Europe's Journal of Psychology
ejop.psychopen.eu | 1841-0413

Theoretical Contributions

A Review and Critique of Obsessive-Compulsive Personality Disorder Etiologies

Reckoning With Heritability Estimates

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Abstract

The present review and critique of extant etiological theories centers on a single finding: Obsessive-compulsive personality is highly heritable (0.78) and not significantly influenced by "common, shared-in-families environmental factors" (Torgersen et al., 2000, p. 424). This finding, though twelve years old, has remained dissociated from existing etiological accounts. Psychoanalytic theories anachronistically maintain that obsessive personality is familially forged. Biological theories, few, unelaborated and weakened by postulating proximate instead of ultimate explanations, fail to seriously reckon with Torgersen's findings. Truly integrating heritability estimates into a functional etiological account of obsessive character, it is argued in the discussion section, will come from an evolutionary model that understands obsessive personality to be an evolved strategy rather than a dysfunctional disorder.

Keywords: obsessive compulsive personality, etiology, heritability, psychogenic

Europe's Journal of Psychology, 2014, Vol. 10(1), 168–184, doi:10.5964/ejop.v10i1.679

Received: 2013-08-27. Accepted: 2013-12-02. Published (VoR): 2014-02-28.

Handling Editor: Vlad Glăveanu, Aalborg University, Aalborg, Denmark

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Introduction

Both the fourth and fifth editions of the Diagnostic and Statistical Manual of Mental Disorders describe ten personality disorders. These ten are divided into three groups. The third group, collectively referred to as the *Anxious and Fearful Cluster,* contains Obsessive Compulsive Personality Disorder (OCPD). Broadly characterized by the American Psychiatric Association (2000, p. 296; 2013) as a pervasive pattern of "preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency," OCPD is comprised of the following symptoms: (1) Preoccupation with details, rules, lists, order, organization, or schedules; (2) shows perfectionism that interferes with task completion; (3) is excessively devoted to work and productivity; (4) is over-conscientious, scrupulous, and inflexible about matters of morality, ethics, or values; (5) is unable to discard worn-out or worthless objects; (6) is reluctant to delegate tasks; (7) adopts a miserly spending style towards both self and others; (8) shows rigidity and stubbornness. This concise list consolidates and operationalizes OCPD, which, more than its Cluster C compeers, has a distinguished hundred year heritage tracing its origins to Freud's 1908 paper entitled *Character and Anal Eroticism* (Gay, 1989). In his seminal paper, Freud explicated the anal

triad, a pattern of orderliness, stinginess and obstinacy that remains compatible with DSM's definition and description. In the approximately one hundred years separating its initial exposition to its most modern formulation, OCPD has not changed in substance.

Although obsessive character[i] is well described across time and theorists, it has yet to be satisfactorily explained. That is, no extant etiology effectively accounts for the existence of obsessive character. Establishing this fact is the aim of the present paper. In attempting to accomplish this aim, OCPD etiologies will be divided into two broad categories: Psychoanalytic etiologies and biological etiologies. Psychoanalytic etiologies of OCPD have a much more distinguished pedigree, enjoy greater diversity, and are more conscientiously articulated. In contrast, biological etiologies of OCPD only appeared in the late 1990s and remain unelaborated adjuncts to larger works. Despite differences in orientation and derivation, both psychoanalytic and biological accounts have an intractable and irresolvable flaw. Psychoanalytic and biological theories alike fail to acknowledge the import of the heritability (Pollak, 1987; Reichborn-Kjennerud et al., 2007; Torgersen et al., 2000) of obsessive character. Furthermore, these theories could not genuinely absorb the implications of obsessive character's heritability without repudiating their foundational tenets.

Psychoanalytic models are irrevocably committed to, and fully predicated upon, psychogenic character formation. In other words, psychoanalytic models find obsessive origins in nurture to the relative exclusion of nature. Long accepted and faithfully perpetuated though these models are, they have become untenable in the face of modern heritability estimates, which find obsessive origins, not in familial and social dynamics, but in mainly in heritable elements. Few, unelaborated, and insufficiently differentiated from their psychoanalytic progenitors, existing biological theories, are works of localization, which provide proximate, rather than ultimate explanations of obsessive origins. Nevertheless, providing proximate rather than ultimate explanations only make biological etiologies incomplete, not wrong. However, as with psychoanalytic theories, existing biological theories are controverted by their inability to absorb heritability estimates without repudiating their common central tenet; namely that obsessive character is a disorder. Though biological etiologies assume that obsessive character is a disorder, none explain why this disorder exists and why it is intergenerationally perpetuated.

Again, the principle aim of the present article is to expose the weaknesses, peripheral and central, of current OCPD etiologies. First, psychoanalytic models will be reviewed and critiqued, and then biological models will be reviewed and critiqued. The discussion section will comment on these weaknesses and examine their implications. Briefly stated here, these implications are 1) that we need to take seriously and assimilate the behavioral genetics estimates that demonstrate the high heritability of obsessive character; 2) we need then abandon psychoanalytic models outright, not as possible adjunctive modifiers of obsessive character, but as primary determinants of it; 3) we need to ask more of biological models, demanding not just localization, but true explanation; 4) we need to either reject or revise those biological etiologies that simultaneously find obsessive character to be both heritable and harmful; 5) we need to consider that the only way to craft a truly convincing model of obsessive origins might be to altogether stop thinking of obsessive character as disorder; 6) instead, we would do better to search for the adaptive logic behind obsessive behavior and posit an evolutionary model on these grounds (Hertler, 2013). The process of this critique, and the conclusions that it comes to, are intended to inspire critical thought about the nature of personality and individual differences. In the rapidly changing field of personality, new information needs, not only to be generated, but assimilated.

A History of Psychoanalytic Etiological Models

In his classic track on obsessive character, *Character and Anal Eroticism,* Freud assumed that the obsessive is born with constitutional anal sensitivities (Gay, 1989). Nevertheless, it was not "anal erotogenicity" (Gay, 1989, p. 294) itself, but parental reactions to it, that truly created obsessive character. Later psychoanalysts would further emphasize parental dynamics to the consummate exclusion of constitutional anal erotogenicity. Thereafter, decades of observation correlated perfectionism and rigidity in childhood with later development of obsessive character (Millon & Davis, 1996). Obsessive character is hypothesized to be environmentally instilled through the mechanisms of parental over-control and strict disciplinary practices. Expressed during the anal phase of psychosexual development and acutely felt throughout toilet training, parental over-control creates lasting characterological defects. The child's natural impulses to elimination are, in a series of overwrought conflicts, supplanted by the parental will to regulate and defer defecation (McCann, 2009). Often, unsuccessful training is initiated before sufficient physiological control can be expected, or is carried out in an exceedingly severe manner, such that it strips the child of control. Control is forever after searched for, but never found.

Psychoanalytic theory, as it passed from Freud to his progenitors, became less sexual and more social. Following this general trend, later etiological explanations of obsessive character would focus on psychosocial, rather than psychosexual factors. Where traditional psychoanalytic explanation focused on the parental wish to precisely regulate the expulsion of feces, modern psychosocial explanation pivots more broadly on control. Representative of such amendments to psychoanalytic canon, Karen Horney thought that the anal character was raised by authoritarian parents that veiled their domineering traits under a façade of "fairness and infallibility" (Pollak, 1987, p. 253). The child of such a parent becomes anxious for security and so conforms obsequiously to every demand of the parent. Parents demand obedience, deference and order without establishing the requisite love, trust and attachment that serve as the foundation of filial allegiance (Eskedal & Demitri, 2006). There is, however, significant anger that is tightly secured within the over-controlled child; anger for his acceptance being made conditional; anger at having to conform; anger at not being able to express his natural emotional reactions. According to Horney, it is the parent that arouses the obsessive child's very perception of the world as unstable and threatening (Pollak, 1987). Likewise, Erikson and ego psychologists follow Freud's mark, but understand toileting to be one battle in a larger war of wills. Obsessive character emerges from tyrannical parental control, which, over time, divests children of any vestige of self-determination (Pollak, 1987). Psychosexual dynamic explanations and psychosocial dynamic explanations, alike, attribute obsessive character formation to controlling, punitive, tyrannical parents that bestow approval only on children that strictly conform to parental ideals. In the refined psychosocial models, however, obsessive character is instilled over a greater span of time and through a greater range of interaction that subsumes the Freudian anal stage within the Eriksonian stage of autonomy versus shame and doubt.

Modern theorists continue to use elements of psychoanalytic and psychosocial explanation as they create ever refined etiological theories. Mallinger (1984), for instance, asserts that the obsessive is "frightened in early childhood by feelings of helplessness and vulnerability stemming at least in part from anxiety-arousing parental interpersonal attitudes including rejection, domination, and intrusiveness" (Pollak, 1987, p. 253). According to Mallinger, early in development, the child constructs a "myth of absolute personal control" in reaction to the unconscious sense of impotence against the perception of the environment as "untrustworthy, hostile and unpredictable" (Pollak, 1987, p. 253). Accordingly, the obsessive's life orientation is thought to be a relentless toil towards minimizing threatening disorder through ever rigorous control of the internal and external environment. Thus early parent-child interaction stamps the child with an obsessive personality. Mallinger, following the intuition of Harry Stack

Sullivan, believes that character is shaped as the child interacts with the environment in the same manner that smooth river stones are shaped by tumbling over one another. This is the essence of psychogenic character formation.

Most recently, Mallinger (2009, p. 106) continues to describe the causal role of "intra-psychic forces and counter-forces." However, more and more frequently psychoanalytic theorists are confronted with behavior genetics research, as Mallinger (2009, p. 107) reports, "several twin studies conclude that specific personality traits and disorders are influenced strongly by heredity." Assuming the role of an apologist, Mallinger quickly provides the following caveat: "This finding, however, invalidates neither the importance of psychological contributors nor the value of psychotherapy. Indeed, the same studies also provide evidence that genetics alone cannot entirely explain 'choice' of personality type" (Mallinger, 2009, p. 107). Mallinger's caveat is duly regarded, but so should the behavioral genetics research that inspired it. Mallinger does now acknowledge genetic variation in basic temperamental features, but maintains that "early experiences and perceptions are hearth, hammer, and anvil—the shapers of these raw possibilities and limitations into coordinated adaptive trait clusters, or styles" (Mallinger, 2009, p. 107). As will be seen in the following section, notwithstanding such a concession, present evidence suggests that nurture must share the field of influence more equitably with nature.

Evaluating Psychoanalytic Etiologies

Inchoate, developmental, dynamic and unconscious, the causal mechanisms posited by psychoanalytic etiologies of obsessive personality have proved difficult to operationalize, measure and study (McCann, 2009). Still, with tenacity and persistence, studies were conducted. After reviewing a series of published studies, Pollak judged predominantly psychoanalytic etiological models to no longer be viable. There is nearly no empirical support, Pollak (1979) states, to the notion that rigid toilet training practices cause obsessive pathology in children. Pollak (1979) cites fourteen independent researchers or groups of researchers who have experimentally investigated the proposed causal relationship. Taken individually or in aggregate, the results of these investigations provide "at best, meager support for the hypothesized relationship between toilet training practices and the development of anal or obsessive-compulsive character structure" [ii] (Pollak, 1987, p. 227). Pollak cites Beloff (1957), who states that, although the character type is well described and the constellation of traits co-occurs, no link between this constellation of traits can be related to how rigidly or permissively toilet training was undertaken by the parent (Pollak, 1979). Pollak (1979) judged the etiological literature, derived through psychoanalytic treatment and theory, to be unsupported and far from compelling. Of course, psychosocial theorists construe toilet training as simply representative of the more general struggle for autonomy and control that characterizes early childhood; so what of them?

Again, for the psychosocial theorist, the struggle over autonomy replaces the struggle over fecal regulation, the Eriksonian phase of autonomy versus shame and doubt subsumes the Freudian phase of anality, and the process of character formation takes place over many years, rather than over many months. Still, even when the scope is thus broadened, supporting data has not been forthcoming (Pollak, 1987). There is only scanty empirical data, according to Pollak, to support the widespread theories that obsessive character pathology is formed in the crucible of childhood. Bettelheims's *refrigerator mother* and Fromm-Riechman's *schizophrenigenic mother* recollect the pernicious trend of unjustly saddling parents with the causal burden of their children's mental illness. It seems that personality psychopathology has sprung the same trap, lured in by the bait of the blank slate (Locke, 1689/1996;

Pinker, 2002). More than two decades ago, Pollak did his part to deliver psychology from this trap, calling for biological explanations of obsessive character pathology. Still, rigidly psychogenic models persisted.

More than the failure to demonstrate the validity of psychoanalytic etiologies, more than the weight of Pollak's judgment, more than the fourteen studies that have failed to support the hypothesized link between elimination and personality formation, psychoanalytic theories, with their psychogenic predilections, suffer from their incompatibility with the established heritability estimates of behavioral genetics research.

Using controlled twin studies, behavioral genetic methods carefully tease apart genetic and environmental influences so that it is now possible for researchers, such as Penke, Denissen, and Miller (2007, p. 550), to state that "one of personality psychology's most important findings in the last three decades has been that virtually every aspect of personality is heritable. This fact is now so well established that Turkheimer (2000; Turkheimer & Gottesman, 1991) even called it a law." Even in the unconditioned infant, the rudiments of traits can be observed (MacDonald, 1995). Further still, allelic variation can now be associated with "variation in novelty seeking and exploration behavior in a range of species, from humans to great tits" (Tschirren & Bensch, 2010, p. 624)." Researchers such as McAdams and Pals (2006, p. 207) who fully appreciate developmental dynamics, identity, narrative construction and lifespan development, nonetheless assert that "at least half of the variability in trait scores appears to be a result of genetic differences between people."

More than this, there are not only general data on the heritability of personality, but data on the *anxious and fearfulness* disposition. Taken together, it seems that cluster C personality disorders are moderately heritable with little evidence of shared environmental effects (Reichborn-Kjennerud et al., 2007)[iii]. However, when isolating obsessive character, when studying it in its own right, heritability estimates become much more pronounced. Indeed, genetic investigations of obsessive compulsive personality disorder, as estimated by Torgersen et al. (2000), yielded a heritability estimate of 0.78 (Torgersen et al., 2000; Reichborn-Kjennerud et al., 2007). In discussing this finding, Torgersen et al. (2000, p. 424) described OCPD as "particularly strongly influenced by genes." This confirms earlier reviews by Pollak, which cite the co-occurrence of compulsivity in monozygotic twins. Further still, this is consistent with the judgment of Reichborn-Kjennerud and colleagues (2007, p. 649), a group that found that obsessive character is familially grouped and that these familial groupings are "best explained by genetic factors alone." Such strong genetic influence exceeds that of nearly all Axis-I disorders, rivaling even the highly heritable bipolar disorder (Torgersen et al., 2000). Such findings place the etiological burden squarely upon a genetic footing.

After so demonstrating the heritability of obsessive character, the principle predicate of psychoanalytic etiologies becomes patently implausible. Again, there is still room for such social and familial dynamics to influence identity, self and the life course; further there is still room for such social and familial dynamics to moderate personality in terms of its expression and degree, but there is no room for social and familial dynamics to either create the individual traits that comprise obsessive character or arrange them into their coherent whole. Continuing to make such claims becomes anachronistic in light of the above proffered information.

Biological Alternatives

Little has changed since Pollak's exhaustive etiological review written in 1987; alternative biological perspectives remain essentially unexplored (McCann, 2009). Some have loosely applied Cloninger's tripartite temperamental dimensions (novelty seeking, reward dependence and harm avoidance) to explain obsessive behavior as follows:

Low novelty seeking elicits routine living; high harm avoidance elicits caution. Others (Joyce et al., 2003), connected the perfectionistic traits of the obsessive to the DRD3 dopamine receptor, but subsequent studies (Light et al., 2006) failed to replicate this association. Such speculation does not amount to an etiological theory. The present review revealed only three accounts that were sufficiently coherent and elaborated to amount to an etiological theory; and truly, even of these, none are prominent authoritative statements of obsessive origins; rather, they are speculations embedded within broader works that treat obsessive character generally, measure it empirically, or evaluate it diagnostically.

First, Theodore Millon's writings on the genesis of obsessive personality are, in almost every manner, psychodynamic in character. Nevertheless, working with Davis in (1996, p. 530) he devoted two paragraphs to "hypothetical biogenic factors." The entirety of their first paragraph is devoted to caution and disclaimer, which begins as follows: "there is little evidence to suggest that biogenic influences contribute in any distinct manner to the development of the compulsive personality." Nevertheless, Millon and Davis, in their second paragraph, go on to speculate upon a connection between obsessive character and limbic organization:

> we might hypothesize that the neurological regions of the limbic system associated with the expression of fear and anger may be unusually dense or well branched among these patients; these conflicting dispositions might underlie the hesitancy, doubting, and indecisive behaviors seen in these patients (Millon & Davis, 1996, p. 530).

Pioneering and imaginative, these were the first cautious forays away from psychoanalytic accounts. However, Millon and Davis quickly ended these speculative advances and, in the very next section, retreated back to psychoanalytic orthodoxy, asserting that "the foundations of the compulsive pattern are rooted primarily in interpersonal experience." More problematically than its being abjured and unelaborated, Millon and Davis' biological theory, even if borne out by research, would only find the physiological correlates of obsessive decision making. Of course, this would be a spectacular advance in and of itself; but it would still leave us etiologically confounded. One would still then ask; why is the obsessive brain structured in this way? In other words, limbic differences are a proximate explanation for which an ultimate explanation would still be wanting. Millon and Davis, however, in later amendments to their original theory, did go on to try to connect their limbic system speculations, which by themselves amount only to a theory of localization, to more fundamental explanations. In this attempt, Millon and Davis invoke evolution, but seem to succeed only in reiterating psychodynamic explanations in modern garb. Millon and Davis speak of *aims of existence, strategies of replication and instrumental modes of adapting to one's environment*, but they do so, at least as far as the obsessive personality is concerned, in a way that would not be recognizable to evolutionary biologists. McCann (1999, p. 592) summarizes:

> The obsessive compulsive personality is viewed as a conflicted style experiencing intense ambivalence over needs for individuation and nurturance. Thus, there is guilt over seeking individual, self-centered needs but resistance to accepting direction from others. Therefore, the individual that is obsessive-compulsive passively adopts strict adherence to external rules and structure to cope with the ambivalence created by attempts to individuate and seek nurturance from others.

As both the tone and content of the above-referenced passage indicate, this theory is not sufficiently differentiated from its early psychodynamic heritage. Evolutionary explanation is not wrought. Explanations remain proximate.

Second, Aycicegi-Dinn, Dinn, and Caldwell-Harris (2009, p. 601) proposed that characteristic obsessive compulsive personality traits arise in response to cognitive deficits. This research group submitted the following hypothesis:

"Individuals demonstrating obsessive compulsive personality traits … would demonstrate cognitive disorganization during neurocognitive task performance and would display working memory deficits." However, the ambitious range of interests incorporated into Aycicegi-Dinn et al.'s research, along with the methodology employed, make it difficult to extract conclusive or convincing inferences about the etiology of obsessive character. For instance, Aycicegi-Dinn et al.'s participants were classified as having obsessive traits simply by placing higher than one standard deviation above the mean on a self-report measure. Still, findings were forthcoming and worth considering.

To test for the hypothesized deficits in organization, Aycicegi-Dinn and colleagues (2009) used Rey-Osterrieth Complex Figure Test (ROCFT), which is primarily a measure of visuospatial memory. Yet the ROCFT, in its recall phase, purportedly measures *organizational strategy;* and it was obsessive organizational strategy on this recall phase that proved aberrant. Obsessive personality traits were associated with "significantly lower copy organization scores" and slower performance during the recall phase (Aycicegi-Dinn et al., 2009, p. 601). After examining their results, Aycicegi-Dinn et al. (2009, p. 607) were able to make the following statement: "findings lend support to the contention that OCPTs [obsessive compulsive personality traits] may represent, at least in part, compensatory tactics that evolve in response to executive control deficits." To be clear, Aycicegi-Dinn et al. (2009) did not seek to explain the origins of obsessive character and their quoted passage to that effect represents closing conjecture as to the results of their cognitive study. Nevertheless, in closing with this interpretation of their results, Aycicegi-Dinn et al. are postulating an etiology that should be considered and weighed.

Third, after skillfully reviewing the diagnosis and conceptualization of the obsessive construct, Hummelen, Wilberg, Pedersen, and Karterud (2008) devote a paragraph of their discussion section to etiological speculation. Although it is herein described as biological, as the authors state, their position is a generous modification of Freudian theory; one that creatively couples past and present, describing an interaction between nature and nurture to explain obsessive character development. Hummelen et al. most markedly diverge from Freud in their use of Baron-Cohen's (2006) theory of systemizing and empathizing, originally developed to describe autistic behavior. To understand Hummelen's etiological theory, it is necessary to understand the rudiments of Baron-Cohen's dichotomy. On one hand, there is the *systemizing mechanism*, an evolutionary solution that enables comprehension for *lawful* and *nonintentional* events. On the other hand, there is an *empathizing mechanism*, which enables comprehension of intentional motivated behavior characteristic of humans. In typically developing persons, the empathizing mechanism and systemizing mechanism are both adequately developed and used appropriately according to the demands of particular situations. Thus, the nature and dangers of gravity are discovered by the systemizing mechanism, while the wiles and cunning of man are discovered by the empathizing mechanism. Using Baron-Cohen's scheme, Hummelen et al. assert that in the obsessive, the empathizing mechanism is blunted and the systemizing mechanism is sharpened. Consequently, obsessives incongruously apply their systemizing mechanism across situations, attempting to subject the vagaries of social life to the order of physical life. This inborn imbalance represents the temperamental seeds of obsessive character, which must be cultivated by parental mismanagement, as described by Hummelen et al. (2008, p. 453) in the following passage:

> We suggest that OCPD develops out of an intersubjective matrix where children with a moderate to high inborn tendency of systemizing mechanism and thus displaying more rigidity, stubbornness, and perfectionism than average, are met by rigid and inflexible countermeasures by parents who may share the same genetic disposition. With regard to temperament, if there is a predisposition toward rage reactivity, this intersubjective matrix may handle the rage less than optimally and fail in containing and transforming the rage into healthy assertiveness as well as failing to modify the archaic grandiose self.

In this way, Hummelen et al. substitute the biology of anal eroticism, as described by Freud, for the biology of systemizing, as described by Baron-Cohen. Significantly, they also endow biological contributions with much more power than Freud allowed for. Freudian theory describes biology essentially as a factor that skewed the nature of parental care, with the parental care itself mostly responsible for obsessive character formation. Hummelen et al. assign a more independent causal role to biology, creating a system that resembles more modern conceptualizations of disorder developing via diathesis and stress or via nurture and nature.

Evaluating Biological Etiologies

To recount, Millon and Davis (1996) first suggested that obsessive character arises from differences in limbic organization; Millon and Davis then suggested that there may be evolutionary underpinnings for such limbic differences. When considering Millon et al.'s basic limbic speculations, we are left with a work of localization, not of explanation; when considering Millon et al.'s integrated theory, we are left with a composite of biology and psychoanalysis that invokes evolution more effectively than it employs evolution. It is a composite of new and old that is certainly an advance, that is certainly more modern; but an explanation which does not explain how the obsessive character, replete with the deficits attributed to it, could have evolved.

To recount, Aycicegi-Dinn, Dinn, and Caldwell-Harris (2009) empirically demonstrated deficits in executive control, which they then interpreted as causally organizing all other obsessive traits through the course of development. They say that obsessive character evolves in reaction to these executive control deficits. In taking the measures of this etiological assertion, the word *evolve* should be scrutinized, as much hinges upon it. At first glance, it seems that Aycicegi-Dinn and colleagues are postulating an evolutionary account of obsessive origins, but this is not the case. Their use of the word *evolve* does not appear to be used in a formal sense; rather it seems to be used to denote a class of developmental dynamics more precisely described as *reactive heritability*. Reactive heritability involves structuring one's strategy of living around an inherited trait (Tooby & Cosmides, 1990); for Aycicegi-Dinn and colleagues that inherited trait is executive control dysfunction and that strategy of living is the entirety of obsessive behavior. In other words, the obsessive personality, during the course of development, is organized around this pre-existing executive dysfunction. To the degree that the present reading of Aycicegi-Dinn et al.'s theory is correct, there are two prominent problems with this explanatory model: First, it is not consistent with the ample evidence that personality traits are themselves, heritable. Heritable traits such as conscientiousness and extraversion are not predominantly instilled as a matter of habit. As suggested by modern personality theory and behavioral genetics research, personality traits are heritable entities in and of themselves. Furthermore, these same sources do not suggest that traits capriciously coalesce into clusters over the course of development. As such, although this is a biological model with a concrete finding, the interpretation of this finding is implicitly predicated upon the psychogenic formation and organization of traits. Second, should this theory be fully correct, it would still answer one question (what is the source of obsessive character traits?) by posing another (what is the source of the executive control deficits?) In other words, it would offer a proximate rather than an ultimate explanation. Truly, this cognitive deficit, being a proximate explanation, must then be explained in turn and of its own accord: Why does it exist and why is it perpetuated? As it currently stands, Aycicegi-Dinn, Dinn, and Caldwell-Harris' work is significant more for the biological nature of its inquiry and for its empirical findings, rather than for the integration of these findings into a functional etiological model of obsessive character formation. Incidentally, Aycicegi-Dinn et al.'s findings are an expected validation of earlier theoretical work by Shapiro on obsessive attention. For decades, Shapiro (1999) has described obsessive attention as sharply focused, but rigidly fixed. Also congruent with Acygegi-Dinn et al.'s findings, Gibbs-Gallagher, South, and Oltmanns (2003) describe the obsessive as having an atten-

tional agenda. It is very likely that the executive control problems described by Aycicegi-Dinn and coauthors are not causal but symptomatic of compulsivity and conscientiousness. A recent study by Fineberg et al. (2010), for example, found compulsivity to be associated with a reduced ability to fluidly refocus attention to different environmental cues. Compulsivity leads one to focus on perceptions, features or cues intently, rendering shifts in set difficult. In conclusion, executive control deficits documented by Aycicegi-Dinn et al. are likely ancillary associated features, rather than primary determinants, of obsessive character.

To recount, Hummelen et al. (2008) posited that obsessive character arises from an indiscriminant application and general overreliance on the systemizing mechanism, leaving the obsessive ill equipped to comprehend human motivation and action. Hummelen and colleagues state that this disposition, this indiscriminate application and general overreliance, is inborn. It is an inborn deficit. Nevertheless, they do not describe from whence this deficit came and why it persists. Like other biologically oriented theorists, Hummelen et al. mention that they will use evolution as part of the explanation of obsessive origins, but this appears to be limited to the use of Baron-Cohen's two evolved mechanisms: Systemizing and empathizing. They do not explicitly say that the inborn overreliance on systemizing evolved in the obsessive, and they do not further comment on its origins. Because of this, Hummelen et al., even if fully correct in other respects, could only provide a proximate explanation of obsessive origins. Again, without specifying the etiological source of the indiscriminate application of systemizing, we are left with a partial explanation that fails to fully satisfy our curiosity.

There are three problems, which at different times and to different degrees plague all three biological etiologies: 1) The substitution of proximate for ultimate explanation, 2) failure to seriously reckon with established heritability estimates, and 3) failure to rigorously apply evolutionary thought and theory. First, whatever the specified biological dysfunction, aberrant limbic functioning (Millon & Davis, 1996), executive control deficits (Aycicegi-Dinn et al., 2009) or underdeveloped empathizing (Hummelen et al., 2008), there is no explanation for its existence. Beyond describing their causal role in the development of obsessive character, there is no compelling description of how the various dysfunctions came to be. Second, heritability is variously acknowledged, but not fully assimilated. General environmental forces or parental dynamics or the process of reactive heritability remain incongruously empowered. None of the above described biological theories, despite their characterization, are sufficiently biological. All, to a great degree, incorporate psychogenic reasoning. All continue to understand developmental dynamics as a potent causal force in obsessive personality formation. In this way they are evolutionary, rather than revolutionary. Because they do not make a marked break with the past, these biological theories, though to a lesser extent, are contradicted by the judgment of behavioral genetics researchers such as Reichborn-Kjennerud et al. (2007, p. 649) who assert that obsessive character is "best explained by genetic factors alone" and Torgersen et al. who assert that obsessive character is not significantly influenced by "common, shared-in-families environmental factors" (Torgersen et al., 2000, p. 422). Third, all three theories mention evolution, but none are sufficiently evolutionary. In none of the three accounts are there descriptions of why the pattern might have evolved and no discussion of the evolutionary pressures that shaped obsessive character.

The last of these three problems merits further scrutiny. Whether in name or in spirit, whether done convincingly or unconvincingly, invoking evolution creates a new problem … at least it does so given that all three of these biological etiologies follow their dynamic predecessors in assuming that obsessive character is pathological, harmful and detrimental. Simultaneously maintaining that obsessive character is dysfunctional and evolved, places a heavy burden on the theorist. It demands that these biological theorists, to the extent that they invoke evolution, answer Keller and Miller (2006) and *resolve the paradox of common, harmful, heritable mental disorders*. For indeed,

this is what they have posited, a paradox. It seems that there is no way to extricate these theories from this paradox. To become free of this explanatory burden, these theories would have to describe how obsessive character, possessing the variously attributed deficiencies, then managed to continue its representation in the population. These theories would have to describe how obsessive persons, in spite of deficits in limbic organization, in spite of deficits in executive control, in spite of deficits in empathizing, still managed to remain as fecund and fit as their unimpaired competitors. In the struggle for life, through competition for reproductive rights and survival, the less fit are eliminated. Unabashed deficiencies are not suffered to continue on.

There are at least two ways to resolve this paradox and continue to maintain that obsessive character is both dysfunctional and evolved: First, one might say that obsessive character is an evolutionary vestige, a pattern that was once adaptive, but, finding itself in a modern context, is now maladaptive. This is the concept of phylogenic inertia (Cachel, 2006). In such a scheme the ancestral environment should be described, followed by how it contrasts with modern environments, which no longer support such a personality organization. Second, one might say that obsessive character is maintained by antagonistic pleiotropy. As the costs of sickle cell anemia are, in malarial regions, offset by the benefits of being a carrier, so too one might say that the variously proposed costs of obsessive character are somehow offset by some unarticulated balancing benefit. One may, in other words, posit a tradeoff. Again, whether using one of these solutions or another, this paradox must be resolved before any of these theories can be given credence.

Discussion and Conclusions

It was in 1908 that Freud wrote the first etiological explanation of obsessive character. In the past century, scientific advances have crowded academic journals, revolutionized thought and matured the Western mind. This is an age of genomics, controlled twin studies, evolutionary psychology, modern personality theory and cognitive neuroscience. Yet, for all such advances, we still have no compelling etiological account of obsessive origins. Though Lockean developmental theory, with its emphasis on nurture to the exclusion of nature, is no longer plausible, psychodynamic etiologies of obsessive character continue to be revised by theorists, published in journals and taught in schools. These psychodynamic theories are relics of a past age, but have not been recognized as such. Furthermore, if the presence of alternative biological theories can be taken as a sign of progress, this progress is minimal, judged both by the number of researchers and the time invested in theory development. Psychodynamic explanations are formal etiological models, but models that have not been supported by research and which cannot hope to be reconciled with the increasingly prominent demonstrations of the heritability of obsessive character. Biological models, aside from not globally explaining obsessive character, have not satisfactorily explained ultimate causes. Consequently, the established etiologies of yesterday are fatally contradicted, while the burgeoning alternatives of today cannot replace them.

Now to take up the six points outlined early in the introduction. First, we need to take seriously and assimilate the behavioral genetics data, which indicates that obsessive character is highly heritable. Behavioral genetics is a more modern field than is psychoanalysis; the two fields have different researchers, practitioners and readers. Still, it is nearly incredible that more than a decade has passed without psychoanalytic theorists seriously reckoning with established heritability estimates. Continued adherence to traditional etiological models is not, most probably, purposefully reactionary, but it is, nonetheless, anachronistic. Psychoanalytic commentary on obsessive origins continues to be uncritically read and written … it continues in Lockean bliss, unimpeded by the heritability of personality and the evolutionary significance of individual differences. Occasionally, contrary evidence is encountered

and addressed. As previously described, Mallinger (2009), after writing about obsessive personality in the traditional psychoanalytic mode for decades, perforce, commented on the heritability of traits. However, he defensively parried the evidence, rather than honestly accounting for it. Ostensibly, Mallinger and all other psychoanalysts writing on obsessive personality have not read Torgersen et al.'s (2000) heritability estimate of .78. Again, Torgersen et al. (2000, p. 424) conclude that "common, shared-in-families environmental factors … seem to be of little importance" in explaining the origins of many personality disorders, among them obsessive personality disorder. It is now twelve years since these findings were published and still they remain dissociated from psychoanalytic and psychosocial etiological accounts.

Second, because of these irreconcilable differences, we need to abandon these psychogenic etiologies. The dynamics described by psychoanalytic and psychosocial models, for instance, the parent-child power struggles within Erkisonian stage of autonomy versus shame and doubt, are quite possibly influential. These dynamics might very well affect identity specifically and the person generally. Indeed, such dynamics could affect obsessive personality itself, modifying its expression and severity. It is just that such dynamics are not the crucible of character; they are not the primary determinants of obsessive personality. Maintaining, as does Mallinger, that traits are predominantly forged and arranged by parental forces contradicts demonstrable data.

Third, we need to ask more of biological models; we need to demand true explanation of them. Rushton (1988, p. 1018) states that the "ultimate aim of science is causally to explain the world around us." In this sense, the reviewed biological theories are not truly fulfilling this aim. They posit proximate, instead of ultimate explanations. Again, an ultimate explanation is one that, when proffered, silences curiosity conclusively; one that admits of no subsequent question. Suggesting differences in limbic organization (Millon & Davis, 1996), describing executive control deficits (Aycicegi-Dinn et al., 2009) or positing an overreliance on a systemizing mechanism (Hummelen et al., 2008) do not causally explain at this most fundamental level. Instead, what they do is offer potentially correct proximate explanations, which, if substantiated, would then themselves require ultimate explanations. In conclusion, while proximate explanations are valuable, we are still left without a compelling etiological theory of obsessive origins that is grounded in ultimate causes.

Fourth, we need to either reject or revise those biological etiologies that simultaneously find obsessive character to be both heritable and harmful. Before being accepted, these biological theories, finding obsessive character to be heritable and harmful, should satisfactorily resolve Keller and Miller's (2006) paradox: *The paradox of common, harmful, heritable mental disorder.* If obsessive character is, on one hand to be understood as a disorder of empathizing, limbic function or of executive control and, on the other hand is accepted as heritable, we must explain how this is so; we must explain the coexistence of these facts. Just as consumers do not continue to buy products that are reliably broken, so disordered individuals do not continue to replicate at the mean reproductive rate. Problems in areas as profound as empathizing, through which we explain the world of motivated behavior, limbic functioning, through which we regulate our emotion, executive control, through which we plan our actions, are significant enough to depress reproductive success. Behavioral prediction, emotional regulation and future planning are essential abilities required across cultures and environments, without any of which the obsessive would be at a significant disadvantage in the competition to survive and reproduce. As a result of this disadvantage, obsessives would eventually be driven from representation within the gene pool. In conclusion, it is not that it is impossible for obsessive character or another disorder to be both heritable and harmful, but if obsessive character or another disorder is portrayed as both heritable and harmful, the etiological theorist assumes the burden of resolving and

explaining this paradoxical coexistence. None of these biological theories have recognized, much less shouldered this burden.

Fifth, we need to accept that the only way to craft a truly convincing model of obsessive origins might be to altogether stop thinking of obsessive character as disorder (Hertler, 2013). Rather than trying to fix a bad job; rather than trying to answer Keller and Miller; rather than trying to resolve the paradox while clinging to existing models, it seems best to start from scratch. In doing this we give up all assumptions. We accept that our descriptions of obsessive character are more or less sound, but we abjure all extant etiological speculation. Most fundamentally, if we are to make headway, we do not even assume that obsessive character is dysfunctional. By finding functionality within obsessive behavior, we fully sidestep Keller and Miller's challenge. Instead of resolving the paradox, we escape it by stating that obsessive character is not a disorder at all (Hertler, 2013). There is ample evidence to support such a position, despite some dissenting empirical studies (Diaconu & Turecki, 2009; Soeteman, Hakkaart-van Roijen, Verheul, & Busschbach, 2008)[iv] and despite its incongruence with established orthodoxy, obsessives 1) manifest a high global assessment of functioning (Jovev & Jackson, 2004), 2) obtain material wealth and worldly success (Ullrich, Farrington, & Coid, 2007; Ryder, Costa, & Bagby, 2007), have sound early attachments and maintain autonomous functioning (Aaronson, Bender, Skodol, & Gunderson, 2006; Kyrios et al., 2007).

Sixth, we would do better to search for the adaptive logic behind obsessive behavior and posit an evolutionary model on these foundations. Unarguably, obsessive behavior is both irregular and extreme. Yet, after surrendering the reflexive prejudice that states that obsessive character is disordered character, we are free to ask an entirely new question: What is the adaptive significance of obsessive behavior? Simply in asking this question, we are oriented towards the development of an etiological theory that simultaneously accounts for established heritability estimates, avoids psychogenic explanation, and renders Keller and Miller's paradox nugatory. We come to think of obsessive character as an intergenerationally transmitted pattern, which persists because of some advantage it confers. We look for the strategic function of the obsessive character. Recognizing that the obsessive is not cunning, dominant or charismatic, we try to find the idiosyncratic characteristics by which the obsessive can otherwise compete (Hertler, 2013). We now know that personality is not, as Tooby and Cosmides (1990) erroneously asserted, selectively neutral; it does indeed have profound consequences for survival and reproductive success. We also understand, as Figueredo et al. (2011) state, that extreme traits can arise within a population through a process of *niche splitting*. As competition intensifies, organisms are driven to abandon straightforward competition and begin to evolve alternative strategies. Might not the obsessive personality be an extreme character type evolved to exploit a previously unoccupied niche within the social landscape? This is precisely how Linda Mealey (1995) explained the psychopath. Mealey shows how the psychopath, an extreme and pathologized character type approximately synonymous with DSM-IV's antisocial personality disorder, might in fact be an evolutionary strategy. Though unwanted by society, the psychopath engages in exploitative exchanges with the larger population for which it has theoretically evolved. Blunted anxiety, circumscribed empathy and prominent sensation seeking are all genetically heritable neurological traits by which the psychopath effectively deceives and manipulates. In this way, for all its appearance of disorder, the psychopath, like the parasitic mosquito, is functioning just as it was designed to function. Perhaps both the antisocial and the obsessive are extremes pathologized, not because they fail to function, but because they fail to function in accordance with the dominant values of the day; the antisocial in terms of honesty, honor and the social contract, the obsessive in terms of balance, spontaneity and the ideal of happiness (Hertler, 2013).

Notes

i) Obsessive character or obsessive personality is generally substituted for Obsessive Compulsive Personality Disorder because the latter is a smaller subset of the former. The terms obsessive character and obsessive personality are more inclusive, subsuming both clinical and non-clinical manifestations of the pattern. The etiological review and the discussion of its implications are meant to apply to both the clinical and non-clinical varieties.

ii) It is important to note that the studies reviewed by Pollak which cast doubt on psychoanalytic etiologies do so more by failing to find support for them; rather than by pointedly disproving them. Because they cast doubt on psychoanalytic etiologies rather than positively falsifying them, many continue to believe in their validity. OCPD was first observed by Freud and has remained firmly ensconced in the psychoanalytic tradition. Even while other psychoanalytic explanations have fell out of favor, those specific to OCPD continue strong; and even those who would not describe themselves as psychoanalytically oriented, often subscribe to psychodynamic etiological accounts of OCPD. Thus, they are highlighted in the present review. Only the heritability estimates cited presently can positively disconfirm these psychoanalytic theories.

iii) Reichborn-Kjennerud et al. (2007) advise against grouping obsessive personality within the anxious and fearful cluster C umbrella because it is much more heritable and distinct from both the avoidant and dependent personalities. As Reichborn-Kjennerud and colleagues state, obsessive personality is truly “genotypically” and “phenotypically” distinct from its two counterparts because the avoidant personality and the dependent personality are “related to the … higher-order factor” of emotional dysregulation, while the obsessive personality is related to “the lower-order trait of compulsivity” (Reichborn-Kjennerud et al., 2007).

iv) Soeteman, Hakkaart-van Roijen, Verheul, and Busschbach (2008) found OCPD to impose more of an overall economic encumbrance than all other personality disorders with the single exception of borderline personality. Likewise, Diaconu and Turecki (2009) found that depressed patients exhibit more non-lethal suicidal behavior when they carry a comorbid diagnosis of OCPD. Such studies do not correspond with those cited above. This is especially true of Ullrich et al’s work which suggests that OCPD is significantly and positively associated with elevated socio-economic standing, high income, managerial positions, enlarged living quarters, and home ownership (Hertler, 2013; Ullrich, Farrington, & Coid, 2007). Nor do they correspond with other studies, as reviewed in Hertler (2013) that show OCPD to have fewer comorbidities than other personality disorders (McGlashan et al., 2000), or studies that show OCPD to be less impairing than any other personality disorder (Barber, Morse, Krakauer, Chittams, & Crits-Christoph, 1997; Skodol, Gunderson, McGlashan, & Dyck, 2002).

Soeteman et al. (2008) and Diaconu and Turecki (2009) use samples that are quite low functioning, and which are comprised of persons combining an array of psychological difficulties from a variety of categories. Rather than representing a particular personality type, these participants most often evidence symptoms from multiple personality disorder categories, while also carrying axis I symptoms. Such participants are drawn exclusively from clinical populations receiving a combination of outpatient treatment, partial hospitalization and inpatient care.

Such samples are not representative of the obsessive type and do not characterize the group as a whole. Clinically identified obsessives represent a less functional subset of the larger obsessive population; and in turn, those in the studies by Soeteman etc. represent a less functional subset of the population of clinically identified obsessives. These are people not only close enough to mental health services to be formally identified as OCPD by a mental health professional, but those that are so impaired as to gain more than one diagnosis or require treatment, and sometimes even hospitalization. In trying to understand the etiology of the obsessive, we are trying to understand their very existence. Thus, we are concerned with explaining all persons of that ilk, those in the community and in the clinic. Since the former far outnumber the latter, one should be careful of basing conclusions on such a select group, especially given the high prevalence of obsessive personality.

Funding

The author has no funding to report.

Competing Interests

The author is one of the Associate Editors of Europe's Journal of Psychology but played no editorial role for this particular article.

Acknowledgments

The author has no support to report.

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Europe's Journal of Psychology
2014, Vol. 10(1), 168–184
doi:10.5964/ejop.v10i1.679