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Nalmefene Reduces Anticipatory Reward in Persons with Alcohol Use Disorder

Nalmefene is a μ- and δ-opioid receptor antagonist, κ-opioid receptor partial agonist that was approved in 2013 in the UK and several European countries for treating alcohol use disorder. In an important double blind, placebo controlled study, Dr. Barbara Mason reported Nalmefene was safe and effective in preventing relapse to heavy alcohol drinking. She described her findings at the first RiverMend Health Scientific Advisory Board meeting (https://www.youtube.com/watch?v=gSYoFzCYNQ4).

It Works Is Like Naltrexone, But with Differences

Nalmefene is a considered an opioid antagonist like naltrexone but may have advantages over oral naltrexone in the treatment of alcohol dependence. Nalmifene is a universal opioid antagonist which may allow treatment without injections with reduced side effects and longer duration of antagonist action after oral dosing. Like naltrexone, nalmefene is a pure opioid antagonist with no agonist activity and no abuse potential that may also give a more sustained mu opioid antagonist effect than naltrexone. But, nalmefene also binds to other opioid receptors which are generally thought to have roles in alcohol reinforcement, drinking, and relapse.

Non-μ receptors (Δ and κ) are thought to reinforce alcohol consumption, and nalmefene binds more competitively with μ, Δ, and κ receptors than naltrexone.

New Study Sheds Some Light

Numerous human studies using functional magnetic resonance imaging (FMRI) have demonstrated activation of the mesolimbic dopamine system during reward, learning, the use of psychoactive substances, and anticipation of reward. However, the formation of blood-oxygen-level dependent (BOLD) responses in the mesolimbic dopamine system during activation in humans is not well understood. Using a randomized, double-blind, placebo-controlled, crossover design, Quelch DR, Mick I, et al.) uses FMRI, to determine whether a single dose of nalmefene would have a measurable effect on striatal BOLD signaling during anticipation of reward. The authors report that nalmefene "blunted" the BOLD response in the striatum during anticipation of monetary reward and alcohol intake. The authors offer their findings as support for nalmefene's mechanism of action, e.g., its effect on opioid receptor modulation in the mesolimbic dopamine system.

Why This Matters

Naltrexone has been successful in reducing drinking and as a medically assisted therapy for alcohol dependence. Double blind, placebo controlled direct comparison of Naltrexone vs Nalmefene might allow for both comparable efficacy, adherence and cost-benefit analysis. The overall goal would be to improve access, reduce side effects and cost to such treatments that can be given orally. Alcohol Use Disorder remains the number one reason people seek treatment in the US, and is causally related to the growing health burden in the US, even after adjusting for its beneficial effects. An estimated 88,000 people (approximately 62,000 men and 26,000 women) die from alcohol-related causes each year, making alcohol the fourth leading preventable cause of death in the United States. Surely prevention remains a priority, but the need for more effective treatment modalities will save lives.

Nalmefene labeling by the manufacturer is a pharmacological treatment for reducing alcohol consumption for people with:

Alcohol Use Disorder

- A high risk drinking level, as defined by the World Health Organization (WHO)
- Individuals without current withdrawal symptoms
- Individuals who do not require immediate detoxification
- Ongoing psychosocial support focused on treatment adherence and reducing relapse

Reference

Quelch DR, Mick I, et al., Nalmefene Reduces Reward Anticipation in Alcohol Dependence: An Experimental Functional Magnetic Resonance Imaging Study. Biol Psychiatry. 2017 Jan 10. pii: S0006-3223(17)30008-2.

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Recent Laboratory Studies Reveal the Potential for Oxytocin as a Treatment for Methamphetamine Addiction

Oxytocin is a naturally occurring hormone, or endogenous peptide, associated with maternal bonding, lactation and human intimacy. Because of its positive effect on social behavior in humans, it has been studied for its psychotherapeutic potential, particularly among those suffering from addictive disease. Recently Cox and colleagues (2016) studied the effect of oxytocin on drug seeking behavior in rats addicted to methamphetamine. Using a behavioral-economic research design, similar to those used in research that predicted drug desire and relapse in humans, the researchers found that oxytocin was associated with predicting drug seeking and relapse behavior in rodents.

The difference in methodology (rodents versus humans) allowed researchers to determine where the therapeutic benefits occur. The effect in rodents were mediated by actions within the nucleus accumbens, which is where many of the rewarding effects psychostimulants and other intoxicants occur in humans.

Why This Matters

Much of the focus of drug-induced changes in addiction or medication development has been on dopamine. Rarely do we hear about hormone changes. We have heard that drugs of abuse may interfere with maternal-child bonding and parenting. This work on oxytocin may allow us to study these drug-related behavioral changes. Additionally, the potential for any additions to the current arsenal of treatment modalities for methamphetamine addiction is good news, because the treatment outcome data for methamphetamine addiction is dismal. The relapse rate among meth addicts is more common than not, in part because both structural and functional changes in the brain caused by chronic use may never substantially improve. As a result, intense cravings, hallucinations, and delusions often reappear and may continue for months and even years.

Given that most patients in treatment for meth addiction relapse, need for novel and more effective treatments has never been higher.

Reference

Cox BM, Bentzley BS, Regen-Tuero H, See RE, Reichel CM, Aston-Jones G. Oxytocin Acts in Nucleus Accumbens to Attenuate Methamphetamine Seeking and Demand. Biol Psychiatry. 2016 Dec 1. pii: S0006-3223(16)33055-4.

Ketamine, Part One:
An Old Anesthetic Offers New Treatment for Severely Depressed Patients

A growing number of psychiatrists have begun to offer Ketamine, a powerful anesthetic associated with dissociative symptoms, as an off-label treatment for depression in patients who have not responded to other, more traditional treatments. (JAMA Psychiatry. March 2017). A number of small well-controlled studies have found that ketamine can relieve and even eliminate depressive states and suicidal depression in a matter of hours in these otherwise non responders. Ketamine can be administered safely, via infusion, by anesthesiologists with close monitoring in a hospital or surgical setting. Because of the potential for adverse reactions, a much needed consensus statement from the American Psychiatric Association task force offers some welcome guidance and recommendations for clinicians.

What is Ketamine?

Ketamine was developed more than 50 years ago as an anesthetic. Ketamine is a safe, effective, fast acting anesthetic with a good safety profile. It is approved for anesthesiology use on both pediatric and adult patients as well as animals. Ketamine is a dissociative anesthetic which describes its attractiveness as a drug of abuse. It was a popular club drug called "Special K" during the Rave movement and currently the number one club drug in Asia today.

Depression is a Major Public Health Problem

Considering that that up to one-third of patients with major depression fail to respond to available treatments, and many more experience only a partial response, psychiatrists and researchers have been looking for improved treatment options. SSRIs, while offering less side effects and more safety over the tricyclics, do not help everyone. Moreover, current treatment regimens have not significantly reduced suicide, disability and despair caused by depression. Remember, the mortality rate for untreated or under-treated depression among adults is over 15%. Accordingly, the need for new, novel effective treatments has never been greater. With the exception of Transcranial Magnetic Stimulation (TMS), the FDA has not approved any new treatment modalities for depression. This is why we are cautiously hopeful regarding the potential of ketamine as a bonafide treatment for major depression.

Ketamine is delivered as a low dose intravenous infusion of 0.5mg/kg over forty minutes. The results are often dramatic. However, a single infusion may last for only one week. Psychiatrists are hoping to extend the benefit by giving repeated infusions. Cognitive impairment from the acute dissociative state, as well as transient hypertension, that can be mediated with clonidine, are a concern, and the long term risks remain unknown. Still, the findings are remarkable. Small clinical studies indicate more than 70 percent of patients with treatment-resistant depression experience significant relief with ketamine infusion therapy. In addition, the fact that ketamine's mechanism of action and efficacy may open a door to a new treatment targets as a novel and effective treatment for depression. Yet, perhaps the most exciting aspect is the nearly instantaneous response, whereas the therapeutic benefit of most current treatments can take at least 3 weeks—usually longer for most SSRI's. With proper training, emergency room physicians can administer ketamine in patients who present with high suicidality, thereby expediting a transition from the emergency department to a psychiatric treatment setting.

Safety Precautions

Because ketamine is a powerful anesthetic, caution is warranted. Respiratory function, CO2 levels, along with vital signs should be monitored during infusion. Those administering ketamine, preferably an anesthesiologist or interventional psychiatrist with training in advanced cardiac life support, can reduce the risk of adverse reactions, as cardiovascular and respiratory emergencies can occur. Because each patient is unique, a thorough work up prior to infusion provides valuable information regarding risks, dosage and treatment frequency to maximize response and minimize adverse events.

Why This Matters

As stated, the suicide rate for depressed adults is now over 15%, and survey data shows that as many as 20% of high school students are depressed and have considered or attempted suicide. So far, the best available evidence suggests that Ketamine produces a very strong and rapid antidepressant effect. Ketamine may be a harbinger of newer and more effective treatments for depression. As a result, some of the elite psychiatric training programs are offering Fellowships in Interventional Psychiatry with specific training in TMS and Ketamine infusion.

The lack of data on long-term efficacy and safety is of concern and demands caution. Clearly more research is needed to address these gaps in knowledge and to provide additional safety guidelines for clinicians.

Reference

Zorumski CF1, Conway CR2. Use of Ketamine in Clinical Practice: A Time for Optimism and Caution. JAMA Psychiatry. 2017 Mar 1.

Ketamine, Part Two:
A Consensus Statement is Issued Regarding Off Label Use of Ketamine for Treatment Resistant Depression and Suicidality

We and others have reported on the misuse, abuse, and dependence associated with the illicit use of ketamine. Ketamine is not new, it entered to public consciousness as "Special K" in the late 1990's at the onset of the Rave movement. However, it is important to keep in mind that although ketamine can be abused as a result of its dissociative properties, it has numerous indications and uses in anesthesiology, dentistry, and veterinary medicine.

Primarily, ketamine has been used in anesthesiology for nearly 50 years. But recently, important research has shown that ketamine hydrochloride can rapidly change mood, reverse suicidal thinking, and produce persistent antidepressant effects in patients with persistent depression, mood and anxiety disorders. Most clinical trials and case reports available have centered on the use of infused ketamine, via IV solution at a dose of 0.5mg/kg per 40 minutes for individuals with treatment resistant depression or anxiety disorder.

Why Does This Matter?

Despite not being an FDA approved treatment for treatment resistant depression or anxiety, increasingly psychiatrists are using ketamine as an "off-label" treatment for depression and suicidality. As a result of the reported success associated with symptom reduction, The American Psychiatric Association (APA) has recently issued guidelines for clinician who wish to use ketamine for this purpose.

Guidelines-From the APA

1. A comprehensive diagnostic assessment should be completed to establish current diagnosis and evaluate history of substance use and psychotic disorders.
2. Assessment of baseline symptom severity should be completed to allow later assessments of clinical change with treatment.
3. A thorough history of antidepressant treatment should be collected and documented to confirm previous adequate trials of antidepressant treatment.
4. A thorough review of systems should be performed to evaluate potential risk factors associated with ketamine treatment.
5. Decisions on the specific physical examination and laboratory screening assessments should be made according to established guidelines and advisories issued by the American College of Cardiology Foundation/American Heart Association and the American Society of Anesthesiologists and should be based on a patient's individual clinical characteristics.
6. A careful review of past medical and psychiatric records and/or corroboration of the past history by family members are strongly encouraged; all current medications and allergies should be reviewed, including histories of opiate and benzodiazepine use; the use of a baseline urine toxicology screen is strongly encouraged to ensure the accuracy of the reported substance use and medication record.
7. An informed consent process, including discussion of the risks associated with the treatment, the limits of the available information pertaining to the potential benefits of the treatment, the fact that this is an off-label use of ketamine, and a discussion of alternative treatment options should be completed; this discussion should be complemented with written materials, and the patient should provide written informed consent before initiating treatment.

Reference

Sanacora G, Frye MA, et al. A Consensus Statement on the Use of Ketamine in the Treatment of Mood Disorders.American Psychiatric Association (APA) Council of Research Task Force on Novel Biomarkers and Treatments. JAMA Psychiatry. 2017 Mar 1. doi: 10.1001/jamapsychiatry.2017.0080. [Epub ahead of print]

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The Unique Genetics of Heroin Addiction

Mu-opioid receptors (MOPRs) are the target of heroin and other prescription opioids, which is the underlying neurobiology responsible for endemic addiction morbidity and mortality in the US.

Opioids in the Brain

The opioid system consists of three receptors, mu, delta, and kappa. Opioid receptors are activated in response to natural rewarding stimuli and by drugs of abuse, which causes neuroadaptation to the opioidergic reward system as addiction develops.

Charbogne and colleagues (Journal of Biopsychiatry, 2016) report that Naloxone reversible, Mu opioid receptors (MORs) mediate analgesic, euphorigenic, and other biological effects of opioids. Yet the aberrant activation and modifications of the mu opioid system associated with drug craving and relapse are not well understood.

Behavioral analysis of OPRM1 mice shows that this population does regulate locomotor and motivational effects of heroin. These receptors do not contribute to heroin-positive reinforcement. Beyond a well-established role in reward processing at the level of local ventral tegmental area neurons, MORs moderate motivation for appetitive stimuli and motivation to obtain heroin and food reward, revealing a yet unreported role for MORs within addiction circuits. Several brain areas responsible for MOR-mediated reward have been identified. Yet more research is needed to establish the key underlying molecular function of the system and to locate neural sites where opioid peptides and receptors contribute to the onset of addictive disease.

Why Does This Matter?

We wonder why do some people exposed to prescription opioids become addicts quickly, others slowly, while others do not. We also have wondered about individual differences in opioid medication response, toxicity, addiction, and relapse. Genetic association studies reveal that the OPRM1 A118G genotype (found in up to 30% of Caucasian and 60% of Asian populations) increases the risk of heroin addiction. Accordingly, more genetic research is needed to further elucidate our understanding of the biology of addiction and for the development of therapeutic interventions to treat the disorder.

Reference

Charbogne P, Gardon O, et al. Mu Opioid Receptors in Gamma-Aminobutyric Acidergic Forebrain Neurons Moderate Motivation for Heroin and Palatable Food. Biol Psychiatry. 2016 Dec 26. pii: S0006-3223(16)33156-0. doi: 10.1016/j.biopsych.2016.12.022.

Alcohol + Marijuana = Lower GPA in College

Current research supports previous findings regarding alterations in the brains of adolescents and young adults who use alcohol or marijuana. These neuroadaptations in function and anatomy are associated with impaired decision making, memory and impulsivity in this population.

Currently, 4 of 5 college students drink alcohol and half of these are binge drinkers. In addition, 58% of alcohol drinking adolescents report using alcohol and marijuana simultaneously. Yet there is a dearth of data regarding the concurrent use of these intoxicants—until now.

Data from the 2-year Brain and Alcohol Research in College Students (BARCS) study included 1142 freshman students who completed monthly marijuana and alcohol consumption surveys. Based on the results, the students were classified into one of 3 data-driven groups, based on their consumption. 1) No/low users of both, 2) medium-high alcohol/no-low marijuana, and 3) medium-high users of both substances.

The Analysis Was Sobering

Compared to sober peers, students using moderate to high levels of alcohol plus low marijuana use had lower GPAs, but this difference becomes non-significant over time. In contrast, students consuming both substances at moderate-to-high levels attained significantly lower GPA at both the outset and across the 2-year investigation period. Follow-up analysis showed significant improvement in GPA when students curtailed their substance use compared to those who continued moderate or high levels marijuana and alcohol over the two year period.

Why Does This Matter?

Today 8,000 more Americans, mostly children and adolescents under age 18, will use an illicit drug for the first time. Substance use, misuse and dependence have considerable effects on development and also academic performance, beyond GPA.

Each Year...

- More than 1,700 students die from alcohol poisoning and alcohol-related injuries.
- 700,000 students are assaulted by classmates who were drinking.
- Almost 100,000 students are victims of alcohol-related sexual assaults and rapes.

As drug and alcohol abuse increase among our young people, we can expect a further decline in school performance, referral to addiction and psychiatric treatment.

Prevention, plus better and more accessible treatment is absolutely necessary if we are to reverse this disturbing trend.

Reference

Meda SA, Gueorguieva RV, et al. Longitudinal influence of alcohol and marijuana use on academic performance in college students. PLoS One. 2017 Mar 8;12 (3): e 0172213. doi: 10.1371/journal.pone.0172213. eCollection 2017.
Califano, J, America magazine, May 28, 2007

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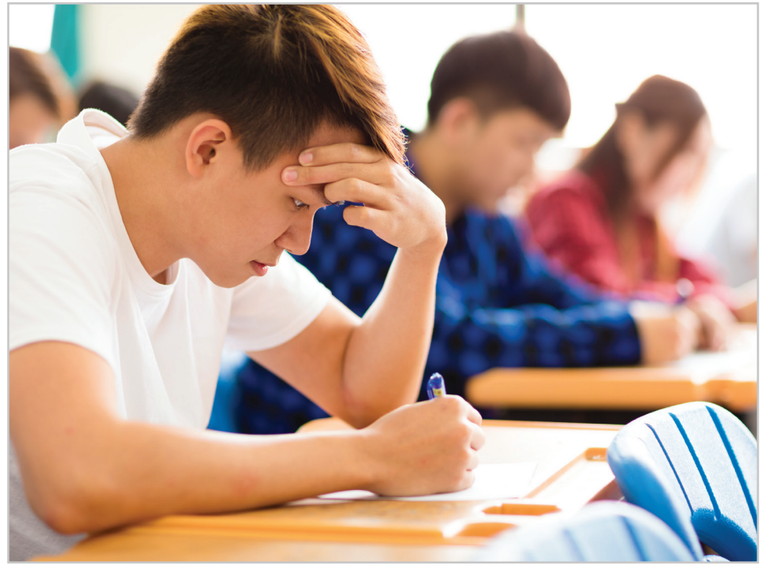

Predictors of Synthetic Cannabis Use Among Adolescents

Synthetic cannabinoids (SCs) are structurally similar to δ-9-tetrahydrocannabinol, the psychoactive constituent in crude cannabis. Many of the street grade synthetics are full CB1 and/or CB2 cannabinoid agonists. As a result the potency, level of impairment and adverse reactions are unpredictable and associated with a variety of negative health effects, including cardiovascular events and death. Yet, little is known about the risk and protective factors associated with the use of SCs among adolescents including psychiatric history, psychological issues and known substance-use and abuse risk factors. Ninnemann, Choi, et al., hypothesized that anxiety, depression, impulsivity, and marijuana use could predict SC use during adolescence. Using cross-sectional methodology, over a 12 month span, with subjects (n=964) from multiple schools in Texas.

The results showed that depressive symptoms, cannabis, alcohol and SC use at baseline were predictive of SC use at 1 year follow-up. Anxiety and impulsiveness at baseline were not predictive of SC use at follow-up. In addition, African Americans and females were less likely to use SCs than males or other ethnic groups. Findings from other studies have demonstrated that lifetime cigarette, marijuana, alcohol, or other illicit drug use dramatically increased the likelihood of past-year SC use. Yet, the frequency of past marijuana use is the strongest correlate of future SC use.

Why Does This Matter?

The use of SC's among high school students has declined over the past few years (Monitoring the Future Survey, 2016) largely because we have identified and outlawed many of the chemicals used to manufacture SC's. However, the deleterious effects and mortality has increased as young amateur chemists have also identified the ingredients to create and sell dangerous, homemade SC's to unwitting teens.

This is the first prospective research of SC use among a high risk cohort. Learning that depressive symptoms, marijuana and alcohol use are predictive of subsequent SC use is the first step in developing much needed, evidenced based prevention strategies.

References

Ninnemann AL, Jeong Choi H, Stuart GL, et al. Longitudinal Predictors of Synthetic Cannabinoid Use in Adolescents. Pediatrics. 2017;139(4):e20163009 Link : At this time the article is embargoed.

Drug Enforcement Administration, Department of Justice Schedules of controlled substances: temporary placement of four synthetic cannabinoids into Schedule I: final order. Fed Regist. 2014;79(27):7577–7582pmid:24605391

Medically Assisted Treatment using Suboxone Improves Participation and Adherence in Addiction Treatment

Studying opioid addicted patients is difficult for numerous reasons, namely high rate of drop out and low participation. It's the nature of addictive disease.

Yet previous studies using patients who came to a local Emergency department for overdose of opioid related morbidity, who were initiated into Suboxone (buprenorphine plus naloxone) and continued to receive suboxone in primary care were more likely to sustain engagement in addiction treatment and reduce their use illicit opioids at 30 days post assessment compared to ED patients who received either brief intervention without Medically Assisted Treatment (MAT), or were referred to an addiction treatment provider. However, 30 days is not a goal or outcome that most researchers would use to determine the efficacy of any treatment for a life threatening, chronic disease. In oncology, for example, the 5 year mortality rate is the "gold standard" for treatment efficacy. With this in mind, the authors recruited and evaluated randomized participants (n=290) at 2, 6 and 12 months post ED initiated intervention. The bench marks included: Self-reported engagement in formal addiction treatment, the number of days of illicit opioid use, and HIV risk behavior (2, 6, 12 months); and urine toxicology testing at 2 and 6 months, which is the best indicator of outcome.

The Results

At 2 months, the MAT group reported fewer days of illicit opioid use versus brief intervention. But, no significant differences in illicit opioid use were observed at 6 or 12 months. There were also no significant differences in HIV risk or rates of opioid-negative urine results between the groups at any time.

Why Does This Matter?

Considering the increasing mortality rate associated with the illicit use of opioids and the resources being expended to curtail this epidemic, better prevention and treatment resources are needed. Emergency intervention with MAT appear to be short-lived. Smaller studies have shown that MAT combined with addiction centered, multiphasic, multimodal and multidisciplinary treatment of appropriate intensity and duration is associated with superior outcomes. The key to successful treatment is continuous supportive care and accountability, including continuous monitoring.

Reference

D'Onofrio G, Chawarski MC, et al. Emergency Department-Initiated Buprenorphine for Opioid Dependence with Continuation in Primary Care: Outcomes During and After Intervention J Gen Intern Med. 2017 Feb 13. doi: 10.1007/s11606-017-3993-2. [Epub ahead of print].

New Sub-Dermal Buprenorphine Implant Approved by FDA for Opioid Addiction

Amid the current scourge of opioid addiction and death occurring throughout the US, treatment employing long-term use of partial opioid agonists such as Suboxone are associated with better outcomes when compared to an opioid taper program or psychological treatments alone without Medically Assisted Treatment (MAT). The superior outcomes include: Less prescription opioid use, better adherence to prescribed medication. Improved adherence to non-MAT modalities for opioid dependence, such multiphasic, multimodal and multidisciplinary treatment programs.

Now, researchers have developed a subdermal drug delivery system for Buprenorphine called Probuphine.

What is Probuphine?

Probuphine is a subdermal implant, consisting of 4 small devices, about the size of a matchstick surgically placed under the skin in the patient's upper arm. The device releases a steady, measured dose of buprenorphine for six months. The stated objective for probuphine is to improve patient compliance, adherence and safety.

Because probuphine requires surgical insertion, only medical professionals who have been trained by the Probuphine Risk Evaluation and Mitigation Strategy (REMS) program can administer the implant.

The efficacy and safety of probuphine has been established in several clinical trials, including a placebo-controlled study of 163-patients, over a 24-week period, and in a study of 287 patients, published in the journal Addiction.

Why Does This Matter

Oral Naltrexone has had adherence challenges. Injectable Naltrexone does not have these problems on a day to day basis, but still needs monthly injections. Similarly, Suboxone has challenges with drop outs and adherence. Probuphine can provide increased stability with longer relief, improve patient safety and adherence, and hopefully improve the patient's quality of life. The gold standard, however, is 5 year outcome studies. Probuphine is more expensive than oral transmucosal (sublingual) buprenorphine, which is similarly effective among highly motivated patients.

Reference

Buprenorphine Implants (Probuphine) for Opioid Dependence. JAMA, 2016

Nov 1;316(17):1820-1821. doi: 10.1001/jama.2016.10899.

Link : http://jamanetwork.com/journals/jama/article-abstract/2576597

Cost Comparison Buprenorphine vs. Probuphine

Drug Name	Dose	Cost
Buprenorphine	Generic 2, 8 mg sublingual tabs, 16 mg once/day	$1671.80
Probuphine (Titan)	74.2 mg subdermal implant 4 implants, for 6 months	$4950.00
Buprenorphine/Naloxone	Generic 2/0.5 mg, 8/2 mg sublingual tabs, 16/4 mg once/day	$2813.90
Bunavail (BioDelivery Sciences)	2.1/0.3 mg, 4.2/0.7 mg, 6.3/1 mg buccal films, 8.4/1.4 mg once/day	$2660.40
Suboxone (Reckitt Benckiser)	2/0.5 mg, 4/1 mg, 8/2 mg, 12/3 mg sublingual films, 16/4 mg once/day	$2660.40
Zubsolv (Orexo)	1.4/0.36 mg, 5.7/1.4 mg sublingual tabs 11.4/2.8 mg once/day	$2660.40

The Psychiatric Risks and Rewards of Bariatric Surgery

For 30 plus years I have researched and written extensively about the global obesity epidemic, often from the point of view that highly palatable foods and specifically sugars, can cause neuroadaptation in much the same way as drugs of abuse in genetically susceptible persons.

The World Health Organization (WHO), which coined the term Globesity, has taken leadership in detailing the global consequences of obesity in terms of rates of metabolic derangement, which includes diabetes, cardiovascular disease, and other morbidities including obstructive sleep apnea, cancers and a high lifetime prevalence of mental illness.

While behavioral and pharmacological treatments have had limited efficacy, surprisingly, bariatric surgery has proven to be a safe and effective tool for Type 2 diabetes reversal and weight loss. Bariatric surgery outcomes, even 5 year outcomes, show important and sustained improvements in medical comorbidities and quality of life. There is, however emerging evidence correlating a range of mental health complications following bariatric surgery that require the assistance of specifically trained psychiatrists and other mental health professionals for providing individualized, patient centered care for such individuals.

Long-term Bariatric Surgery Outcomes

Our group and others have reported on Roux-en-Y gastric bypass surgery outcomes that increase the risk for alcohol abuse and dependence. Others research has have shown de novo substance use disorders after bariatric surgery.

Postsurgical changes in alcohol absorption and metabolism can cause impairment in both cognitive functioning and of course reaction time to physical stimuli, e.g., impaired driving, behavioral loss of control, nutritional deficiencies, or increased caloric consumption limiting weight loss. Consequently, a complete psychiatric and addiction evaluation before surgery with followed by strict postsurgical follow up is an essential component in our clinical practice guidelines.

Why Does This Matter?

Bariatric works but complications and emerging problems can compromise success. Early on we hypothesized that the brain's reward and reinforcement mechanisms produced by food, and the anticipation, preoccupation of eating highly palatable food is ground zero in the battle to reduce mortality, morbidity and to improve one's quality of life.

Postsurgical depression, thoughts of self-harm and suicide are statistically significant complications following weight loss surgery. The reasons for this are poorly understood. But, the Psychosomatic Council of the American Psychiatric Association (APA) suggests the following might be causal:

- Postsurgical neurohormonal changes
- Behavioral loss of control
- Altered absorption of alcohol and/or psychiatric medication
- Lack of improvement in one's quality of life
- Weight regain, and continued or recurrent physical limitations.

Additionally, excess skin folds after surgery may create comorbid body image concerns leading to lowered self-image and distressed psychological health.

Many experts think bariatric surgery is far and away the best treatment for morbid obesity and obesity with T2 diabetes. We have come a long way from telling obese persons to simply diet and exercise, but we have miles to go to make a dent in the globesity pandemic.

Reference

Sockalingam S, Micula-Gondek W, et al. Council Psychosomatic Medicine. Bariatric Surgery and Psychiatric Care. Am J Psychiatry. 2017 Jan 1;174(1):81-82. doi: 10.1176/appi.ajp.2016.1731001.

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