6-1-2013

Schizophrenia: Causes, Crime, and Implications for Criminology and Criminal Justice

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Abstract

This paper is aimed at criminologists and criminal justicians seeking to understand their role in educating law enforcement and correctional personnel who must deal with the mentally ill. It is motivated by William Johnson's (2011) recent call for rethinking the interface between mental illness, criminal justice, and academia, and his call for advocacy. We concur with his concerns, and insist that this rethinking must necessarily include grounding in the etiology of mental illness (specifically, with schizophrenia) as it is currently understood by researchers in the area. Advocacy must go hand in hand with a thorough knowledge of the condition of the people for whom we are advocating. We first examine major etiological models of schizophrenia, emphasizing the neurodevelopmental model that incorporates genetics, neurological functioning, and immunological factors guided by the assumption that the typical criminologist/criminal justician has minimal acquaintance with such material. We then address the link between schizophrenia and criminal behavior, and conclude with a discussion of the implications for criminology and criminal justice.

Key Words: Schizophrenia. Dopamine. Epigenetics. The two-hit neurodevelopmental model. Comorbidity.

1.1 Introduction

William Johnson (2011, p. 21) recently highlighted the troubling nature of mental illness, particularly schizophrenia, the relative lack of concern for individuals suffering from it, and urged us to "rethink the interface between mental illness, criminal justice, and academia." He indicates that because of the "cult of curability" (the optimistic idea that any human malady can be cured) and the deinstitutionalization movement that followed it, the largest providers of psychiatric care in the United States are jails in New York, Chicago, and Los Angeles. Because jails have become surrogate psychiatric hospitals, Johnson pleads for a greater understanding of mental illness within criminal justice system, and offered proposals about what we in criminology/criminal justice can do to help deal humanely with those who suffer these afflictions. As academic disciplines educating future practitioners who will have to deal with the mentally ill, and as scholars who will have to explain it to them, we need to gain a deeper understanding of it as it is currently understood by disciplines most engaged in that line of research.

Fisher, Silver and Wolff (2006, p.11) call for a "criminological–informed" framework for mental health services because mental health workers of often not aware of the risks for criminal behavior that do not involve aspects of the individuals' mental health. Likewise, we call for a "mental health" informed criminology and criminal justice because criminal justice workers and their mentors are often not aware of the risks for criminal behavior that are not a part of the individuals' environmental circumstance alone. This paper is thus an effort to move criminology and criminal justice in the direction a better understanding of mental health. There are many mental disorders but we concentrate on schizophrenia because it is the illness most strongly associated with criminal behavior (Fazel et al., 2009).

2.1 Schizophrenia and its Symptoms

Schizophrenia is the most widespread of the psychotic disorders for which there is an estimated lifetime prevalence of 0.30-0.60% (van Os & Kapur, 2009). It a progressive disorder with symptoms that may be relatively mild and often unnoticed by others when they first appear. For some sufferers symptoms may be episodic and may improve with age. Many subtle manifestations of the disease such as motor and attentional problems, as well as social difficulties, are noted in the childhood behavior of many individuals who are later diagnosed with schizophrenia (Fatemi and Folsom, 2009). It affects males and females about equally, but has an earlier age of onset in males (around mid-adolescence versus late 20s – early 30s for females). As a rule, the earlier the onset of the disease the more severe the impairments, and like other psychiatric disorders that emerge during adolescence, probably reflects aberrations in the maturational changes that normally take place in the brain during that period (Paus, Keshavan, & Giedd, 2008). Schizophrenia comes in a variety of subtypes that are categorized according to the symptoms manifested. Some individuals are unkempt, illogical, and frenetic (disorganized), some are rigid, unresponsive, slow moving and sometimes totally immobile (catatonic), and some are hostile and distrusting (paranoid). It is the latter type that is most prone to overt physical aggression (Volavka & Citrome, 2011). We need to emphasize that people with schizophrenia are more like ourselves than different for us, and that while there is no cure for schizophrenia, the symptoms are manageable, and with the proper medication and care people with schizophrenia can lead productive lives (Cannon, 2009).

Schizophrenia symptoms are both positive and negative. Positive symptoms include delusions (beliefs with no basis in reality), thought disorders (using nonsense words and sentences), hallucinations (hearing, seeing and smelling things that are not there) and movement disorders (slow movements and repetitious gestures). Negative symptoms include anhedonia (the inability to experience pleasurable emotions), low energy levels, social withdrawal, and poor hygiene. The severity of these symptoms varies widely across individuals, which is why schizophrenia is seen as a spectrum disorder rather than a discrete disease which one either has or has not (Garrett, 2009).

The mental life of persons with schizophrenia is so far removed from the everyday experience that it requires an analogy to obtain a subjective understanding of it. We have all experienced vivid scary dreams from which it is a relief to awaken. Once awake, we are aware that we were dreaming, and we begin to respond normally to stimuli outside our private worlds. When dreaming our neurons are making random connections ("noise") unrelated to any external stimuli. During sleep there are no perceptions of external stimuli, so the brain confabulates in an attempt to generate order from noise input by drawing on past experiences stored in its memory banks (Garrett, 2009). The brains of people with schizophrenia seem to engage in the same sort of haphazard synaptic connections when awake that brains not afflicted with schizophrenia brains do in the dream state. With all this disconnected neural activity buzzing around in their heads persons suffering from schizophrenia experience delusions and hallucinations, and as a consequence have difficulties filtering information and focusing their attention on real environmental stimuli (Dixon, 2005).

2.2 Genetics, Epigenetics, and Schizophrenia

The complex multi-determined nature of schizophrenia has spawned many different etiological models. Some models emphasize distal causes (e.g., genetics, viruses, fetal teratogenic insults) and others more proximate mechanisms such as aberrant neural signaling. These models are non-mutually exclusive and reinforce one another while recognizing that any one nominated model does not explain all cases. It is not clear if any of the proposed causes are necessary and/or sufficient to account for the syndrome; it is most likely that all the putative causes aggregate and interact to bring about the onset of the disease (Cannon, 2009). One thing that we have known for a long time, however, is that genetics play a large part in the development of schizophrenia. Behavior genetic studies of schizophrenia show an average heritability across many studies of 0.80 (Keshavan, Nasrallah & Tandon, 2011). Although this is indicative of a large genetic effect, given that monozygotic (MZ) twins share 100% of their genes and (most generally) 100% of their rearing environment, one would expect to see a higher concordance rate. Because we do not, something other than raw DNA and shared environment must be playing a significant role. Concordance rates for schizophrenia are almost identical for MZ twins regardless of whether they are reared together or apart, which suggests that any environmental influences occur prenatally or perinatally.

Because of the lower than expected concordance rate for schizophrenia in MZ twins, and because the search for specific genes predisposing a person to schizophrenia has not been productive, the search has shifted to looking at epigenetic processes. A broad definition of epigenetics is "any process that alters gene activity without changing the DNA sequence" (Weinhold, 2006:163). Epigenetic modifications affect the ability of the DNA code to be read and translated into proteins by making the code either more or less accessible (Gottleib, 2007). Epigenetic regulation is accomplished by two main processes: DNA methylation and histone acetylation. DNA methylation occurs when a group of atoms called a methyl group are attached to a cytosine base (one of the four "letter" bases, ATCG) which prevents the translation of DNA into messenger RNA (mRNA), and hence the protein the gene codes for is not manufactured (Corwin, 2004). Histone acetylation involves acetyl group of atoms attached to histones, which are the protein cores around which DNA is tightly wound. This has the effect of "loosening" or "relaxing" the DNA making the code easier to read, thus increasing the likelihood of genetic expression (Lopez-Rangel & Lewis, 2006). To apply criminal justice metaphors to these processes, acetylation is a mechanism that aids and abets gene expression and methylation arrests it. Methylation can produce stable, even permanent changes in genetic functioning, but acetylation is labile and reversible (Powledge, 2009).

One study of phenotypic discordance for a number of traits among healthy MZ twins found that as twins got older they diverged epigenetically, with 50 year-old twin pairs averaging four times the epigenetic differences than 3 year-old twin pairs, indicating that epigenetic alterations occur and accumulate throughout life (Fraga, et al., 2005). Kaminsky et al. (2009) examined methylation patterns in MZ and dizygotic (DZ) twins and found discordance in both groups, although there was significantly greater discordance among DZ twins, suggesting that the DNA sequences themselves can affect methylation patterns. *In vivo* and postmortem studies of brain tissue have demonstrated that a number of genes associated with schizophrenia are particularly susceptible to DNA methylation and acetylation (Gavin & Sharma, 2010).

Wong and his colleague's (2010) longitudinal study concentrated on epigenetic modification of genes most often hypothesized to operate at the interface of nature and nurture, such as dopamine receptor genes, serotonin transporter genes, and enzymes such as MAOA that degrade neurotransmitters. Wong et al, (2010) found a number of interesting methylation patterns over a five-year period, with both MZ and DZ co-twins diverging. Most interestingly, they found that while gene expression was changing with age, MZ twins were changing in the *same* direction but DZ twins were diverging in the *opposite* direction. This phenomenon may partially account for the consistent finding that the correlations between pairs of MZ twins across a variety of traits (e.g., IQ and various personality traits) remain quite stable with age while the correlations between DZ pairs fall rather dramatically (Beaver, 2009; Moffitt, 2005). That divergence occurs at all in MZ twins reared in the same home indicates also that many epigenetic events are stochastic (Feinberg & Irizarry, 2010).

There is a consensus among schizophrenia researchers that although no single genetic polymorphism or set of polymorphisms have been identified as either necessary or sufficient to cause schizophrenia, the condition cannot occur without genetic vulnerability (Fatemi & Folsom, 2009).[1] Polymorphisms of 9 genes have been repeatedly associated with schizophrenia, but frustratingly no single gene has been replicated in every study, which underlines the causal heterogeneity of the condition (Faremi & Folsom, 2009). The first plausible etiological model of schizophrenia, and still most robustly supported, is the dopamine (DA) hypothesis.

Over the past three or four decades it has been convincingly shown that persons with schizophrenia show abnormalities of prefrontal cortex (PFC) functioning, namely a disturbance of the signal-to-noise ratio (S:N) during information processing. Signaling is the inter-neuronal communication of meaningful information; noise is the random (nonsense) firing of neurons as in dreaming. If the S:N is beyond normal ranges, communication between brain regions is interrupted, leading generally to aberrant responses. Dopamine signaling in the PFC is a critical factor in modulating the S:N, and consequently in how persons interpret synaptic activity. Variation in DA signaling is mediated by the enzyme catechol-O-methyltransferase (COMT) that degrades DA (see note 1). Because DA has a significant effect on PFC signal-to-noise ratios, the COMT gene is a susceptibility gene for schizophrenia (Winterer & Weinberger, 2003). Differences in methylation patterns between MZ twins who are discordant for schizophrenia are evident in the COMT gene and the dopamine D2 receptor gene (Rutten & Mill, 2009). Further strengthening the dopamine hypothesis is the fact that DA antagonist drugs work in alleviating positive symptoms (delusions, hallucinations, movement disorders) in most brain of people with schizophrenia by blockading D2 receptors, and that the effectiveness of different drugs is directly related to how well they block DA (Garrett, 2009).

However, DA antagonists fail to work in about one-third of cases, even though these cases receive as much DA blockage as those for whom the drug works (Garrett, 2009). This suggested that more than DA is at work to produce the aberrant S:N. Glutaminergic functioning became a major research focus after it was found that the drug PCP (phencyclidine) caused schizophrenic-like symptoms by inhibiting the glutamate receptor N-methyl-D-aspartate (NMDA) (Keshevan, Nasrallah & Tandon, 2011). Because neurotransmitter systems interact, changes in one system are expected to result in changes in another. It is thus not surprising that neurotransmitters other than DA also play a role. Furthermore, because glutamate is by far the most common of all neurotransmitters and plays a pivotal role in neural communication, and because NMDA receptors play a key role in the development of neural pathways and in pruning neural connections, glutamate has to play a large part in the schizophrenic condition (Javitt et al, 2008).

Administering glutamate agonists (drugs that bind to receptors and mimic the action of glutamate) helps to relieve both positive and negative symptoms whereas DA blockers only alleviate positive symptoms. Positron emission tomography (PET) studies show that positive symptoms are associated with *hyper*stimulation of D2 receptors, hence DA antagonists will alleviate positive symptoms. On the other hand, negative symptoms are associated with *hypo*stimulation of D1 receptors. Dopamine functioning thus results in different symptoms depending on which receptors it activates in what areas of the brain (Patel et al, 2010). Further evidence of the pivotal role of glutamate is found in the fact that about 75% of persons with schizophrenia smoke (and usually cigarettes with high nicotine content) as opposed to about 23% of the general population. This is seen as an effort at self-medication because nicotine increases glutamate release, as well as increasing DA in the dorsolateral PFC where DA is depleted in schizophrenia sufferers (Garrett, 2009). The lower DA activity in this frontal area is known as hypofrontality and can be seen in neuroimaging and assessed by attention tasks on which people with schizophrenia do very poorly, such as the Wisconsin Card Sorting Test (WCST). The WCST measures the ability of a subject to display neural flexibility in the face of rule changes over the course of the testing period. As the rules change the subject patient must adjust, with test scores based on the mistakes made.

Other neurotransmitters and receptors have also been implicated to one degree or another in accounting for schizophrenic symptoms, particularly the GABAergic system. Gamma amino butyric acid (GABA) is the major inhibitory neurotransmitter; i.e., prevents over excitement of the synapses and is a major factor in reducing anxiety. Too little GABA and the excitatory neurotransmitters run wild and the person suffers

from high anxiety. The promiscuous multiplication of all these putative causal factors is frustrating to those who want definitive answers. Noting that neural circuitry and neurotransmitter systems are fully integrated and that focusing on one transmitter system alone is misguided, Benes (2009:1004) writes that: "If one asks whether it is dopamine, GABA, or glutamate that is responsible for disturbances in cortical information processing in schizophrenia, the rational response should be 'All of the above'!"

3.1 The One-Two Hit in Schizophrenia: The Neurodevelopment Model

The neurodevelopmental model unites genetics, epigenetics, immunology, and the two critical periods of brain development (prenatal/perinatal and adolescence) in a comprehensive attempt to show how these many factors combine to produce the symptoms associated with schizophrenia. The model asserts that schizophrenia results from abnormal in utero brain development due to any number of factors; this is hit number 1. Hit number 2 comes when the brain is being remolded into its eventual adult form during adolescence. Fatemi and Folsom (2009, p. 528) write: "According to this model, early developmental insults may lead to dysfunction of specific neural networks that would account for premorbid signs and symptoms observed in individuals that later develop schizophrenia. At adolescence, excessive elimination of synapses and loss of plasticity may account for the emergence of symptoms."

The fact that premorbid behavioral abnormalities could be present years before schizophrenic psychosis develops meant that there had to have been a "first hit" of some kind, and the transition from childhood premorbid features to overt psychosis occurring around adolescence meant that there also had to be a "second hit." The first hit that "primes" the psychotic pump is most likely to occur during embryonic development. There are a number of environmental candidates for delivering the first hit. For instance, there is a 10% increase in the risk of developing schizophrenia among children born during January through March in the northern hemisphere, a time when many viral infections are present (Krause et al, 2010). Mednick, Machon and Huttunen (1988) documented that the risk for schizophrenia increased 50% for individuals whose mothers were exposed during the second trimester to a virulent type of influenza during the 1957 epidemic in Finland. Another study found a 10 to 20-fold increase in risk for schizophrenia in a New York City cohort who were born to mothers who contracted rubella during pregnancy (Brown, 2006).

Because a wide variety of infectious agents have been linked to schizophrenia, a good deal of recent research has focused on the immune response (Meyer & Feldon, 2009). When the immune system detects a foreign substance in the host body it mobilizes a horde of specialized cells to launch an attack on it. These cells release a class of proteins called cytokines that carry signals from cell to cell near the location of the antigen (the foreign body) and alter cell functioning to initiate the immune response. The overall effect of this immune process is to trigger inflammation. If a pregnant woman contracts an infectious disease or ingests noxious substances, her immune system will release cytokines that can enter fetal circulation. The importance of this observation is that cytokines play a role in the proliferation, differentiation, and survival of neurons and they also influence neurotransmission by modulating neuronal and glial cell functioning. As Depino (2006, p. 7777) explains: "cytokines released by the maternal immune system (and/or the placental or fetal immune system) in response to infection may be responsible for the interaction between maternal infection during pregnancy, altered neuronal development, and mental diseases."

Muller and Dursun (2010) note that many genes of interest to schizophrenia researchers are related to immune system functioning, further indicating that a dysfunctional immune response plays a role in priming the schizophrenia pump. Muller and Dursun (2010, p. 1) further note that many studies of immunological response in schizophrenics have shown "a blunted activation of type-1 immune response and a reciprocal overactivation of the type-2 response (in certain cases switching to an autoimmune response)." Type-1 and type-2 immune responses are hypersensitivity responses; type-1 being largely associated with allergies, and type-2 being a delayed response. Type-2 hypersensitivity results in antibodies which identify and attack antigens on the hosts own cells and destroy them, but they also

destroy the host cell as well (the autoimmune response). Of course, numerous individuals have been exposed during gestation to pathogens that initiated the same immune response without developing lasting effects. Only those unfortunate few with a genetic vulnerability will go on to develop schizophrenia.

The mechanisms of the second hit are more speculative and more complex. One theory posits that infections acquired during gestation and associated with neuro- inflammatory processes lie dormant (or semi-dormant in cases where premorbid symptoms are in evidence) until puberty when immune surveillance is weakened (Muller & Schwarz, 2006). The immune system can keep the latent problem in abeyance until puberty when the thymus gland starts to undergo a major reduction in tissue volume and function (Kinney et al, 2010). The thymus gland is essential to the maintenance of the immune response. It increases rapidly in size from infancy to puberty, slowly building up the body's stock of T-cells (thymus lymphocytes—white blood cells originating in the bone marrow) which are vital to immunity. After reaching its maximum size at puberty, the thymus decreases by about 3% each year to become almost indistinguishable from surrounding tissue in old age (Barton et al., 2000).

The second aspect of the second hit is exaggerated synaptic pruning. Synaptic pruning occurs with a vengeance during adolescence and is a normal part of brain maturation that refines neuronal connectivity and function (Steinberg, 2005). When pruning goes beyond the norm, however, it results in the kinds of grey matter loss observed in the brains of persons with schizophrenia brains (Rosenthal, 2011). Thus, through a tangle of intricate chemical activity, the processes initiated by the first hit combine with excess synapse losses in adolescence and the reduction in thymus structure and function to produce the symptoms of schizophrenia. This combination initiates or contributes to the dysregulation of the neurotransmitter systems that cause the aberrant signal-to-noise ratio of schizophrenia. According to Kinney and his colleagues (2010, p. 555), the neurodevelopmental theory of abnormal immune system response, "may help explain roles of prenatal hazards, post-pubertal onset, stress, genes, climate, infections, and brain dysfunction." This comprehensive model may have finally provided a unified theory of schizophrenia that ties all of its many correlates together in one bundle.

3.2 Schizotypy: Subclinical Schizophrenia

Schizotypy is part of the schizophrenia spectrum and is a subclinical manifestation of many of the same underlying factors that produce full-blown schizophrenia. Even while not displaying clinical-level signs of the disorder, we would expect close relatives (particularly a MZ co-twin) of individuals with full-blown schizophrenia to harbor a greater genetic risk for schizophrenia than people in general, and they do. These individuals have a life-time vulnerability to developing full-blown schizophrenia, with the odds of a first-degree relative being diagnosed with the disorder being 10 times greater than the odds of someone in the general population being diagnosed (McDonald et al. 2009).

Because symptoms are not sufficient to seriously disrupt their lives, schizotypal individuals are usually identified by the Schizotypal Personality Questionnaire (SPQ). High scorers on the SPQ evidence sub-psychotic signs indentified by behavioral, psychometric and neuroimaging techniques. High scorers on the SPQ were twice as likely to smoke as control subjects (Stewart, Cohen, and Copeland, 2010). Another study using the SPQ found that high schizotypy was related to reduced empathy, increased negative affect, and poorer social functioning (Henry, Bailey, & Rendell, 2008). On the other hand, Nettle and Clegg (2005) found individuals high on schizotypy to be more creative than controls.

Brain imaging studies also show schizotypal individuals to have neural profiles intermediate between persons with full-blown schizophrenia and controls. A review of 20 fMRI studies of nonpsychotic relatives of schizophrenia patients matched with controls showed consistent activational differences (increases or decreases in activation) between the groups in the same brain areas associated with schizophrenia. The most consistent finding across the 20 studies was increased activity in the right ventral PFC, followed by increased activity in right parietal cortex. In studies that included increases or

decreases from either brain hemisphere, the most consistently findings were impairments of the cerebellum, dorsal prefrontal, lateral temporal and parietal cortices, and thalamus. The authors concluded that the overall lesson of the 20 studies: "suggests a very broad impact of liability genes, consistent with findings in the illness itself" (McDonald et al, 2009, 1159).

A meta-analysis of 25 studies imaging brain volume comparing 1065 nonpsychotic first-degree relatives of persons with schizophrenic with controls also found significant differences (Boos et al., 2007). The largest effect (d = .31) found was reduction in hippocampal volume among the first-degree relatives, followed by smaller third ventrical (d = .21) and gray matter (d =.18) volume. Taken together, all lines of evidence indicate that nonpsychotic first degree relatives share the genetic risk for developing full-blown schizophrenia but have somehow managed to avoid clinical-level symptoms. However, many do manifest behavior, personality, and neural anatomy and physiology profiles that suggest they are mildly affected.

4.1 Schizophrenia and Criminal Behavior

According to Elbogen and Johnson (2009) after decades of denying that there was any link between mental illness and crime, the psychiatric community has reversed its stance. Prior to the period of deinstitutionalization, researchers found little or no link between mental illness and crime because seriously afflicted individuals were routinely institutionalized, sometimes for life (Ditton, 1999). As noted by Johnson (2011), the deinstitutionalization movement returned many such persons back to the community, resulting in greater visibility and higher arrest rates. There is still some reluctance to affirm the link between crime and schizophrenia out of fear of further stigmatizing an already highly stigmatized group, but the evidence cannot be dismissed or ignored (Elbogen & Johnson, 2009).

A review of 47 studies of the schizophrenia/criminal behavior relationship found 42 to be positive, 3 non-significant, and 2 were negative (Ellis and Walsh 2000). Researchers in Denmark looking at more than 300,000 individuals followed to age forty-three found that persons with histories of psychiatric hospitalization (90% schizophrenic) were three to eleven times more likely to have criminal convictions than people with no psychiatric history (Hodkins et al. 1996). A Swedish study reported that people with psychosis are about four times more likely to have a criminal record than members of the general population (Tuninger et al. 2001). Finally, a meta-analysis of 20 studies including over 18,000 subjects found that the odds ratios for risk of violence among persons with schizophrenia compared to the general population ranged between 1 and 7 for men and 4 and 29 for women (Fazel et al., 2009). When calculated for persons with schizophrenia comorbid for substance abuse disorders, the odds ratios increased from between 3 and 25. In five studies in the meta-analysis that compared persons with schizophrenia to the general population, the pooled odds ratio for homicide was19.5. The combined weight of numerous studies leads to the conclusion that the risk for committing a violent crime for persons with schizophrenia is considerably greater than that of the population at large. However, it is important to emphasize that psychosis-driven violence is still a very small part of society's violence problem (Taylor, 2008).

Researchers debate the degree to which schizophrenia per se is responsible for violent behavior. Elbogen and Johnson (2009, p. 159) assert that it is "simplistic as well as inaccurate to say the cause of violence among mentally ill individuals is the mental illness itself." Researchers of this persuasion attribute violence committed by the mentally ill to be the result of comorbid factors such as substance abuse, antisocial personality disorder, and homelessness, and noncompliance with medical regimens. Others maintain that a modest but significant relationship exists between violence and mental illness independent of these factors (Van Dorn, Volavka & Johnson, 2011). Whatever the case may be, lifetime substance abuse/dependence among persons with schizophrenia is estimated to be at 50% versus 15% of the general population (Mueser et al., 2013). As is the case with cigarette smoking, the abuse of alcohol and drugs may be an attempt to self-medicate. Attempts at self-medication may then lead to dependence, which then puts persons with schizophrenia at even greater risk for offending.

Nevertheless, the majority people with schizophrenia are nonviolent and are more likely to be victims of violence than perpetrators (Taylor, 2008). Those most at risk for victimization are also those most at risk for committing violence. A meta-analysis of 9 studies of individuals with schizophrenia or some other psychosis found that they were 2.3 to 140.4 times more likely to be victimized than the non-mentally ill in the general population. The large odds ratio differences among the various studies were attributable to variance in the severity of psychotic symptoms, criminal activity and substance abuse among subjects, and whether or not they are homeless (Maniglio, 2009).

The realization that individuals with schizophrenia are more likely to be sinned against than to sin, has led to attempts to differentiate between thoise who are and who are no antisocial along a number of dimensions. Schug and Raine (2009) conducted a meta-analysis of 43 studies that compared neurophysiological performance (a total of 98 different neurological assessment tools were reported across the 43 studies) among antisocial and non-antisocial people with schizophrenia and non-schizophrenic antisocial individuals. Antisocial persons with schizophrenia demonstrated numerous deficits across many domains of cognitive functioning compared to antisocial individuals without schizophrenia. However, in comparison to non-antisocial people with schizophrenia, antisocial persons with schizophrenia only demonstrated deficits in general intellectual functioning and memory dysfunction. The authors suggest that their findings imply that orbital frontal prefrontal cortex functioning is a common denominator in schizophrenia and antisocial behavior and that dorsolateral prefrontal cortex functioning differentiates antisocial persons with schizophrenia from non-schizophrenic antisocial subjects.

Further adding to our knowledge of the heterogeneous nature of offending by people with schizophrenia is the life course typology developed by Sheilagh Hodgins (2008). Using data from around the world, Hodgins identifies three distinct types of offenders defined by the age of onset of antisocial behavior that is reminiscent of Moffitt's (Moffitt et al., 2001) typology of life course persistent (LCP), adolescent limited (AL), and late-onset (LO) offenders. Similar to LCP offenders, Hodgins' Type I offenders begin offending in childhood prior to the onset of the identifiable illness, and offending remains stable across the life course. Type II offenders are the largest group of schizophrenic offenders. As with Moffitt's AL offenders, Type IIs do not evidence antisocial behavior until adolescence, which typically coincides with the onset of the illness. Unlike AL offenders who desist with age, however, Type II offenders continue to offend across the life course. A small group of Type III offenders are those who display no violence for two or three decades after disease onset, but then suddenly engage in some serious act of violence, which may involve killing a caregiver. Hodgins' typology covers only persons with schizophrenia who engage in violent behavior, not those who do not.

4.2 Implications for Criminal Justice and Criminology

We have premised the preceding discussion on the notion that one should have at least a rudimentary understanding of what one has to work with. While we agree with Johnson (2011, p. 21) that it is time for criminologists and criminal justicians to "come out of the academic closet" and get involved in advocacy, we also believe that we should be able to provide our students with a basic understanding of the causal processes involved with the schizophrenia spectrum, and not just with a discussion of criminal justice practices related to it. Advocacy is taken most seriously when advocates know what they are talking about. The burden of advocacy has been placed upon us by the de facto criminalization of many forms of mental illness following the deinstitutionalization movement. Police, probation/parole, and correctional officers must come to understand mental illnesses associated with increased vulnerability to crime as diseases for which the individual is no more responsible than is the cancer patient (perhaps even less so in some cases of cancer). This understanding should lead to the destigmatization of mental illness among criminologists, criminal justicians, and the criminal justice professionals they educate, as well as a more benevolent attitude toward those who suffer from it.

While there is limited evidence that members of the general public are less likely to blame, be angry with, or socially avoid the mentally ill after they are educated about its biological origins (Corrigan and Watson, 2004), there is also evidence that focusing on a biological etiology can actually be more stigmatizing (Mehta and Farina, 1997; Read & Law, 1999). Studies of this kind tend to find weak results from university undergraduates, who may well share the general public's (and even that of many academics) belief that biology is destiny, and so more powerful than putative psychosocial causes. According to Mehta and Farina (1997, p. 416) "The disease view may incline us toward an awareness that we ought to have generous feelings toward the afflicted but this view may also induce us to view those with mental disorders as set apart from the rest of humanity." This is an unfortunate social construction based on little or no knowledge of the causes of schizophrenia, and we are less concerned with public attitudes than we are with informing criminologists and criminal justicians. Our point is that a more complete understanding of the part of people (criminologists and criminal justicians) charged with educating the criminal justice system's practitioners can go a long way to assuaging this stigmatizing construction and lead to better methods and practices in their dealings with persons with schizophrenia.

The police are the first responders to public appeals for assistance, and sometimes mentally ill individuals are involved as either victims or perpetrators. As former law enforcement officers, both present authors have been involved in confrontations with offenders suffering from schizophrenia and consider it one of the most difficult tasks officers have to face, a position shared by most serving officers (Lamb, Weinberger & DeCuir, 2002). With the unpredictable and possibly violent nature of calls involving the mentally ill, police department have initiated programs aimed at minimizing the danger to both officers and mentally ill persons. Ideally, officers should be able to control the situation without resorting to violent means, and to resolve a minor situation without having to arrest a mentally ill suspect or transport him or her to a mental health facility. This is more like to occur if officers have a better understanding of schizophrenia and can allay their fears generated by the often bizarre behavior exhibited by person with schizophrenia. Officers must possess an understanding of schizophrenia so that they realize that individuals suffering from schizophrenia may not readily understand or comply with police commands, or be able to communicate details of an offense where they are victims rather than perpetrators.

The typical police response model to mental illness calls in the United State is the Crisis Intervention Team (CIT). This involves police officers trained in the causes, signs, and symptoms of mental illness and in crisis intervention and de-escalation skills. These officers are charged with forging strong relationships with community mental health agencies so that they are aware of what resources are available to the mentally ill (Watson, et al., 2008). A study of CIT trained and non-CIT trained police officers found that CIT trained officers less likely to use physical force than non-CIT trained officers across a variety of scenarios involving psychotic offenders (Compton et al., 2009). CIT officers are not necessarily the first on the scene, which is why all officers should have some training in dealing effectively and humanely with the mentally ill.

Another model which is more popular in Canada than the US is the Mobile Crisis Team (MCT). This model has mental health professionals (nurse, social worker) respond with police officers to mental health calls. In such cases, mental health professionals can carry the heavy load of crisis intervention with the protection of a police officer on the scene. This model can result in a more accurate assessment of mentally ill individuals as well as suggesting to most appropriate police response (Cotton & Coleman, 2010). Both models mean that additional resources have to be allocated to policing the mentally ill so that it can be accomplished more humanely while still recognizing that the primary focus of police work is the safety of the community and of responding officers.

It is perhaps even more important for probation/parole officers to be trained in mental health issues because they are in more frequent contact with the same individuals (their probationers and parolees) than are police officers. Supervising persons with schizophrenia is disconcerting to even experience probation-parole officers not trained in mental health issues (Skeem & Louden, 2006). Most mentally ill offenders on probation or parole have mandatory treatment orders, and although treatment is primarily a medical concern, correctional worker must be involved as community resource brokers and medication monitors. Most clients with schizophrenia are manageable and cooperative while on medication; the difficulty is

making sure that they take it, because non-compliance with medical regimens is the norm rather than the exception (Patterson & Leeuwenkamp, 2008). The reluctance to take antipsychotic drugs is understandable given their significant side effects, and officers who gain and understanding of these effects may respond to clients more compassionately and usefully about non-compliance issues. This reluctance to take medications may be circumvented by negotiating an agreement with offenders that they be treated with the long acting drug risperidone. This drug is injected every two or three weeks, thus alleviating the "forgetting" problem, with the medication gradually released over that time (Buckley 2004).

However, antipsychotic drugs no more cures schizophrenia than insulin cures diabetes. Antipsychotic drugs do for schizophrenia patients does what insulin does for diabetics; they stabilize biological functions by reducing or eliminating delusionary thoughts and by reducing anxiety. Pharmacotherapy does not help them to find a job or increase their social and coping skills, however. What they do is stabilize the brain so that sufferers can take advantage of adjunctive psychosocial therapies that help with these problems, including adherence to medical regimens. The four psychotherapeutic modalities that are considered most useful in this regard based on meta-analyses of treatment results with schizophrenia patients (effect sizes ranging from .20 to .60) are cognitive behavioral therapy (CBT), family intervention therapy (FIT), social skills therapy (SST), and cognitive remediation therapy (CRT) (Patterson & Leeuwenkamp, 2008). An examination of these modalities is beyond the scope of this paper, but each one has its strengths and weaknesses, and each works best for certain cases. However, Mueser and colleagues (2013) provide a comprehensive review of extant psychosocial treatment programs.

Community correctional workers cannot themselves provide these services; they are more advocates and brokers of than counselors. In terms of the emerging case management emphasis in corrections, correctional workers should find themselves as a member of a team of professionals bringing their skills and expertise to bear on creating treatment and service plans for mentally ill offenders. The probation/parole officer, having legal authority over the offender, must be the lead person and coordinator of these services. Officers' ability to provide extended and effective services to mentally ill offenders is proportional to the scope of their knowledge of mental illness and of the available resources in the community. The combination of appropriate medication and individual and family counseling has shown good results with schizophrenia patients who have been properly assessed as able to benefit from such a regimen (Lamberti 2007). Unfortunately, it has been found that probation/parole agencies that emphasize a law enforcement approach to supervision are less likely to involve their officers with community mental health agencies in a positive manner, which again emphasizes the need for education across all areas of criminal justice (Lamberti et al., 2011).

Conclusion

Schizophrenia is associated with an elevated risk of committing criminal acts, but those afflicted with the condition are also much more vulnerable that the average person to be victimized as well. This is especially true for those with the most severe symptoms, the homeless, and those comorbid for drug and/or alcohol abuse. If we as a society can come to view schizophrenia as a disease of the brain circuitry that results in unusual behavior rather than a "mental" disease or an eccentric abuse of free will we should find less stigma attached to the condition among CJ personnel.

Genes play a large role in the etiology of schizophrenia, but they are not the whole story. With the emergence of the neurodevelopmental two-hit model researchers have been about to tie together many correlates of the disease from the most proximate cause of positive symptoms to the most distal. We can now "see" schizophrenia in the very structure and function of the brain thanks to advances in brain imaging techniques, and we can effectively treat all but the worst cases with a combination of pharmaco- and psychosocial therapies. This is a huge improvement over the "cold mother" notions prevalent not too long ago when our only "solution" to the mentally ill was to lock them up for life. This is the bounty we have reaped from coming to understand schizophrenia scientifically, and is a bounty that criminologists and criminal justicians cannot afford ignore. As Fisher, Silver and Wolff (2006) point out, the common goal of the mental health and criminal justice systems is to reduce recidivism and victimization among this highly vulnerable group, and to do it humanely, but the first step is to understand the problem.

Endnotes

1. A genetic polymorphism is a minute difference in DNA sequences among individuals and groups at the same locus on an allele (an alternate form of a gene; one paternally and one maternally inherited). The two major polymorphisms are single nucleotide polymorphisms (SNPs) and micro- and mini-satellites (referred to collectively as variable number of tandem repeats—VNTRs). A difference in just one nucleotide is all that differentiates one allele from another in a SNP. These minute genetic differences may make an enormous difference at the phenotypic level. For instance, The Val158Met SNP of the enzyme catechol-O-methyltransferase (COMT) that degrades a variety of neurotransmitters has the tri-nucleotide sequence *ATG* that produces methionine and a *GTG* sequence that produces valine. In this case we say that there are two alleles for COMT—A and G. The valine variant degrades dopamine at about four times the rate of the methionine variant (Beaver, 2009).

VTNRs differ from one another in the length of contiguous nucleotide bases that are repeated a different number of times. The more times the sequence of nucleotides is repeated, the longer the allele. An example of an important VNTR is the 7-repeat allele of the dopamine receptor DRD4 gene. The number of repeats this gene has determines the receptor's sensitivity to dopamine, with shorter repeats being very sensitive and longer repeats being much less so. Thus, it is in allelic variants of the same genes such as SNPs and VNTRs that individual and group genetic differences are found.

Conflict of Interest:

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