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EPIGENETIC PERSPECTIVE ON BEHAVIOR DEVELOPMENT, PERSONALITY, AND PERSONALITY DISORDERS

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SUMMARY

After 30 years of clinical work and research based on categorical criteria for personality disorders (Diagnostic and Statistical manual of Mental Disorders – DSM IV TR) and (International Classification of Diseases - ICD 10th revision), a solid conceptual understanding and treatment of these disorders have not been established. For the field to move forward, it is imperative that future classifications introduce major revisions of the concept, diagnosis, and classification of personality disorders. This paper proposes one such revision.

Based on recent advances in molecular biology and epigenetics, we define personality disorders as maladaptive syndromes developed trough person-environment interaction. We conceptualize maladaptation as a failure of integrative functions of personality (i.e., those that carry out adaptive processes) caused by strong biogenetic dispositions or by pathological environmental effects, or both. Hence, accurate diagnosis of personality disorder depends upon neurobiological (innate) and adaptive (interactive) etiological factors. We propose a 2-step diagnostic algorithm for personality disorders: adaptive processes (i.e., character) are used to diagnose maladaptation, whereas biological aspects (i.e., temperament) are used to specify dominant clinical presentation and for differential diagnosis. We suggest that the term "Personality Disorder" be replaced by a more appropriate term "Adaptation Disorder" as the latter reflects more accurately the real nature of the disorder and distributes the causality of maladaptive syndromes more evenly, between the person and the environment.

Diagnostic, research, and treatment advantages of the proposed solution are discussed in some detail.

Key words: *epigenetics – psychiatric classification – diagnosis – personality disorders- adaptation*

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INTRODUCTION

After 30 years of clinical work and research based on categorical criteria for personality disorders (Diagnostic and Statistical manual of Mental Disorders – DSM IV TR (APA 2000)) and (International Classification of Diseases - ICD 10th revision (ICD 1992)), our conceptual understanding and treatments of these disorders have not substantially advanced. Recent survey of 400 experts demonstrates a major disapproval of the categorical concept of personality disorders (Bernstein et al. 2007). Clearly, the field is stagnating, with no clear directions. For the field to move forward, it is imperative that future classifications revise both the concept and the diagnosis of personality disorders.

Future classifications: imperative for change

There is a growing pressure to "dimensionalize" personality disorders, i.e., to describe them as quantitative variants along a number of behavior dimensions or traits (Livesly 2007, Cloninger & Svrakic 2009). Some (Livesly 2007) are advocating that the focus ought to be on extreme ("pathological") behavior traits, which is intended to separate syndromes of personality disorder from normal personality variants. However, it is of critical importance to recognize that personality disorders cannot be accurately defined solely on the basis of extreme behavior dimensions for the following reasons:

- One of the most robust findings in personality research is that four broad dimensions underlie normal temperament and symptoms of personality disorders (Cloninger et al. 1993, Ignjatovic & Svrakic 2003, Trull & Durrett 2005, Livesly 2007, Cloninger & Svrakic 2009). With respect to normal temperament, these are (a) Harm Avoidance, (b) Novelty Seeking, (c) Reward Dependence, and (d) Persistence (Cloninger & Svrakic 2009). With respect to personality disorders, the dimensions have been variously labeled in the literature: (a) Anxiety / Neuroticism (b) Antisocial / Dissocial (c) Social Withdrawal / Asocial, and (d) Conscientiousness / Compulsivity (Trull & Durrett 2005, Livesly 2007). Note that the dimensions identified for personality disorders are in fact extreme variants of normal temperament traits (specifically, the continuous variants are: high Harm Avoidance with Anxiety / Neuroticism, high Novelty Seeking with Antisocial / Dissocial, low Reward Dependence with Social Withdrawal / Asocial, and high Persistence with Conscientiousness / Compulsivity). In conclusion, no traits, extreme or otherwise, have been identified as discriminative of personality disorders. These disorders are not de novo personality manifestations, but continuous (although extreme) variants of

normal temperaments. As discussed later, the defining aspect of personality disorder is poor adaptation to the environment, not extreme dimensions or traits.

- The robustness of the 4-factor structure of temperament throughout history, across normative studies, clinical and nonclinical samples, and measuring instruments, indicates that it reflects natural building units of personality that "carve the nature at its joints": these four dimensions are general determinants of fundamentally different behaviors patterns (see (Cloninger & Svrakic 2009) for review) – stable over time, across situations, and cross culturally (Cloninger et al. 1994, Miettunen et al 2006). Of note, the Five Factor Model (FFM) (Costa & McCrae 2008) defines four factors (i.e., Neuroticism, Extraversion, Conscientiousness, and Openness) which correspond to the four dimensions described above. A closer review of the fifth factor in the FFM (Agreeableness) reveals that it actually corresponds to adaptive behaviors referred to as character traits in this work.

a) No rational (i.e., non-arbitrary) cut off points on the continuum between normal and extreme positions on behavior dimensions have been found. For illustration, behavior traits typical of personality disorders (e.g., compulsivity) are continuous with normal traits (e.g., persistence) with no clear cut-off points to mark the transition from normal persistence to pathological compulsivity. Hence, diagnostic models defined only by extreme traits must use arbitrary cutoff decisions to break a continuous dimension and extract only its extreme aspects. This presents a strong argument against artificially separating the study of normal personality from that of maladapted personality.

b) Personality disorder phenotypes defined solely by extreme traits are diagnostically imprecise as normal individuals may be included as well. For example, a very shy person, who is otherwise well adapted and functional, would qualify for the diagnosis based only on extreme traits, but would not qualify if other aspects of their personality (e.g., quality of adaptation) were considered. Indeed, a number of authors agree that an independent evaluation of impairment is required to diagnose personality disorders because high or low levels of traits are not necessarily indicative of pathology (Cloninger et al. 1993, Ignjatovic & Svrakic 2003, Trull & Durett 2005, Cloninger & Svrakic 2009). The core deficit is frequently described as a "failure to adapt" (Trull & Dutrrett 2005, Livesly 2007, Cloninger & Svrakic 2009) which is created through person environment interaction and cannot be captured by diagnostic models based on extreme temperament traits alone.

To summarize, we agree with the expected dimensionalization of personality disorders but argue i) against using only extreme ("pathological') traits to define the personality disorder phenotype but rather continuous dimensions that account for individual differences in both normal and extreme behavior variants, ii) for a conceptual revision that includes maladaptation to the environment as an integral component in the diagnosis, and iii) for the distinction between biogenetic and adaptive personality processes – which is critical to guide more productive research, more reliable diagnosis, and more efficient treatment of personality disorders (discussed later in the text).

Personality and behavior development: beyond the Nature-Nurture dichotomy

Recent breakthroughs in the field of epigenetics have advanced our understanding of molecular mechanisms underlying gene – environment (GxE) interaction. It has become increasingly clear that genetic influences on the phenotype are not direct and invariable. DNA is no more considered to be the master blueprint for physical and behavioral features that operates in an ecological vacuum. Rather, DNA outlines the overall adaptive potential of an organism through broadly outlined ("uncommitted") physical and behavior dispositions which serve as building material for the final phenotypic outcome in response to specific environmental stimuli (Templeton 2006).

We refer the reader to (Sato et al. 1998, Impey et al. 2004, Pruessner et al. 2004, Fraga et al. 2005, Colvis et al. 2005, Champagne 2008, Champagne & Curley 2008, Svrakic et al. 2009) for details. Here, we provide a review of epigenetic influences on behavior development that are most relevant to this paper. These epigenetic findings are of fundamental importance for future research, conceptualization, and treatment of personality disorders and psychiatric disorders in general.

The field of epigenetics studies changes in gene expression that are mitotically and/or meiotically heritable and do not involve changes in the DNA sequence. As shown by a number of authors (Impey et al. 2004, Fraga et al. 2005), the substrate for GxE interaction is not directly DNA, but rather the activity status of a gene – which is regulated ("marked" or "programmed") to be either active or silent depending on the nature of the modulating stimulus. Such regulation of gene expression is referred to as "epigenetic".

Recall that DNA is wrapped around a complex of histone proteins forming the chromatin globe, where DNA is either accessible ("euchromatin") or inaccessible ("heterochromatin"). In order for DNA to be expressed, chromatin must be in its active state, with nucleic acid sequences exposed to transcription factors. Recently, genome wide analysis of transcription factor binding sites in vivo has been reported (Impey et al. 2004). Transcription factors bind in a coordinated way ("transcriptome") to networks of genomic targets involved in a particular adaptive response ("regulons"). The targets for transcription factors are not always gene promoters, but also other transcription factors and/or non-coding RNA, adding to the complexity and multiplicity of possible GxE interaction outcomes

(Colvis et al. 2005). The long-term pattern of epigenetically marked genes creates the epigenome (or, as it were, a "programmed" genome) which ultimately determines physical and behavioral phenotypic outcomes.

Chromatin configuration is considered to be the link between external environment and cellular DNA. Extracelluar signals can reach the intracellular nucleus (one example is the MAPK/ERK pathway) and change chromatin configuration, mostly through histone tail (de) acetylation and (de) methylation. This in turn recruits DNA modulating enzymes and proteins (e.g., DNA methyl transferase – DNMT and methyl CpG binding protein 2 - MeCP2) and regulates gene expression. In other words, environmental effects can influence the transition between active and inactive chromatin and thus control gene expression (Colvis et al. 2005).

Of note, epigenetic processes are involved in normal development (e.g., cell differentiation, silencing of the X chromosome. etc), pathological processes (e.g., cancer, schizophrenia, etc) and adaptive response to the environment (e.g., resistance to stress, personality traits, etc). Here we focus on adaptive environmental effects only.

Epigenetic regulation of gene expression is stable, but reversible

Early epigenetic DNA markings tend stabilize into adulthood (Champagne 2008), but are reversible, even in adulthood, through sustained environmental effects, which can be either chemical (Fraga et al. 2005) or social / situational (Nithianantharajah & Hannan 2006, Champagne 2008) or both. In rodents, for example, natural variations in maternal behavior are associated with high anxiety in novel situations, increased stress reactivity, and increased corticosterone response to stress in the offspring (Colvis et al. 2005, Champagne 2008). At the molecular level, this involves altered histone acetylation, increased DNA methylation, and reduced Nerve Growth Factor binding (NGFI-A is a transcription factor), which results in decreased expression of the glucocorticoid receptor ($GR1_7$) in the hippocampus (Sato et al. 1998). These behavioral and molecular effects are reversed by early postnatal cross-fostering and/or by pharmacological manipulations in adulthood (Colvis et al. 2005, Champagne 2008).

Environment – Environment interaction: guidelines for psychotherapy

Epigenetic regulation of DNA expression is not limited to early infancy or parenting. Postweaning environments have also been shown to modulate phenotypic features via epigenetic mechanisms (Champagne 2008). As noted above, variations in maternal behavior influence vulnerability to stress in the offspring (Colvis et al. 2005, Champagne 2008). If these offspring are placed in socially isolated or enriched postweaning housing conditions, the group differences disappear (Champagne & Meaney 2007). Therefore, social environment in adulthood can alter phenotypic features developed earlier in life through GxE interaction. These results provide evidence for environment x environment (ExE) interaction (Champagne 2008) in which already developed epigenetic features change under the modifying influence of different environmental conditions. Although the precise mechanism is still unclear, there is evidence that these ExE effects are also mediated through DNA methylation (Weaver et al. 2004, 2005, 2006). Evidence for ExE interaction provides important guidelines for psychotherapy theory and practice: psychotherapy should be rehabilitative, or, as it were, a human equivalent of "care" in animal models in order to be able to trigger epigenetic processes and lasting, neurobiological mechanisms for behavior changes.

Epigenetic modulation of personality traits

There is a growing consensus that most, if not all, behavior traits develop epigenetically, through GxE interaction (Tempelton 2006). If observations of children are any indication, behavior development starts out with few broad dispositions (e.g., distress, content) which branch into successively more specific traits in response to specific environmental requirements and tasks (Tempelton 2006). This developmental branching follows a self-organizing pattern to satisfy multiple, complex, and changing internal and external demands (Cloninger & Svrakic 2009). In other words, behavior development reflects not only GxE interactions with the external environment (across different gradients of environmental specificity and complexity) but also internal modifications by growing cognitive capacities and self-awareness. Hence, manifest behavior involves numerous layers of epigenetic modulations, both vertical (i.e., successively more specific variants of a single trait) and horizontal (i.e., alternative adaptive solutions). These specific, socialized personality traits are increasingly less guided by innate biogenetic determinants and more by rational adaptive solutions, not nearly as stable over time or as predictable as their antecedent biogenetic dispositions.

However, even complex social traits (e.g., character traits), expected to be modulated mostly by the environment, show heritability estimates similar to those observed for temperament (Gillespie et al. 2003). As we discuss later in the text, temperament and character traits either develop independently, but from the same underlying biogenetic dispositions, or character crystallizes from temperament, via conceptual transformation of temperament traits. Either way, biogenetic roots of temperament and character are strong and their heritability estimates are expected to be similar.

DNA susceptibility to environmental influences continues throughout lifetime: MZ twins are roughly concordant for the degree of DNA methylation and

histone H3 and H4 acetylation of genes in peripheral blood lymphocyte and other non-neural tissues at 3 years of age, but at the age of 50 years there is a four-fold difference (Fraga et al. 2005). It is unlikely that this magnitude of difference in gene expression between 50-year-old MZ twins compared to 3-year-old MZ twins is entirely accounted for by random stochastic effects and thus may also be related to the degree of discordance in environmental variables between the twins (Champagne & Curley 2008), i.e., to environmental effects that alter the expression of their otherwise identical genetic makeup. Clearly, MZ twins may be genetically identical, but are very different epigenetically (especially with aging) which has major ramifications for research.

Epigenetic modulations and GxE interaction have been implicated in adult psychiatric disorders in humans. For example, poor maternal care coupled with over-protection in childhood (the co called "affectionless control") increase the risk of antisocial traits, addictions, attention deficit, OCD, anxiety disorders, and depression (Sato et al. 1998) in adulthood. In contrast, good maternal care correlates with high self-esteem, decreased trait anxiety, and decreased salivary cortisol in response to stress (Pruessner et al. 2004). Clearly, early care in humans sets the stage for a wide spectrum of disorders or mental health, via epigenetically modulated expression of initial genetic susceptibilities, much more so than previously believed. This has major implications for the etiological understanding and prophylaxis of psychiatric disorders in general, as well as for the promotion of well-being and mental health since early childhood.

Non-genomic (Lamarckian) inheritance of phenotypic features

In addition to traditional genetic transmission, there is growing evidence of nongenomic inheritance of adaptive phenotypic features. Acquired behaviors are incorporated into the epigenome, primarily via DNA methylation, and are subsequently transmitted onto multiple generations trough incomplete removal of epigenetic marks during meiosis – all without changes in the DNA sequence (Fraga et al. 2005, Champagne 2008).

The above findings challenge some of the methodologies traditionally used in personality research. Specifically, the non genomic, Lamarckian transmission of acquired features makes it impossible to determine the exact genetic roots of behavior traits by methods of quantitative genetics. If, as it certainly seems to be the case, an environmentally induced, acquired trait can be transmitted to the next generation by incomplete removal of chromatin remodeling without changes in the DNA sequence - this trait will appear to be genetic in twin or family studies even though it is environmentally caused - because quantitative genetics can not discriminate between non-genomic and genetic inheritance.

Maladaptation: the distinguishing feature of personality disorder

Official classifications (DSM, ICD) do not define personality disorders based on extreme traits, but require that the diagnosis be based on behaviors that "deviate markedly from the expectations of the society". In other words, deviation from the expected normative standards (which is another way of saying poor adaptation) is a decisive diagnostic point. Accordingly, a number of authors argue that an independent evaluation of impairment is needed because high or low standings on behavior traits are not ipso facto pathological (Trull & Durrett 2005, Livesly 2007, Wakefield 2008, Cloninger & Svrakic 2009). For example, Livesley (2007) describes personality disorders as a … "failure to solve adaptive life tasks relating to identity or self, intimacy and attachment, and prosocial behavior". (p. 203). This and similar suggestions recognize the fundamental aspect of the personality disorder diagnosis: the core deficit is maladaptation, i.e., deviant or poor adaptation to the environment, not extreme behavior traits (although extreme traits may have interfered with successful adaptation).

Maladaptation is frequently defined as a failure of integrative functions of personality, i.e., those that carry out adaptive processes (Wakefield 2008). These integrative, adaptive functions (referred to as character in this work) are not developmentally simultaneous with biogenetic dispositions to temperament, but emerge later, through person-environment interaction and conceptual learning, as an adaptive "interface" to optimize the fit between the internal needs and external normative pressures (Cloninger et al. 1993, Svrakic et al. 2009). In other words, these integrative, adaptive functions determine whether an individual with very high Novelty Seeking develops into a well adapted researcher, explorer, or hobbyist, or into a poorly adapted antisocial personality.

Although several experts have recognized the central importance of maladaptation for the concept of personality disorder, only a few proposals to measure it have been reported. The two noteworthy examples are described below.

The psychoanalytic concept of defense mechanisms has been used for this purpose (Mulder et al. 1996, 1999). Specifically, primitive defense mechanisms centered around splitting, but not mature defenses centered around sublimation, correlate with immaturity and symptoms of personality disorders (Mulder et al. 1996, 1999). The feasibility of assessing defense mechanisms for widespread, routine clinical use to diagnose personality disorders is open to debate.

Another alternative is to use operationalized behavior traits reflective of poor adaptation such as character traits of Self-deirectendness and Cooperativenss (Cloninger et al. 1993) as discussed later in detail. Suffice here to say that low scores on Self-Directedness and Cooperativeness account for

fragmented self-concept, reduced ability to work, and reduced ability to get along with people typical of personality disorders. In clinical research, these two character traits correlate consistently with symptoms of personality disorders (Svrakic et al. 1993). To the best of our knowledge, no study has repudiated this finding out of cca. 350 cited replication reports in peer-reviewed journals. Of note, high (low) character scores also correlate with mature (immature) defenses (Mulder et al. 1996) supporting the concurrent validity of both concepts.

Maladapted personality: a self-organizing pathological system

As described elsewhere (Svrakic et al. 1996, Cloninger et al. 1997, Cloninger & Svrakic 2009), personality development reflects a complex, self-organizing, adaptive effort to maximize the fit between internal needs (defined by emotional temperament traits) and external demands (defined by social norms). Even a maladapted personality reflects this self-organizing effort which, in these cases, results in suboptimal personal and social adaptive solutions. Specifically, this adaptation in the "wrong direction" can be caused by extreme, inflexible early behavior dispositions or by pathogenic environmental effects, or both. On one hand, strong genetic dispositions influence one's perception of the environment and one's choice of activities and relationships (the so called "gene-environment correlation") (Saudino et al. 1997). For example, individuals with constitutionally high aggression have a tendency to engage in aggressive, antisocial behaviors and to surround themselves with antisocial, aggressive individuals. On the other hand, pathogenic environmental factors modulate maladaptive behavioral and cognitive styles (e.g., antisocial behaviors are frequently learned in one's family – mostly from the father). As shown in humans (Sato et al. 1998) and in cross-fostering studies of animals (Maestripieri 2005, Champagne & Curley 2008), environmental effects are powerful enough to modify behavior traits in any direction: "good" environments (i.e., affectionate care) can ameliorate "bad" epigenomes (i.e., high disposition to aggression) and "bad" environments (i.e., those provoking fear) can pathologize "good" epigenomes (e.g., can turn confidence into fearfulness). In animal models, these effects have been shown to be independent of parental care and possible in adulthood as well, indicating that the critical period for shaping lasting behavior features can be extended beyond early experiences and parenting (Nithianantharajah 2006, Champagne 2008). With this in mind, adult social environments and rehabilitative psychotherapy may have significant corrective power in psychiatric treatments and correctional facilities trough both GxE and ExE interaction.

Personality disorder does not mature with time (except for some of the most drastic symptoms such as violence or severe narcissism); other symptoms are typically chronic and unchanged over many years. At the peak of their deviant development, maladapted personality eventually reaches the point of relative stability (Svrakic et al. 1996, Cloninger et al. 1997). Spontaneous change towards better adaptation is rare, because efforts to achieve a new developmental peak (a new point of stability) are discouraged by a period of initial instability associated with change. In dynamical systems' parlance, maladapted patients go through a period of temporary decrease in fitness before reaching a better adapted fitness peak in a wider epigenetic landscape. This "U" shaped developmental curve explains the treatment resistance seen with many individuals with personality disorders and more generally resistance to change observed in many normal individuals.

Disorders of personality or disorders of adaptation?

Adaptation to the environment is a process that begins very early in life, as early as in utero. As noted, one's failure to adapt can reflect either extreme, inflexible behavior dispositions or pathogenic environmental effects, or both. So far, however, only the person took the "blame" for maladaption - as implied in the term "Personality Disorder".

With the above in mind, we suggest that the diagnosis "Personality Disorder" be replaced by a more appropriate diagnostic term "Adaptation Disorder". There are several reasons to suggest this change in nomenclature.

First, the term "Adaptation Disorders" reflects more accurately the etiological reality of the problem as it distributes the causality more fairly, between the person and the environment.

Second, the term "Adaptation Disorder" reflects the essential feature of the disorder, i.e., chronic mal-adaptation to the environment. Most other psychiatric disorders are termed after dominant symptoms (e.g., Anxiety Disorders), or impaired function (e.g., Sleep Disorders), or after historically assumed etiology (i.e., Schizophrenia = "split mind"). No other disorder involves terminology that singles out a higher order entity, a general denominator of psychic life (i.e., personality), but not its affected component, i.e., impaired adaptation.

Finally, it is clearly less stigmatizing to convey to the patient the diagnosis of adaptation disorder vs. personality disorder. It is also more positive and motivating to direct treatment towards "adaptation problems" than towards "personality disorder".

An analogous solution to the one suggested here already exists for the group of Adjustment Disorders, which are defined as an acute failure to adapt (Ignjatovic et al. 2003). In contrast, personality disorders (or as we suggest Adaptation Disorders) reflect a chronic failure to adapt (APA 2000). Incidentally, Adjustment Disorders are not called "Acute Personality

Disorders" because the diagnosis here appropriately focuses on the failed function (i.e., failure to adjust) not on the higher order entity of personality, although one's acute response to stress may reflect either underlying personality factors (e.g., one's emotionality) or objective aspects of stress, or both (much like the etiology of chronic maladaptation).

A specifier to differentiate between four clinical subtypes of mal-adapted behavior is based on the dominant emotional / temperament symptoms (Svrakic et al. 1993). As advocated in prior work (Svrakic et al. 2009), Adaptation Disorder can be sub-classified as: a) *anxious type*, b) *impulsive type*, c) *inhibited type*, d) *obsessive / anankastic type* (each subtype reflects relative dominance of one temperament trait in the overall temperament profile of the patient), and e) *mixed type* (in cases when more than one of the four underlying traits is prominent). Analogously, in DSM IV, Adjustment Disorders are divided into subtypes based on dominant clinical symptoms (depressed subtype, anxious subtype, disorder of conduct subtype, etc) (APA 2000).

We suggest that the diagnosis of Adaptation Disorder be graded into *"mild", "moderate", and "severe"*. These quantifiers of severity are needed to reduce the problem of diagnosing only severe prototypical cases which is inherent in categorical decisions. Most clinicians find it easier to choose from a scaled, more flexible, four-point graded choice (no case, mild, moderate, severe) than between two categorical choices ("case" or "no case"). Character scores are applied to quantify the degree of severity of maladaptation (Cloninger et al. 1993, 2009, Svrakic et al. 1993).

Traditional categories of personality disorders are not completely lost…

The above proposal does include traditional categories of personality disorders (e.g., Narcissistic, Borderline, Schizoid, etc). These traditional personality disorder categories convey vivid, but not always accurate clinical information about patients because they are not clearly separated (due to the overlapping DSM criteria). Indeed, most clinicians prefer to use more general DSM Clusters (e.g., Dramatic, Eccentric, Fearful) to describe their patients. Moreover, individual categories of personality disorder are neither discrete taxons nor permanent configurations of dimensions, but rather meta-stable, quasi-discrete combinations of component processes that interact as expressions of a nonlinear dynamical system (Miettunen et al. 2006). Consequently, categorical diagnoses may alternate in time and across situations. For example, Narcissistic personality may at times alternatively present with a dominant Antisocial or Histrionic façade (Svrakic & McCallum 1991) and vice versa, making these categorical diagnoses variable or, as it were, "a moving target". Our model provides for the continuity with the categorical system: traditional categories of personality disorder can be retrieved from dimensional data as unique configurations of temperament dimensions (Cloninger & Svrakic 2009) (see below).

Proposed solution: The Psychobiological Model of Personality ("The Seven Factor Model")

At present, the most frequently used dimensional models of personality and personality disorder are the Five Factor Model – FFM (Costa & McCrae 2006), the Psychobiological Model of Temperament and Character (Cloninger et al. 1994, Cloninger & Svrakic 2009) and Dimensional Assessment of Personality Pathology – DAPP (Livesley 2007). Out of these 3 models, the Psychobiological model is most congruent with the current epigenetic understanding of behavior development and ethiopathogenesis of personality disorder. By no means do we suggest here that this model is final and flawless. Further revisions, even major ones, may be necessary to keep up with scientific advances. At the time of its introduction in 1987 and 1993, it was certainly an avant-garde model appealing to many for its visionary hypotheses. In the meantime, many of these hypotheses have been tested and empirically supported in the US and worldwide (see (Cloninger & Svrakic 2009) and (Cloninger et al. 1994) for review).

Other two available dimensional models, the FFM and the DAPP, conceptualize personality disorders based on extreme behavior dimensions only (DAPP), confound biological and adaptive personality processes (FFM), do not consider maladaptation as an integral diagnostic component of personality disorders (FFM) and do not provide tools / ways to assess maladaptation in clinical work (DAPP and FFM).

In what follows, we briefly describe the Psychobiological Model of Temperament and Character (also known as the Seven Factor Model). For details on see Cloninger and Svrakic (Cloninger & Svrakic 2009).

According to the Psychobiological Model, personality structure consists of two distinct but interacting components of temperament and character, each reflecting different forms of learning and memory and each providing unique developmental and functional significance in human life (Cloninger et al. 1993, 1997, Svrakic et al. 1996). The model describes four temperament dimensions (i.e., the biological "core" of personality) and three character dimensions (i.e., the "adaptive interface" of personality), but allows for their behavioral variability and alternative activation based on reciprocal causality between biogenetic factors, experience, and adaptation. Each dimension is defined as a bipolar continuum from low to high expression, capturing both normalcy and extreme presentations. Temperament and character dimensions and their facet scales are presented in Table 1.

Table 1. TCI Temperament and Character scales, subscales, and descriptors of high and low scores

I TEMPERAMENT	High scorers	Low scorers
HARM AVOIDANCE (HA)		
HA1: worry and pessimism vs. uninhibited optimism	pessimistic	optimistic
HA2: fear of uncertainty vs boldness	fearful	daring
HA3: shyness with strangers vs outgoing	shy	outgoing
HA4: fatigability and asthenia vs energetic	fatigable	energetic
NOVELTY SEEKING (NS)		
NS1: exploratory excitability vs. stoic rigidity	exploratory	reserved
NS2: impulsiveness vs. reflection	impulsive	deliberate
NS3: extravagance vs. reserve	extravagant	thrifty
NS4: disorderliness vs. orderliness	irritable	stoical
REWARD DEPENDENCE (RD)		
RD1: sentimentality vs aloof	sentimental	cold
RD2: sociability vs reclusive	open	isolative
RD3: attachment vs. detachment	warm	detached
RD4: dependence vs. independence	affectionate	independent
PERSISTENCE (PS)		
PS1: eagerness of effort vs procrastination	industrious	lazy
PS2: work hardened vs spoiled	determined	spoiled
PS3: ambitiousness vs underachieving	enthusiastic	underachiever
PS4: perfectionism vs pragmatism	perfectionist	pragmatist
II CHARACTER		
SELF-DIRECTEDNESS (SD)		
SD1: responsibility vs. blaming	responsible	blaming
SD2: purposefulness vs. lack of goal direction	purposeful	goalless
SD3: resourcefulness vs. helplessness	resourceful	passive
SD4: self-acceptance vs. self-striving	confident	self-conscious
SD5: spontaneity vs. lack of congruent second nature	spontaneous	conflicted
COOPERATIVENESS (CO)		
C1: social acceptance vs. social intolerance	tolerant	intolerant
C2: empathy vs. social disinterest	compassionate	revengeful
C3: helpfulness vs. unhelpfulness	empathic	insensitive
C4: compassion vs. revengefulness	helpful	selfish
C5: fairness/principles vs. self-serving opportunism	principled	opportunistic
SELF-TRANSCENDENCE (ST)		
ST1: self-forgetful vs. self-conscious	acquiescent	alienated
ST2: transpersonal identification vs Self-differentiation	insightful	concrete
ST3: spiritual acceptance vs rational materialism	transpersonal	avoiding
ST4: enlightened vs objective (optional subscale)	creative	conventional
ST5: idealistic vs practical (optional subscale)	spiritual	skeptical

Temperament traits and related behavior habits develop early in life, via associative learning and synaptic strengthening which creates stable percepts, affects, and procedural memory. Specifically, temperament traits (Harm Avoidance, Novelty Seeking, Reward Dependence, and Persistence) are conceptualized as behavioral derivatives of primary emotions (i.e., fear, aggression, joy, persistence) and develop through associative learning (e.g., inhibition, activation, continuation, perseveration of behavior) in response to specific environmental stimuli (e.g., danger, novelty, reward, and frustrative non-reward, respectively) (Cloninger & Svrakic 2009). Temperament dimensions have a number of dissociable biogenetic correlates (see (Cloninger & Svrakic 2009) for review). If these correlates are assumed to be genetic in nature, then temperament traits correspond to the concept of "endophenotypes" which are easier to study given their state-independence and temporal and phenomenological stability.

Character traits (Self-Directedness, Cooperativeness, and Self-Transcendence) develop later in life, through person-environment interaction, and involve conceptual and insight learning and higher cognitive processes of symbolic representation, logic, propositional memory, etc. These traits are much more influenced by adaptive epigenetic mechanisms, and thus less stable in time, more culturally molded, and more prone to change with the changing environment. Exact etiological origins of character are difficult to identify with currently available methodologies. The two most likely scenarios are: i) character and temperament develop independently,

from the same underlying biogenetic dispositions, but via different learning styles, i.e., conceptual learning (character) vs. associative learning (temperament); or, ii) character crystallizes from temperament, through cognitive processing and conceptual transformation of antecedent temperament traits during person - environment interaction. Each of these scenarios accounts for the observed high heritability of character traits (Gillespie et al. 2003).

From an adaptive perspective, the developing character traits serve as a conceptual "interface" which optimize adaptation of temperament (i.e., early emotionality) to the environment by reducing discrepancies between one's emotional needs and norm-favoring social pressures (Cloninger & Svrakic 2009). Some of the crucial differences between temperament and character are presented in Table 2.

Table 2. Key differences between temperament (associative or procedural learning) and character (conceptual or semantic learning)

	Temperament	Character
Learning type	Procedural	Propositional
Awareness level	Automatic	Intentional
Memory form	Percepts, concepts	Procedures, propositions
Learning principles	Associative conditioning	Conceptual, Insight
Key Brain System	Limbic system, Striatum	Frontal/Temporal Neocortex, Hippocampus
Manifesting traits	Habits, emotional	Adaptive concepts, socialized
Associated emotions	*Primary emotions* (anger, fear, perseverance, attachment)	*Secondary emotions* (pride, compassion, ethics, altruism)
Role of subject in mental activity	Passive, reproductive	Active, constructive
Form of mental representation	stimulus-response sequences varying additively in strength	interactive networks (conceptual schema) varying qualitatively in configuration

The Psychobiological Model is a quantitative model of normal and maladaptive personality *development* as a self-organizing multidimensional dynamical system created through GxE interaction. Such systems are defined as complex adaptive system in biology (Cloninger et al. 1997). Based on a sophisticated mathematical model (Svrakic et al. 1996, Cloninger et al. 1997) we have shown that character traits develop as a nonlinear function of underlying temperament traits, socio-cultural factors, and random life events.

In summary, character and temperament are *etiologically* related (either share the same biogenetic roots, or character develops from temperament), developmentally related (final character outcomes are limited by antecedent temperament traits) and *functionally* related (through bidirectional interaction, mature internalized concepts regulated by character modify the significance and the salience of sensory percepts and affects regulated by temperament and *vice versa*).

The Temperament and Character Inventory (TCI) (Cloninger et al. 1994) is a family of tests, self-reports and interviews, designed to measure temperament and character traits described above (Table 1). The TCI has been validated across normative, clinical, and non clinical samples, cross culturally, and internationally (Cloninger et al. 1994, Miettunen et al. 2006).

Diagnosing maladaptive syndromes: a two step process

Diagnosis of personality disorder includes biological and adaptive aspects: character is essential to assess maladaptation, temperament to distinguish the dominant symptomatic presentation. This is achieved as a two step process:

Step 1.

Maladaptation is assessed by two essential features of character - low Self-Directedness and low Cooperativeness – which indicate a fragmented (immature) self-concept, problems with identity, reduced ability to work, and reduced ability to get along with people (Svrakic et al. 1993). This is highly congruent with proposals by other leading authors in the field (e.g., Livesely) who conceptualizes the core deficit in personality disorder as a "three-level", multifaceted adaptive failure: at the *individual* level, personality disorder involves poorly developed or fragmented representations of self and others, at the *interpersonal* level, it involves difficulties resolving attachment problems and developing the capacity for sustained intimacy, and at the *group* level, it involves problems with prosocial behavior, altruism, and maintaining the cooperativeness needed for effective social functioning (Livesley 2007). Livesley's concepts of adaptive failure are captured by low scores on the TCI character traits of Self-Directedness and Cooperativeness (for descriptors see Table 1).

In a study of 136 patients (Svrakic et al. 1993) we used logistic regression to predict the likelihood of personality disorder diagnoses, based on the subject's Self- Directedness and Cooperativeness scores. With lower character scores, the predictions became markedly improved, reaching >85% for the lowest scores. This finding suggests that some compound

variable, encompassing both Self-Directedness and Cooperativeness, may be a useful measure of adaptation. One obvious such measure is a weighted sum of the two character scores, with weights assigned according to their observed correlations with personality disorders. Since the two correlations are nearly equal (0.10 and 0.11, respectively, as calculated from logistic regression), the measure of adaptation in this case reduces to a simple sum of the two

scores. With this in mind, and without introducing arbitrary categorical cutoffs, one can then speak of mild, moderate, and severe maladaptation within the existing measures of character, by dividing this compound score into three equal intervals. This makes the concept of adaptation proposed in this work readily available for research and clinical use.

An alternative way is to recognize that the compound measure of adaptation is normally distributed in populations, as confirmed by our (Cloninger et al. 1993, Svrakic et al. 1993) and other studies. One can then use the standard statistical procedure to extract regions of mild, moderate, and severe maladaptation from the character scores. This can also be implemented in research and clinical practice in a straightforward manner.

Whichever method one opts for, it is clear that a simple, practical measure of adaptation, both specific and quantitative, can be construed from readily available personality scores.

Step 2.

After the diagnosis of personality disorder is established based on character, temperament is used for sub-classification and differential diagnosis. As shown in prior work (Svrakic et al. 1993), temperament traits efficiently discriminate DSM Clusters of personality disorders: Harm Avoidance is dominant in Cluster C ("fearful" personalities), Novelty Seeking in Cluster B ("impulsive" personalities), low Reward Dependence in Cluster A ("asocial" personalities), and high Persistence in Obsessive Compulsive personality disorder (no DSM Cluster). Moreover, individual categories of personality disorder are described by composite configurations involving all four temperament dimensions, i.e., by a profile composed of high and/or low scores on Novelty Seeking, Harm Avoidance, Reward Dependence, and Persistence (Svrakic et al. 1993). This dimensional approach preserves most traditional, categorical diagnoses described in DSM and ICD which are still occasionally used by clinicians. Namely, unique combinations of high and/or low scores on temperament dimensions create eight composite configurations which correspond to eight categorical DSM diagnoses of personality disorders. For instance, Histrionic personality is characterized by high Novelty Seeking, low Harm Avoidance, and high Reward Dependence (Svrakic et al. 1993). Antisocial personality has the same profile except that Reward Dependence is low (see Table 3 for more detail).

Table 3. Traditional Categories of Personality Disorders and TCI Dimensions

Categorical Diagnosis	TCI TEMPERAMENT DIMENSIONS			
	Harm Avoidance	Novelty Seeking	Reward Dependence	Persistence
Antisocial	Low	High	Low	High
Histrionic	Low	High	High	Low
Borderline	High	High	Low	Low
Narcissistic	High	High	High	High
Avoidant	High	Low	High	Low
Dependent	Low	Low	High	High
Schizoid	Low	Low	Low	Low
Obsessive/Anxious	High	Low	Low	High

"Composite" 2-step diagnosis: clinical and research advantages

Consistent (or defining) and variable (or discriminating) features of personality disorder incorporated in the 2-step diagnostic algorithm are summarized in Table 4. As already noted, extreme temperament dimensions are associated with long term personal, social, and/or occupational impairments described as personality disorder *only* when accompanied by low character traits. In other words, poorly developed character (i.e., low responsibility, low resourcefulness, selfishness, lack of purpose, hostility, etc) is what makes extreme behavior traits maladaptive.

This two-step, composite definition of the personality disorder phenotype has several advantages over other dimensional and categorical approaches. First, it provides specific guidelines for research on both biogenetic processes (procedural and associative learning and habit forming associated with temperament) and adaptive processes (insight learning, conceptualization of self and environment associated with character) in the etiopathogenesis of maladaptation. Second, it provides treatment guidelines in

clinical work: affective instability, dysphoria, impulsivity, and other symptoms associated with temperament are primarily treated with pharmacotherapy, whereas maladaptive behavior styles and concepts (i.e., character) are more amenable to psychotherapy. In other words, medication is used to tone-down extreme temperament traits which set the stage for a more efficient psychotherapy to facilitate character change. Lastly, the 2-step diagnostic approach increases the specificity of diagnosis by reducing the likelihood of diagnosing as personality disorder individuals with extreme traits only or individuals with anxiety disorders, depression, or bipolar disorder, who are otherwise well adapted and functional.

Table 4. 2-Step Diagnosis Quantifiable (Dimensional) Features of Personality Disorders (Adaptation Disorders)

CONSISTENT FEATURES
low Self-directedness
irresponsible, blaming
no mature goals
resourceless, helpless
poor self-esteem
undisciplined
low Cooperativeness
intolerant of others
lack of empathy
unhelpful
revengeful
unprincipled
VARIABLE FEATURES
high persistence (obsessive-compulsive symptoms only)
low reward dependence (odd cluster only)
high novelty seeking (erratic cluster only
high harm avoidance (anxious cluster only)

Addendum: DSM-5 draft

Since the original first version of this paper was formulated, the DSM-5 draft has become available on line, for comments and suggestions (APA 2010). We are pleased to report that the proposal to revise DSM-5 (APA 2010) largely corresponds to the proposals in this paper. Specifically, that the Work Group has recommended a significant reformulation of the approach to the assessment and diagnosis of personality disorders, most importantly the revised definition of personality disorder, the provision for clinicians to rate dimensions of personality traits, a limited set of personality types, and the ratings of the overall severity of personality dysfunction.

DSM-5 definition of personality disorder. We are especially pleased that the Work Group provides a *new definition* of personality disorder which is focused on the failure to adapt, rather than on extreme traits, consistent with our proposals in earlier work (20, 31) and in this paper. In the DSM-5 draft, personality disorder is defined as "the failure to develop a sense of self-identity and the capacity for interpersonal functioning that are adaptive in the context of the individual's cultural norms and expectations" (37). This adaptive failure is manifested as an impaired sense of self-identity (e.g., poor identity integration, poor integrity of self-concept, and low self-directedness) and/or as failure in interpersonal functioning (e.g., lack of empathy, intimacy, low cooperativeness, and incomplete integration of representations of others). Clearly, this is highly in accord with our proposal that personality disorder be defined based on character traits of Self-Directedness (i.e., one's Self concept) and Cooperativeness (i.e., one's capacity for interpersonal functioning) (Cloninger & Svrakic 2009, Svrakic et al. 1993, 2009).

Specifically, the DSM-5 defines personality disorder as the adaptive failure and deficits in one or both of the following two areas: A. *Impaired sense of self-identity*, and B. *Failure to develop effective interpersonal functioning.*

A. *Impaired sense of self-identity* evidenced by one or more of the following:

i **Identity integration:** poorly integrated sense of self or identity, such as limited sense of personal unity and continuity, shifting self-states, beliefs that the self presented to the world is a façade;

ii **Integrity of self-concept:** impoverished and poorly differentiated sense of self or identity - such as difficulty identifying and describing self attributes, sense of inner emptiness, poorly delineated interpersonal boundaries, definition of the self changes with social context;

iii **Self-directedness:** low self-directedness, such as inability to set and attain satisfying and rewarding personal goals, lacks direction, meaning, and purpose to life;

All of the above aspects are adequately captured by low Self-Directedness in the TCI (see Table 1), including the psychodynamic concepts of identity and self-integration (as noted earlier, low scores on the TCI Self-Directedness correlate highly with primitive defense mechanisms, which typically underlie confused identity and fragmented self concept) (Mulder et al. 1996, 1999).

B. *Failure to develop effective interpersonal functioning* as manifested by one or more of the following:

i **Empathy:** impaired empathic and reflective capacity such as difficulty to understand the mental states of others;

ii **Intimacy:** impaired capacity for close relationships such as inability to establish or maintain closeness and intimacy, to function as an effective attachment figure or to establish and maintain friendships;

iii **Cooperativeness:** failure to develop the capacity for prosocial behavior, e.g., failure to develop the capacity for socially typical moral behavior, lack of altruism;

iv **Complexity and integration of representations of others:** poorly integrated representations of others, such as poorly related images of significant others.

Again, these interpersonal aspects are adequately captured by low Cooperativeness in the TCI (see Table 1), including the psychodynamic concepts of object-represenations (as noted earlier, low scores on Cooperativeness and Self-directedness correlate with primitive defense mechanisms and partial object relations) (Mulder et al. 1996, 1999).

Level of personality functioning. The Work Group provides an independent assessment for severity of the above adaptive impairments. Each of the two impairments is rated on a 5 point scale (0=absent, 4=severe) and specific criteria are provided for these ratings. In our prior work, we have used scores on the TCI character dimensions to assess the severity of maladaptation (Svrakic et al. 1993, Cloninger & Svrakic 2009).

Personality trait domains

In an attempt to dimensionalize personality disorders, the Work Group identifies 6 personality *trait domains* each comprised of several lower order, more specific *trait facets*. The six dimensions are: 1. ***Negative Emotionality*** (e.g., anxiety, depression, guilt/ shame, worry, etc.), 2. ***Introversion*** (e.g., withdrawal from other people, restricted affective experience and expression, limited hedonic capacity, etc), 3. *Antagonism* (antipathy toward others and an exaggerated sense of self-importance including narcissism, histrionism, antisociality, etc), 4. ***Disinhibition*** (behavior is driven by current internal and external stimuli, rather than by past learning and consideration of future consequences including impulsivity, recklessness, irresponsibility, etc), 5. ***Compulsivity*** (tendency to think and act according to a narrowly defined and unchanging ideal, and the expectation that this ideal should be adhered to by everyone – includes perfectionism, perseveration, rigidity, orderliness), and 6. ***Schizotypy*** (odd or unusual behaviors and cognitions, including both process (e.g., perception) and content (e.g., beliefs) such as unusual perceptions, beliefs, cognitive dysregulation, dissociation proneness, etc. For the four of the proposed six personality domains empirical evidence is robust across different personality models (Livesley, Costa and McCrae, Cloninger) and they correspond to the four fundamental dimensions of normal temperaments and personality disorders described earlier: negative emotionality (conceptually similar to high Harm Avoidance and Neuroticism), disinhibition (conceptually corresponds to high Novelty Seeking and Antisociality), introversion (conceptually similar to low Reward Dependence and Social Withdrawal), and compulsivity (conceptually similar to high Persistence and Obsessionality). Therefore, the decision to use six (and not four) traits is somewhat puzzling. Nevertheless, the selected traits may well be useful for describing prominent features in people who do not qualify for a diagnosis of personality disorder. For example, it is often useful to describe someone as introverted, compulsive, or disinhibited, etc, even if they do not qualify for a personality disorder diagnosis. These terms are all familiar to clinicians, which will make their application easy. Unfortunately, there is no consensus in the field about the number or the content of traits describing personality. As a result, the proposed six domains are tentative, labeled by the DSM-5 Work Group as "pending empirical validation" which means that the number could be revised in the final version. The most questionable of the proposed traits is the domain of schizotypy. In accord with ICD 10, we advocate that Shizotypy with its cognitive, perceptual, and behavioral impairments etiologically belongs to Axis I, where it could be studied more productively, together with other schizophrenia spectrum disorders.

Some of the popular models of personality (e.g., Livesley, Cloninger) can be used without major revisions to study personality disorders as defined by DSM-5. This is even more the case should the six proposed domains / traits be reduced to four in the final version of the manual.

Personality disorder types

In order to keep continuity with the traditional categories of personality disorder, the Work Group recommends five personality disorder types (Antisocial /Psychopathic, Avoidant, Borderline, Obsessive-Compulsive, and Schizotypal) and provides symptomatic description for each type. As a novel feature, each type is rated for its typicality on a dimension of graded membership ranging from 5=TYPICAL to 1=NO MATCH. In addition, these personality types are also defined as *trait composites* using the above described 6 personality domains / traits. A list specifying component domains / traits for each type (e.g., component traits of Borderline Type are negative emotionality, schizotypy, disinhibition, and antagonism). Finally, the extent to which the domains / traits are descriptive of the particular type of personality disorder is rated on a 4 point scale (from not descriptive to extremely descriptive). Of note, these five personality types are also tentative, pending empirical validation.

CONCLUSION

The reader may get confused with all the different names, contents, and descriptions of numerous personality traits in the literature. As noted, there is no

consensus in the field about the number or the content of traits describing personality. Statistically, no factor analytical solution for personality traits is better than others. However, the central problems in personality research is the question of natural units ("natural joints") to define individual differences in personality, and this question is not answered by linear factor analysis. Linear statistical methods are inadequate to study complex non linear dynamical systems such as personality. Such methods take only a snap shot of underlying interacting traits, can not discriminate between distinct and sequential developmental strata in personality structure (e.g., emotional vs. adaptive), can not detect their dynamic interaction or their coordinated activation by specific environmental stimuli. Rather, linear approach flattens out all behaviors into an artificial plane and lumps all correlated behaviors (based on their average proximity to the axis) into higher order "factors"– regardless of their motivational priority or underlying learning processes, specific eliciting stimuli, and unique motivational power. If factor analysis is used, it should be used separately for biogenetic and adaptive personality processes to avoid misleading results. TCI measures were developed rationally, based on a number of complementary perspectives – such as ethology, evolution, genetics, neuroscience, behavior science, and biology to better measure individual differences in learning and personality development and structure. Statistical analyses, including factor analyses, were used to fine tune the psychometric properties of the TCI, not to formulate the model or to determine its contents. Psychometrically, the TCI provides a coherent body of data to account for alternative approaches to personality assessment, including the proposal for DSM-5 and has predictive validity as good as or better than other available tests (Grucza & Goldberg 2007). The TCI measures are compared to those of Eysenck and Zuckerman (Zuckerman & Cloninger 1996) in Table 5 and to those of Costa and McCrae in Table 6 (Cloninger 2006).What is important for a clinician to know is that the same name does not always mean the same thing: different tests with the same name measure different things, as seen by examining the TCI correlates of the Neuroticism measures of Eysenck, Zuckerman, and Costa and McCrae. In addition, the constructs in the TCI provide a way to evaluate the personality disorder as proposed for DSM-5 (illustrated in Table 7). The TCI character traits directly measure the features pivotal for the definition and the diagnosis common to all personality disorders (Self-directedness and Cooperativeness). The temperament measures allow distinguishing the types and traits proposed for DSM-5 as well.

Table 5. Correlations (r x 100) between the Temperament and Character Inventory (TCI) scales and those of the Eysenck Personality Questionnaire (EPQ-revised) and the Zuckerman-Kuhlman Personality Questionnaire ZKPQ (correlations over .4 in bold, significant correlations only shown, n=207, adapted from Zuckerman & Cloninger, 1996)

	HA	NS	RD	P	SD	CO	ST
EPQ Neuroticism	**59**				**- 45**		
EPQ Extraversion	**-53**	**44**	23		18		
EPQ Psychoticism		**41**	**-45**	-29	-31	**-42**	
EPQ Lie		-21			25	34	
ZKPQ Neuroticism	**66**				**-49**		
ZKPQ Impulsive Sensation	-39	**68**	-20				28
ZKPQ Hostility			-27		-32	**-60**	
ZKPQ Sociability	-38	37	31				
ZKPQ Activity	-29			**46**	36		

Legend: HA- Harm Avoidance; NS – Novelty Seeking; P – Persistence; RD – Reward Dependence
SD – Self-directedness; CO – Cooperativeness; ST – Self-Transcendence

Table 6. Correlations between the scales of Temperament and Character Inventory-Revised (TCI-R) and the NEO-PI-Revised (correlations over .4 in bold, significant correlations only shown, multiple correlation also shown, n=662, adults in USA)

	HA	NS	RD	P	SD	CO	ST	mR
NEO Neuroticism	**63**			-20	**-62**	-28		75
NEO Extraversion	**-55**	40	**52**	40	25		22	77
NEO Openness	-25	**43**	25				37	54
NEO Conscience	-26	-34		**51**	**41**			70
NEO Agreeability		-23	40		31	**61**	20	66
mR	76	65	68	60	67	65	45	

Legend: HA- Harm Avoidance; NS – Novelty Seeking; P – Persistence; RD – Reward Dependence
SD – Self-directedness; CO – Cooperativeness; ST – Self-Transcendence/ NEO – Neuroticism, Extraversion, Openness

Table 7. Relationship of TCI dimensions to DSM-V proposal of Personality Trait Domains

DSM-V Proposal	SD	CO	ST	HA	NS	RD	PS
General Features							
Self-identity impaired	Low						
Interpersonal function poor		Low					
Types							
Antisocial	Low	Low		Low	High	Low	
Avoidant	Low	Low		High	Low	High	
Borderline	Low	Low		High	High	Low	
Obsessive	Low	Low		High	Low	Low	High
Schizotypal	Low	Low	High	High	Low	Low	
Traits							
Negative Emotionality	Low	Low		High			Low
Introversion	Low			High	Low	Low	
Antagonism	Low	Low	Low		High	Low	
Disinhibition	Low			Low	High		
Compulsivity				High	Low		High
Schizotypy	Low		High				

Legend: HA- Harm Avoidance; NS – Novelty Seeking; P – Persistence; RD – Reward Dependence
SD – Self-directedness; CO – Cooperativeness; ST – Self-Transcendence

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