Dr. B. Duncan McKinlay
Psychologist

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

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

Publishing:
Nix Your Tics! Eliminate Unwanted Tic Symptoms: A How-To Guide for Young People

NOTE — THIS PAPER WAS WRITTEN AS PART OF A MIDTERM EXAM IN PSYCHOPATHOLOGY DURING THE FIRST YEAR OF MY MASTERS PROGRAM. HENCE IT IS SOMEWHAT DATED AND BASED ON THE LEVEL OF KNOWLEDGE I POSSESSED AT THAT TIME. PLEASE READ WITH THIS IN MIND AND A GRAIN OF SALT!

Question: Anxiety Disorders co-occur and/or are strongly related to a number of other disorders. Describe similarities or areas of overlap with other disorders and in describing these areas speculate on a model that might account for such overlap.

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Anxiety disorders are a heterogeneous mix; a myriad of behaviours, symptoms, problems, and disorders overlap with and within this category. It is obvious that some reconceptualizations of current classifications of anxiety and related disorders are necessary. Most individuals with Obsessive Compulsive Disorder experience some degree of Generalized Anxiety Disorder and Depression. Other anxiety disorders such as Social Phobia, Specific Phobia, Panic, Agoraphobia, and associated disorders such as Impulse-Control Disorders and Eating Disorders revolve around obsessive concerns and ritualistic or avoidance behaviours. If an individual is diagnosed with OCD, (s)he has a higher probability of diagnosis of (in descending probability): Tourette's Syndrome, Major Depression, other Anxiety Disorders (GAD, panic, Social phobia), Substance Abuse, Conduct Disorder, Attention-Deficit-Hyperactivity Disorder, or an Eating Disorder (Rapoport, 1992 from notes). This degree of comorbidity is cause for raising suspicion against our current "valid" diagnostic system.

Emil Kraepelin suggested over a hundred years ago in his book Psychiatry (Kraepelin, 1885 from Roberts) that diagnostic validity should be based on five factors: common etiology, biological mechanisms, symptoms, prognoses, and treatment. That is, two presentations should be considered the same disorder if they agree on all five previous dimensions. To highlight the extent to which other disorders resemble certain anxiety disorders, evidence will be cited along each dimension. Particular emphasis will be placed on the similarities between Obsessive Compulsive Disorder and Tourette's Syndrome. Etiology will be discussed last, as it pertains to a newly proposed model of psychopathology.

The symptomatology of OCD is hardly unique. In one study, 45.4% of TS'ers exhibited obsessive-compulsive behaviours in contrast to only 8.5% of controls (Comings, 1990). Furthermore, the obsessions and compulsions of TS are virtually indistinguishable from OCD. Touretters are similarly plagued with intrusive thoughts of contamination, sexuality, symmetry, precision, and aggression. Checking, washing, counting, symmetrical, behavioural repetition, and touching compulsions also overlap. For illustration, the following two captions are from two different individuals suffering from TS describing obsessions and compulsions, respectively:

> At age 21 I began to develop obsessive thoughts about many things. These included a) constant dwelling on math especially long division, b) constant thoughts of how traffic lights work…..a desire to keep my teeth in perfect order….worrying about whether traffic lights really went green and backing up to check……I would find myself climbing up to the top of buildings to measure the dimensions of the structures, taking exact measurements for hours (Comings, 1990).

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> I am brushing my teeth. The need to use my right hand to hold the toothbrush causes me to begin jerking my right hand. This in turn triggers my whole right arm to begin flinging outward. While this is happening I am blinking my right eye and opening my eye wide. Also I am bouncing my head up and down while I am repeating, often times aloud and in groupings of four, the words, 'Amen, Amen, Amen, Amen'….I also am tapping my right temple with my second and third fingers, again in some series of four…..(Comings, 1990).

Both OCD and TS obsessions and compulsions are ego-dystonic. Both types of patients harbour an inner sense of incompleteness, and feel imperfect, and a need for control (most probably a voluntary compensation for the lack of control involuntarily). Difficulty in focusing concentration, due to the cognitive expenditures involved with obsessional thinking, is a hallmark of not only OCD and TS, but ADHD as well, a disorder with higher occurrence rates in both populations. Depression is high amoung both disorders; the supposed "unusually high requests for reassurance" is a natural response if one considers that, in these disorders, there is an unusually high degree of self-deprecation. Both individuals try to hide their disorders, and can suppress the symptoms in particular environments (ex., the doctor's office) for limited amounts of time, and both disorders increase in severity immediately after such efforts. Lack of sleep and stress exacerbate both conditions, and onset for both is approximately 7-10 years of age. Finally, up to 80% of TS'ers are also diagnosed with OCD, and although only 6-7% of OCD patients are **diagnosed** with TS, up to 30% demonstrate tics of insufficient severity to call TS, and up to 70% of patients have tics or a diagnosis of TS in their family pedigree (Comings, 1990).

With respect to physiological mechanisms, the basal ganglia, and in particular the caudate nuclei, have been implicated in both OCD and TS. Both disorders have similar EEG abnormalities, and involve a serotonin/ dopamine interaction problem. Finally, frontal-lobe dysfunction is definitive of both conditions.

Identical treatments can be considered for OCD and TS. Clonidine, a drug which decreases norepinephrine levels, not only minimizes the symptomatology of OCD and TS, but also Panic, ADHD, phobias, depression, and aggressive/oppositional behaviours. Clomipramine, which inhibits re-uptake of serotonin, thus "increasing" it, is effective for OCD, TS, panic, and depression. Lastly, Fluoxetine (Prozac) also helps OCD, depression, panic, and coping with TS. The fact that OCD and TS respond similarly to drugs suggests similar neurochemical problems (i.e., it would appear that the decrease in serotonin -- thought to create the obsessions, compulsions, and low mood -- disinhibits production of both dopamine and norepinephrine -- thought to be involved with the more motoric aspects of the disorders -- in both cases).

The prognoses for OCD and TS are a lifelong course with waxing and waning. Both are involuntary, intrusive, "ego-alien entities". It is interesting to note that as TS'ers age, motoric tics tend to decrease, and more "cognitive" tics (i.e., O/C behaviour) increases. This makes biochemical sense if one considers that it is a natural development for human dopamine levels to decrease as we grow older (Cavanaugh, 1993).

In discussing a shared etiology for OCD, TS, and a whole host of associated disorders, including depression, anxiety disorders, ADHD, substance abuse, conduct disorder, learning disorders such as dyslexia, and impulse-control disorders, a new model for consideration of such disorders will be outlined. Comings (1990) has postulated the existence of the Gts (or generalized disinhibition) gene. This gene is considered to be semi-dominant, semi-recessive and, based on the assumption that TS'ers are homozygous, and based then on the prevalence of TS (about 1%), it is thought to be carried by a full 18% of the population. The disorders associated

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with TS (for example, OCD) are thought to be the result of only one Gts gene.

Where do these assumptions come from? Before Comings (1990) suggested that TS might be genetically associated with other disorders, the relatively low incidence of TS led most to believe that the disorder was a dominant trait. After taking the pedigrees of hundreds of TS'ers, however, Comings noticed that OCD, learning disorders, ADHD, and so forth within the family was the rule rather than the exception. In one study he compared the immediate and distant relatives of TS'ers on both sides with control families, and found considerable substantiation for his assumptions:

> -if TS'ers are homozygous, one would expect to see evidence of heterogeneous carriers on both sides of the family, as evidenced by learning disorders, OCD, ADHD, and such. There were significant differences between the numbers of these disorders in the TS'ers family and controls (on both sides).
>
> -if the gene is semi-dominant, semi-recessive, one would expect that, on average, the gene would manifest as OCD, tics, dyslexia, ADHD, etc. 50% of the time on average. When both tics and associated behaviours on both sides of the family were considered, 45.3% of the TS'ers families demonstrated evidence of the gene, compared to 5.7% of controls.
>
> -if the parents of TS'ers are both carriers of this Gts gene, and there is a 50% probability of this gene manifesting in the parent, one would expect that there is a 25% probability of both parents displaying tics or associated behaviours, neither displaying tics or associated behaviours, and a 50% possibility that one will demonstrate signs. 29.4% of TS'ers had parents who both showed signs of the gene, 20.6% had neither parent demonstrate signs, and 48.2% and 60.6% had mothers and fathers with signs of the gene, respectively (Comings, 1990).

It seems at first intuitively strange that evolution would select for such a gene, but closer consideration fosters a possible explanation. First, the concept of "heterosis" is invoked – in this scenario, this genetic principle reads as follows: if the selection of ONE Gts gene is of GREATER adaptiveness than TWO Gts genes are of LESSER adaptiveness, then selection will still occur. Put another way, having one Gts gene (a "little bit" of disinhibition) is such an advantage, that it is worth the gamble of possibly ending up with 2 Gts genes, which is not adaptive itself at all. Why is disinhibition adaptive? Many possibilities can be suggested, but Comings focuses on sexual disinhibition. In a study by Behar and Stewart (1984) it was found that mothers who gave birth to children with conduct disorder (carriers of the Gts gene) were significantly younger than control mothers (18.7 vs. 23 years, $p>.001$). Even assuming that everything else is equal, and that each mother only has one child, the Gts gene would represent an 18.7% increase in the number of offspring by the conduct disorder group (Comings, 1990).

By this logic, a gene can be selected for even if it ISN'T adaptive to the organism who carries it: this is analogous to the "Driving Y" or the "Son Killer" SK gene which can actually exterminate populations simply because up until extinction there still exists a selective advantage for the individual gene (Daly). Lastly, just because the expression of a single Gts gene is not adaptive in contemporary society does not mean that it was not adaptive historically: because civilization "evolves" at a much faster rate than does genetic evolution, often the environment which we are optimally suited for is "mismatched" with today's lifestyle. Cleanliness rituals ("OCD) were doubtlessly vital before medications were known about, and a quickly shifting focus of attention ("ADHD") would be beneficial to a hunter if he wished to avoid becoming the prey. Another line of reasoning would suggest that these "disorders" only manifest due to the way our culture is shaped – for instance, there have

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always been many "Attention-Deficit" individuals, but they were undetectable until the present educational system was developed and demanded an extent and duration of attention which surpassed these individual's "thresholds".

Now that the model has been outlined, some of the previously stated findings can be re-conceptualized. It was mentioned previously that far more TS'ers are diagnosed with OCD than the reverse, however oftentimes individuals with OCD display tics of insufficient severity to warrant a diagnosis of TS. This now makes perfect sense if one considers OCD to be an "incomplete" expression of TS. Also, to return to the neurochemical aspects of these associated disorders, one might hypothesize that one Gts gene lowers serotonin levels somewhat, and the additional Gts gene affects serotonin sufficiently to disinhibit dopamine and norepinephrine. This would explain why, for example, depression (which is affected by serotonin levels) is highly correlated with both TS and associated disorders such as OCD, and why ADHD (affected by dopamine levels) is more highly correlated with TS than it is with anxiety disorders such as OCD. Finally, due to the "disinhibition" terminology, one begins to conceptualize TS, OCD, affective disorders, anxiety disorders, and ADHD as different manifestations of disinhibition – TS is predominantly recognized for it's motoric disinhibition, however OCD is a cognitive disinhibition (uncontrollable, intrusive thoughts), ADHD is an attentional disinhibition (the chief problem isn't so much an inability to CONCENTRATE, as it is an inability to FOCUS that concentration), and mood disorders and panic are different disinhibitions of emotional thought.

Finally, to tie this discussion into question #1, this model fits well with a dimensional approach to classification (ex. Hudson & Pope's affective spectrum), as the degree of disorder would be due to the environment one was in. The Gts gene(s) represent(s) the "diathesis", while the stressors impinging on one's life would determine the extent of expression. Dimensions like "Mood", "Anxiety", "Concentration", and "Motor" could be ranked by degree of disinhibition, and each individual would demonstrate unique combinations and severities of disinhibition depending not only on what genes they have, but how the disinhibitions have manifested because of particular learned events, culture, occupation, personality, age, and so on.

In conclusion, while much of the foregoing discussion is based on a mythical Gts gene yet to be marked, it should at least be obvious that there exist significant problems with the current method of categorizing anxiety and overlapping disorders. If nothing else, it has been shown that there are alternative ways of considering these disorders with at least as much plausibility as the present system.