Creativity and Psychopathology: A Shared Vulnerability Model

Shelley H Carson, PhD

Creativity is considered a positive personal trait. However, highly creative people have demonstrated elevated risk for certain forms of psychopathology, including mood disorders, schizophrenia spectrum disorders, and alcoholism. A model of shared vulnerability explains the relation between creativity and psychopathology. This model, supported by recent findings from neuroscience and molecular genetics, suggests that the biological determinants conferring risk for psychopathology interact with protective cognitive factors to enhance creative ideation. Elements of shared vulnerability include cognitive disinhibition (which allows more stimuli into conscious awareness), an attentional style driven by novelty salience, and neural hyperconnectivity that may increase associations among disparate stimuli. These vulnerabilities interact with superior meta-cognitive protective factors, such as high IQ, increased working memory capacity, and enhanced cognitive flexibility, to enlarge the range and depth of stimuli available in conscious awareness to be manipulated and combined to form novel and original ideas.


Highlights

- Cognitive systems that underlie creative ideation may be dependent primarily on irregularities in both serotonin and dopamine neurotransmitter systems.
- Creative people may depend on vulnerability factors to enhance their work.
- As creativity and psychopathology may be different outcomes of biological vulnerability factors, art, music, or writing therapies may improve symptoms of psychopathology by increasing protective factors associated with creativity.

Key Words: alcoholism, creative achievement, bipolar, creativity, divergent thinking, genetics, IQ, latent inhibition, novelty seeking, psychosis proneness, schizotypy

John Forbes Nash, mathematician, Nobel Prize winner, and person diagnosed with schizophrenia, was once asked why he believed that aliens from outer space had recruited him to save the world. He responded, “because the ideas I had about supernatural beings came to me the same way that my mathematical ideas did. So I took them seriously.”, p 11 While creative ideas may certainly be produced by deliberate mental effort, Nash’s statement suggests that, at least in some instances, creative insight may share phenomenological elements with psychotic experiences, by appearing to arrive suddenly and fully formed into conscious awareness.

Biographies of creative people, and an array of empirical studies, provide an ever-growing catalogue of evidence for a relation between creativity and psychopathology. This relation appears to be best described by a model of shared biological vulnerability. Genetic vulnerability factors, related to the functioning of DA and 5-HT in theprefrontal and subcortical brain, may predispose certain people to experience altered mental states that provide access to—and interest in—associational material typically filtered out of conscious awareness during normal waking states. These vulnerability factors may manifest themselves as severe psychopathology or as creative ability, depending on the presence of additional cognitive factors that act to protect a person from the most severe consequences of mental disorder.

Is There a Relation Between Creativity and Mental Illness?

Scholars in the Humanist tradition have viewed creativity as an aspect of the fully functioning personality and as a
facet of self-actualization. Maslow described the creative person as one who was living a fulfilled life. Others have noted that creativity is correlated with desirable personality constructs, such as openness to experience and self-confidence. Creativity is a highly valued trait in fields as diverse as business, sports, and the military, as well as the arts and sciences. Despite the association of creativity with desirable personal features, the tendency for creative people to suffer from mental illness has been noted since the time of the ancient Greeks. Plato, for example, suggested that poets, philosophers, and dramatists suffered from “divine madness” p 51 while Aristotle was among the first to associate poets with melancholia.

Empirical evidence for the connection between creativity and psychopathology began to emerge in the latter half of the last century, when 2 separate studies reported that the children of mothers with schizophrenia were found to be more creative than the children of matched control subjects. Such findings prompted a new generation of researchers to empirically examine the incidence of psychopathology within the population of high creative achievers. In general, this research has noted a higher risk for 3 types of disorders among creative people: mood disorders (especially bipolar spectrum disorders), SSDs, and substance abuse disorders.

**Creativity and Mood Disorders**

In the 1980s, 2 studies based on modern diagnostic methods indicated that prominent creative people incurred a greater risk for mood disorders than their less creative counterparts. Andreasen compared writers from the prestigious Iowa Writers Workshop and their first-degree relatives to a matched control group. She found that fully 80% of the writers had suffered from a mood disorder, and that the writers were 4 times more likely to suffer from BD than the control subjects. Andreasen also found that both mood disorders and creative interests tended to run in families, concluding that “affective disorder may be both a ‘hereditary taint’ and a hereditary gift.”

Meanwhile, Jamison compared award-winning artists and poets from the United Kingdom to population norms and found that they were more than 5 times more likely to be diagnosed with any mood disorder and 6 times more likely to be diagnosed with BD. She further reported that rates of creative productivity seemed associated with upswings in mood both in disordered and in nondisordered subjects.

Two influential studies examined mood symptoms in deceased creative luminaries, rendering psychiatric diagnoses based on available biographical sources. Post studied the lives of 291 world-famous men in different creative professional categories. Using general population demographics as control subjects, he found that his subjects, in all professional categories, demonstrated higher rates of undifferentiated mood disorder. Ludwig, analyzing the biographies of more than 1000 deceased luminaries, found significantly higher rates of psychopathology, including mood disorders, among people in the creative arts (artists, musical composers and performers, and writers) than among those pursuing other professions.

While these studies indicated a connection between creativity and mood disorders, Richards et al found that the degree of dysfunction was an important component of this connection. They noted that subjects with cyclothymia and the first-degree relatives of subjects with manic depression (BD) had higher creativity scores than either nondisordered control subjects or the subjects with BD themselves. The authors concluded that either hereditary risk or milder, subclinical variations of bipolar pathology may enhance creativity, but that full-blown BD may interfere with creative activity—a set of conclusions known as the inverted U hypothesis of creativity and psychopathology.

In general, research on creativity and mood disorders suggests:

1. creative people may carry a risk for BD that is greater than that of the general public
2. mild forms of bipolar pathology or genetic risk for BD are more beneficial to creative output than more severe forms of the illness.

**Abbreviations**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>5-HT</td>
<td>serotonin</td>
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<tr>
<td>5-HTR2A</td>
<td>serotonin receptor 2A gene</td>
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<tr>
<td>5HTT</td>
<td>serotonin transporter</td>
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<td>5HTTLPR</td>
<td>promoter region of the human serotonin transporter</td>
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<tr>
<td>A779C</td>
<td>single nucleotide polymorphism (SNP) of the TPH1 gene</td>
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<tr>
<td>BD</td>
<td>bipolar disorder</td>
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<tr>
<td>COMT</td>
<td>catechol-O-methyltransferase</td>
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<td>DA</td>
<td>dopamine</td>
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<td>DRD2</td>
<td>dopamine D2 receptor gene</td>
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<tr>
<td>DRD4</td>
<td>dopamine D4 receptor gene</td>
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<td>LI</td>
<td>latent inhibition</td>
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<td>NRG1</td>
<td>neuregulin 1</td>
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<tr>
<td>SLC6A4</td>
<td>serotonin transporter gene</td>
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<tr>
<td>SSD</td>
<td>schizophrenia spectrum disorder</td>
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<tr>
<td>T102C</td>
<td>single nucleotide polymorphism (SNP) of the 5-HTR2A gene</td>
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<tr>
<td>Taq1A</td>
<td>polymorphism of the DRD2 gene</td>
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<tr>
<td>TPH1</td>
<td>tryptophan hydroxylase gene 1</td>
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<td>Val158Met</td>
<td>polymorphism of the COMT gene</td>
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3. creativity appears to run in families,\textsuperscript{11,12} and
4. shifts in mental states associated with mood may facilitate creativity.\textsuperscript{12}

These findings appear to indicate the presence of shared vulnerability between creativity and BD—one that is facilitated by altered states of cognition brought about by mood changes.

\textbf{Creativity and SSDs}

There is a rich literature describing psychotic and odd or eccentric behaviour in creative people. For example, William Blake claimed that his poetry and his paintings resulted from visions of visiting spirits who sometimes jostled him while competing for his attention,\textsuperscript{10} a claim reminiscent of that of Nash. The composer Robert Schumann suffered from frequent hallucinations and delusions, and at one point believed that Beethoven and Mendelssohn were channeling musical compositions to him from their tombs.\textsuperscript{17} Studies of creative achievers at Berkeley’s Institute for Personality Assessment and Research (commonly referred to as IPAR) found that creative writers and creative architects had elevated scores on the Minnesota Multiphasic Personality Inventory scales of Schizophrenia and Paranoia.\textsuperscript{18,19} and noted that creative people in both categories frequently reported unusual perceptual occurrences and odd mystical experiences.\textsuperscript{20}

Reviews by Prentky\textsuperscript{21} and Brod\textsuperscript{22} associate schizotypy and psychosis proneness with creative ability, and demonstrate that creativity and schizotypy tend to run in families. In addition, several recent studies have examined schizotypy and creativity, making the distinction between positive schizotypy (characterized by the unusual perceptual experiences and magical thinking) and negative schizotypy (characterized by low sociality and anhedonia).\textsuperscript{23} Two studies by separate research groups compared British art students to students in non-arts disciplines. Both Burch et al\textsuperscript{24} and O’Reilly et al\textsuperscript{25} found that art students scored significantly higher than the control group on measures of positive, but not negative, schizotypal traits.

Two additional studies\textsuperscript{26,27} have found that positive and negative schizotypal traits may differentiate types of creative people. Nettle\textsuperscript{26} found that poets and artists, along with psychiatric patients, had elevated levels of positive schizotypal traits. However, mathematicians reported levels of positive schizotypy that were actually lower than those of the general population, as well as high levels of negative schizotypal traits.\textsuperscript{26} These results were replicated by Rawlings and Locarnini,\textsuperscript{27} who found that their group of professional artists and musicians scored higher on measures of positive schizotypy, hypomania, and a tendency to make loose associations than their group of biologists and mathematicians. The latter group received higher scores on an Asperger scale that included many of the symptoms of negative schizotypy. The authors conclude that the relation between creativity and psychopathology may be divided by domain into a propensity to oversystematize stimuli in the science–math domains and a psychosis prone tendency toward undersystemization and loose associations in the fine arts domains.\textsuperscript{27}

Replicating the inverted U pattern of creativity and psychopathology noted with patients with BD in their earlier study, Kinney et al\textsuperscript{28} found that peak creativity levels were higher in subjects with schizotypal personality disorder or 2 schizotypy signs (such as magical ideation or illusion experiences) than in subjects with no schizotypal signs or with full-blown schizophrenia.

In general, the SSD studies have provided evidence to support 3 conclusions:

1. there is an elevated level of schizotypy and psychosis proneness in divergent thinkers and creative people;\textsuperscript{21–28}
2. schizotypy and psychosis proneness appear to run in families;\textsuperscript{21,22,28} and
3. milder symptom sets are more conducive to creativity than more severe forms of the SSDs,\textsuperscript{28} as is the case with bipolar spectrum disorders.

The tendency toward schizotypal thinking appears to constitute an altered state, in which the person becomes aware of material that is typically suppressed before entering consciousness. Sass\textsuperscript{29} has suggested that the break with reality associated with such schizotypal cognition may enhance creativity, under some circumstances, by allowing the affected person to view situations from a totally new perspective.

\textbf{Creativity and Alcoholism}

Creative luminaries have long associated alcohol ingestion with creative inspiration. Alcohol ingestion may induce an altered state that allows unusual ideas to enter consciousness as a consequence of the relaxing of normal inhibitory mechanisms. Over 2000 years ago, the Roman poet Horace wrote, “No poems can please for long or live that are written by water drinkers.”\textsuperscript{30, p 4255} Results of the research on creativity and alcoholism support a greater prevalence of alcoholism among creative groups.\textsuperscript{11,13,14}

For example, Andreasen\textsuperscript{11} found that 30% of the writers in the Iowa Writers Workshop suffered from alcoholism, compared with 7% from the control group, a significant difference. Post,\textsuperscript{13} in his biographical review of famous men, found that 14% of the group of writers, composers, and artists met diagnostic criteria for alcoholism. Ludwig\textsuperscript{11} reported, likewise, an elevated mean level of alcoholism (19% to 41%) among artists, musicians, fiction writers, and poets, but low rates (1% to 2%) among natural scientists. Levels of alcoholism appear particularly elevated among fiction writers. Among the 8 American novelists who have
won the Nobel Prize, 5 have been alcoholics. As with other disorders, it appears that although creative people may find that drinking inspires creativity, full-blown alcoholism is detrimental to creative efforts.32 Biographical accounts of the lives of famous writers such as Hemingway, Poe, and Fitzgerald provide dramatic evidence that progressive alcoholism diminishes both the creative ability and the creative productivity of writers.32

Because rates of alcoholism are extremely elevated, not only among creative people but also among the populations with BD and SSDs as well,33 the tendency toward alcoholism may be indicative of an underlying shared vulnerability.

The Shared Genetic Vulnerability Model

Because the disorders associated with creativity, as well as creativity itself, are bothheritable2,34,35 and polygenetic,36,37 several investigators have suggested that creative thought processes may share genetic factors with such disorders but may also include other genetic elements that discourage the expression of mental illness.38,39 Such a shared vulnerability model explains numerous findings, including the greater risk for psychopathology found in some creative people and the findings of increased creativity in the first-degree relatives of people with serious psychopathology. The model also accounts for the stable rate of disorders such as schizophrenia, despite evidence that people with schizophrenia reproduce at a lower rate than the general population.40 The role of creativity in maintaining the adaptability of the species may provide a reproductive advantage for the transmission of at least some portion of the schizophrenia genotype.41

Based on available results from neuroimaging and genetic studies, it seems reasonable to propose a shared vulnerability model in which factors common to both creativity and psychopathology act to increase access and attention to material being processed below the level of conscious awareness, while protective cognitive factors allow for executive monitoring and control of such enhanced access. Protective editing factors thus allow creatively productive people to exert meta-cognitive control over bizarre or unusual thoughts, enabling the person to take advantage of such thoughts without being overwhelmed by them.42 Candidates for vulnerability factors include reduced LI, increased sensitivity to novelty salience, and neural hyperconnectivity. Candidates for protective factors include high IQ, enhanced working memory capacity, and cognitive flexibility (Figure 1).

Reduced LI as a Shared Vulnerability Factor

LI is the capacity to screen from conscious awareness stimuli previously experienced as irrelevant. When LI is reduced, information that would typically be categorized as irrelevant is allowed into conscious awareness.43 Reduced LI has been observed in schizophrenia and psychosis-prone people,44,46 and can be induced by ingesting psychoactive substances.47 Reduced LI has also been reported in nondisordered subjects who score high on the personality variable of openness to experience, a trait often associated with creativity. Carson et al48 have proposed that reduced LI may enhance creativity by enlarging the inventory of unfiltered stimuli available in conscious awareness, thereby increasing the odds of synthesizing novel and useful combinations of stimuli. These researchers have demonstrated that LI was in fact reduced in high-functioning people who had high scores on measures of creativity and openness to experience.48–50

Novelty Seeking as a Shared Vulnerability Factor

While reduced LI may increase the information available in conscious awareness that can be combined to form novel ideas, enhanced novelty seeking may provide the intrinsic motivation to attend to such novel ideas. Creative people tend to seek out novel or complex stimuli over familiar or simple stimuli.51–53 Internal rewards (via the DA system) for seeking novel aspects of the environment or novel stimuli may provide the creative person with intrinsic motivation and intellectual curiosity.54 However, novelty seeking is also associated with alcohol abuse and addiction,55 and with bipolar states of hypomania and mania.56 People with both BD and substance abuse disorders have demonstrated significantly higher scores on measures of novelty seeking.57 Therefore, novelty seeking may be both an incentive for creative work and, simultaneously, constitute a risk factor for psychopathology.

Neural Hyperconnectivity as a Shared Vulnerability Factor

A third potential shared vulnerability factor, neural hyperconnectivity, includes an abnormal neural linking of brain areas that are not typically functionally connected. Hyperconnectivity, generally supposed to be caused by irregularities in synaptic pruning during development, has been detected among both people with schizophrenia and their first-degree relatives, and may account, in part, for the bizarre associations often reported by people with schizophrenia.58 Hyperconnectivity has also been noted in neuroimaging studies of synesthesia, the tendency to make cross-modal sensory associations.59 Synesthesia runs in families, and is 7 to 8 times more prevalent among highly creative people than among the general population.60 Brain-imaging studies have also reported more alpha synchronization, both within and across hemispheres, in the brains of highly creative, compared with less creative, subjects during creativity tasks,51 suggesting unusual patterns of connectivity. Ramachandran and Hubbard60 have speculated that patterns of hyperconnectivity may form the basis of human metaphorical thinking, a type of thinking often described both by creative people and by those experiencing hypomania, psychotic episodes, and drug intoxication. Mednick60 theorized that the ability
to synthesize remotely associated elements of thought into new and useful combinations constitutes the basis of creativity. The connection of (normally) functionally unrelated parts of the brain as a consequence of decreased synaptic pruning may provide the neurological mechanism for remote associations between stimuli that are the basis of creative thought.

**High IQ as a Protective Factor**

Increased IQ helps protect otherwise vulnerable people from several serious psychopathologies. A body of research indicates that IQ is correlated with measures of creativity up to an IQ score of 120, suggesting an threshold score for IQ that is necessary but not sufficient to explain creativity. However, it should be noted that the IQ threshold is somewhat dependent on the domain or creative endeavour, with visual artists demonstrating a lower IQ threshold than theoretical physicists. Carson et al hypothesized that if reduced LI increases the stimuli available to conscious awareness, then high IQ may allow a person to process and manipulate the additional stimuli rather than becoming confused or overwhelmed by it. In the presence of low IQ, reduced LI may increase the probability of psychosis. In the presence of very high IQ, however, reduced LI may allow people to visualize novel connections between internally and (or) externally generated stimuli. In Carson et al's study of eminent creative achievers, the combination of reduced LI and high IQ predicted 30% of the variance in creative achievement scores (Figure 2).

**Enhanced Working Memory as a**

**Protective Factor**

Enhanced working memory capacity may also constitute a protective factor in a shared vulnerability model of creativity and psychopathology. People with higher working memory capacity may be more able to process additional stimuli resulting from altered states of consciousness, such as those produced by reduced LI, mood disorders, or SSD symptoms. Support for this hypothesis emerged in a study of a high-functioning group of subjects in which LI deficits combined with high scores on a measure of working memory for abstract forms to predict over 25% of the variance in creative achievement scores. Working memory for abstract forms has also been shown to predicted the ability to solve insight problems (a type of creative task) in a sample of college undergraduates. If, as suggested by Mednick, creativity is based on the ability to combine aspects of remotely associated constructs, then the ability to hold and process a large number of constructs in mind simultaneously without becoming confused or overwhelmed should predispose the individual to creative rather than disordered cognition.

**Cognitive Flexibility as a Protective Factor**

A final protective factor, cognitive flexibility, is the ability to switch attentional states by disengaging attention from one stimulus or concept and re-engaging it on other stimuli through conscious mental control. Deficits in this ability have long been considered a hallmark of schizophrenic thought. Cognitive flexibility allows a person to entertain information from more than one perspective and is associated with the trait of openness to experience, the personality trait most often associated with creativity.
If creative people are experiencing magical thoughts or unusual perceptions, cognitive flexibility may provide them with a method of either disengaging attention from the psychoticlike experiences or interpreting them in a benign manner rather than as a sign of madness. Recent research indicates that psychoticlike experiences are more prevalent in the general population than previously expected and that the interpretation of such events (rather than the events themselves) may determine the extent of the associated mental illness. Therefore, cognitive flexibility, allowing one to move in and out of altered states of consciousness and allowing one to interpret anomalous experiences in a healthy manner, may constitute a protective factor in the interface between creativity and psychopathology.

**Genetic Research**

Molecular genetic studies have begun to hone in on a set of genes, many of which are related to DA and 5-HT transmission, that appear to be associated with the creativity-relevant mental illnesses: BD, schizophrenia, and alcoholism. Some possible genetic links to these illnesses and to the previously described protective factors for creativity are listed below. However, studies that link genetic variations to psychological factors are often difficult to interpret; in many instances conflicting results have been obtained, and in other instances initial studies have not been replicated. Therefore, the following examples must be considered as potential areas for further research rather than as established fact.

For example, Reuter et al. found that the A1+ allele of Taq1A was linked to creativity in a sample of German university students. The A1+ allele has also been associated with novelty seeking, schizophrenia, and alcohol addiction. D2 receptor sensitivity has also been linked to reduced LI in mice and humans; however, the precise DRD2 alleles associated with decreased LI have not been reported. Additional genes related to DA, including DRD4 and SLC6A3 (the dopamine transporter gene) have been linked to both risk for schizophrenia and BD and to novelty seeking. As novelty seeking is important in creative cognition, variations in the availability of, and sensitivity to, DA may determine shared vulnerability to creative cognition and to the types of psychopathology associated with creativity.

COMT, an enzyme responsible for degrading catecholamines, including DA, has been implicated in various cognitive processes. The Val158Met polymorphism of the COMT gene appears to exert particular influence over the availability of DA in areas of the frontal cortex. The Val allele of the Val158Met polymorphism allows greater expression of COMT in the prefrontal areas, thus...
diminishing the availability of DA, while the Met allele increases availability of prefrontal extracellular DA. The Val+ alleles (including both the Val/Val and the Val/Met variants) have been linked to risk for schizophrenia,\textsuperscript{80,81} while the Val– allele (Met/Met) has been associated with higher IQ, working memory, and cognitive flexibility.\textsuperscript{81–83} Variants of the COMT gene may, therefore, distinguish the person with psychosis from the poet, with the Val+ variants conferring risk for psychopathology and the VAL– variant acting as a protective factor that enhances creativity.

The link between 5-HT availability, mood, psychosis, and altered states of consciousness is well-established.\textsuperscript{84} Variations in 3 genes involved in 5-HT availability, 5-HTR2A, SLC6A4, and TPH1, may also confer shared vulnerability among psychiatric disorders and creativity, or confer protective factors that favour creativity. The T102C polymorphism of the 5-HTR2A gene has been linked to schizotypy and risk for schizophrenia, although which alleles confer this risk have been disputed.\textsuperscript{85} The T/T allele of T102C has been associated with higher levels of absorption, a measure of the propensity to experience altered states of consciousness.\textsuperscript{86} Because absorption is associated with creativity,\textsuperscript{87} variants of the T102C polymorphism may establish either a genetic link or a genetic distinction between creativity and psychotic risk. Brang and Ramachandran\textsuperscript{88} have identified 5-HTR2A as the gene which may underlie the expression of synesthesia, further connecting this gene location to the shared vulnerability model, although these researchers have not identified a specific polymorphism.

SLC6A4 (also denoted as 5HTT) has been widely studied in relation to psychiatric disorders. The short allele of the 5HTTLPR promoter region of the SLC6A4 gene has been associated with several constructs related to creativity, including the personality traits openness to experience\textsuperscript{89,90} and absorption,\textsuperscript{91} as well as creative dance performance.\textsuperscript{92} SLC6A4 regulates the concentration of 5-HT in the synapses, with the short allele of the 5HTTLPR region apparently reducing the reuptake of 5-HT. Because alteration in levels of 5-HT are implicated in hallucinogenic and unusual perceptual experiences, it is possible that variants in this gene are also related to a propensity for altered states of consciousness.\textsuperscript{93} Studies have also linked the short allele of the 5HTTLPR polymorphism with risk for BD.\textsuperscript{92–94} The short variant of this polymorphism, therefore, may be important in both creative ideation and psychopathology associated with creativity.

The A779C polymorphism of the TPH1 (the enzyme that regulates levels of 5-HT) gene has been linked to risk for schizophrenia,\textsuperscript{95} suicide,\textsuperscript{96} and smoking addiction.\textsuperscript{97} Reuter et al\textsuperscript{92} found that carriers of the A allele of A779C polymorphism scored higher on measures of creativity, especially figural and mathematical creativity, than carriers of the C allele in a large group of university students.

Finally, the T/T genotype of a functional promoter polymorphism of the NRG1 gene (involved with neuronal development, glutamate transmission, and glial cell functioning) has been linked to an increased risk for psychosis as well as decreased activation of the frontal lobe during cognitive tasks.\textsuperscript{98} This genotype has also recently been found to characterize highly creative people within a high IQ sample.\textsuperscript{99} Although NRG1 is not directly associated with DA or 5-HT transmission, it may be another link in the shared genetic vulnerability of creativity and mental disorder.

Again, these examples of genetic variation and their possible roles in creative ideation must be considered speculative at this time. Further, many genes and their polymorphisms have been implicated in the risk for serious psychiatric disorders. The variants of the genes mentioned above, most of which act on the neurotransmitters DA and 5-HT, comprise only a small segment of this risk. Nevertheless, these neurochemicals may be important in mediating cognitive states associated with creative cognition, as well as psychosis, mood disorders, and addiction. However, individual genes do not confer creativity, psychosis, or addiction upon a person. The complex interactions of multiple genes, with each other and with the person’s environment, are important in determining a tendency toward either creativity or toward psychopathology. Neuroscience, psychology, psychiatry, and molecular biology all have a role to play in our growing understanding of these interactions.

Conclusions

Highly creative people are at greater risk for certain types of psychopathology, including mood disorders, SSDs, and alcoholism, than are members of the general population. However, creativity is also a valued trait associated with positive, and sometimes exceptionally positive, personal characteristics. Although these 2 sets of descriptions appear at odds with one another, a shared genetic vulnerability model of the relation between creativity and psychopathology can account for the paradox.

Creative cognition may share common biological vulnerabilities with psychopathologies that grant access to altered states of consciousness. These vulnerabilities may include a tendency toward transient cognitive disinhibition, which appears to be, to date, associated with variation primarily in various DA- and 5-HT–related genes. Cognitive strengths, such as high IQ, good working memory capacity, and cognitive flexibility, may interact with these vulnerabilities to enhance creativity and to act as protective factors against severe forms of the relevant psychopathologies. The shared vulnerability model currently includes only factors for which there is some corroborating support from molecular biology. However, there are likely additional shared vulnerabilities and protective factors that warrant inclusion. Future research will extend this model.
Implications for Treatment

The shared genetic vulnerability model of creativity and psychopathology suggests that psychopathology outcomes may be reduced by one or more of the following: treating symptoms associated with vulnerability factors; enhancing protective factors associated with creativity; or, enhancing overall creativity. Because highly creative people may rely on the cognitive manifestations of shared vulnerability to access altered states of consciousness that inform their work, they may be better served by treatment goals that aim for partial rather than complete neutralization of psychopathology symptoms. Creative people may prefer to tolerate higher levels of symptomatology in exchange for lower dosages of creativity-killing pharmaceuticals. They may also respond well to cognitive-behavioural interventions that target the interpretation of psychotic symptoms rather than the removal of such symptoms (see O’Connor70 for a discussion of the reappraisal of psychotic symptoms).

People with mood disorders, SSDs, and addiction disorders who have not displayed creative propensities may benefit from the addition of one of the many creative therapies, such as art, creative writing, drama, or music therapy, to their treatment regimen. Because several factors that may predispose a person to these types of psychopathology may also predispose them to creative modes of thought, redirecting patient interest into creative fields may help them to find a voice for their suffering and provide productive activity. With good fortune, such interventions may help protect them against the demons of psychopathology while enhancing their access to the muse.

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References


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Résumé: Créativité et psychopathologie: un modèle de vulnérabilité partagée

La créativité est considérée comme un trait personnel positif. Cependant, les gens très créateurs ont démontré un risque élevé de certaines formes de psychopathologie, notamment les troubles de l’humeur, les troubles du spectre de la schizophrénie, et l’alcoolisme. Un modèle de vulnérabilité partagée explique la relation entre la créativité et la psychopathologie. Ce modèle, soutenu par de récents résultats des neurosciences et de la génétique moléculaire, suggère que les déterminants biologiques porteurs d’un risque de psychopathologies interagissent avec des facteurs cognitifs protecteurs afin d’améliorer l’idéation créatrice. Les éléments de la vulnérabilité partagée sont notamment la désinhibition cognitive (qui permet plus de stimu à l’éveil conscient), un style d’attention mû par le caractère distinct de la nouveauté, et une hyperconnectivité neurale qui peut accroître les associations parmi des stimuli disparates. Ces vulnérabilités interagissent avec des facteurs protecteurs métacognitifs supérieurs, comme un QI élevé, une capacité accrue de la mémoire de travail, et une flexibilité mentale améliorée, afin d’élargir la gamme et la profondeur des stimuli que l’éveil conscient peut manipuler et combiner pour former des idées nouvelles et originales.