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Naltrexone Can Control Binge Eating and Binge Drinking of Alcoholic Beverages

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Abstract: ***Given the extensive research on the influence of both exogenous opioid agonists and antagonists on durations of ingestion, it seems prudent to use that information to control diseases associated with binge drinking and eating. Doses of naltrexone, an opioid antagonist, can be used to manage binge drinking of alcoholic beverages and binge eating of palatable foods. Here, details are provided on what might be optimal procedures using naltrexone to manage binge drinking characteristic of alcoholism and binge eating disorders.***

Keywords: binge drinking of alcoholic beverages, binge eating, naltrexone, Alzheimer's disease

Hunger is experienced via signals from a nearly empty stomach & small intestines and habits involving cultural-meal-times, e.g., pending breakfasts, lunches, and dinners (Schachter, 1968). Hunger motivates foraging for food and fluids and when ending well begins a bout of ingestion.

Injections of opioid antagonists routinely reduce intakes of a variety of ingesta, opioid agonists extend a meal. Those findings support the idea there is an endogenous opioidergic circuit sustaining ingestion sufficiently often to achieve adequate nutrition. This functionality is sensitive to the release of gut-hormones whose cumulative effects, when processed via circuits in the brain, induce satiation (Livovsky, Pribict, & Azpiroz, 2020). The cumulative effect of processes of the gut eventually inhibits the influence of the endogenous opioidergic (s) sustaining ingestion.

A factor in the regulation of ingestion is palatability (taste, smell and look) of available ingesta. Palatability happens in the oral part of the gut (Rockwood& Reid, 1982) and vision. The functionality of preparedness to learn, for humans, favors sweet, mildly salty, and savory ingesta, such food can provide much of an adequate nutrition. Humans, concurrently, are prepared to not fully ingest foods and drinks that are bitter, extremely sour, and ingesta emitting foul odors signaling an ingesta is not wholesome and maybe poisonous (Kessler, 1951).

Commercial enterprises providing food and drink fill the shelves of grocery stores and menus of restaurants with products enhancing pleasurable palatability (often due to a large loading of sucrose and animal fat). Much of a modern diet in prosperous nations is so centered about highly pleasurable food and drink (and inexpensive to provide) to be less than optimal as described by scientifically derived standards of what is nutritious. Those circumstances eventually lead to habits of what to ingest on an on-going, daily schedule, hence can induce chronic unhealthy eating. In brief, many common daily diets are just too much of a "good thing." Healthy meals limit intake of sweet, heavily salty, and savory ingesta.

Binge eating is less-than-optimal eating. Binge eating (with and without purging) is significantly problematic to be classed as a disease by various diagnostic manuals. Previously, binging on food and drink was called gluttony and often thought of as an original sin by various religions. Binging eating has deleterious effects, e.g., obesity, inadequate nutrition, and the uncomfortable sense of loss of control. Both obesity and inadequate nutrition can aid and abet one or more diseases of the organs of the trunk and features of the brain, hence shortening life-spans.

Given the extensive research indicating that exogenous opioid agonists can extend a bout of eating and that exogenous opioid antagonists can reduce a bout of eating such opens the possibility that exogenous opioids might be useful in controlling eating disorders (including obesity) and maybe anorexia. More on that after a discussion of binge drinking of alcoholic beverages. There is little value to reiterating that alcohol use disorder (AUD or alcoholism) is instrumental in causing many diseases, accidents, and asocial behaviors, hence a significant detriment to a culture. The salient issue is how to reduce the harm caused by regularly drinking alcoholic beverages; particularly, periodic binge drinking done within less drinking on most days of a month.

When laboratory rats are given opportunities to drink alcoholic beverages, they develop a habit of drinking the beverages. When injected with opioid agonists, rats drink more alcoholic beverage and when injected with opioid antagonists they drink less of an alcoholic beverage (Hunter & Reid, 1984; Reid, 1990). Under the influence of opioid antagonists, the pattern of drinking an alcoholic beverage is some initial intake upon an opportunity to drink, but the rats end their drinking quickly and such leads to much less consumption of ethanol. Stated differently, a dose of an opioid antagonist significantly slows or stops (dose sensitive) the endogenous opioidergic physiology sustaining ingestion.

The usual first step toward ending an AUD is to endure the misery of stopping all drinking of alcoholic beverages, which induces miserable withdrawal effects for usually 4 to 7 days. Effective therapists try to minimize the misery of withdrawal, but nevertheless it is surely not a pleasant process. After some days post withdrawal and not taking ethanol, an appetite for alcoholic beverages (associated with

well-engrained habits) is usually sustained and there is a struggle to not relapse to drinking.

An apparent consensus: alcoholism is a chronically relapsing disease. That consensus is probably too extreme because some individuals with a diagnosis of AUD do seek treatment from therapists or attempt to control their drinking on their own and with effort and persistence successfully sustain abstinence or near abstinence. Nevertheless, even after a period of abstinence, there are many relapses to habitually drinking alcoholic beverages. Within most citizens' surroundings, there are numerous stimuli that can, often unconsciously (but can be experienced as temptations), elicit the habit of drinking ethanol which is counter to the rational goal of sustained abstinence. A collapse in ability to avoid drinking is perceived by many citizens, including those trying to sustain abstinence, to be a weakness of "will-power" despite alcoholics often having "will-power" to sustain other difficult tasks and challenges.

USA's Center for Disease Control provided a document on binge drinking of alcoholic beverages. The following is a lead sentence: **"**Binge drinking is the most common, costly, and deadly pattern of excessive alcohol use in the United States. " They also provided potential activities that might limit binge drinking (e.g., increasing taxes on the sale of alcoholic beverages). None of their listed remedies *could directly* limit binge drinking.

For those who have a period of abstinence and are still having strong urges to drink alcoholic beverages, there may be a safe way to overcome binge drinking. If an individual is struggling trying not to engage the first drink of an opportunity to drink an alcoholic beverage, it would be prudent for that individual to take an oral dose of naltrexone prior to being in a situation with opportunities to drink. An oral dose of naltrexone is not apt to stop the first drink of a potential relapse to binge drinking (but would be good if such happened) but rather blocks an opioidergic system sustaining ingesting, i.e., preventing binge drinking. This approach has similarities to the Sinclair Method (TSM) of treating alcoholism, i.e., naltrexone counters the propensity to drink alcoholic beverages once an individual has begun a session of drinking (Sinclair, 2001).

The rationale for TSM is based on the idea that naltrexone reduces ethanol's ability to induce pleasure via a surge of endorphins and/or dopamine. That rationale is based on the theory that dopamine-surges, manifest as pleasure, sustains taking of addictive drugs, a salient feature of the generally accepted theory of addictions. The rationale is also based in the idea that at least some endogenous opioids directly induce positive affect (we do know that exogenous opioid agonists do induce positive affect initially). Sinclair's theory is based on the idea that *if there* is a marked reduction in the pleasurable effects of doses of ethanol due to naltrexone that would eventually lead to extinction of the habit of drinking mimicking the data from classic studies on extinction of habits. It should be noted that the classical studies on extinction of enduring habits take many instances of the habit's activity not being reinforced before the end of the behaviors in question. Those classical studies also indicate that the return of a reward will usually reinstate the activity in question.

The Sinclair Method encourages drinking under the influence of naltrexone. That routine is to be the setting condition for the eventual extinction of the habit of drinking alcoholic beverages. The alternative explanation is that naltrexone can reduce ingestion usually sustained by endogenous opioids. Both theories do not rely on individuals having a constant supply of opioid antagonists circulating to be effective, hence reducing the side-effects that can accumulate with sustained-release-preparations of naltrexone. Month-long-circulations of an opioid antagonist is likely to produce deleterious side-effects because endogenous opioids are involved with a goodly proportion of the circuitry of the nervous system.

In the service of managing alcoholism, I posit that the better approach to using opioid antagonists, specifically naltrexone, is to first have the patient with alcoholism undergo withdrawal from all drinking of alcoholic beverages. Such should be under the influence of those with request medical training and within a well-equipped treatment center.

Once completion of withdrawal effects (usually from 4 to 7 days) and some days of abstinence, there is a prescription for the patient to take a capsule containing an effective oral dose of naltrexone (a moderate dose is probably adequate, e.g., 10 mg per capsule) before any pending opportunity to drink an alcoholic beverage. Multiple studies using rodents as subjects indicates that a single dose of naloxone or naltrexone will dramatically reduce intakes of ingesta including alcoholic beverages. For future research, there should be assessments for what range of oral doses of naltrexone will be optimal to stop people from binging. Once such is known, walking about with a little pill box with capsules of naltrexone can be a convenient reminder to take a pill that will aid and abet "will power."

The supposition is that few, if any, start drinking alcoholic beverages in order to be a drunk driver or losing control of usually adequate behavior. The usual circumstance is, at the outset of an opportunity to drink, there is a plan to have only one or maybe two servings of an alcoholic beverage and then end the drinking. However, far too often the circumstance of having drunk ethanol is a setting condition for dinking more (often as many as 4 or 5 serving of an alcoholic beverage within the space of a couple of hours and subsequently even more which will be manifest by obvious signs of drunkenness).

The extensive studies testing for conditioned place preferences and conditioned place aversions indicate the first dose of ethanol can induce a more pleasant affect than what was previously extant (experienced as relief). However, that pleasant affect is temporary and as the ethanol of the first drink wanes, there is a return to the affect before the drink and probably some unpleasantness due to mild withdrawal effects. Such are the setting conditions for another drink of an alcoholic beverage and binge drinking. However, with naltrexone circulating there is a good chance that the naltrexone will reduce the opioidergic functionality usually

sustaining ingestion hence little motivation for continued intake of ingesta.

The plan to stop binge drinking of alcoholic beverages or to stop binge eating of palatable food is for a person trying to control their ingestion to have in their pocket a small pill box with capsules containing an adequate oral dose of naltrexone that will mute the motivation to extend ingestion. Prior to that readiness to take a dose of naltrexone there should be considerable education on how naltrexone is to be used in the service of controlling binge ingestion. For example, if the person is taking an opioid agonist for relieve of pain, then the person should know that taking naltrexone will induce abrupt withdrawal effects manifest as stomach pains and a return to pain.

The patient needs to understand the dynamics causing the motivation to drink excessively. Yes, the first effects of a drink of an alcoholic beverage does provide some relief from troublesome affect, but, unfortunately and almost certainly, as the ethanol is being metabolized there is a return to the initial unpleasantness and some additional mild withdrawal effects. The emergent negativity is a motivation to take another drink of an alcoholic beverage. In other words, a vicious circle is established: first pleasant affect, then a return to pre-drinking affect and mild withdrawal effects, then there is the motivation to take another drink to mute the unpleasantness but that is temporary and again a return to unpleasantness and so on. The effects may be subtle, nevertheless they are sufficient to sustain a bout of binge drinking which, as stated, is poisonous.

To prevent binging, when there is an opportunity drink alcoholic beverage, the individual should merely take their readily available naltrexone-pill with the recognition that drinking their favorite alcoholic drink is available while also reminding themselves that the first drink of a day may also lead to excessive drinking. If their reminder to not start drinking fails, they will have the safeguard of naltrexone reducing the endogenous opioidergic sustaining ingestion, hence limiting the drinking sufficiently to stop binge drinking. There is probably some value achieved in experiencing the ability to say no to drink an alcoholic beverage. There is probably some value to experiencing a sense of control of over a circumstance that was previously, seemingly not controllable even knowing that it was both "me and naltrexone" that was making life easier.

I posit that *without good education* about the use of naltrexone, a prescription of naltrexone will not be the most efficient way of "curing" alcoholism.

There should also be some consideration of using small doses of an opioid agonist to aid and abet better eating habits among those suffering for anorexia. Also, one might consider a very low dose of naltrexone.

Summary: there is an opioidergic system sustaining ingestion to ensure adequate nutrition. That endogenous system can be modified by exogenous doses of opioid agonists (enhanced consumption) or opioid antagonists (reduce consumption). Providing an inexpensive dose of naltrexone in pill form can be used to help manage binge drinking of alcoholic beverages and the binge eating of palatable ingesta. Such will reduce the instances of extensive drunkenness and need to purge food to avoid weight gain. Also, the same approach can be used to treat obesity (take an oral dose of naltrexone just before two meals a day and any snacks that are tempting) and the result will be less food-intake hence weight loss to healthy levels.

We know that there is an endogenous opioid system sustaining a bout of ingestion of food and drink. We know that the endogenous opioid system sustaining ingestion can be modulated by exogenous opioidergic administrations. What we do not know is the optimal dosing for managing binge ingestion and the optimal education that will encourage the use of naltrexone in the service of better health. These limitations are amenable to being resolved.

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