Dr. B. Duncan McKinlay
Psychologist

London, ON, Canada
url. www.lifesatwitch.com

Website:
Tourette Syndrome • OCD • ADHD
Sensory • ODD • IED/'Rage'

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"Getting Stuck":
Relationships between Eating Disorders, Obsessive-Compulsive Disorder, and Tourette Syndrome

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It is not uncommon in the world of clinical psychology to recognize and diagnose two or more comorbid conditions presenting concurrently in the same individual. Sometimes these comorbids may be bona fide independent conditions, each with their own unique etiology, symptoms, mechanisms, treatments, and prognosis (as delineated by Emil Kraepelin). In these instances, multiple and different genetic predispositions, unrelated environmental insults and/or triggers, or dissimilar neurochemical imbalances and/or neurobiological differences have resulted in the simultaneous manifestation of numerous disorders, and clinicians were correct in identifying them as separate and distinct problems. This scenario supports the notion that the current Diagnostic and Statistical Manual (DSM-IV; APA, 1994) has indeed carved nature up at its appropriate joints.

However, it is possible that sometimes our bible misleads us – maybe sometimes our carvings have missed the mark, so that while indeed a person may have two different disorders, our conceptions of where one disorder ends and another begins is incorrect. Or maybe our present level of understanding sometimes leads us to mistakenly believe that different manifestations of the same condition represent two or more distinctive disorders. In this instance, we are blinded by surface differences. We erroneously label the person as having comorbid conditions when really we should be stepping back and recognizing how a single problem is having multi-faceted impacts on the individual.

These musings are not trivial – recognizing that we have misdefined certain disorders, or have accidentally divided one disorder into many, can have major ramifications for treatment, and future emphases on research. In this paper, the multiple and intertwining links between obsessive-compulsive disorder (OCD), anorexia, bulimia, and Tourette syndrome (TS) will be explored. Each of these disorders co-occurs at a higher level than expected by chance alone. Identical pharmaceutical treatments can be used in each disorder, foreshadowing the discovery that each of these disorders involves similar neurological mechanisms. A common underlying genetic etiology will be suggested. Finally, a hypothetical model will be outlined which will bring together the cited research into a unifying conception of the symptoms seen in each of these disorders. This model will illustrate how various environmental circumstances and varying severities may take a single problem and cause it to manifest as different "disorders".

If OCD, the eating disorders, and TS were all truly independent conditions, one might expect that these disorders should co-occur at chance levels only. In fact, the research on these conditions does not support this expectation.

Firstly, the literature linking OCD and TS is extensive. OCD is identified in 46-71% of patients presenting with TS (Pauls et al., 1986; Frankel et al., 1986; Comings and Comings, 1987). Most recently, an international effort to combine statistics from multiple TS clinics has placed the comorbidity of OCD with TS between 28 and 67 percent (Freeman & Fast, 1997). Neurologically, the basal ganglia have been implicated in both disorders (Luxenberg et al. 1988, Singer et al., 1993). Phenomenologically, some researchers have argued that the tics and twitches seen in TS are simply motor compulsions (Silvestri, 1994), while others have described gray areas between TS tics and OCD compulsions as "impulsions" (Shapiro et al., 1992). In summary of both phenomenological and neurological research, Van Ameringen, Mancini, and Oakman (submitted for publication)

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suggest that OCD and TS are both frontal-subcortical circuit disorders, with OCD being more "cognitive" in presentation, and TS being more "motor" in presentation. This conclusion is drawn from some evidence that slightly different areas of the basal ganglia are affected in each disorder (the caudate and putamen, respectively; Van Ameringen et al., submitted).

Secondly, OCD also presents at above-chance levels among those diagnosed with eating disorders, both bulimia (Aragona & Vella, 1998) and anorexia (for a review, see Mash & Barkley, 1996). Jarry and Vaccarino (1996) noted an elevated presence of obsessive compulsive symptoms, particularly those which would indicate an Obsessive-Compulsive Personality Disorder (OCPD), in individuals diagnosed with both anorexia and bulimia. 32% of bulimics (aged 22-57 years) met criteria for a diagnosis of OCD, with an additional 24% in sub-threshold levels in a study by Rubenstein et al. in 1993. Kasvikis, Tsakiris, Marks et al. (1986) reported that 10.6% of female OCD patients in their sample had a history of anorexia. In another study, conducted by Lilenfield et al. (1998), the lifetime prevalence of OCD in restricting anorexics and bulimics was higher than that seen in the normal population. Finally, Pigott et al. (1992) asked 59 adult subjects diagnosed with OCD to complete the Eating Disorders Inventory (EDI). He found that this group scored significantly higher than a normal control group on the Eating Disorders Inventory (EDI), and shared some of the same psychopathological eating attitudes and behaviours common in eating disorders (Pigott et al., 1992).

Lastly, TS is often comorbid with eating disorders. Gillberg and Rastam (1992) found that 6% of an AN sample had TS. Although low, this percentage is still noteworthy if one notes that TS is typically diagnosed more often in boys than in girls (4:1; Freeman & Fast, 1998), and is currently considered to be of only .5% prevalence in the normal population (Bruun, Cohen, & Leckman, 1998). Ballard et al (1992) suggests that binge eating can be a feature of TS, and discusses a 15-year old boy with TS as a case study example. Finally, Comings (1990) notes through numerous cases that many TS patients have eating disorders. Obesity is common within TS families, and a common compulsion in the TS child, reported by the parents, is eating (Comings, 1990).

There is also a body of research to support the idea that trichotillomania, the recurrent pulling out of one's hair, may be a form of tic, and best conceptualized within the TS family (Van Ameringan, Mancini & Oakman, submitted; Swedo & Leonard, 1992). As trichotillomania occurs much more often in females than does TS (Van Ameringen, Mancini, & Oakman, submitted), one might predict to see a higher correlation between trichotillomania and eating disorders than between TS and eating disorders. Indeed, Tattersoll (1992) suggests that the comorbidity between eating disorders and trichotillomania may be quite high, although this had been disputed (Christenson & Mitchel, 1991); Pryor (1996) also reports a case where an 18-year old girl presents with OCD, trichotillomania, and AN.

OCD, the eating disorders, and TS often present together. In addition, they may be treated by similar medications. In a review article, Hale (1996) points out the use of serotonin reuptake inhibitors (SSRIs) for OCD, TS, and trichotillomania. Messiha (1993) notes that fluoxetine, one particular SSRI, has potential use in management of OCD, anorexia, bulimia, TS, and trichotillomania. Trimble (1990) notes that clomipramine, which also inhibits the uptake of serotonin, has been proven effective with OCD. He cites 11 double blind comparative trials in which patients with OCD have experienced significant symptom improvement after administration of clomipramine. He also notes benefits in TS, and AN (Trimble, 1990). This begs the question, have problems with the neurotransmitter serotonin (5-HT) been found to be associated with each of these conditions. The answer to this is yes (Comings, 1990; Wilson, Heffernan & Black, 1996).

Could OCD, the eating disorders, and TS all have common genetics? Researchers have accumulated evidence to

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suggest possible genetic components to each of these disorders (Mash & Barkley, 1996; Comings, 1990), and there are some indications of shared genetics. First-degree relatives of anorexic and bulimic probands can be at higher than average risk for eating disorders, as well as OCD (Lilenfield et al, 1998). While no family studies in the literature have looked at whether OCD and anorexia may share common genetics (Black & Noyes, 1990), Hecht, Fichter, and Postpischil describe an intriguing case of identical twins, one diagnosed with OCD and the other with anorexia (1983). With regards to the TS/OCD link, a study by Pauls, Towbin, Leckman, et al. (1986) suggested that a single genetic factor might be common to both TS and OCD. A familial loading for OCD in the first-degree relatives of individuals diagnosed with TS has been documented (Montgomery, Clayton, & Friedhoff, 1982). Comings also presents pedigree analyses which point to shared genetics in TS and OCD (Comings, 1990). He further notes that the Gts gene(s), the gene(s) proposed to cause TS and OCD, appear to result in low serotonin. Low serotonin is integral to appetitive compulsions, and was mentioned in the preceding paragraph as being a problem in OCD, eating disorders, and TS. This low serotonin theory is further elaborated upon below in a proposed model for understanding the relationships between these disorders.

A thread common to each of these disorders is the idea of disinhibition. Whether one is stuck on a movement, thought, compulsion, or binge, the point is that aspects of the individual are out of control. The problem of lowered levels of the neurotransmitter serotonin (5-HT) appears to be shared by each of these conditions. Called "the civilizing neurohormone" (Comings, 1990), 5-HT is responsible for inhibiting all types of behaviours (Solanto, Urrutia, & Morales, 1995). Deficiencies in 5-HT are associated with a whole host of disorders, including ADHD, bulimia, TS, impulsivity, aggression, alcoholism, and depression (Comings, 1990). 5-HT inhibits both motor actions and appetite (Comings, 1990). If 5-HT is low, inhibition of the production of the neurotransmitter dopamine is released. Dopamine is the "movement" neurotransmitter, and is implicated in various movement disorders, including TS (Comings, 1990). Thus motoric disinhibition is the result. Low serotonin increases carbohydrate cravings, and appetite (Comings, 1990). Thus, appetitive disinhibition, or what Wilson, Heffernan, and Black (1996) describe as counter-regulation, is the result. Decreases in serotonin cause postsynaptic serotonin receptors to become hypersensitive, which is the suggested problem in OCD (Comings, 1990). Thus, thought disinhibition is the result. Low serotonin can result in a person getting stuck in tics, getting stuck in bingeing cycles, or getting stuck in thoughts and rituals without the ability to reinhibit these impulses.

If lowered levels of 5-HT are responsible for each of these conditions, why is there not perfect comorbidity between all of them? Based on the above hypothesizing, one would expect that OCD, the eating disorders, and TS would be the consistent and predictable result whenever 5-HT levels fall. This is obviously not the case. Hence, other factors must be involved in determining which manifestation of this disinhibition presents. The next pages will be devoted to exploring what these other factors may be.

If one were to accept the proposed model, namely that a genetic predisposition towards lowered levels of 5-HT is the diathesis for OCD, the eating disorders, and TS, various stressors or triggers might be necessary for each of these types of disinhibition to appear. If this is true, perhaps the disinhibition disorder that one is diagnosed with says more about his/her past environment and experiences than what (s)he was born with.

For example, TS research in recent years has begun to focus quite heavily on the role of the alpha-beta hemolytic streptococcal infection ("strep throat") in triggering tics (Kirshner and Kiessling, 1996). It is believed that the antibodies released against this infection will autoimmunize the basal ganglia in an individual who carries the Gts gene, thus leading to TS symptoms (Kirshner and Kiessling, 1996). Strict dieting is a widely recognized trigger in eating disorders (Wilson, Heffernan, & Black, 1996). It is possible that when one diets (thereby further decreasing their 5-HT levels and increasing postsynaptic serotonin receptor sensitivity), (s)he simultaneously

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increases his/her probability of counter-regulating and becoming obsessive compulsive (in, not surprisingly, their current focus – namely dieting efforts). Increased weight and family dieting are also documented triggers of an eating disorder – within the context of the above outline, these may be secondary in that they bring attention to the primary trigger (i.e. the need to diet).

The elegance of this model lies in it's explanation of why one would expect to see both the higher-than-normal comorbidity between these disorders as cited in the research (for example, Freeman & Fast, 1998; Mash & Barkley, 1996; Gillberg and Rastam, 1992), but not perfect correlations. In essence, a person's presentation would be molded by the presence or absence of each trigger – if the individual only "stumbled across" one trigger in his/her lifetime, the disinhibition of the decreased levels of 5-HT would be fully "channeled" into only one disorder (for example, OCD). If, on the other hand, one were exposed to multiple triggers, his/her disinhibition would be divided among two or three disorders (for example, an eating disorder and TS). Assuming that these trigger experiences are fairly commonplace, but not universal experiences to everyone (as strep-throat, or dieting behaviours are) then the pattern of high comorbidity without perfect correlation is expected.

This model may help to explain other findings in the literature as well: Silvestri et al. (1994) found that when they treated the TS symptoms in one client, he became anorexic. Cantwell, Sturzenberger, Burroughs, et al. (1977) found that OCD symptoms persisted even after normal weight was restored in a sample of anorexics. It has also been established that some TS'ers medicated for OCD can become more behaviourally disinhibited (Barnhill, 1997). What seems to be occurring is that one symptom of disinhibition is substituting for, or persisting in the absence of, another. It is possible that if the low levels of 5-HT are denied expression in one way, a different subclinical condition could rise to surface. If this were true, then findings that initially appear to debunk a possible link between these disorders (such as the study by Cantwell, Sturzenberger, Burroughs, et al., 1977) may in fact be providing support for this link instead.

Differing triggers may result in differing disorders from the same underlying cause. Another factor that could affect the presentation of disinhibition could be the strength of the diathesis. Many researchers have described a so-called obsessive-compulsive spectrum (Goldsmith, 1998; Aragona, 1998; Van Ameringen et al., submitted; Swedo & Leonard, 1992); perhaps this spectrum concept could be broadened to a more general disinhibition spectrum: as one rises the spectrum, more severe, disruptive, and irrepressible the symptoms become.

For example, assuming that the individual is triggered for motoric disinhibition, less and less of the neurotransmitter 5-HT would mean more and more dysregulation in dopamine production, and so one would expect involuntary movements to increase in number, frequency, intensity, and duration. The DSM-IV does in fact recognize various tic disorders, increasing in severity from transient motor or vocal tics (Transient Tic Disorder) to chronic motor or vocal tics (Chronic Tic Disorder) to multiple chronic motor and vocal tics (TS; APA, 1994). As well, it is suggested by Comings (1990) that there may be numerous Gts genes, and that their effects may be additive -- to be Tourettic, he suggests, one must be homozygous (Comings, 1990).

Assuming instead that the individual is triggered for appetitive disinhibition, lower and lower levels of 5-HT would mean stronger and stronger bingeing behaviour. Finally, in both OCD and eating disorders lower and lower 5-HT means stronger and stronger obsessions. This could mean a spectrum spanning from some rigidity and perfectionistic thinking (OCPD) to severe OCD. It could also mean a spectrum spanning from obsessive purging/abstaining sufficient to maintain average weight (bulimia), to obsessive purging/abstaining at dangerous extremes (anorexia).

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In conclusion, based on the high comorbidities between OCD, the eating disorders, and TS, similar treatments, and suggestions of common etiologies, a model was put forward which links each of these conditions as various expressions of neurochemical disinhibition. It was postulated that low levels of 5-HT, a neurotransmitter used by the body to inhibit behaviour, is the general difficulty in each of these disorders. Which set(s) of symptoms one displays is dependent on both which environmental triggers one is exposed to, and the degree of disinhibition displayed.

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