

INTERPRETATION OF SCHIZOPHRENIA

The Biochemistry of Schizophrenia

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The Biochemistry of Schizophrenia

I

Introductory Remarks

A large number of researchers have carried out biochemical investigations of schizophrenia, pursuing the hypothesis that a biochemical or metabolic error is at the basis of the disorder (see Himwich, Kety, and Smythies, 1966; Weil-Malherbe and Szara, 1971). Because life is characterized by an incredibly large number of metabolic processes, it has not been an easy task to isolate the hypothetical one responsible for schizophrenia. The search has so far not been successful but continues along certain directions illustrated in this chapter.

In order to demonstrate that a toxin, a by-product of a faulty metabolic process, is the main etiologic factor of the psychosis, some of the following empirical verifications are necessary:

1. The metabolite should be isolated and should be found only in schizophrenics.

2. If found also in conditions other than schizophrenia, it must be found in schizophrenic patients in quantities that are significantly different from those of nonschizophrenics.
3. The metabolite should reproduce the disorder when introduced into nonschizophrenic persons in certain quantities.
4. A greater sensitivity, allergy, or other kind of abnormal reaction to a special metabolite should be apparent in schizophrenic patients.

Many authors have indeed found biochemical alterations in schizophrenic patients. However, these findings do not seem to satisfy any of these four requirements. Moreover, it will appear from the content of this chapter that at the present stage of research we cannot ascertain whether these alterations are (1) inherent in the basic etiology of the disorder, (2) the *result* of the physiological or psychological changes which occur in the disorder, or (3) the result of the way of living of most patients who have been the object of the biochemical studies. The vast majority of these patients have been institutionalized. Thus the alterations that they presented may have been caused by the limitations in activity of institutional life, by the

diet of institutions, especially vitamin deficiency, or by drug therapy.

In the United States the person who has made one of the most extensive studies of the biochemistry of schizophrenia is Semour S. Kety. His writings (1959, 1966, 1969, 1972) will be one of our main guidelines.

II The Transmethylation Hypothesis

In 1952 Osmond and Smythies stressed the fact that on one hand there are some similarities between mescaline psychosis and schizophrenia and on the other hand there are similarities between mescaline and epinephrine. They quoted Harley-Mason, who had stated that it was extremely probable that the final stage in the biogenesis of epinephrine is a transmethylation of norepinephrine, the methyl group arising from methionine or choline: “It is just possible that a pathological disordering of its transmethylation mechanism might lead to methylation of one or both of its phenolic hydroxyl groups instead of its amino groups” (Osmond and Smythies, 1952).

In 1959 the transmethylation of norepinephrine to epinephrine

was confirmed (Blaschko, 1959). Since that time many authors have tried to prove that pathological transmethylation occurs in schizophrenia. Consequently the delicate balance between the sympathetic nervous system (which is mediated by norepinephrine) and the parasympathetic nervous system (which is mediated by acetylcholine) would be disrupted. Derivations of epinephrine, such as adrenochrome and adrenolutin, or other endogenous metabolites, would cause psychotic symptoms.

The hypothesis that pathological methylation may occur in schizophrenia finds some support in the fact that known psychotomimetic substances include in their formula one or more methyl groups attached to an oxygen or nitrogen atom. When such methyl donors as methionine and betaine are given jointly with a monoamine oxidase inhibitor, they produce exacerbation of psychotic symptoms in chronic schizophrenics (Kety, 1966). The hypothesis would have obtained additional support, according to some authors, if large doses of nicotinamide, which are supposed to divert the methylating processes, would have been able to ameliorate the psychotic condition. Hoffer claims that nicotinamide has therapeutic value, but others have denied such claims. However, if Baldessarini

(1966) is right that nicotinamide does not reduce s-adenosylmethionine in the brain, the transmethylation hypothesis is not ruled out.

In conclusion, although the transmethylation hypothesis suggests the possibility of a metabolic disorder in schizophrenia, it lacks as yet all four of the above-mentioned empirical verifications.

III

The Ceruloplasmin, Taraxein, and Related Hypotheses

In 1957 the Swedish biochemist Akerfeldt reported that he had discovered a new test for diagnosing schizophrenia. The test relied on the oxidation of N-N-dimethyl-p-phenylenediamine. This news aroused some enthusiasm, which soon subsided. The oxidizing substance was recognized as ceruloplasmin, a copper-protein. The hypothesis was thus that this copper-protein was increased in the serum of schizophrenics and would increase the oxidizing activity of the serum. It was soon found, however, that ceruloplasmin is consistently increased in all tested institutionalized patients who had been on a diet poor in ascorbic acid. Normal diet would lower the level of ceruloplasmin.

Under the leadership of Heath, some investigators felt that the high oxidizing power of the serum of schizophrenics on epinephrine *in vitro* could not be attributed solely to a higher level of ceruloplasmin. They isolated a special type of ceruloplasmin to which they gave the name taraxein (from the Greek *tarassein*, “to disturb”) (Heath et al., 1957). They reported that when this substance was injected into monkeys, behavioral changes occurred. Also electroencephalographic abnormalities in focal subcortical regions of the brain were found that, according to the authors, resembled those of schizophrenic patients. When the substance was intravenously injected into volunteer prisoners, some symptoms characteristic of schizophrenia, such as thought disorders, hallucinations, and paranoid ideas, ensued (Heath, 1963). Robins, Smith, and Lowe (1957) tried to duplicate Heath’s findings but could not. Since then the hypotheses of Heath and his school have not received confirmation.

Lief (1957) reported that a volunteer in Heath’s experiments was a psychiatric resident in analysis who knew that he was going to get either saline or taraxein. Lief described in detail the psychological concomitants that may have been responsible for the results. Kety (1959) concludes that the possibility exists that the reported effects of

taraxein are “the result of a combination of suggestion, nonspecific toxic reactions from ammonium sulfate or other contaminants, and reinforcement of these cues by the unconscious biases of subject and observer. . . .” Heath and Krupp (1967) continue to think that taraxein is important in the etiology of schizophrenia. They have now accepted Fessel’s ideas (1962) that schizophrenia might be an autoimmune disease. Taraxein would be a component of the immunoglobulin (IgG) fraction of the serum and would be an antibody specific for the brain. In their experiments Heath and Krupp (1967) used the fluorescent antibody technique of Coons and various serum fractionation methods.

Kety (1959) listed a series of experiments done to demonstrate that the serum of schizophrenia has a toxic effect (1) on larvae of *Xenopus laevis*; (2) on tadpoles; (3) on cells in tissue culture; (4) on rope-climbing speed in rats. Other experiments purported to demonstrate that certain extracts of the urine of schizophrenic patients produce behavioral changes in rats or disturbance in web construction in spiders. According to Kety all these experiments have neither been confirmed nor contradicted.

IV The Serotonin Hypothesis

Woolley and Shaw (1954) hypothesized that mental changes may be attributed to a cerebral deficiency of *serotonin*, caused by a metabolic disorder. Serotonin is a derivative of tryptophan and is known to exist in high concentration in the brain, especially in the limbic system. It was speculated that lysergic acid had an inhibitory effect on serotonin and thus produced a psychosis.

Other experiments, however, led to contradictory results. Iproniazid is a monoamine oxidase inhibitor that causes an increase of the serotonin level of the brain, and yet it may also produce a toxic psychosis. Moreover, other psychotomimetic drugs, or lysergic acid derivatives, do not have an antiserotonin activity. Reserpine lowers the concentration of norepinephrine as serotonin; chlorpromazine instead has no effect on serotonin.

The conclusion reached by Kety is that serotonin and its metabolism play an important role in the central nervous system. What this role consists of, however, we do not know as yet. Some researchers believe that schizophrenia is due to too little serotonin,

while other researchers blame too much serotonin.

V

The DMPEA Hypothesis and Serum Proteins

Friedhoff and Van Winkle (1967) reported the presence of 3, 4-dimethoxy-phenylethylamine (DMPEA) in the urine of schizophrenics. Bourdillon and coworkers (1965) found a “pink spot” having some characteristics of DMPEA in 55 percent of tested schizophrenics. A large number of investigators have obtained contrasting results. Some authors have found an increase in macroglobulins in the serum of schizophrenics (Fessel, 1962; Kopeloff and Fishell, 1963), which other authors (Lovegrove and Nicholls, 1965) have denied. Kety (1969) concluded that up to the present time no abnormal characteristic of schizophrenia has been evidenced by the available techniques. For further studies on the biochemistry of schizophrenia in reference especially to blood proteins the reader is referred to the important works by Beckett and Gottlieb (1970) and by Frohman and Gottlieb (1974).

VI

Other Metabolic Changes

Numerous metabolic changes have been described in schizophrenia. Many early authors have proved in a convincing manner that in many schizophrenics there is a deficiency in the intake of oxygen (Hoskins, 1932, 1937). Several explanations have been given: for instance, that the blood of the patient leaves the lungs in an undersaturated condition; or that the tissues fail to absorb an adequate quantity of oxygen; or that the sluggishness, or hypoplasia, of the circulatory system does not permit an adequate oxygen supply. Other authors have tried to correlate this low consumption of oxygen with hormonal changes (Gomall et al., 1953). Here many questions come to mind. Is the low intake of oxygen what compels the schizophrenic toward low activity? Are the psychological manifestations of schizophrenia similar to those described by McFarland (1932) in state of oxygen deprivation? Or is the opposite true, that is, does the psychological condition of schizophrenia, with its withdrawal characteristics, reduce the oxygen requirements of the patient?

The carbohydrate metabolism in schizophrenia has also been studied by numerous early authors. The reader who is interested in this subject is referred to the review by Shattock (1950). Shattock

states that many of the reports have been contradicted by other authors; however, the following three findings have seldom been disputed: (1) the presence of a normal fasting blood-sugar curve; (2) a tendency to sustained hyperglycemia in early or acute cases; (3) a normal intravenous glucose tolerance test in many patients who, instead, had showed prolonged hyperglycemia after oral administration of glucose. Although these findings are undisputed, their interpretation is very controversial. Frohman and Gottlieb (1974) conclude that there are indications of some abnormalities in carbohydrate chemistry in schizophrenics, but “most data suggest that these irregularities merely reflect abnormality in other phases of metabolism.”

Hoskins (1946) found that the *output of urine* was twice as large in the schizophrenic as in the normal population, but he attributed this to the fact that, generally, schizophrenics drink more water than the average person. A low hippuric acid excretion, suggesting dysfunction of the liver, was found by many authors, but the Quick test, on which these works were based, cannot be considered a reliable method. Buscaino (1952), too, incriminates the liver. Buscaino is one of the staunchest European supporters of the theory that schizophrenia is

the result of a metabolic disorder. In a number of papers, summarized in his report at the First International Congress of Neuropathology, he tried to prove that there is, in schizophrenia:

1. Evidence of functional insufficiency of the liver (bilirubinemia, detoxication power impairment, as shown by deamination test, hippuric acid test, santonin test, and so on). Buscaino states that other researchers failed to get the same results because they relied on only one test, whereas, in order to study the liver properly, one must use a battery of tests (“hepatic constellation” or “instantaneous picture of liver”).
2. Evidence of insufficient reactivity of the hepatic and extrahepatic reticuloendothelial system.
3. Evidence of gastroenteric function, as shown by findings of gastroenteritis.

Buscaino reports that he has found pathologic amines in the blood and urine of schizophrenics and concludes that schizophrenia is an “amino-toxicosis.” In a recent publication (1970) Buscaino has reaffirmed his points of view. One of the disorders of metabolism investigated by Buscaino concerns aromaturia. McGeer and co-workers (1957) have also reported that schizophrenics excrete a

higher than normal concentration of aromatic compounds in the urine.

In the psychiatric literature of the last part of the nineteenth century, reference was often made to an unusual odor of schizophrenic patients, particularly catatonic ones, who also presented greasiness of the skin. Recently Smith and Sines (1960) studied again this phenomenon. They felt that “if there is a unique ‘odor’ in the apocrine sweat or in the sebaceous secretion of schizophrenic patients, the identification of this odoriferous substance may give a clue to an inborn error of metabolism” in the psychosis. The authors trained rats to discriminate between the schizophrenic sweat with the strange odor and nonschizophrenic sweat without this odor. A human odor-testing panel was also able to discriminate between the same samples with greater accuracy than the rats did. According to the authors, this odor can be recognized reliably and bears some relationship to the psychosis.

Smith (1954) studied the functions of the liver of seventy-six schizophrenics and obtained consistently negative findings. Graetz, Reiss, and Waldon (1954) found that the excretion rate of hippuric acid was diminished in chronic cases of schizophrenia but not in acute

cases. Persky, Gamm, and Grinker (1952) found a decreased excretion of hippuric acid in ten catatonic patients. Numerous volumes could be written on the studies of metabolism that have been carried out on schizophrenic patients. McFarland and Goldstein (1938), who reviewed the work in this field up to 1938, concluded that the results of all these investigations were almost entirely negative, so conflicting were the reports. Beliak and Willson (1947) reviewed the literature on etiology of dementia praecox and concluded the sections on the biochemical factors with the remark that the only consistent difference of biochemical findings "from normals lies in the greater variability of values for almost any factor investigated."

Recently Kety (1969) concluded that although substantial progress has been made in this field, "it would be difficult to demonstrate that a definitive increase in our knowledge of biochemical mechanisms in the schizophrenic psychoses has occurred in the past decade."

Perhaps it is worthwhile to mention here Horwitt (1956), who, although writing more than a decade before the last quoted work by Kety, expressed the following notes of cautiousness:

We are now entering a period of renewed interest in biological research on schizophrenia. Nothing can be more harmful to this rejuvenation of the investigation of the biology of mental health than the publication of reports based on techniques of patient selection which do not meet the minimum standards accepted by other disciplines. The manner in which the patient chooses to manifest his difficulties may not be a function of his physiological status.

Symptoms that are usually considered artifacts in other studies are often erroneously accepted as biological aberrations in the evaluation of the schizophrenic patient. Year after year, papers appear which purport to distinguish between the state of schizophrenia and that of normalcy. The sum total of the differences reported would make the schizophrenic patient a sorry physical specimen indeed: his liver, brain, kidney, and circulatory functions are impaired; he is deficient in practically every vitamin; his hormones are out of balance, and his enzymes are askew. Fortunately, many of these claims of metabolic abnormality are forgotten in time with a minimum of polemic, but it seems that each new generation of biologists has to be indoctrinated—or disillusioned—without benefit of the experience of its predecessors.

Horwitt gave the following examples of procedural mistakes made in organic studies of schizophrenia. Basal metabolism during emotional stress is not basal at all but may be a reflection of

metabolism during intense adrenergic stimulation due to the stress itself. Comparing data from blood samples of schizophrenics and normal people is like comparing samples from soldiers on the battlefields and people in a relaxed, basal condition. He also stressed that the vagaries of psychotic behavior are such as to affect food intake. In many cases this abnormal food intake is responsible for nutritional disorders and metabolic changes. He wrote, "Having studied such changes in mental patients for more than fifteen years, I am at a loss to understand how some studies of urinary excretion or of blood constituents can be performed without recourse to nutritional controls." In the same excellent article he gave many more illustrations, which cannot be reported here, of mistakes commonly made in biological studies of schizophrenia.

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