The Adrenochrome Hypothesis

Differences arose between Sen and Prousky on the validity of Hoffer's adrenochrome hypothesis of schizophrenia.[1,2] Two points are noteworthy in this context:

1. Hoffer himself called adrenochrome a "hypothesis" although he concluded that the adrenochrome hypothesis was the best fit hypothesis among other competing hypotheses of the schizophrenia syndrome.[3]

2. We should not overlook the fact that schizophrenia is really schizophrenias (plural). For example pellagra, still around in many parts of the world, may be mislabeled as schizophrenia. Similarly, cerebral allergic reactions to gluten (gluteomorphin) can give rise to clinical features that would be indistinguishable from schizophrenia, and therefore would be overlooked by most psychiatrists.

Hoffer himself sometimes moved away from the adrenochrome hypothesis. For example, he advised us to think of alternate treatments (i.e., hypotheses) if a patient had not responded to therapeutic doses of niacin. Hoffer considered hypothyroidism as a possible cause of schizophrenia.[4] He even quoted a study by Danziger that showed recoveries in 80 schizophrenic patients that were administered natural desiccated thyroid for at least 100 days, and who were ill for six months or less.[5]

Foster,[6] Pataracchia,[7,8] Campbell-McBride,[9] and others have summarized the alternate causes or hypotheses of schizophrenia, such as subclinical hypothyroidism, cerebral allergy to gluten, sugar and petro chemical inhalants, heavy metal toxicity, and candida/gut dysbiosis. In any individual case, any combination of causes might be implicated in the genesis of the schizophrenia syndrome.

Although, at least to my knowledge, adrenochrome has not been measured and compared between schizophrenic, non-schizophrenic and normal control groups of population, we can draw conclusions in favour of niacin and hence, indirectly for the adrenochrome hypothesis, from Hoffer's studies and reports that spanned many decades. Outside of Hoffer, there have been publications giving indirect credence to the adrenochrome hypothesis. For example, Wittenborn published data demonstrating that acute-onset patients (ill for six months or less) were having intact inter-personal relations as a result of niacin treatment.[10]

Hoffer's writings also stressed the value of psychosocial factors when he talked of food, shelter, and respect as critically valuable factors in recovery without psychiatric drugs.[11] In the case of chronic schizophrenic patients, who are likely to develop negative symptoms, much like the well known symptoms of "institutionalization or hospitalization syndrome," the useful hypothesis may be something other than the adrenochrome hypothesis. There is even evidence of gluten sensitivity in chronic schizophrenic patients. They may have become chronic because the treating physician didn't consider gluten as a hypothesis of schizophrenia. A 2009 publication described a lifelong schizophrenic patient that recovered following a gluten-free, low-carbohydrate ketogenic diet.[12]

This leads to another point raised in the Sen and Prousky correspondence. Prousky argues in favour of integration and some kind of cooperation between orthomolecular and orthodox mainstream medicine. If orthomolecular medicine is integrated with the mainstream, then the former will lose its identity and vitamins will become available on "physician's prescription only." The public at large will lose the right to use vitamins as food supplements and the cost of health maintenance will become unaffordable. Let both streams of medicine grow, thereby giving people the right to choose the therapy they want.

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References

1. Sen DR: Does vitamin B_3 really reduce adrenochrome? *J Orthomol Med,* 2012;27:93.
2. Prousky JE: Does vitamin B_3 really reduce adrenochrome? Author responds. *J Orthomol Med,* 2012; 27: 93-94.
3. Hoffer A: The adrenochrome hypothesis of schizophrenia revisited. *J Orthomol Med,* 2009; 24: 160-182.
4. Hoffer A: Thyroid and schizophrenia. *J Orthomol Med,* 2001; 16: 205-212
5. Danziger L: Thyroid therapy of schizophrenia. *Dis Nerv Syst,* 1958; 19: 373-378.
6. Foster HD: *What Really Causes Schizophrenia.* Victoria, BC. Trafford Publishing. 2003.
7. Pataracchia RJ: Orthomolecular treatment for schizophrenia: a review (part 1). *J Orthomol Med,* 2008; 23: 21-28.
8. Pataracchia RJ: Orthomolecular treatment for schizophrenia: a review (part 2). *J Orthomol Med,* 2008; 23: 95-105.
9. Campbell-McBride N: Gut and psychology syndrome. *J Orthomol Med,* 2008; 23: 90-94.
10. Wittenborn JR: A search for responders to niacin supplementation. *Arch Gen Psychiatry,* 1974; 31: 547-552.
11. Hoffer A: Treating chronic schizophrenic patients. *J Orthomol Med,* 2002; 17: 25-41.
12. Kraft BD, Westman EC: Schizophrenia, gluten, and low-carbohydrate, ketogenic diets: a case report and review of the literature. *Nutr Metab* (Lond), 2009; 6: 10.