and origins of personality

disorder. Our initial study demonstrated the heritability of most of the 18 basic traits (Livesley et al., 1994), including constructs such as “narcissism” that were considered to be the product of defensive reactions to adversity. Subsequent, more detailed analysis of all levels of the trait hierarchy based on a larger sample confirmed these findings and showed that all components of personality disorder were highly heritable (Jang et al., 1996). Subsequent analyses suggested that for most personality disorder traits, the genetic influences affecting women were the same as those affecting men (Jang et al., 1998), and that heritable influences were more important in adolescents and young adults than in older adults, where non-genetic factors play a greater role on more traits (Jang et al., 1999).

Having established the heritability of all traits delineating personality disorder, our attention then focused on our second research question, namely, the genetic architecture underlying these traits. We began by investigating the phenotypic structure and genetic architecture of the 18 basic dimensions of personality disorder in three samples: a general population sample ($N = 939$), a clinical sample of patients with personality disorder ($N = 656$), and a twin sample ($N = 686$ twin pairs). Factor analyses of the phenotypic structure of the 18 traits in these three samples identified the same four-factor structure described in earlier studies. This structure was highly stable across the three samples (Livesley et al., 1998). Corresponding analyses of the genetic correlations among these traits revealed a congruent factor structure. These results suggested that the phenotypic structure of personality disorder closely corresponds to the underlying genetic architecture. The implication is that genetic, not environmental factors, primarily contribute to trait covariation (Livesley et al., in press).

The results indicated that four broad genetic dimensions contribute to the phenotypic variation in personality disorder. However, additional analyses in which the variance unique to each of the 18 scales was estimated by regressing the common variance due to the four higher-order factors revealed the residual heritable component for many of these traits. The implication drawn from this finding is that personality disorder is influenced by a small number of genetic dimensions that have a broad impact on personality structure and a more specific set of genetic dimensions that have more specific effects on phenotypic traits. The importance of these results for the clinical treatment of personality disorder, specifically in regards to psychosocial approaches, is explored in Livesley (2000).

The Relationship Between Normal and Abnormal Personality Function

A feature of the UBC Twin Project is that data was also collected using measures of normal personality such as the EPQ-R and NEO-PI-R. This has permitted several studies of the biometric structure of personality that have addressed such long standing issues as the number of basic dimensions required to represent personality variation, and the etiological basis of higher order traits such as neuroticism or extraversion and their relationship to abnormal personality function. What has been striking is the similarity in findings across the two domains of personality

function. For example, Jang et al.'s (1998) study of the NEO-PI-R scales regressed out the influence of the Big Five personality dimensions from each of the facet scales and revealed a substantial heritable residual component to most of the 30 facets delineating the five domains, supporting the hypothesized hierarchical nature of personality. Factorial analysis of the genetic and environmental correlations between the 30 NEO-PI-R facet scales yielded the familiar Five-Factor Model (McCrae et al., 2001), demonstrating that phenotypic structure is a reflection of the genetic structure. However, like the Livesley et al. (1998) paper, environmental factors have a different influence on personality structure. This general finding was replicated in Jang et al. (2002) but was also shown to be consistent across culturally diverse samples (see also Jang et al., 2001), and gender (Jang et al., 2002).

These findings are of interest because evidence of the differential effects of genetics and the environment on personality structure and function has important implications for assessment and clinical intervention. The consistent finding that the higher order structure of personality disorders resembles the higher order structure of normal personality lends support for the argument that personality disorder merely represents the extremes of normal personality variation that throws into question the validity of current approaches to classifying personality disorder. This conclusion was further investigated by showing that the reason why scales of normal personality function, such as the NEO-PI-R, and abnormal function like the DAPP correlate is because they share a common genetic basis (Jang & Livesley, 1999), and this has led us to explore the relationship between normal and disordered personality in more detail (Livesley & Jang, 2000).

Studies in Comorbidity and Implications for Classification

Over the past 11 years, we have also collected data on common psychiatric conditions. This has allowed us to explore the etiology of these disorders and their relationship to personality function. For example, we have studied mood changes with the seasons (Jang et al., 1998; Jang et al., 1997a, 1997b), and alcohol and drug misuse (Jang et al., 1995, 1997; Jang et al., 2000). Several collaborative projects with other twin and family studies have allowed studies of anxiety disorders (Stein et al., in press; Stein et al., 2001; Stein et al., 2001; Jang et al., 1999; Stein et al., 1999) and psychological variables such as attitudes (Olson et al., 2001), leadership styles (Johnson et al., 1999), adult attachment styles (Brussoni et al., 2001), and the relationship between personality and cognitive ability (Ashton et al., 2000). The findings from these projects illustrate the central role of personality in different psychological and psychiatric processes. This is allowing us to take a different perspective on comorbidity. It is also causing us to question the value of classifying mental disorders on two axes (Livesley et al., 1994; Livesley & Jang, 2000; Livesley, 2001, in press).

Current research includes reconstructing the DAPP to reflect the genetic architecture of personality more closely. Multivariate genetic analyses are being used to identify the specific genetic dimensions underlying personality.

Scales are being redefined such that each scale assesses a single genetic dimension and items are genetically homogeneous units as opposed to the factorially homogeneous units employed in traditional scale development. Several approaches to achieving this goal are being experimented with, such as computing weights to index specific genetic and environmental influences on personality measures (e.g., Jang et al., 2001). Our research is also following some general trends in the literature, such as following up earlier research identifying factors in the environment influencing personality (Vernon et al., 1997), and preliminary research explicating the mechanism of their action, specifically gene-environment correlations (Jang et al., 2001) and gene-environment interactions. Other research directions include studies designed to identify putative loci in personality. Unlike many molecular genetic influences on a single trait, a recent collaborative project focused on identifying loci responsible for the covariance of traits (Jang et al., 2001).

In summary, our twin research has introduced biological/genetic criteria into the study of personality function. The results have several implications for personality and personality disorders, most notably a rationalized diagnostic/nosological system to enhanced measurement instruments that index and reflect the influence of specific genetic and environmental influences.

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