# Dopamine, Noradrenalin and Adrenalin Metabolism to Methylated or Chrome Indole Derivatives: Two Pathways or one?

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Indoles and their derivatives, the melan-ins, are among the most important compounds in the body, and perhaps the most neglected (Barr, 1983). This is not because they are uninteresting — they are very reactive, are present in every cell and may be involved in most reactions in the body. (Barr, 1983) summarized the data from which he concluded melanins were the organizing molecules which control growth and development and continuing function of the body. Perhaps they have been neglected because they are so common. We can hardly avoid seeing our own and everyone else's melanin. More likely melanins have been neglected because they are too stable, while their immediate precursors are too unstable. Chemists did not have the techniques, skills or patience to work with either the precursors or the final polymers. Simple indoles are derived from dopamine, noradrenalin and adrenalin. These oxidized indoles — dopachrome, noradrenochrome, adrenochrome and adrenolutin — are very reactive, as are all free radicals. Before they can be extracted from body fluids they will have to be trapped or stabilized by converting them into stable compounds. Adrenochrome forms a stable derivative with semi-carbazone. In fact, the first workers who attempted to repeat our adrenochrome psychological studies, not knowing the difference between adrenochrome-semicarbazide and adrenochrome, and not knowing where to obtain adrenochrome, used the stable derivative which is not an hallucinogen. On the contrary, it has very mild anti-anxiety properties (Rinkel, Hyde and Solomon, 1954; Rinkel and Solomon, 1957). Naturally they did not find it to be an hallucinogen. Until these stable derivatives are isolated, the proof of their presence exists in the extraction of another class of stable derivatives, the melanins.

The sympathomimetic amines and their chrome indole derivatives are involved in the schizophrenias, in coronary disease, in aging, in some neurological diseases such as Parkinsonism, and perhaps the organization and control of life itself.

Research scientists in fields other than psychiatry are leading the field (Mason, 1955, 1959). Research dermatologists interested in skin pigment and its diseases have been the pioneers in determining the structure and precursors of melanin. A few clinicians have become excited about the connection between arrhythmias and adrenochrome metabolism, but psychiatric

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interest remains very low. It was killed by two main factors: one political, one scientific. After NIMH, Washington, took a hostile attitude toward the Adrenochrome Hypothesis, it became clear to American investigators there would be little point in applying for grants. One of the major directors of research was advised personally during an on-site visit he could expect no funds unless he changed the direction of his research.

Scientifically, two lines of evidence were used to discredit the adrenochrome work. It was claimed it was not an hallucinogen because Rinkel, Hyde and Solomon (1954) had found adrenochrome semicarbazide not to be an hallucinogen. All the positive studies including the double blind studies in Czechoslovakia were ignored. It was further claimed that all the adrenalin was metabolized by other pathways leaving none to be oxidized into adrenochrome. In one "devastating" study using crude tracer techniques, it was proven that up to 120 percent of the adrenalin was accounted for by non indolic compounds.

In this report I will review briefly the history of the Adrenochrome Hypothesis of schizophrenia and show how it led to a number of interesting developments in medicine and psychiatry.

In 1952 Humphry Osmond joined me in Saskatchewan, bringing with him the observations he and John Smythies had made (Osmond and Smythies, 1952). They had compared the experiences induced in normal subjects by mescaline with the experiences schizophrenia induced in its victims. There were many remarkable similarities and, of course, differences. They realized that if a molecule as simple in structure as mescaline could produce an experience which mimicked something as complex as schizophrenia, a similar compound produced in the body could be the schizophrenic toxin. Using somewhat similar reasoning Osmond and I induced non schizophrenic subjects to excrete a mauve factor when given LSD. Later, the same factor was identified as kryptopyrrole (Irvine, Bayne and Mijashita, 1969; Sohler, Beck and Noval, 1970), and a rapid screening method was developed by Sohler, Hosztynska and Pfeiffer (1974).

Pfeiffer, Sohler, Jenney and Iliev (1974) described the syndrome pyroluria (which Hoffer (1963, 1965, 1966), Hoffer and Mahon (1961), Hoffer and Osmond (1960, 1961, 1963) had termed malvaria) and its treatment with large doses of Pyridoxine and zinc. Kryptopyrrole is toxic (Walker, 1975; Wetterberg, 1972).

Mescalin is a methylated derivative of a phenylethylamine similar to adrenalin. Adrenalin became even more likely as a source when one of their subjects reported that deteriorated (discolored) adrenalin which he had inhaled for his asthma induced a mescalin type experience in him. They suggested that methylated derivatives of adrenalin played a role in the genesis of schizophrenia. This idea was brilliant for it immediately provided a guide to the search for the elusive toxin. Until then, and since, most biochemists devoted their research to examining body fluids of schizophrenics to find anything which could sort out patients from controls. The odds of finding such a substance from the thousands present in the body were and are very low.

But in 1952 the idea was too far in advance of biochemical technology. There were no methods for measuring these methylated derivatives, and none were being sought because it was "known" that methylation could not take place in the body.

The Osmond-Smythies hypothesis pointed toward methylated derivatives as a general class. With Osmond I began to examine the literature for all known hallucinogens; fortunately there were few: d-lysergic acid diethylamide, ibogaine and harmine, all indoles, mescalin, which was an amine which could be indolized, and pink adrenalin, whose structure remained unknown to us. Soon after, Prof. D. Hutcheon, a pharmacologist, joined the Saskatchewan Committee on Schizophrenia Research. He told us it was adrenochrome. Of the five hallucinogens, four were indoles and the fifth might become an indole. We now had another guideline. Search the body for an hallucinogen, an indole derived from adrenalin. Was it adrenochrome, adrenolutin or one or more of their derivatives (Hoffer, Osmond and

Smythies, 1954)?

From the beginning, the idea aroused an inordinate amount of rage among our psychiatric colleagues. Our first grant request from Ottawa was vetoed by Canada's three most distinguished chairmen of departments of psychiatry. They were overruled by an outside referee, Prof. Nolan D.C. Lewis, of Columbia University. The adrenochrome hypothesis directed our attention to three main research areas. If the predictions derived from the hypothesis were confirmed, this would provide support for the hypothesis. Naturally, adrenochrome or its derivatives must be formed in the body, either only in schizophrenics which would make this disease unique, or in everyone but to a greater degree in schizophrenics. It would also follow that adrenochrome would be hallucinogenic. Finally it would follow that reversing the reaction, i.e. preventing the formation of adrenochrome, would be therapeutic.

To examine the first idea we assembled a small team of chemists led by Dr. R. A. Heacock (1959,1965). We discovered how to synthesize crystalline adrenochrome which was stable at room temperature and also its derivative, crystalline adrenolutin. Our group was the first to have available pure adrenochrome but we were not able to isolate and crystallize it from body fluids. In our book, The Hallucinogens**,** (Hoffer and Osmond, 1967), we summarized the evidence which showed all the conditions essential for the formation of adrenochrome were present. These include the substrate, the catecholamines, the enzymes and catalysts necessary for oxidation, and the products of oxidation, the melanins. These are chrome indole polymers.

Recently, Hegedus, Kuttab, Altschule and Nayak (1981) demonstrated the presence of a particular type of melanin, rheomelanin, in serum. It is made from adrenochrome. Most melanins are made from tyrosine and require the enzyme tyrosinase. However reddish neuromelan-ins are present in the brain even in albinos. They are found in areas of the brain rich in noradrenalin, adrenalin, dopamine and serotonin. These chrome indoles may also be formed from aldehydes formed by amine oxidase. These amines may be metabolized by three pathways, but the chrome indoles may come from any one of these pathways. A deficiency of amine oxidase or a surplus of amines would force the amines more directly into the indole pathway. Areas richest in these amines would be most apt to accumulate neuromelanins.

Serotonin interacts with catecholamines and may modulate the oxidation of adrenalin to adrenochrome. VanderWende and Johnson (1970) reported that substantia nigra and caudate nucleus were high in dopamine and serotonin. Dopamine is in the soluble fraction and serbtoninis bound. An excessive release of or failure to bind serotonin would lead to a reduction in formation of melanin. Less melanin is found in Parkinsonism disease. When the concentration of serotonin is lower than the concentration of adrenalin, adrenochrome formation is accelerated. When the ratio is reversed, oxidation is inhibited. VanderWende and Johnson (1970a) point out both must be measured. The ratio is critical. At normal ratios no adrenochrome is formed. When too little serotonin is present, formation of adrenochrome is accelerated.

The amount of amine oxidized to aminochromes is less important than the time during which oxidation occurs. Even a slight conversion will be pathological if long continued and if detoxifying measures are defective. The ratio of amount formed to amount inactivated is critical. If the body lacks antioxidants (free radical scavengers) more adrenochrome will be formed. Increased quantities of melanin would accumulate in all areas where melanin is normally deposited and in some where it is not. Hair and skin should be darker and in many schizophrenics this darkening is quite evident. One of my female patients deposited a yellow-brown pigment in her fingernails and toenails. As she recovered, a clear area appeared in every nail as new, normal nails grew. the demarcation between distal discolored nail and proximal clean nail was the same in every nail. Deposition of melanin had stopped simultaneously in each nail. Chronic schizophrenics do not develop grey hair as often and do develop pigment in their skin as

do pellagrins, especially when treated with chlorpromazine, in sun exposed areas. The relation of pigment in schizophrenic brains relative to normal brains is not known.

A small number of schizophrenics treated with Vitamin B-3 go through a phase when melanin is deposited in their skin, particularly in the flexor surfaces. It resembles an old suntan which begins to fade. The discoloration of skin is not pathological. Parsons (1974) also saw this in a few patients treated with niacin for hypercholesterolemia. He described it as a localized, velvety thickening and tanning of the skin. It is not acanthosis nigricans, a term used by Lipton et al. (1973) who erroneously concluded this after misreading a paper published by Wittenborn, Weber and Brown (1973). The discolored skin is easily removed by gently rubbing the wet skin. It leaves normal skin underneath. It proves excess melanin is deposited in skin, primarily in schizophrenics. No cases were reported by the coronary study on thousands of patients.

Modern scientists investigating melanin no longer dispute its source. Barr (1983), in his thorough review of the evidence, concluded the melanins play a unique and most important role in growth and development. Beamish, Dhillon, Sinsal and Dhalla (1981); Karmazyn, Beamish, Hiesel and Dhalla (1981) and Sinsal, Yates, Beamish and Dhalla (1981) are convinced adrenochrome plays a most important role in heart function. Old age pigment accumulates in heart muscle at a rate of 1/2 percent of the cell volume per decade, and older people may contain 10 percent of pigment by volume in their cells (Strehler, Mark, Mildwan and Gee, 1959). Graham (1978) and Graham, Tiffany, Bell and Gutknecht (1978) discuss the oxidative pathways of catecholamines in the formation of neuromelanins and cytotoxic quinones. Dopamine and 6 hydroxydopamine are toxic after they are converted into their chrome indoles.

Melanin is a remarkably versatile substance; for example, it is a very efficient photon absorbing molecule which responds also to sound. This is why it is present in skin, eyes and the inner ear. Maybe this is how the day/night light ratio controls mood in some cyclothymic individuals. It is a free radical scavenger binding organic and metallic molecules. Metals bound to melanin in hair and skin are removed from the body as hair and skin are shed, much as deciduous trees eliminate unwanted minerals by dropping their leaves. It is an organizing substance present at every stage from the oocyte into the highly pigmented neural crest from which it is dispersed to the central nervous system and the autonomic nervous system. In the growing rat, neuromelanin reaches a stable adult level in 30 days while dopamine and noradrenalin increase from day 1 to day 60. Neuromelanin is not a metabolic waste-basket.

There are three types of melanin: (1) eumelanins derived from tyrosine, black-brown in color as seen in skin, (2) neuromelanin derived from catecholamines, from serotonin and from melatonin, and (3) phaemelanins which contain cysteine and glutathione and are red, yellow, green, blue or violet.

The oxidizing enzymes are tyrosinase, peroxidase and monoamine oxidase. The latter deaminates amines and indole amines to aldehydes which form melanins. Auto-oxidation also occurs, catalyzed by copper and iron. The type of melanin varies with the site. In substantia nigra, dopamine is the major source. In the locus coeruleus, noradrenalin, and in the diffuse brain stem raphe system, serotonin is the chief precursor. These melanins are found in strategic, highly functional areas of the brain. I am puzzled how theorists who support the dopamine hypothesis of schizophrenia can ignore one of the most important and most toxic metabolites, dopachrome, and its melanin.

Absolute proof a substance is present in the body is to extract that substance and to prove it is identical with a synthetic substance of known structure, or to convert it in the body into a stable derivative which can be crystallized and identified. The body has, in fact, carried out the second step and whenever we extract melanin we are proving the presence of adrenochrome in the body, even if it has a transient life.

Hegedus and Altschule (1968, 1970, 1970a), in a series of reports concluded that

adrenalin, adrenochrome and adrenolutin incubated in blood formed rheomelanins and hemolyzed erythrocytes. They concluded the indole pathway was available in blood and, even more, that erythrocytes from schizophrenics were abnormally susceptible to the hemolytic effect of rheomelanins or precursors formed from aminochromes. Blood contains cells which metabolize adrenalin to adrenochrome as the major pathway.

Matthews, Hallett, Henderson and Campbell (1985) reported that over 80 percent of the adrenalin was oxidized to adrenochrome by polymorphonuclear leukocytes stimulated by latex beads or by the peptide N formylmetleuphe. Oxidation began in five minutes and continued for four hours. This was the favored pathway over amine oxidase and catechol O methyltransferase. They also found that serum from patients drawn after myocardial infarction induced more oxidation than control serum. This provides a cellular mechanism for adrenochrome formation in inflammatory conditions. Perhaps the oxidized catecholamine free radicals are used to destroy bacteria. Adrenochrome is also an antimitotic poison.

It is likely the proportion of catecholamines converted to aminochromes varies enormously from 1 to 2 percent to over 80 percent, depending upon the tissues where this oxidation occurs. In the synapse this oxidation is dangerous, as quinones, an intermediate, are toxic. Liang, Plotsky and Adams (1977) found that 6 hydroxy dopamine reacts with central nervous system tissue in vivo to form paraquinone. Tse, McCreery and Adams (1976) reported catecholamines were oxidized in vitro using conditions comparable to human brain, and Cheng (1984) suggested that paraquinone from 6 hydroxy dopamine was an important toxin causing destruction of neural tissue. Is adrenochrome formation a way of removing more toxic paraquinones?

It is possible the aminochromes, once formed, increase the formation of more by inhibiting catechol O methyl transferase (Borchardt, 1975).

The adrenalin/adrenochrome pathway has become an important area for study in heart disease. Yates, Beamish and Dhalla (1981), Beamish, Dhillon, Sinsal and Dhalla (1981) and Yates, Taam, Sinsal, Beamish and Dhalla (1980) showed adrenochrome is partly responsible for myocardial necrosis and failure following massive injection of adrenalin. Sulfinpyrazone (Anturan) was reported by the Anturan Reinfarction Trail investigators to decrease cardiac mortality after the first infarction. It does so by abolishing adrenochrome induced arrhythmias.

Altieri and Inchiosa (1980) reported adrenochrome was a potent inhibitor of myosin, actomyosin and myofibrillar ATPase at physiological levels and pH. There was no effect on cerebral cortex ATPase. It is formed in smooth muscle and is part of the mechanism by which adrenalin relaxes certain smooth muscles.

Wortsman, Frank and Cryer (1984) recorded extraordinarily high plasma levels of adrenalin in patients resuscitated from cardiac arrest. Adrenalin levels rose 300 times while noradrenalin rose 10 times. Increases also occur in hypoglycemia and hemorrhagic shock.

The exact connection between schizophrenia and oxidized catecholamine metabolites has not been studied. It is unlikely they are specific to schizophrenia. Most likely due to a combination of factors such as a deficiency of reducing substances or anti oxidants, an excess of oxidizing metals or enzymes and an overload of catecholamines. The increased amount of adrenochrome and like substances can not be detoxified fast enough, leading to an accumulation of adrenochrome and its metabolites. One does not even need to postulate higher concentrations of adrenochrome will be found. The increased amount of adrenochrome may be removed or destroyed just as quickly, leaving damaged molecules or an accumulation of its metabolites such as melanin. The oxidation of activated adrenalin at the synapse will destroy the receptors. This is why dopamine is toxic. If dopamine were stabilized so it could not be oxidized to dopachrome, I doubt it would destroy neuron receptors. The destruction occurs by the interaction between the oxidized aminochrome and the chemicals on the surface of the receptors. Walaas (1967) and

Walaas and Walaas (1961) have shown that the oxidation of adrenalin to adrenochrome is accelerated in synapses when there is a deficiency of Vitamin B-3 as nicotinamide adenine dinucleotide.

Niacin is a broad-spectrum hypocholesterolemic substance which decreases cholesterol (Altschul, Hoffer and Stephen, 1955), triglycerides, low density and very low density lipoprotein cholesterol, and elevates high density lipoprotein cholesterol. The huge coronary drug study (1975) compared five substances: placebo, estrogen, thyroid, clofibrate and niacin. Patients were treated for nine years, the study ending in 1976 or so. Niacin, at the end of the study, was superior to the other four treatments, but there was no difference in the death rate. The state of the patients was reexamined beginning in 1982. Recently, Canner (1985) reported that niacin decreased the death rate by 11 percent. Clofibrate and placebo were the same and estrogen and thyroid had increased it and had been discontinued during the original study. What was surprising was this beneficial effect even after treatment had been discontinued many years before. In sharp contrast, Dr. E. Boyle (1968) found that his patients on niacin continually for ten years after their first coronary suffered only 10 percent mortality rather than the expected 90 percent. Niacin could prevent the excessive formation of adrenochrome and would antidote the toxic effect of adrenochrome on myocardial tissue, much as it antagonized the cerebral arrhythmia measured on the electroencephalograph when adrenochrome was given intravenously.

**Is Adrenochrome an Hallucinogen?**

We have reviewed the evidence adrenochrome is an hallucinogen (Hoffer, 1962; Hoffer and Osmond, 1967). This has been established by open clinical studies and by double blind controlled studies; especially noteworthy are studies by Grof, Vojte-chovsky and Horackova (1961), Vojte-chovsky, Grof and Vitek (1962) and Grof, Vojtechovsky, Vitek and Prankova (1963). It is also active in altering animal behavior. We saw this in 1952 when we measured the toxicity of adrenochrome in rats. Weckowicz (1962) did his Ph. D. thesis on the effect of adrenochrome on learning in albino rats. It has altered behavior in other animals as well, including spiders, cats, monkeys and pigeons. Taborsky (1968) found adrenochrome and adrenolutin reduced the work rate of rats. Anything which blocked the 3 hydroxyl group such as 5, 6 dihydroxy N methyl indole removed toxicity.

Adrenochrome produced psychotomimetic reactions which mimicked schizophrenia more closely than does LSD. The usual perceptual changes were illusions but with heavier doses hallucinations were present. Thought disorder was common with loss of insight and paranoid delusions. Depression was very common. When I last took adrenochrome I was depressed for two weeks. I have never been depressed before or since.. We concluded adrenochrome was a dangerous hallucinogen. It did not produce any psychedelic reactions and will never be the darling of the streets as was LSD.

There is evidence LSD will not produce its typical effect in the absence of enough adrenochrome. We had given LSD as a treatment to several thousand alcoholics. Our standard initial dose was 200 to 300 micrograms. Yet many alcoholics needed a lot more. In a small series of cases we gave alcoholics this amount of LSD and when the only response after two hours was increased anxiety, we gave them intravenous adrenochrome. Within ten minutes they had the psychedelic reaction we wanted them to have. There were no other changes in the test situation. The most recent reviews are by Heacock (1971) and Hoffer (1981).

It is known that up to 25 percent of patients with Parkinsonism given 1-dopa in larger quantities become psychotic, i.e. develop perceptual symptoms and thought disorder. With lower doses a smaller proportion become psychotic. Graham (1978,1979) and Graham, Tiffany, Bell and Gutknecht (1978) are convinced a proportion of catecholamines were oxidized to' quinones to aminochromes to neurome-lanin. This is so slow that little is found in brain until age 6. 6 hydroxy dopamine is toxic because it is converted to trihydrox-

yphenylalanine (TOPA) which is converted to aminochrome. Thus, while it benefits the patient by moderating the symptoms, it accelerates the destruction of dopamine receptors. Yaryura-Tobias and Diamond (1971) found that niacin protected Parkinsonism cases against 1-dopa psychosis; I have also found this, but it did not help their neurological symptoms. L-dopa decreases the conversion of tryptophan to NAD. Bender, Eall and Lees (1979) found that patients on 1-dopa were as low in Vitamin B-3 and in its metabolites as were pellagrins.

Adrenochrome increases the arrhythmia normally found in epileptics when measured by the surface EEG, and this is reversed by niacin (Szatmari, Hoffer and Schneider, 1955). It also changes the normal patterns of the depth electroencephalogram as do mescalin and LSD (Schwarz, Sem-Jacobsen and Peterson, 1956).

**Adrenochrome Hypothesis and Treatment of Schizophrenia**

In 1951 there were only three acceptable treatments for schizophrenia: (1) psychotherapy based upon psychoanalysis, which did not work, (2) insulin coma which was rapidly being displaced by electroconvulsive therapy and, (3) ECT which aborted many acute psychotic reactions but in the long run did not change the course of the illness very much. There was nothing else on the horizon except for a few workers who were investigating steroids, histamine, thyroid and the newly discovered antihistamines. Pellagrologists had found that a few patients in southern mental hospitals recovered when given niacin, but they were promptly rediagnosed pellagrins, thus retaining the sacred principle that only pellagrins responded to this vitamin, Vitamin B-3. We confirmed that histamine treatment helped many acute patients, but it was no more practical to use it on a sustaining basis than it was to use insulin coma or ECT for chronic control of psychosis.

Humphry Osmond and I agreed we should base any new treatment on our adrenochrome hypothesis for two reasons. If such a treatment were successful it would bolster the hypothesis, but much more important, we would have a way of helping our patients. We decided to use compounds which were safe, available, could be taken easily and which would prevent or decrease the oxidation of adrenalin to adrenochrome. This was the first example of antioxidant therapy applied to medicine. We hoped that Vitamin B-3 would decrease the production of adrenochrome or prevent its toxic effect on the respiratory enzymes. Adrenochrome was known to be a powerful inhibitor of these enzymes. We also hoped that ascorbic acid, as one of nature's best water-soluble reducing substances, would act in a like manner.

Both vitamins were and are safe and fit our requirements for a treatment which could be taken for a lifetime if necessary. But in order to be effective we concluded we would have to use doses larger than those already reported, for if smaller doses had been effective this would have been reported. We decided to begin with 3 to 10 grams per day of each vitamin.

A few preliminary studies soon showed these doses were safe and acceptable to patients. Our first pilot study included eight severely ill acute cases, two under my care and six under Dr. Osmond's care at a mental hospital. All had not responded to previous treatment. All recovered on these vitamins. I am still surprised. Perhaps in 1952 schizophrenics were not as badly damaged by high-tech nutrition as they are today. Pritchard (1967) found no difference in outcome between patients treated before and after tranquilizers were introduced, but Bockoven and Solomon (1975) concluded schizophrenics treated between 1947 and 1952 had a better long-term outlook then patients treated with tranquilizers between 1967 and 1972. They concluded, "psychotropic drugs may not be indispensible to the success of community-based mental health services and that their extended use in aftercare may prolong the social dependency of many discharged patients." They were, in fact, made as dependent then as they are now on tranquilizers alone. It is impossible to function normally in any activity requiring energy, initiative and concentration on tranquilizers alone. Would you allow a heavily tranquillized surgeon to operate on your heart? Two

factors have made the outcome worse: (1) malnutrition and, (2) tranquilizers alone.

We started a double dummy experiment to test our ideas. These experiments had been done in England but had not yet come across to North America. In fact the first report in North America that tranquilizers were helpful in the treatment of manic states were open clinical experiments. According to modern clinical scientific dogma, these results should not have been published, should not have been read and should not have been believed because they were anecdotal — that evil way of making observations. Yet H. Lehmann became the North American pioneer in introducing tranquilizers because of one anecdotal report. Thank God double blind theorists did not exist then. By the time we finished our experiment it had become "double blind," overthrowing the British term "double dummy."

In our experiment the addition of 3 grams per day of either niacin or niacinamide to the treatment then available doubled the one year cure rate of acute patients from the placebo rate of 35 percent, to 70 percent. Three more double blind controlled experiments gave us similar results. We also found and reported in 1955 that chronic patients did not respond to Vitamin B-3 even in doses of 10 grams per day (Hoffer and Osmond, 1963, 1964, 1966; Hoffer, Osmond, Callbeck and Kahan, 1957; Osmond and Hoffer 1962, 1967, 1978). Our method was described by Clancy, Hoffer, Lucy, Osmond, Smythies and Stefaniuk (1954).

Our main conclusions were:

1. That acute patients doubled their one year recovery rate.
2. That chronic patients did not.

These findings have been confirmed by every study reported since then. Thus the Montreal group using only chronic patients labelled newly admitted obtained mixed results. Generally they did not respond, but they ignored evidence of improvement. The New Jersey studies were also mixed. The initial report on chronic patients was negative. However, Dr. J. Wittenborn (1974) later reviewed his data, pulled out 24 patients who were less chronic, less ill, and found that compared to placebo about 70 percent had responded, i.e. double the placebo rate. Every psychiatrist since 1960 has been able to find similar responses, provided they took the pains to follow the basic treatment protocols. So far I have not known any psychiatrists who, having used the nutritional approach for at least one year, have given it up, in spite of massive harassment by local, state and national psychiatric establishments. There is one exception — a northeastern psychiatrist gave up all use of vitamins after six months because if he had not, he would have lost his hospital privileges, his practice, his friends and his family. He, in the end, merely lost his self-respect, for in his sign-off letter to me he bemoaned the fact he was not permitted to treat his schizophrenic patients in the most efficacious way.

Vitamin therapy is advancing very quickly because it works, even when there are no drug companies to push it, and arrayed against it are all the establishment organizations including nutritionists, psychiatrists, psychologists and social workers. Our main support has come from the thousands of families hit by schizophrenia who have compared results when their relatives were on tranquilizers only and then treated more effectively with Orthomolecular therapy. Politics, not science, has inhibited the advance of Orthomolecular therapy, and it is politics as practiced by our patients which will force you to eventually become Orthomolecular psychiatrists. Until then, our poor schizophrenics will be treated in tranquilizer institutions or condemned to the streets, while the wealthy will be able to seek out Orthomolecular hospitals and practitioners and be given their chance for recovery.

Treatment today is much more than Vitamin B-3 and ascorbic acid, even though these two vitamins remain basic components. Treatment today is to our original treatment as a Japanese Cressida is to a Model T Ford. This does not mean the Adrenochrome Hypothesis is proved, but it does provide further support; it is a viable theory and must continue to be examined.

**Conclusion**

The Adrenochrome Hypothesis has been tested experimentally by a large number

of scientists since it was first reported in 1952 in New York before the Dementia Praecox Committee of the Scottish Rites Masons. Three major sub-hypotheses have been confirmed: it is made in the body, it is an hallucinogen and its antidotes are therapeutic for schizophrenia. What must still be done is to determine how schizophrenics and normals differ in the way they deal with the catecholamine/aminochrome pathways.

Schizophrenia is a syndrome, the Adrenochrome Hypothesis may account for the final clinical picture but the abnormal changes are triggered by a number of factors such as cerebral allergies, vitamin dependencies and deficiencies, and so on. The essential fatty acids (Rudin, 1981,1982) and the prostaglandins are involved (Horrobin, 1977,1979). One day all these factors will be examined and co-related so that we will have a clear picture of what happens when a person becomes schizophrenic (Hoffer, 1981).

All three known pathways by which the catecholamines are metabolized play a role in the body. What we must know is how they are related to each other and what are the factors which direct these pathways.

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# Letters

**Dear Dr. Hoffer:**

What a wealth of information the Second Quarter 1985 issue of the *Journal* contains! From your editorial about the Mayo Clinic study of vitamin C as an anti-cancer factor and article about desiccated thyroid and breast cancer, to A.R. Patton's comment that, in testing the effect of glutamine, researchers substituted glutamic acid, this issue cautions that investigators must adhere to correct protocols.

> Similar carelessness is evident throughout medical literature. For example, Bennett et al. (1983) published negative results in his replication of Dr. Ruth Harrell's study (1981) of the GTC Formula to treat mentally retarded children. The GTC Formula, based on a nutritional combination developed by Dr. Mary Allen, and on Dr. Roger Williams' genetotrophic concept, was supplemented with thyroid if indicated by the Barnes test. However, Bennett et al. decided to exclude thyroid because all the Down syndrome children were "euthyroid" — although 25 percent of them had elevated serum TSH, indicating a hypothyroid condition, and although the investigators noted that Harrell had "given thyroid hormone [to nearly all subjects] without first ascertaining thyroid function by currently standard blood measures." Others who tried to replicate the Harrell study likewise eliminated thyroid. Yet, during my 40 years of work with Down syndrome patients I have found that they almost invariably benefit from thyroid supplementation (1962,1963).

However, I was surprised and disappointed not to find my work or Dr. Harrell's mentioned in the text or 112 references of an article, "Effect of Nutrient Supplements" in the same issue. No doubt the authors realized the need to end their extensive list of references. Nevertheless, I believe that ongoing therapeutic methodology of more than 40 years' duration in the treatment of borderline retarded (1978,1984) as well as Down syndrome patients (1975) should have been mentioned. In fact, it appeared in Dr. Bernard Rimland's chapter (1973), which was cited.

I began to treat retarded children, especially those with Down syndrome, in the early 1940's. Their massive metabolic accumulations, now attributed to the trisomy, were readily apparent to me. I used the nutritional/medical method of the "U" Series to help remove them and to provide nutrients to malnourished, underdeveloped tissues. The article by Dr. Boggs and

coworkers pointed out that nutrients can remove deleterious substances stored in tissues, and this is the rationale underlying the "U" Series treatment. The "U" Series was also, as far as I know, the first treatment method to use vitamin C for its effect on the immune system, to reduce levels of histamine, and as a mild diuretic to reduce brain swelling in certain types of retardation. When they were developed, antihistamines were added to the "U" Series.

In short, while I do not wish to fault this impressive work, I do think that an article about nutrients and retardation should at least. mention the "U" Series and GTC Formula.

Sincerely,

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## To the Editor

I have been in the practice of preventive and Orthomolecular medicine for the past ten years. In 1975, I visited the medical clinics of renowned Orthomolecular practitioners, notably Drs. Elizabeth Rees, Theron Randolph, Allan Cott and Abram Hoffer. I also went to the Brain Biocenter run by Dr. Carl Pfeiffer and his associates, the Huxley Institute for Biosocial Research with Drs. David Hawkins, Mollie Shriftman, and the American Biologies in Tijuana, Mexico.

I cannot thank them enough for opening the doors for me into this fascinating field of medicine. Up to the present, I am still the only Orthomolecular practitioner and sole member of the international Orthomolecular organizations from the Phillippines. My patients tell me that they are worried that I do not have a follower or an understudy to take over my work. While there are a number of therapists who may be interested in this field, they have not tried prescribing megadoses of vitamins and supplements to their patients. And we all know the reasons why.

My contacts with Orthomolecular pioneers gave me much encouragement and confidence to deal with my patients. Because of my experience seeing my patients recover with megavitamin therapy, my life has been more fulfilled and enriched. In the beginning, I just could not possibly believe how people could hear strange voices, see uncanny visions and have paranoid delusions, and how, within a short span of meganutrient treatment, these could disappear. And yet, the patients themselves give oral and written testimonies as to their recovery. These experiences make me and my patients' families very happy.

As I go along in my practice, I meet all kinds of diseases. Each day brings me new learning from my patients, new sufferings, new ideas, new treatments which are both challenging and fulfilling. What interested me most are the patients who were diagnosed as epileptics. I discovered that most of these patients were either hypoglycemics or vitamin B6 and magnesium dependent, or were allergic to some food

or chemicals which cause seizures that were not even grand or petit mal.

My technique of diagnosis is very simple. I only use my stethoscope, the blood pressure apparatus, the Vitamin C test deficiency. I use the HOD test to identify symptoms common among hypoglycemics and schizophrenics. To verify if the patient is telling the truth, I give Dr. Tavel's symptometer. This takes a lot of time but the patients enjoy answering these tests. Physical examination is quite simple and I give emphasis to the nail, hair, skin, tongue, stretch marks, weight, and blood pressure. If after one month of mega supplements the patient only has very little improvement, I advise the patient to have some laboratory work such as the Oral Glucose Tolerance Test (OGTT), thyroid or adrenal studies and CBC. Very seldom do I recommend that patients take an ECG or EEG or scanning and X-rays. I send some samples of the patient's hair to America for mineral toxicity and imbalance tests. If patients are very ill and have relatives or friends in North America, I refer them to our associates there to help me and the patients.

I am indeed very happy to tell my colleagues abroad that I have handled a lot of degenerative diseases like arthritis, allergy, post MI or CVA, cancer, psoriasis, multiple sclerosis, learning disabilities of children, kidney or gall bladder stones that have been dissolved successfully with meganutrient therapy.

One of my most gratifying successes in this field is the rapid improvement of the behaviour and speech of the children who are in special schools. The teachers refer them to me and many more are coming because the teachers as well as the parents are so pleased with the change of the behavior, speech and habits of these kids.

My practice is not all roses, however. I have been insulted by my colleagues, particularly the psychiatrists and neurologists even in the presence of the patient. One even said "who believes in the vitamins and the tests that Verzosa is giving? Who believes in Linus Pauling?" My answer at that time was — "What is wrong if I can help? I am not encroaching on your practice. If patients like me, let them have me; if they like you, you can have them." One nice incident was about a famous neurologist who was a columnist on health in one of our daily papers. One time he attacked the use of vitamins. I negated his arguments; he fought back. We had three printed rebuttals in the daily. Instead of continuing the fight, I sent Dr. Abram Hoffer the prints. Dr. Hoffer wrote the publisher to clarify our stand, and had his letter published. That ended our rather unpleasant exchange. That somehow clearly defined the polarity between our traditional therapists and me, as an advocate of meganutrient therapy.

I do not advertise as a therapist. I have often been invited, however, to speak on television and radio broadcasts, and to speak before big and small groups like the Rotarians and the Lions Club. Several reporters have interviewed me and what I do has been published in several daily papers and magazines. It is from these sources that I get patients, other than the referrals from recovered patients.

What more can I ask of my seventy years of life and ten years as an Orthomolecular therapist? I have one more dream — to be able to put up a center for living, loving, learning and sharing, a health institute where I can have patients rehabilitated from their chronic or painful ailments, teach them to be more useful and self-sufficient even in their illness, and when the time comes, to learn to die a happy death with dignity and acceptance.

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