Recurrent Fallacy of Some Critics of Orthomolecular Psychiatry

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of September, 1975, C. H. Hollenberg , M.D., formulates the following question, "Why is it that such an eminent biochemist as Linus Pauling should advocate megavitamin therapy for certain diseases?", and he answers:

"The reasoning is based upon a well-known biochemical principle: Under certain circumstances, one of the processes involved in enzyme function is rendered abnormal by genetic mutation or disease. The rate of that process is very much slowed. Administration of large quantities of the material to be transported, transformed or bound, will drive the reaction toward a more normal reaction rate."

And he continues:

"There are situations in which large vitamin doses represent a rational and effective approach to diseases due to specific genetically determined disorders of vitamin metabolism," and he mentions megaloblastic anemia and central nervous system abnormalities as

1 Antonio J. De Liz, M.D., Ph.D., Psychiatrist in Private Practice, Former Assistant Professor of Psychiatry at the University of Georgia. 218 Stewart Avenue. Garden City. N.Y. 11530. inherited disorders due to folic acid metabolism.

Then he concludes:

"What the advocates of megavitamins have done is to extrapolate from such situations. They reason that, because of the frequency of genetic mutation in humans, there must be many instances in which abnormalities of mental and physical function are due to inherited abnormalities in enzyme function."

Following a comparable trend of thought, Monica D. Blumenthal, M.D., Ph.D., states:

"The idea that there is a relatively straightforward relationship between chemical processes and behavior is an interesting one which probably represents an extrapolation the great wave of enthusiasm accompanying popularization of human inborn errors of metabolism. The dramatic association between certain known metabolic defects and mental retardation seemed to present a clear and readily comprehended demonstration of the relation-ship between metabolism and behavior. It seemed a simple step to extrapolate from mental retardation to mental illness, and so the challenge was rung that 'for every crooked thought there must be a crooked molecule."

The Orthomolecular scientist knows that even in inborn diseases of metabolism linear causality does not obtain between genetic, molecular defects and forms of mental retardation. For between molecular events and subsequent retarded behavior or thinking processes, there are intervening chains of enzymatic transactions. For example, in phenylke-tonuric children secondary and tertiary enzymatic reactions culminate in the formation of inhibited enzymes which are the direct outcome of the autosomal genetic defect. These inhibited enzymes, dopadecarboxy-lase, are generated by the action of phenylpyruvic acid and lead in turn to a cybernetically modulated mechanism causing deficiency in norepinephrine metabolism and tyrosine hydroxylase. One has to remember the enormous complexity of the cell with its several thousand genes in a process of interaction and co-action with thousands of environmental variables process culminating in phenotype. Innumerable enzymes formed in this process which give rise to an inextricable maze of biochemical pathways, thus making impossible to identify the biological consequences of an inborn genetic defect, and a fortiori of the biological and environmental structural sequences of transformations, compositions, and integrations that culminate in schizophrenic behavior. This is tantamount to saying that strict determinism would be absolutely useless when used as the main causal principle of scientific explanation here. In the scientific study of schizophrenia, one has rather to insightfully envisage hypotheses that can be tested. Research scientists in schizophrenia are here reminded of Albert Einstein's words:

"There is no logical path leading to universal laws in science from which a true picture of facts can be obtained by pure deduction. Those laws can only be reached by intuition, based upon something like an intellectual love (Einfuh-lung) of the objects of experience."

M. Blumenthal, M.D., assumes that the Orthomolecular and/or biochemical scientists endorse strict determinism as the basic

interpretative model of causation in inborn diseases of metabolism. Such an assumption is unwarranted. Linus Pauling, for example, never countenanced such a principle, no matter how indestructible it appeared in the bygone era of Newtonian mechanics.

Therefore, all those psychiatrists who share his views cannot be blamed for the extrapolation of an idea in which they have never believed.

In his profound study of vitamin C, Linus Pauling tells us that the human organism, the other primates, and the guinea pig have lost their previously probable capacity to make the synthesis of that vitamin during phylogenetic evolution. He assumes that such a loss was due to mutation, and it is quite clear in his writings that for him such a loss was due to an alteration of the molecular structure, or its symmetry that followed some kind of change of the cellular environment. There is no evidence at all in his thinking that he postulates absolute determinism or, for that matter, teleologic vitalism or blind accidental causation in vital processes. From what premises then does Dr. Blumenthal deduce her statement, namely, "that for every crooked thought there must be a crooked molecule"?

I would conjecture that Linus Pauling would rather endorse as a paradigm of causality one consonant of the basic principles of cybernetics. This principle postulates directive correlated purposeful adaptations of the organism to a changing environment, either in health or in disease.

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