

# The Treatment of Alcoholism with Vitamin B<sub>3</sub>

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**Abstract** *The consequences of excessive and prolonged use of alcohol leads to mild-to-severe forms of pellagra (i.e., some combination of diarrhea, dermatitis, dementia, and possibly death). The author describes the work of many notable individuals who reported on the clinical effectiveness of vitamin B<sub>3</sub> treatment for compulsive drinking behaviour, alcohol withdrawal delirium, and for improving sobriety. There is definitely a significant therapeutic role that vitamin B<sub>3</sub> could play (mostly, niacin) if it was part of the mainstream approach to treating alcoholism and its related complications.*

## Introduction

The global prevalence of alcohol use disorders (AUD; also denoted as alcoholism in the published literature) in adults up to 65 years of age over a 12-month period of time is 3.6%; males and females accounting for 6.3% and 0.9%, respectively.<sup>1,2</sup> Heavy drinking is defined as consuming more than four drinks daily or 14 drinks each week for males, and for females it is defined as more than three drinks daily or seven drinks each week.<sup>3</sup> The estimates show that one out of four heavy drinkers has alcohol-related dependence or abuse problems.<sup>3</sup> Some 18 million people in the United States have AUD, which are characterized by symptoms that include: (1) craving – having a strong need or urge to drink; (2) loss of control – not being able to stop drinking once it has begun; (3) dependence – withdrawal reactions like nausea, sweating, shakiness, and distressing emotional states such as anxiety, once drinking has been stopped; and (4) tolerance – needing increasing amounts of alcohol to feel the same desired effects.<sup>4</sup>

There are numerous whole-body impairments that result from AUD. Two of the

most affected include the nervous system and nutrient metabolism, even though the cardiovascular (e.g., arrhythmia, hypertension, and stroke), immune (e.g., pneumonia), and gastrointestinal systems (e.g., chronic pancreatitis) might also be significantly affected by the excessive and prolonged use of alcohol. The nervous system problems involve the following: intoxication (poor physical coordination); memory loss (due to alcohol-inhibition of the glutamate receptor, known as the N-methyl-D-aspartic acid/NMDA receptor) leading to blackouts and amnesia; tolerance (loss of the ability to become intoxicated); uncontrolled craving (addiction); and withdrawal symptoms leading to seizures and tremors.<sup>5</sup>

Among patients with AUD, nutrient metabolism problems result from the replacement of normal calories from foods with alcohol, which can induce malnutrition, liver damage (e.g., fatty liver, hepatitis, fibrosis, and cirrhosis), as well as hypoglycemia.<sup>5</sup> Proper nutritional treatments are needed to compensate for the numerous micronutrient deficiencies resulting from the alcohol-induced metabolic problems. While

there are undoubtedly many micronutrients that would be required to assist individuals with AUD, vitamin B<sub>3</sub> (i.e., niacin/nicotinic acid or niacinamide/nicotinamide) appears to be among the most important because the consequences of excessive and prolonged use of alcohol leads to mild-to-severe forms of pellagra (i.e., some combination of diarrhea, dermatitis, dementia, and possibly death). In this article, I will summarize and comment on the following: (1) research showing a connection between AUD and pellagra; (2) hypothetical research that has linked a deficiency of the vitamin to compulsive drinking behaviour; and (3) clinical research pertaining to vitamin B<sub>3</sub> and AUD.

### Connection between AUD and Pellagra

Pellagra is a disease caused by a cellular deficiency of the nicotinamide coenzymes due to inadequate dietary supply of tryptophan and vitamin B<sub>3</sub>. Diarrhea, dermatitis and dementia characterize the 3 Ds of this deficiency disease. Although is it not usually fatal, when all of these deficiency manifestations are present death can occur if the disease remains untreated, so in a sense pellagra could be characterized by having 4Ds or four defining clinical manifestations.

The adult intake of vitamin B<sub>3</sub> necessary to prevent pellagra is likely somewhere between 12 and 16 mg per day since these doses have been shown to normalize the urinary excretion of niacin metabolites.<sup>6</sup> The body can manufacture approximately 1 mg of niacin equivalents from 60 mg of tryptophan obtained mostly from dietary protein.<sup>7</sup> This *in vivo* conversion makes it rather difficult to develop frank pellagra in affluent, industrialized countries where food supply is seldom scarce unless there are mitigating factors like the excessive and prolonged use of alcohol.

Pellagra causes some very notable whole-body system changes (as mentioned previously), which explains why any person with AUD would eventually develop some combination of skin, gastrointestinal, and neuropsychiatric signs and symptoms. Badawy<sup>8</sup> has written an extensive report discussing pellagra and alcoholism, and notes that the

essential reason why AUD causes pellagra is simply the result of malnutrition, gastrointestinal damage, and B-complex vitamin deficiencies. In Badawy's report, the nutritional, biochemical, and physiological mechanisms in alcoholic pellagra are described, and are summarized (as adapted from his work) in **Table 1** (p.125).

There is nothing particularly novel about the association between pellagra and AUD (e.g., see references 9-12). I have included two reports to demonstrate how pellagra can present when due to AUD (**Table 2**, p.125).

It is worth highlighting some additional mechanisms that account for the psychiatric symptoms that often result from pellagra associated with AUD. The symptoms of depression and dysphoria are linked to impaired cerebral serotonin synthesis due to less tryptophan being available (i.e., from protein malnutrition), and possibly from reduced decarboxylation of 5-hydroxytryptophan (i.e., secondary to a functional vitamin B<sub>6</sub> deficiency).<sup>8</sup> Anxiety is also believed to be partly mediated by impaired cerebral serotonin synthesis, but also from 5-ALA inhibition of GABA neuronal release.<sup>8</sup> Psychosis is believed to be the result of neurotoxicity, NMDA-receptor antagonism, and the inhibition of GABA neuronal release.<sup>8</sup>

Even though dementia is often considered one of the hallmark features of pellagra, its neuropsychiatric presentation when associated with AUD is likely to be more consistent with delirium, in addition to the psychiatric symptoms discussed previously. One report suggested that the 3Ds of pellagra should really be considered diarrhea, dermatitis, and delirium (and not dementia).<sup>13</sup> Other neurological features of pellagra associated with AUD include an assortment of extrapyramidal symptoms.<sup>13</sup>

### Vitamin B<sub>3</sub> Deprivation and Compulsive Drinking Behaviour

The late John P. Cleary, MD, believed that AUD were among a category of diseases known as the NAD Deficiency Diseases (NAD-DD).<sup>14-15</sup> NAD-DD result from long-term, sub-optimal intake of vitamin B<sub>3</sub>,

**Table 1.** Alcoholic Pellagra and Associated Mechanisms

Nutritional	Biochemical	Physiological
<ul style="list-style-type: none"> <li>* Deficiencies of B-complex vitamins, zinc, and protein</li> <li>* Defective absorption and metabolism of nutrients</li> </ul>	<ul style="list-style-type: none"> <li>* Dysfunction of glutamate and gamma-aminobutyric acid (GABA) neuronal function</li> </ul>	<ul style="list-style-type: none"> <li>* Impaired liver tryptophan 2,3-dioxygenase and synthesis of niacin precursors</li> <li>* Deactivation of pyridoxal 5'-phosphate by acetaldehyde</li> <li>* Diminished synthesis of NAD<sup>+</sup> cofactors</li> <li>* Build-up of the porphyrin precursor, 5-aminolaevulinic acid (5-ALA)</li> </ul>

**Table 2.** Two Case Reports of Patients that Developed Pellagra from Alcohol

Reference	Brief Case Description	Treatment	Outcome
9	40-year-old female was admitted to the neurological department with "rapidly evolving ataxia of gait and diplopia." Prior to this, she had 3 episodes of dizziness and a sun-induced rash on her lower legs, which eventually spread to her arms, hands and head. She reported drinking large amounts of wine.	Intramuscular injection containing vitamins B <sub>1</sub> , B <sub>6</sub> , and B <sub>12</sub> (the specific dose of each B vitamin and the duration of treatment were unspecified). The patient was given an oral multiple vitamin supplement that contained vitamin B <sub>3</sub> (doses were unspecified).	The patient's neurological symptoms disappeared within 24 hours. Her rash improved and was discharged (length of stay not mentioned) with instructions to continue with the multiple vitamin supplement.
12	40-year-old female with alcohol-dependent syndrome presented with "well-demarcated hyperpigmented dry scaly lesions with a burnt appearance on the dorsa of both hands extending to the forearms" for the past 2 months. She also had anorexia, nausea, cheilitis, glossitis, and redness and swelling of the buccal mucosa. Other complaints included burning feet sensations causing insomnia and a phobia related to walking, tiredness, and a loss of interest in daily activities.	500 mg once daily of oral niacin, in combination with unspecified amounts of multivitamin injections, iron, and protein	The cutaneous and mucosal lesions began clearing with treatment, and the neuropsychiatric symptoms improved within a couple of weeks. The patient was then referred to the cancer institute for management of carcinoma of the cheek.

which leads to a deficiency of NAD, and results in diseases or unwanted behaviors and addictions geared towards the filling of unoccupied NAD receptor sites. The principle treatment for the NAD-DD is the administration of optimal amounts of vitamin B<sub>3</sub> in order to cover the NAD receptor sites and shut-off the vicious addiction-withdrawal cycle.

Cleary reported on various aspects of AUD, such as the craving for alcohol, the relationship between opioids and NAD receptor sites, and the need for niacin as a means to withdrawal and abstain from alcohol.<sup>14</sup> According to Cleary, alcohol consumption increases the formation of acetaldehyde, which then combines with dopamine to form morphine-like compounds that fill brain NAD receptor sites and temporarily shut-off withdrawal symptoms. When these receptor sites become unoccupied or unbound, withdrawal symptoms occur, and the craving for alcohol begins once again. Cleary reports that it is possible to stop the addiction-withdrawal cycle by substituting alcohol with optimal amounts of niacin. The net effect is a reduction of drinking behaviors, cravings and withdrawal symptoms via the occupation of the NAD receptor sites as well as the reduction of acetaldehyde levels. Overall, he believed that niacin could cure the patient's addiction to alcohol by correcting the underlying NAD deficiency.

In one of these reports, Cleary noted that, "...nicotinic acid given orally in dosages of 500 mg per day will not only relieve acute intoxication but permanently relieve alcohol addiction."<sup>14</sup> In one of Cleary's last report on the subject, he provided the following findings from his own clinical experience:

"In 1980, I was treating a 60-year old man for hyperlipidemia with niacin 500 mg daily and he came back after one month and said he felt much better and had stopped drinking three to four pitchers of beer every night to get to sleep. A pilot study of 12 patients was done with 11 out of 12 getting off alcohol painlessly in three or four weeks on 500 mg daily."<sup>16</sup>

Unfortunately, Cleary never published the specific details of the pilot study, or detailed patient reports describing his clinical experiences with niacin for AUD. From my perspec-

tive and based on the paucity of evidence, his views are more hypothesis-generating than proof that niacin arrests compulsive drinking behaviour. I actually doubt that niacin can "cure" alcoholism since maintaining sobriety depends on so many variables that extend well beyond biochemical alterations.

## **Clinical Research Pertaining to Vitamin B<sub>3</sub> and AUD**

### **Alcohol withdrawal delirium**

The mortality and morbidity associated with alcohol withdrawal delirium is significant.<sup>13</sup> Dr. Abram Hoffer wrote about the "Toxic Psychosis Associated With Alcoholism" in 1962, and described niacin deficiency encephalopathy as a syndrome "characterized by clouding of consciousness, cogwheel rigidity of the extremities and uncontrolled grasping and sucking reflexes."<sup>17</sup> In the data cited by Hoffer, the mortality rate dropped from 95% to 15% when pure niacin was included as part of the standard treatment.

With respect to the treatment of delirium tremens, Hoffer noted the following about using niacin:

"I have been able to bring many alcoholics with severe tremor out of the tremor very quickly by giving them one gram of nicotinic acid only, by mouth. This has helped several alcoholics to terminate a severe alcoholic bout"<sup>17</sup>

Hoffer, in collaboration with Dr. Humphry Osmond, developed a protocol for patients in 1953 that were either experiencing delirium tremens or were in a "pre-delirium" (i.e., niacin deficiency encephalopathy) state (Table 3, p.127).<sup>17</sup>

Hoffer published some detailed cases of recovery. I have included one complete case description to demonstrate the effectiveness of this approach (Table 4, p.127).<sup>17</sup>

Hoffer also published a summary of the treatment results obtained from niacin and ascorbic acid for niacin deficiency encephalopathy and delirium tremens (Table 5, p.127).<sup>17</sup>

Treatment was maintained until the patient's symptoms ameliorated. The best outcomes were achieved when optimal doses

**Table 3.** The Hoffer and Osmond Treatment for Delirium Tremens or for Patients in a Predeliroid State

On Admission	The First Day
Niacin, 400 mg intravenously Niacin, 3 grams oral Ascorbic acid, 2 grams oral	Niacin, 3 grams three times daily Ascorbic acid, 3 grams three times daily Chloral hydrate for sleep if necessary

**Table 4.** Case of Niacin Deficiency Encephalopathy

Mr. T.F. (November 1 to December 7, 1955) (Age 49, Male, Married)

This man began to drink in 1939 and had had three bouts of delirium tremens. In 1950, he was abstinent for four years while in AA but he started drinking again three weeks before admission and was brought in by friends drunk and delirious. He has been having visual hallucinations for about four days. These consisted of peculiar men and women but also an unusual animal which he had never seen before which bedded down with him. He was irritable and tense and had been beaten up receiving two black eyes. He was given two grams of nicotinic acid and two grams ascorbic acid t.i.d. by mouth at 6:00 p.m. and by 10:30 p.m. he was cooperative, pleasant and related well. The next day, although jittery and tense, he ate a good lunch, and described how badly he felt on the day of admission. He has stayed well from this day on.

**Table 5.** Treatment Results from Niacin and Ascorbic Acid for Niacin Deficiency Encephalopathy and Delirium Tremens

Patient Identifier	Age	Diagnosis	Clinical Outcome
T.F.	49	Delirium tremens	Recovery in 24 hours
M.M.	26	Delirium tremens	Recovery in 4 days
A.T.	38	Alcoholic tremor	Recovery in 24 hours
E.S.	42	Alcoholic intoxication	Recovery in 24 hours
S.D.	31	Delirium tremens	Recovery in 4 days
A.M.	34	Alcoholic intoxication	Recovery in 24 hours
N.C.	34	Alcoholic intoxication	Recovery in 24 hours

of niacin were given soon after admission, which tends to diminish delirium tremens or niacin deficiency encephalopathy within twenty-four hours. This method has the added advantage of not causing sedation or

toxic complications that can happen when other medications are used, such as the major tranquilizers (i.e., neuroleptics).

A more modern report has confirmed the findings of Hoffer and Osmond. In this

report, the authors stress the importance of considering niacin deficiency encephalopathy (a.k.a., pellagrous encephalopathy) when other measures have not been successful (e.g., large doses of benzodiazepines and other sedatives) in resolving alcohol withdrawal with associated delirium.<sup>13</sup> They describe three patient cases in which measures to resolve alcohol withdrawal delirium remained unsuccessful until the patients were given niacin as treatment. **Table 6**, (below) describes each of the cases and how niacin (in addition to other measures, such as intravenous lorazepam infusion, high-doses of oral benzodiazepines, and possibly neuroleptics) was used to successfully resolve the alcohol withdrawal delirium.<sup>13</sup>

This report demonstrates that clinicians

ought to seriously consider pellagra in their differential diagnosis when patients present with alcohol withdrawal delirium. Apparently, current guidelines for the management of this condition excludes niacin deficiency encephalopathy, despite the fact that it expediently assists in the resolution of mental status changes, ataxia, and rashes.<sup>13</sup> Even though niacin was the form of vitamin B<sub>3</sub> used in two of the three cases, the authors' recommended the oral administration of niacinamide, 100 mg orally given three times daily, for 3-4 weeks to facilitate the resolution of alcohol withdrawal delirium.<sup>13</sup> I am not certain why the authors recommended niacinamide instead of niacin, but my suspicion is that they consider both forms of vitamin B<sub>3</sub> to be effective for resolving pel-

**Table 6.** Three Patient Cases Demonstrating Successful Resolution of Alcohol Withdrawal Delirium with Niacin

Patient Identifier	Age/Gender	Diagnosis, Brief Description	Description of Clinical Outcome
Mr. A.	51, Male	Alcohol intoxication associated with increasing agitation and combativeness, autonomic instability, and rash in a photodistribution manner on patient's scalp, face, neck, upper chest, and dorsal lower arms.	On day 4 of his admission, a diagnosis of pellagra was suspected and he was given 100 mg of niacin orally every 6 hours. By day 6, his rash had almost cleared, lorazepam was tapered, and his mental status improved to its baseline status by day 18 when the patient was discharged.
Mrs. B.	54, Female	Alcohol intoxication with diarrhea, vomiting, weight loss, and altered mental status. Clinical evaluation also revealed a resting tremor and marked ataxia.	By day 26 or more (unclear in case description), a diagnosis of pellagra was considered and the patient was started on 100 mg of niacin orally three times daily. Within 3 days, her delirium had resolved and her ataxia had significantly improved. Her affect became normal and was discharged to a rehabilitation facility.
Mr. C.	61, Male	Alcohol intoxication with altered mental status, an ataxic gait, and a rash on sun-exposed areas.	By around day 7 (unclear in case description), a diagnosis of pellagra was suspected and the patient was given 100 mg of niacinamide orally three times daily. Within 2 days the patient's mental status significantly improved and the rash on his nasal bridge had cleared.

lagra, and by using niacinamide the cutaneous flush would be avoided.

### Treatment of Alcohol Use Disorders (AUD)

Mr. Bill Wilson, the cofounder of Alcoholics Anonymous (AA), met Abram Hoffer in 1958.<sup>18</sup> Their relationship evolved and Wilson became an ardent proponent of vitamin B<sub>3</sub> for the treatment of AUD. Wilson wanted every alcoholic to take vitamin B<sub>3</sub> as part of their AA experience. He even produced three seminal manuscripts (published in 1965, 1968, and 1971), "The Vitamin B<sub>3</sub> Therapy," in well-researched communications to AA's physicians.<sup>19-21</sup> In the first report, Wilson included research involving 30 friends who had taken niacin or niacinamide for three months to a year or more.<sup>19</sup> He noted that 10 of the 30 cases "showed prompt and usually spectacular recovery from sometimes long-standing depression, exhaustion, heavy tension and even troublesome paranoid behavior."<sup>19</sup> These cases involved people with histories of AUD, or mental health problems, or both.

In the second communication to AA's physicians, Wilson described the progress that had been made in the last couple of years since the first publication on "The Vitamin B<sub>3</sub> Therapy."<sup>20</sup> In this second communication Wilson stated the following about vitamin B<sub>3</sub>: "It is increasingly clear that B<sub>3</sub> is becoming a valuable adjunct to the treatment of alcoholism, because such a large majority of problem drinkers are beset with these conditions which, since they can cause depression, anxiety, tension and exhaustion, often make it difficult if not impossible to achieve sobriety."<sup>20</sup> This report also included brief updates by prominent clinicians that had been using vitamin B<sub>3</sub> for a variety of different medical conditions, and not just for AUD. There was even a report by the late David R. Hawkins, MD, about his clinical experiences in using vitamin B<sub>3</sub> in which he noted the following:

"I would say that as far as straight alcoholics go, we have used B<sub>3</sub> in well over a hundred cases. I do not think the experience

with the use of B<sub>3</sub> in alcoholism can be reported in percentages. In other words, it is not like Antabuse where you could have a control and a treatment group. I use it clinically in the following ways:

1. Routinely for all alcoholics until they are sober for six months at least. We feel that it helps the patient clear up faster mentally and emotionally.

2. We use it in all alcoholics who are already sober, but who come in because of continuing emotional difficulties. A great many of these clear up on B<sub>3</sub>. Of course, a lot of these people are unrecognized borderline schizophrenics.

To date, we have not seen serious side effects from the use of B<sub>3</sub> in any alcoholic, sober or otherwise. Because of our satisfactory experience with it, we are planning to continue using it indefinitely."<sup>20</sup>

Another inclusion in this second communication involved a report discussing how vitamin B<sub>3</sub> helps to overcome the hypoglycemia issues that are common among individuals with AUD. The report mentioned that some 70 percent of all individuals with AUD have hypoglycemia that can be effectively treated with therapeutic doses of vitamin B<sub>3</sub>.<sup>20</sup>

In the third communication to AA's physicians, Wilson had passed away but a number of notable clinicians reported on their clinical experiences with the vitamin.<sup>21</sup> Among them was a Dr. Russell Smith, MD, who conducted the only clinical study (to my knowledge) that evaluated the therapeutic effects of vitamin B<sub>3</sub> when administered to some 500 alcoholics over a five year period.

In Smith's five year clinical study, niacin was administered to three different groups of alcoholics (n=507) at dosage levels of 3,000 mg or more each day.<sup>22</sup> Group 1 consisted of an alcoholic outpatient population, and all subjects had extensive histories of withdrawals, complications, and numerous failed treatment attempts. Group 2 consisted of hospitalized alcoholics who were primarily seeking treatment voluntarily. The subjects in this group were repeated treatment fail-

ures, with severe withdrawal reactions and complications of alcoholism. Group 3 were subjects who had moderately advanced alcoholism, with good educational resources and lifestyle habits, and were highly motivated. All subjects from the three groups were followed for five years by mail, telephone, and on-site evaluations. At the end of five years, 24 percent of the entire study population had an “excellent” response (total alcohol abstinence for two or more years and stable moods) from niacin treatment. The side effects documented in this study involved the uncomfortable and persistent flush reaction, blocking of the Antabuse reaction, occasional visual disturbance, periodic gastroenteritis, and alteration of diabetes mellitus status. Among the individuals that had an “excellent” response, they had the following notable characteristics:

1. Average age, 55–65 years;
2. Long history of alcoholism with documented delirium tremens, seizures, severe withdrawals, and evidence of advanced organic alcoholism;
3. Long episodes of toxic brain syndrome;
4. Severe, persistent insomnia; and
5. Serious depressions and euphoria.<sup>22</sup>

It makes sense that the most severe alcoholics had the best treatment responses to niacin. I assume that these “excellent” responders were more markedly in need of vitamin B<sub>3</sub>, and therefore, their responses would be more pronounced owing to their pre-treatment niacin insufficiency or deficiency encephalopathy. The results of this study also showed that some 75 percent of all patients benefited from niacin therapy and demonstrated substantial strength in their ability to remain abstinent.<sup>21</sup> The study also revealed that niacinamide produced no significant beneficial effect.<sup>21</sup> Smith remarked that niacin provides an opportunity to strike “...at the heart of the physiologic mechanisms underlying alcohol tolerance, withdrawal, and perhaps even the alcoholic disease process...” and “...seems to make a significant difference

in the ability to obtain and maintain alcohol abstinence.”<sup>21</sup>

## Conclusions

There is definitely a significant therapeutic role that vitamin B<sub>3</sub> could play (mostly, niacin) if it was part of the mainstream approach to treating alcohol withdrawal delirium, and even as a putative treatment to curb drinking behaviour. At the very least, niacin offers individuals with AUD the opportunity to feel more mentally stable, likely from the correction of either niacin insufficiency or deficiency (if the alcohol consumption is significant and prolonged) and/or from the anti-tension, anti-anxiety<sup>23,24</sup> and antidepressant<sup>25</sup> effects that the vitamin possesses. Even though smaller doses have been used successfully for alcohol withdrawal delirium, I believe that the optimal doses of niacin as advocated by Hoffer, i.e., one gram three times daily with meals, should be the therapeutic dose prescribed to all individuals that have AUD, and even to individuals that present to hospitals or medical facilities with alcohol intoxication.

## Competing Interests

The author declares that he has no competing interests.

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