Antisocial Behaviours from a Primal Health Research Perspective

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Primal Health Terminology

The Primal Health Research Institute proposes this practical vocabulary adapted to the new scientific context first articulated in Odent (1986), *Primal Health*. London: Century-Hutchinson.

Primal – first in time and first in importance
Primal period – the time which includes fetal life, the perinatal period, and early infancy. It is during this primal period that the adaptive systems involved in what we commonly call health reach maturity. It is the time of close dependence on the mother. One can anticipate that any kind of event happening during this period can have irreversible effects.
Primal Adaptive System – the subcortical nervous system, the endocrine system and the immune system should no longer be separated and should be understood as a whole (e.g., the brain is a gland, insulin is a neuromediator, lymphocytes can release endorphins, etc.). We call this network the "primal adaptive system." Phrases used in the medical literature, such as "psychoneuroimmunoendocrinological system," "psychoneuroimmunology," and "immunoendocrinology" etc., should be expressed in simpler terms. A review article in the *New England Journal of Medicine* gave a perfect updated description of what we call the "primal adaptive system:" Seymour-Reichlin. 1993. Neuroendocrine-immune interactions. *New Engl J Med 329:* 1246-53

As human beings we are endowed with gregarious tendencies. Like most other primates we need to live in groups. Since we are also endowed with the capacity to communicate in sophisticated ways, particularly through language, we create cultural milieus. Cultural milieus prescribe and proscribe behavior, that is, what people should do and should not do given their social surroundings and circumstances. We establish norms, with at least some degree of consensus, that are enforced through social sanctions.

Because antisocial behavior is contrary to the standards of the particular society, this framework is imprecise. Furthermore, the degrees of deviation from what is considered normal are numerous and varied. In other words, gregarious tendencies are not equal among the members of a given human group.

The primal health research perspective will help us to overcome such difficulties and to include diverse personality traits, behaviors and diseases under the same umbrella. When studying antisocial behaviors, the conventional barriers between medicine, criminology and the study of personality traits become blurred, even misleading. Nevertheless, they serve as reference points. We'll first consider deviations that are usually studied within the framework of psychiatry.

Deviations Usually Classified as Pathological

Schizophrenia is a productive keyword when exploring the Primal Health Data Bank. Increasing and converging evidence suggests that cerebral anomalies in this condition develop pre-and perinatally. An accumulation of data published after 1990 points to the conclusion that the pathogenesis of schizophrenia is a chapter of "womb ecology."

The first clues provided by data concern head circumference at birth. "Pre-schizophrenic" newborn babies have a disproportionately smaller head circumference (in relation to body length) than controls. Low birth weight and length, as well as low placental weight, are also risk factors. Furthermore, the frequent minor physical anomalies (particularly anomalies of the mouth in males) associated with schizophrenia are obvious consequences of prenatal developmental defects.

Studies of schizophrenia after prenatal exposure to famine are highly convincing. Those who spent the first half of their fetal life during the Dutch famine of winter 1944-1945 were at increased risk of developing schizophrenia. Rates of adult schizophrenia following prenatal exposure to the Chinese famine of 1959-1961 confirmed the Dutch data.

Among diseases in pregnancy, we must give particular importance to influenza, which has been widely studied as a risk factor. (Only one study failed to detect a correlation between schizophrenia and influenza prevalence during prenatal life.) Serologic evidence of prenatal influenza in the etiology of schizophrenia confirms the epidemiological data. The existence of maternal influenza in pregnancy as a risk factor for schizophrenia can help interpret the numerous studies relating the keywords "seasonality of birth" and "schizophrenia." A significant excess of winter/spring births among schizophrenic patients is already known to occur in the Northern hemisphere. This is confirmed by an overview of the medical literature. The quarterly birth distribution of patients with schizophrenia appears to be reversed in the Southern hemisphere, and season-of-birth effects are difficult to demonstrate in tropical and equatorial countries.

A relationship has also been demonstrated between maternal antibodies to toxoplasmosis and the risk of schizophrenia and other disorders of the schizophrenia spectrum in offspring.

The most common pregnancy complications emerge as risk factors. A study covering all psychiatric hospital admissions and all hospital births in Scotland identified obstetric records of people born 1971-1974 who were subsequently admitted to hospital with a diagnosis of schizophrenia and then compared their standardized obstetric records with those of matched controls. A total of 115 schizophrenic and control pairs were involved. The former showed a highly significant excess of pregnancy complications. In particular, there was a significant excess of pre-eclampsia (10 versus two).

All Swedish children born during 1973-1979 were involved in a study linking data from the Swedish Birth Register with data from the Swedish Inpatient Register. Among boys whose mothers suffered bleeding during late pregnancy, the risk was multiplied by four.

Drugs to treat diseases in pregnancy may cause more harm than the disease itself. A Danish study of 7999 individuals that included 116 cases of schizophrenia found a significant association between

second-trimester exposure to analgesics and increased risk of schizophrenia. In a previous study of 7866 individuals and 84 cases of schizophrenia, the same team of researchers had found that drugs prescribed to treat hypertension (particularly diuretics) in the third trimester conferred a 4.01-fold elevated risk.

The risk of schizophrenia in relation to intrauterine pollution, particularly pollution with heavy metals, is a new and promising avenue for research. Lead levels of stored blood samples were collected from expectant California mothers between 1959 and 1966. A comparison was made between 44 women whose children went on to develop schizophrenia and 75 mothers whose children did not. Children of mothers whose blood contained more than 150 micrograms of lead per liter were twice as likely to develop schizophrenia as those whose blood levels were below this threshold.

In spite of methodological difficulties, certain maternal emotional states in pregnancy have been demonstrated to be risk factors. In a Finnish study, data were collected prospectively in a cohort of 11,017 individuals born in 1966. In the sixth or seventh month of pregnancy mothers were asked whether the pregnancy was wanted, mistimed but wanted, or unwanted. Schizophrenia diagnoses were obtained from the Finnish hospital discharge register. The risk of later schizophrenia among unwanted children was higher when compared with wanted or mistimed children, even after adjustments for socio-demographics, pregnancy and perinatal variables (risk multiplied by 2.4).

Animal experiments can help interpret epidemiological studies. Prenatal lesions of the hippocampi of rats apparently remain silent until adult life when there is an abnormal dramatic response to stress and challenge from amphetamines. It seems highly probable, when taking animal experiments into account, that a developmental defect of the hippocampus during fetal life is one of the main components of the chain of events that lead to schizophrenia.

Several studies detected such risk factors in the perinatal period as fetal distress during labor, abnormal presentations and “complicated” c-sections, while they could not detect any association between obstetric complications and family history of schizophrenia. In fact, the inclusion of multiple variables suggests that these complications may be partly secondary to earlier events. However, we must keep in mind that in animal models dopaminergic systems appear to be particularly vulnerable to a wide range of perinatal insults and that schizophrenia is associated with alterations of dopaminergic functions.

One noticeable aspect of most studies is that they found no protective effect of breastfeeding. However, according to an Italian study, breast milk might postpone the onset of the illness without reducing the risk. We must pay special attention to a Danish study involving 6841 individuals of whom 1671 had been breastfed for two weeks or less (early weaning) and 5170 had been breastfed longer. Breastfeeding for two weeks only (or no breastfeeding at all) was associated with elevated risk of schizophrenia. When comparing this study with others, the protective effect of breastfeeding appears to occur only if the cut-off for weaning is two weeks instead of one month. This study tends to confirm that the risks of schizophrenia are established during an early phase of development that ends soon after birth.

The study of schizophrenia from a primal health research perspective is an ideal opportunity to illustrate the importance of timing when considering the effects of gene-environment interactions on

human development. Epidemiological studies reveal a great diversity of environmental factors that can increase the risks of schizophrenia. However, the timing of an insult is more important than its nature. The fact that maternal-fetal blood incompatibility also appears as a risk factor confirms that the chain of events leading to schizophrenia starts at an early phase of development.

Autism is also considered a pathological deviation from the usual gregarious human tendencies. An accumulation of data (included in the Primal Health Research Data Bank) suggests that the timing of the gene-environment interactions is different for autism than for schizophrenia. Several authoritative studies indicate the paramount importance of what happened at birth in the genesis of the different autistic spectrum disorders.

My interest in autism started in 1982, when I met Niko Tinbergen, one of the founders of ethology, who shared the Nobel Prize with Konrad Lorenz and Karl Von Frisch in 1973. An ethologist familiar with the observation of animal behavior, he researched the non-verbal behavior of autistic children in particular. As a "field ethologist" he studied the children in their home environments. Not only did he offer detailed descriptions of his observations, but he also listed factors that predispose to autism or can exaggerate the symptoms. These factors in the perinatal period included: induction of labor, "deep forceps" delivery, birth under anesthesia and resuscitation at birth. We must also save from oblivion a report by Ryoko Hattori, a psychiatrist from Kumamoto, Japan. She evaluated the risks of becoming autistic according to the place of birth, such as a certain hospital where children were significantly more at risk. In that particular hospital, the routine was to induce labor a week before the expected date of birth and to administer a complex mixture of sedatives, anesthesia agents and analgesics during labor.

Among the three recent large and authoritative studies of autism from a primal health research perspective, the Australian one will convince anyone that the main risk factors occur in the perinatal period. The 465 subjects born in Western Australia between 1980 and 1995 and diagnosed with an autism spectrum disorder by 1999 were compared with the birth records of 481 siblings of the individuals and 1313 controls. No differences in gestational age at birth (including the proportion of premature infants), weight for gestational age, head circumference or length were observed between the individuals with autism and the control subjects. Pre-eclampsia did not appear as a risk factor. These negative findings lend more importance to perinatal factors. Compared with their siblings, individuals with autism were more likely to have been induced, to have experienced fetal distress and to have been born with a low Apgar score. Compared with control subjects, they were more likely to be born after induction and to be born by elective or emergency c-section.

Similar conclusions can be drawn from a study involving all Swedish children born from 1974 to 1993. No association was found between autism and head circumference, maternal diabetes, being a twin or season of birth, while c-section appeared to be a risk factor. This study could not consider labor induction as a possible risk factor, since this term did not appear in the Swedish birth registers until 1991.

A recent report from Israel also found no prenatal differences between autistic children and controls, but the rates of birth complications were higher among the population with autism. In addition, we must consider data indication that perinatal factors may play a lesser role in autism in "high-functioning" individuals compared with studies of autism associated with severe retardation, as well

as data suggesting that anesthesia during labor is a risk factor for the development of dyskinesia among autistic children.

Although the risk factors for autism seem to occur mostly in the perinatal period, we must keep in mind the association of autism with fetal valproate syndrome (anti-epileptic medication) and with thalidomide embryopathy.

Attention Deficit Hyperactivity Disorder is also considered a pathological deviation from the norm. Children with this condition cannot socialize like others. An overview of the studies introduced by the key word "ADHD" suggests the main risk factors occur during fetal life. The best-documented risk factors are low birth weight, premature birth, smoking in pregnancy, alcohol and drugs in pregnancy, maternal iodine deficiency, and also the degree of anxiety of the mother, particularly between 12 and 22 weeks gestation.

In fact, all mental diseases interfere with the process of socialization, including manic-depressive psychosis, which has not yet been widely studied from a primal health research perspective.

Deviations Usually Classified as Criminal Behavior

When exploring the Primal Health Research Data Base, the keyword "criminality" leads to research indicating the importance of prenatal factors. Two Finnish studies suggest that certain maternal emotional states in pregnancy are risk factors. In one of these studies the authors identified 167 children whose fathers had died before they were born. Also identified were 168 children whose fathers had died during the children's first year of life. The medical records of all 335 of these children were followed for 35 years. In both groups, the parents were of comparable age and from comparable social classes. All of the children grew up fatherless. However, only those who lost their fathers while in the womb were at increased risk of criminality (plus alcoholism and mental diseases). The results of this study suggest that the emotional state of the mother during pregnancy has more long-term effects on the child than during the year following birth.

The other Finnish study researched 12,059 children born in 1966 and followed to the end of 1998. The pregnant mothers were asked at the antenatal clinic if they felt themselves to be depressed. The Finnish Ministry of Justice provided information on criminal offences for all descendants. Male children of prenatally depressed mothers showed a significant increase in criminality.

Smoking in pregnancy is a well-documented risk factor for criminality. In one study, involving a cohort of 4169 male and 3943 female subjects born between 1959 and 1961, a dose-response relationship was found between the amount of maternal prenatal smoking and criminal arrest in male and female subjects.

More than 4000 male subjects born in the same hospital in Copenhagen were followed until age 18 and then assessed again at the age of 34. The authors looked in particular at the interaction between birth complications an dearly maternal rejection. The main risk factor found in these studies for being a violent criminal is the association of birth complications with early maternal rejection. Early maternal rejection by itself is not a risk factor. We can conclude once more that very early influences are implicated in violent criminality.

At the Crossroads of Psychiatry, Criminality and Psychology

Antisocial personality disorder refers to individuals who lack regard for the moral or legal standards of the local culture and who exhibit a marked inability to get along with others or abide by societal rules. They may be termed psychopaths or sociopaths. Many eventually end up imprison or die by violence or in accidents caused by risk-taking.

We must bear in mind that psychiatrists themselves are not in unanimous agreement on the existence, content and diagnosis of antisocial personality disorders. In spite of these difficulties, the keyword "antisocial personality disorder" leads to epidemiological studies that are within the framework of primal health research. The largest study looked at the consequences of the blockade of food supplies in the Netherlands during the winter of 1944-1945. The participants were 100,543 Dutch men born in large urban areas in 1944-1946 who were given psychiatric examinations for military induction at age 18 years. They ere classified by the degree and timing of their prenatal nutritional deficiency based on their date and place of birth. Men exposed to severe maternal nutritional deficiency during the first and/or second trimesters of their fetal life exhibited increased risk for antisocial personality disorder. Third-trimester exposure to severe nutritional deficiency and prenatal exposure to moderate nutritional deficiency were not associated with increased risk.

The Eccentrics and the Geniuses

Certain deviations from the typical human gregarious tendencies are classified as personality traits. Among these deviations we must mention the case of the eccentrics and the geniuses. Most highly-creative, legendary geniuses had unusual, eccentric personalities and manifested many schizotypal traits. Isaac Newton never married and lived most of his life alone. Albert Einstein had poor grooming and hygiene and well-documented interpersonal deficiencies. Bertrand Russell had been an aloof, lonely and somewhat insecure child, before leading an unstable adult life.

The link between "genius and insanity" has been widely studied since the nineteenth century, after Cesare Lumbroso, an Italian psychiatrist, published his book *l'ouomo di genio* in 1864, and Francis Galto published *Hereditary Genius* in 1869. The personality traits of geniuses might simply be written off as unimportant details, but for the fact that their family histories indicate strong connections between genius and schizophrenia. J.L. Karlsson, a psychiatrist in Iceland, looked into the genetic relationship between schizophrenia and creativity by examining the relatives of individuals listed in Iceland's *Who's Who.* He reported that the relatives of these successful people suffered an increased rate of schizophrenia. Of notice is that James Joyce's daughter was schizophrenic. The family pedigree of Bertrand Russell was loaded with schizophrenic people: his uncle William was "insane," his aunt Agatha was delusional, his son John was diagnosed with schizophrenia and his granddaughter Helen also suffered from schizophrenia and committed suicide by setting fire to herself. As well, Albert Einstein's son by his first marriage suffered from schizophrenia. The son of John Nash, the gifted schizophrenic mathematician and Nobel Laureate in Economics, suffers from schizophrenia too. A few schizophrenic individuals, but many first-, second- or third-degree relatives, who share part of the schizophrenic genome, are some of the most creative individuals around. The close relationship between madness and creativity led David Horrobin to assume the "schizophrenia shaped humanity."

For obvious reasons, studying imprecise concepts such as social behaviors and creativity from a primal health research perspective is difficult. It will probably be easier in the near future to improve our understanding of the gene-environment interactions, particularly during fetal life. We are learning today that in some cases a parent's gene is silenced in one organ, which can be the brain. This is why the concept of imprinted genes might help us to interpret the relationship between antisocial behaviors, schizophrenia, and creativity.

I tried in the past to review the primal period of famous geniuses such as Galileo, Newton, Pascal, Darwin, and Einstein. It is noticeable that these highly-creative people were born premature and before the age of intensive care units. Their survival was likely due to exceptionally loving, vigilant and stimulating environments that exposed them to a great variety of sensory stimulations at an age when others are still in the womb. During a critical phase of development the gene-environment interactions occurred in unusual conditions.

Is there a link between prematurity and creativity? Who will study sociability and creativity in relation to "Kangaroo care?"

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