

Mayor Robert Garcia, City Council Members and The City

I am Frances Emily Dawson Harris, a reside in District #1 and a pediatric nurse

In regards to Hearing: 18-0492: I am reluctant to support this ordinance amending the Long Beach Municipal Code (by adding Chapter 5.92; approving ZCA 18-003; LCPA 17-009 and amending Title 21 Zoning Ordinance) which according to the Planning Commissioners recommendation (as per their May 17, 2018 Agenda) " to define and designate Adult-Use Cannabis uses as permitted, conditionally permitted or prohibited within specific zoning districts in the City. (Citywide)

Why?

Note: Exhibit A: "Governor Jerry Brown's June 27, 2017 signed law, the Medical and Adult-Use (Recreational) Cannabis Regulation and Safety Act (MAUCRSA) grants local governments the ability to regulate and/or prohibit commercial marijuana activity within their jurisdictions. Thus, cities are not required to permit recreational marijuana collective dispensaries." If you do not believe me; then, ask Attorney Michael Mais.

Note: Alhambra, Burbank, Beverly Hills, Glendale, Inglewood, Pasadena and West Covina do not permit recreational marijuana sales..

I believe if Adult-Use Cannabis reads as Adult Recreational Cannabis; then, this will prevent confusion and eliminate any belief of being mislead.

I believe that notifying the public regarding "hearings" for applicants for a CUP: conditional use permit; particularly, for adult use recreational cannabis needs to be mandatory.. Surely, ^{opinion} Public is essential.

What is the Long Beach Police Department's position regarding Item 18-0492? Please refer to **Exhibit B**: Lt. Kevin Coy's march 22, 2018 response including marijuana smoking at the

L.B. Bus stops; on the Metro Platform and while riding on the Metro Line. Over 70 smoking tickets

since July 1, 2017 have been issued. He confirmed today via phone this problem is ongoing.

Secondhand marijuana smoke emits cancer causing substances and toxic chemicals.
Youths

will find a ways to use recreational cannabis which when combined with alcohol, prescription drugs, over the counter drugs and or "street drugs" is potentially deadly. The serious effects of acute marijuana intoxication is shockingly alarming. Usage by people with neurodevelopmental disorders and mental illness has a probable detrimental effect.

If you feel you must permit approve this "ordinance"; then, please consider at the most only permitting adult use cannabis dispensaries within a medical cannabis dispensary.

City Councilmembers please vote for what is right not what is popular.

Thank you very much for the opportunity to speak.

Frances Emily Dawson Harris

June 19, 2018

Exhibit B: Articles used regarding Long Beach Recreational Marijuana Collective Dispensaries:

***** copies of articles provided to the City Clerk *****

Marijuana acute intoxication

<https://www.uptodate.com/contents/cannabis-marijuana-acute-intoxication>

Developing a roadside test for marijuana intoxication isn't as easy as it sounds

<https://www.sciencedaily.com/releases/2018/01/180125135606.htm>

Pot 101: Facts you should know about California's legal marijuana by Chris Nicholas

<http://www.politifact.com/california/article/2018/jan/05/pot-101-facts-you-should-know-about-californias-le/>

Legalizing Recreational Marijuana by Kody Kershner

<https://khtoday.com/news/2018/05/07/legalizing-recreational-marijuana/>

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LONG BEACH



FINANCIAL MANAGEMENT

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ADULT-USE (RECREATIONAL) MARIJUANA

On November 8, 2016, California voters approved Proposition 64, the Adult Use of Marijuana Act (AUMA). AUMA created a statewide regulatory and licensing system for adult-use marijuana businesses. On * June 27, 2017, Governor Jerry Brown signed into law the Medicinal and Adult-Use Cannabis Regulation and Safety Act (MAUCRSA), which merged regulations for medical and adult-use cannabis into a single regulatory framework. MAUCRSA grants local governments the ability to regulate and/or prohibit commercial marijuana activity within their jurisdictions.

* On November 14, 2017, the Long Beach City Council voted to request the City Manager to work with affected City Departments to develop recommendations to legalize and regulate commercial adult-use (recreational) marijuana businesses in Long Beach. The City Attorney will report back to City Council with a draft ordinance to allow, license, and regulate the retail sale, cultivation, manufacture, distribution, and laboratory testing of adult-use marijuana in Long Beach by June 2018.

TEMPORARY MORATORIUM ON ADULT-USE MARIJUANA BUSINESSES



Frances Dawson Harris <fsoulconnection2@gmail.com>

B

* **MARIJUANA USAGE AT THE LB TRANSIT MALL, ON THE METRO LINE PLATFORM AND WHILE RIDING THE METRO LINE**

4 messages

Kevin Coy <Kevin.Coy@longbeach.gov>

Thu, Mar 22, 2018 at 6:26 PM

To: "fsoulconnection2@gmail.com" <fsoulconnection2@gmail.com>

Cc: Luke Klipp <Luke.Klipp@longbeach.gov>, Alex Avila <Alex.Avila@longbeach.gov>

Hello Ms Harris,

I have received the concern you sent regarding marijuana smoking at the Long Beach Transit Mall, on buses, and the Metro Trains and platforms. I am Lieutenant Kevin Coy and I am in charge of the Long Beach Police Department Metro Blue Line law enforcement contract.

Since the Long Beach Metro Transportation Section began the Blue Line contract on July 1, 2017, our officers have written over seventy (70) smoking tickets to smoking violators on the Blue Line trains and platforms in Long Beach. This includes violations for the smoking of marijuana. I know that we have advised many, many violators as well. I am checking with LA Metro to see how many Code of Conduct smoking violations have been written by their Security Officers during the same period and I should have that number soon. I am sure it will be multiple numbers. Our Long Beach Transit officers also continuously advise and cite smoking violators at bus stops around the city.

B

Frances Emily Dawson Harris

June 19, 2018

Research articles used regarding Long Beach Recreational Marijuana Collective Dispensaries:

***** copies of articles provided to the City Clerk *****

Marijuana acute intoxication

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Developing a roadside test for marijuana intoxication isn't as easy as it sounds

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Contributor Disclosures

All topics are updated as new evidence becomes available and our [peer review process](#) is complete.

Literature review current through: Apr 2018. | **This topic last updated:** Apr 25, 2018.

INTRODUCTION — This topic discusses the epidemiology, pharmacology, toxicity, clinical manifestations, and management of acute intoxication with cannabis (marijuana).

The clinical manifestations and management of toxicity from synthetic cannabinoids, medical uses of cannabinoids, and the manifestations and treatment of cannabis use disorder are provided separately:

- (See "[Synthetic cannabinoids: Acute intoxication](#)".)
- (See "[Cancer pain management: Adjuvant analgesics \(coanalgesics\)](#)", section on 'Cannabis and cannabinoids'.)
- (See "[Symptom management of multiple sclerosis in adults](#)", section on 'Cannabinoids'.)
- (See "[Palliative care: Assessment and management of nausea and vomiting](#)", section on 'Cannabinoids and cannabis'.)
- (See "[Overview of the management of epilepsy in adults](#)", section on 'Alternative therapies'.)
- (See "[Cannabis use and disorder: Epidemiology, comorbidity, health consequences, and medico-legal status](#)" and "[Cannabis use and disorder: Clinical manifestations, course, assessment, and diagnosis](#)" and "[Treatment of cannabis use disorder](#)".)

EPIDEMIOLOGY — According to the World Health Organization (WHO), 147 million people, or 2.5 percent of the world population, use cannabis (marijuana), making it the world's most widely cultivated, trafficked, and abused illicit substance [1]. Use is high in the adolescent age group. As an example, among surveyed adolescents in the United States, approximately 7 percent of 8th graders, 15 percent of 10th graders, and 21 percent of 12th graders reported cannabis use in the past month [2].

Several countries, including Canada, Switzerland, and the United States are contemplating or actually liberalizing laws governing cannabis use [3]. At the United States federal level, cannabis products are classified as Schedule 1, ie, no currently accepted medical use and a high potential for abuse [3-5]. However, over 20 states have decriminalized medical marijuana or are reviewing legislation to allow low-dose delta-9 tetrahydrocannabinol (THC) products for specific medicinal indications. Colorado, Washington, Oregon and Alaska have allowed the retail sale and possession of recreational marijuana [6].

The public health impact of decriminalization or legalization of recreational cannabis use include:

- Both decriminalization and legalized recreational use have been associated with increased unintentional pediatric ingestions [7-9]. As an example, after legalization of recreational marijuana use in Colorado, annual calls to the regional poison control center for pediatric marijuana exposure increased 34 percent on average to 6 cases per 100,000 population, which was almost twice the rate for the rest of the United States [9]. Exposure to recreational marijuana accounted for about half of cases. Rates of hospital visits at a large regional children's hospital system also increased significantly during the period of the study, although the total number of presenting patients (81) was small.
- In regions with medical marijuana availability, diversion of drug from registered users may also encourage adolescent abuse [10].
- In other countries where cannabis can be used legally, rates of usage vary. For example, in the Netherlands, the overall annual prevalence of cannabis usage is 23 percent among young adults compared with 5 percent annual usage reported by persons 12 to 64 years of age in Uruguay [11,12]. Thus, the impact of decriminalization or legalization on the subsequent prevalence of cannabis usage is not easily predicted and varies depending upon the specifics of regulatory enactment [3].

PHARMACOLOGY AND TOXICITY

Site of action — The cannabinoid receptor is a G-protein linked receptor, which inhibits adenylyl cyclase and stimulates potassium conductance. There are two known cannabinoid receptors: CB1 and CB2 [13-15]:

- CB1 is found in the central nervous system including the basal ganglia, substantia nigra, cerebellum, hippocampus, and cerebral cortex. It acts presynaptically and inhibits release of several neurotransmitters including acetylcholine, L-glutamate, gamma amino butyric acid (GABA), norepinephrine, dopamine, and 5-hydroxytryptamine.
- CB2 is found peripherally in the immune system tissues (eg, splenic macrophages and B lymphocytes), peripheral nerve terminals, and vas deferens. It is postulated that it plays a role in regulation of immune responses and inflammatory reactions. Anandamide and palmitoylethanolamide are known endogenous cannabinoid receptor ligands.

Pharmacokinetics — The pharmacokinetics and pharmacodynamics of delta-9 tetrahydrocannabinol (THC) vary by route of exposure as follows [13,15-23]:

- **Inhaled marijuana** – After inhalation of marijuana smoke, onset of psychoactive effects occurs rapidly with peak effects felt at 15 to 30 minutes and lasting up to four hours. These effects mirror plasma delta-9 tetrahydrocannabinol (THC) concentrations. Approximately 2 to 3 mg of inhaled THC is sufficient to produce drug effects in a naïve user. Pulmonary bioavailability varies from 10 to 35 percent of an inhaled dose and is determined by the depth of inhalation along with the duration of puffing and breathholding.
- **Ingested marijuana** – When compared to inhalation, cannabis ingestion has a delayed onset of psychoactive effects that ranges from 30 minutes to three hours. Clinical effects may last up to 12 hours. Orally administered cannabis has low bioavailability (5 to 20 percent) because of chemical degradation in gastric acid and substantial first-pass metabolism in the liver. In naïve users, psychotropic effects occur with 5 to 20 mg of ingested THC.

THC is lipid soluble, highly protein-bound (95 to 99 percent), and has a volume of distribution of 2.5 to 3.5 L/kg [13,19].

THC metabolism occurs via hepatic cytochrome oxidases, CYP2C9 and 3A4. The primary active metabolite is 11-hydroxy THC, and the inactivated metabolite is THC-carboxylase [13]. THC crosses the placenta with fetal

plasma concentrations 10 to 30 percent of maternal concentrations. It also accumulates in breast milk at a concentration as high as eight times maternal plasma concentrations [13,24].

After metabolism, THC is mostly excreted as hydroxylated and carboxylated metabolites via feces (65 percent) and urine (20 percent) [13]. Although difficult to measure, the elimination half-life of THC is slow, ranging from 25 to 36 hours [13]. This lengthy half-life is likely due to slow release from lipid storage compartments and enterohepatic circulation. Elimination half-life is longer in regular cannabis users.

Cannabis formulations — There are a variety of subspecies and strains of cannabis (marijuana); *Cannabis sativa* is one of the most commonly used for recreational purposes. Cannabis sativa contains over 500 different chemical compounds, and over 60 known cannabinoids; of these, delta-9 tetrahydrocannabinol (THC) is the most psychoactive and is responsible for most symptoms of intoxication [13,17].

The dried flower of the marijuana plant has a large range of THC content, ranging from 1 to 20 percent of the total weight; however, much variability exists among marijuana samples [25]. In general, marijuana potency has increased over the past 20 years [26]. Common slang terms for marijuana include "pot," "grass," "dope," "MJ," "Mary Jane," "doobie," "hooch," "weed," "hash," "reefers," and "ganja."

Chemical analogues of THC, called "synthetic cannabinoids" may have been available in Europe as early as 2004, and were first reported in the United States in December 2008. The clinical effects can be similar to natural marijuana intoxication, but may also result in more severe life-threatening symptoms. Acute intoxication from synthetic cannabinoids as discussed separately. (See "[Synthetic cannabinoids: Acute intoxication](#)".)

Recreational use — Recreational marijuana use often consists of smoking the dried flower in the form of rolled cigarettes (joints) and water bongs [27]. THC is also extracted using various solvents (butane, ethanol, hexane, isopropanol) to create highly concentrated products (60 to 99 percent of weight) including oils and tinctures called "wax," "dabs," "budder," and "shatters" [28]. In addition to being smoked, these highly concentrated products are also vaporized (eg, using electronic cigarettes [29,30]) or mixed in food products (such as brownies, cakes, candies, and beverages) and ingested.

In regions where marijuana use is legal, ingested forms are popular and may pose a risk of unintentional ingestion by children or excessive ingestion by adults. As an example, in Colorado, some companies have produced packaging for marijuana products that mimic popular candy [31]. Furthermore, many of these products contain up to four times the suggested dose of 10 mg.

Medicinal use — Medicinal marijuana is supplied as dried flowers of the *Cannabis sativa* plant that are smoked as described for recreational cannabis use (see '[Recreational use](#)' above). Derivatives of cannabinoids are also available as pharmaceuticals in some countries including oral preparations ([dronabinol](#) and [nabilone](#)) and a spray for buccal use ([nabiximols](#)).

Marijuana and its components have been proposed for various medicinal purposes, such as chronic severe pain (eg, due to cancer), refractory nausea and vomiting, anorexia and cachexia, glaucoma, and seizures [14]. However, none have been proven to have greater efficacy than other currently available medications.

Of these indications, medical marijuana is most frequently prescribed for severe or chronic pain. An oromucosal spray containing THC and cannabidiol (Sativex), also called [nabiximols](#), has been shown to have some efficacy as a multipurpose analgesic in combination with traditional therapy and is approved for use in Canada and elsewhere but not in the United States. No controlled studies demonstrate the efficacy of inhaled marijuana as an adjunct to traditional pain medications for patients with cancer-related pain (see "[Cancer pain management: Adjuvant analgesics \(coanalgesics\)](#)", section on '[Cannabis and cannabinoids](#)'). Trials in patients with multiple sclerosis have failed to show consistent pain reduction. (See "[Symptom management of multiple sclerosis in adults](#)", section on '[Cannabinoids](#)'.)

Although inhaled, buccal, or ingested marijuana has shown some efficacy for refractory nausea and vomiting or glaucoma [32,33], consensus expert guidelines do not support its use. (See "[Cancer pain management: Adjuvant analgesics \(coanalgesics\)](#)", section on 'Cannabis and cannabinoids' and "[Palliative care: Assessment and management of nausea and vomiting](#)", section on 'Cannabinoids and cannabis'.)

Cannabinoids demonstrate anticonvulsant properties in animal models, but no randomized controlled human trials have proven efficacy. (See "[Overview of the management of epilepsy in adults](#)", section on 'Alternative therapies' and "[Evaluation and management of drug-resistant epilepsy](#)", section on 'Cannabinoids' and "[Seizures and epilepsy in children: Refractory seizures and prognosis](#)", section on 'Cannabinoids'.)

Toxic effects — Recreational cannabis intake to achieve psychoactive effects can often result in adverse effects because there is no clear demarcation between doses that achieve symptoms desired by a marijuana user and noxious effects.

In adolescents and adults, inhaled doses of 2 to 3 mg of delta-9 tetrahydrocannabinol (THC) and ingested doses of 5 to 20 mg THC impair attention, concentration, short-term memory and executive functioning [13,15-24,34-39]. More severe adverse effects may occur at doses >7.5 mg/m² THC, including nausea, postural hypotension, delirium, panic attacks, anxiety, and myoclonic jerking [34,35]. Psychosis has also been associated with use of higher potency/concentrated marijuana products [40,41].

Toxicity in children is most often reported after ingestion of a highly concentrated food product [7,8,42-47] or hashish resin [48]. Estimated oral doses from 5 to 300 mg in pediatrics have caused a range of symptoms such as mild sleepiness, ataxia, behavior changes, excessive and purposeless motor activity of the extremities (hyperkinesia), coma, and respiratory depression with more severe intoxication correlated with higher estimated doses. For example, in a small cohort of 38 children presenting to an emergency department for acute marijuana intoxication after ingestion, degree of symptoms corresponded to an estimated dose as follows: 3.2 mg/kg of THC led to observation and minimal medical intervention, 7.2 mg/kg of THC led to admission to an inpatient floor and moderate medical intervention, and 13 mg/kg of THC led to admission to an intensive care unit and major medical interventions [49]. Patients without prior THC exposure more commonly had lethargy or somnolence and had a longer duration of clinical symptoms. Similarly, as concentrated hashish resin has become more available in France, a corresponding increase in the number and severity of annual admissions has occurred among infants and young children [48].

CLINICAL MANIFESTATIONS — The clinical manifestations of acute cannabis (marijuana) intoxication vary according to age.

Neurologic abnormalities are more prominent in children and include ataxia, excessive and purposeless motor activity of the extremities (hyperkinesia), lethargy, and prolonged coma, which may be life-threatening [7,8,42-48].

Acute marijuana intoxication is an unusual primary complaint in adolescents and adults. Patients who come to medical attention are more likely to have hyperemesis or behavioral problems (eg, dysphoria or agitation) caused by adverse cannabis effects or medical emergencies (eg, bronchospasm or pneumothorax) associated with the method of inhalation. Chest pain with myocardial infarction in young adults without any prior history of coronary artery abnormalities has also been rarely described [50-53]. The issue of causation is unclear, however, in light of the frequency of cannabis use in the general population and the presence of unsuspected atherosclerosis or other cardiac condition in some of these patients.

Children — In children, acute marijuana intoxication typically occurs after exploratory ingestion of marijuana intended for adult use [7-9,43-47,54]. Less commonly, intentional exposure of children by caretakers, including encouragement of cannabis inhalation to promote sleepiness and to decrease activity, has been reported [55].

Pediatric ingestions of marijuana products happen more frequently in regions with decriminalization or legalization of cannabis use [7-9,56]. (See 'Epidemiology' above.)

After limited exposures, children may display sleepiness, euphoria, irritability, and other changes in behavior [7,8,43-47,54-57]. Vital signs may show sympathomimetic effects (eg, tachycardia and hypertension) or, in patients with depressed mental status, bradycardia. Nausea, vomiting, conjunctival injection, nystagmus, ataxia, and, in verbal children, slurred speech may also be present. Dilated pupils have frequently been reported, although miosis has also been described [57-60].

In large overdoses (eg, ingestion of edible products, concentrated oils, or hashish), coma with apnea or depressed respirations can occur [7-9,43-48,57].

Although not typical of pediatric cannabis intoxication, seizures have also been reported [44,54,59]. In one instance, cocaine was also found on urine screening [59]. In one retrospective series of 29 children under age 3 admitted with documented cannabis exposure, seizures occurred in four patients, all of whom had ingested hashish resin [54].

Adolescents and adults — The physiologic signs of cannabis intoxication in adolescents and adults include [13,15,16]:

- Tachycardia
- Increased blood pressure or, especially in the elderly, orthostatic hypotension
- Increased respiratory rate
- Conjunctival injection (red eye)
- Dry mouth
- Increased appetite
- Nystagmus
- Ataxia
- Slurred speech

Complications associated with inhalation use include:

- Acute exacerbations and poor symptom control in patients with asthma [61].
- Pneumomediastinum and pneumothorax suggested by tachypnea, chest pain, and subcutaneous emphysemas caused by deep inhalation with breathholding [62].
- Rarely, angina and myocardial infarction [63-65].

The risk for myocardial infarction among regular cannabis users has been found to be as high as 4.8 times baseline [63].

Cannabis intoxication in adolescents and adults also results in the following neuropsychiatric effects:

- **Mood, perception, thought content** – Ingestion typically leads to feeling "high," marked by a euphoric, pleasurable feeling and a decrease in anxiety, alertness, depression, and tension. However, first-time cannabis users, as well as anxious or psychologically vulnerable individuals, may experience anxiety, dysphoria, and panic [16]. Increased sociability usually occurs during intoxication, although dysphoric

reactions may be accompanied by social withdrawal. Inexperienced users who ingest cannabis products may not be aware that effects may not be felt for up to three hours which may cause them to continue to consume high potency products with an increased likelihood of dysphoria. (See '[Pharmacokinetics](#)' above.)

Perceptual changes include the sensation that colors are brighter and music is more vivid [16]. Time perception is distorted in that perceived time is faster than clock time. Spatial perception can also be distorted, and high doses of potent cannabis products may cause hallucinations. Mystical thinking, increased self-consciousness, and depersonalization may occur, as well as transient grandiosity, paranoia, and other signs of psychosis [16,66].

- **Cognition, psychomotor performance** – Cannabis use decreases reaction time and impairs attention, concentration, short term memory, and risk assessment. These effects are additive when cannabis is used in conjunction with other central nervous system depressants [16]. Acute cannabis use also impairs motor coordination and interferes with the ability to complete complex tasks that require divided attention.

Impairment of cognition, coordination, and judgment lasts much longer than the subjective mood change of feeling "high." Psychomotor impairment lasts for 12 to 24 hours due to accumulation of marijuana in adipose tissue, slow release of THC from fatty tissue stores, and enterohepatic recirculation. However, a marijuana user may think that he or she is no longer impaired several hours after the acute mood altering effects have resolved. As an example, a placebo controlled trial with licensed pilots found that smoking marijuana impaired performance on a flight simulator for up to 24 hours, although only one of the nine subjects possessed self-awareness of this [67].

Acute psychomotor impairments interfere with the ability to operate other heavy machinery, such as automobiles, trains, and motorcycles. A meta-analysis of nine studies found an association between cannabis intoxication and an increased risk of a motor vehicle collision involving serious injury or death [16,38,68,69]. Drivers using cannabis are two to seven times more likely to be responsible for accidents compared to drivers not using any drugs or alcohol [70]. Furthermore, the probability of causing an accident increases with plasma levels of delta-9-tetrahydrocannabinol [38].

Drug testing for cannabinoids — Hospital testing for cannabis typically consists of urine drug screen. (See '[Diagnosis](#)' below.)

Standard urine drug screens that are available in most healthcare facilities consist of immunoassays that detect delta-9 tetrahydrocannabinol (THC) metabolites, primarily THC carboxylase. The lower limits of detection range from 20 to 100 ng/mL, depending upon the specific assay [71]. The Substance Abuse and Mental Health Services Administration (SAMSHA) standard is 50 ng/mL, with confirmatory testing using 15 ng/mL, as the lower limits of detection [72].

In situations where a positive screen for cannabis has legal implications or may impact school attendance or sports participation, individuals may claim that the test results from passive inhalation of marijuana smoked by others. In adolescents and adults, it is difficult to achieve sufficient concentrations from secondhand smoke from typical cannabis cigarettes to detect metabolite concentrations above most urine drug screen limits [73-77]. However, studies using products with higher THC content (typical of what is more commonly used since 2005) have not been performed. Also, the potential for second-hand cannabis exposure to cause a positive screen in children has not been studied.

False positives for cannabinoids are rare, because the chemical structure is unique and immunoassays are targeted toward metabolites of THC. Reported false positives for THC include: [dronabinol](#), [efavirenz](#), proton pump inhibitors, hemp seed oil, nonsteroidal antiinflammatory drugs (NSAIDs), and baby wash products in infants [78-83]. However, most package inserts for commercially available immunoassays will list possible false

positives for their cannabinoid assay. If required for clinical or social indications, confirmatory testing of urine, blood or serum can be sent to reference labs by gas chromatography and mass spectrophotometry. However, results of confirmatory testing do not return quickly enough to affect clinical care.

Other ancillary studies — Most adolescents and adults do not warrant any testing for the diagnosis or treatment of acute cannabis (marijuana) intoxication. Patients with chest pain suggestive of myocardial ischemia or infarction warrant a 12-lead electrocardiogram and possibly cardiac biomarkers (eg, troponin T or I). (See "[Troponin testing: Clinical use](#)", [section on 'Diagnosis of acute MI'](#).)

Chest radiograph may assist in the diagnosis of stable patients with chest pain indicative of a spontaneous pneumothorax. However, patients with signs of a tension pneumothorax should undergo decompression prior to chest radiography. Bedside ultrasound may assist with rapid diagnosis of pneumothorax in these unstable patients. (See "[Primary spontaneous pneumothorax in adults](#)", [section on 'Imaging'](#) and "[Thoracic ultrasound: Indications, advantages, and technique](#)".)

Children may warrant testing for other potential causes of altered mental status depending upon whether the exposure is known and based upon specific physical findings including rapid blood glucose, electrolytes, blood gas analysis, and neuroimaging (eg, computed tomography of the head). Neuroimaging should be avoided in known cannabis exposures unless focal neurologic findings are also present or concerns for other etiologies such as head trauma exist. (See "[Differential diagnosis](#)" below.)

DIAGNOSIS — Regardless of age, acute cannabis intoxication is a clinical diagnosis. However, diagnosis in the pediatric population can be difficult because a history of exposure is often lacking and the symptoms of marijuana exposure are nonspecific. Thus, urine drug screens can be helpful in confirming the diagnosis because any positive result in children identifies acute exposure. (See "[Drug testing for cannabinoids](#)" above.)

Urine drug screens are less helpful in adolescents and adults for the diagnosis of acute intoxication. Although testing is usually positive several hours after acute exposure it can also be positive well after symptoms have resolved. As an example, positive results for delta-9 tetrahydrocannabinol metabolites have been reported up to 10 days after weekly use and up to 25 days for after daily use [84]. Thus, cannabis testing does not provide any specific information on the timeline of exposure or correlate with severity of intoxication.

DIFFERENTIAL DIAGNOSIS

Children — The differential diagnosis for cannabis exposure in children is broad because toxicity most commonly presents as altered behavior, lethargy, or coma. When history of exposure is lacking, a positive rapid urine drug screen for cannabinoids is helpful for identifying cannabis as the likely culprit in children too young to be using marijuana recreationally. (See "[Drug testing for cannabinoids](#)" above.)

Some common medical causes of lethargy and coma and differentiating features from cannabis (marijuana) intoxication include (see "[Evaluation of stupor and coma in children](#)", [section on 'Etiologies'](#)):

- **Hypoglycemia** – Low rapid blood sugar (see "[Causes of hypoglycemia in infants and children](#)")
- **Electrolyte imbalance** (eg, hyponatremia or hypocalcemia) – Low serum sodium or calcium (see "[Hyponatremia in children](#)", [section on 'Etiology'](#) and "[Etiology of hypocalcemia in infants and children](#)")
- **Central nervous system infection** (eg, meningitis or encephalitis) – Fever and/or meningismus
- **Traumatic brain injury, especially abusive head trauma** – Child abuse may present with intracranial injury (eg, subdural hematoma) without an appropriate mechanism by history, retinal hemorrhages, skin bruising, and/or fractures (see "[Child abuse: Evaluation and diagnosis of abusive head trauma in infants and children](#)", [section on 'Clinical features'](#))

A positive urine drug screen for metabolites of cannabis also helps differentiate cannabis (marijuana) intoxication from poisoning with other agents but is not immediately available in most facilities. Toxicologic causes of lethargy and coma in children are extensive ([table 1](#)). The following agents and important clinical features that distinguish them from cannabis (marijuana) intoxication include:

- **Opioids** – Lethargy and coma following cannabis ingestion does not respond to [naloxone](#), which differentiates it from toxicity caused by opioid analgesics. (See "[Opioid intoxication in children and adolescents](#)", [section on 'Clinical manifestations'](#).)
- **Sedative/hypnotic agents** – Cannabis toxicity typically causes more tachycardia or seizure-like activity than sedative/hypnotics. (See "[Benzodiazepine poisoning and withdrawal](#)", [section on 'Clinical features of overdose'](#) and "[Meprobamate poisoning](#)", [section on 'Clinical features of overdose'](#).)
- **Ethanol** – A sickly sweet breath odor, hypoglycemia (when present), and an elevated blood alcohol concentration are important findings of ethanol intoxication. (See "[Ethanol intoxication in children: Clinical features, evaluation, and management](#)", [section on 'Clinical features'](#).)
- **Oral hypoglycemic agents** – Patients typically have a normal mental status and examination unless hypoglycemia is present. Cannabis intoxication is **not** associated with hypoglycemia. (See "[Sulfonylurea agent poisoning](#)", [section on 'History and physical examination'](#).)
- **Clonidine** – Some patients with coma from clonidine poisoning will respond to [naloxone](#) administration. Patient will also show more signs of hemodynamic instability such as bradycardia and hypotension. (See "[Clonidine and related imidazoline poisoning](#)", [section on 'Clinical features and diagnosis'](#).)
- **Antihistamines** – Significant toxicity is often associated with anticholinergic findings (dilated pupils, flushing, dry skin and mouth, decreased breath sounds, and delirium with seizures). (See "[Anticholinergic poisoning](#)" and "[Anticholinergic poisoning](#)", [section on 'Clinical features of overdose'](#).)
- **Carbon monoxide** – Other members of the household may have flu-like symptoms in patients with carbon monoxide poisoning and carboxyhemoglobin will be elevated. (See "[Carbon monoxide poisoning](#)", [section on 'Clinical presentation'](#).)
- **Psychotropic agents (eg, antipsychotic or antidepressant agents)** – These agents often are associated with anticholinergic findings as described above and may be associated with arrhythmias or electrocardiographic abnormalities (eg, prolonged QRS or QT intervals). (See "[First generation \(Typical\) antipsychotic medication poisoning](#)", [section on 'Clinical features of overdose'](#) and "[Second generation \(atypical\) antipsychotic medication poisoning](#)", [section on 'Clinical features of overdose'](#).)

Adolescents and adults — Several other commonly used recreational drugs have some overlapping clinical features with cannabis intoxication in adolescents and adults including:

- Cocaine (see "[Cocaine: Acute intoxication](#)", [section on 'Clinical manifestations'](#))
- Amphetamines and methcathinones (bath salts) (see "[Acute amphetamine and synthetic cathinone \("bath salt"\) intoxication](#)", [section on 'Clinical features of overdose'](#) and "[Methamphetamine: Acute intoxication](#)", [section on 'Clinical features'](#))
- Lysergic acid diethylamide (LSD) and other hallucinogens (eg, phencyclidine [PCP], [dextromethorphan](#), or psilocybin) (see "[Intoxication from LSD and other common hallucinogens](#)", [section on 'General clinical features of intoxication'](#))
- MDMA (ecstasy) (see "[MDMA \(ecstasy\) intoxication](#)", [section on 'Clinical features'](#))

- Synthetic cannabinoids (see "[Synthetic cannabinoids: Acute intoxication](#)", section on 'Clinical manifestations')

Cannabis is frequently used recreationally with these drugs or may serve as a vehicle for use (eg, lacing cannabis cigarettes with PCP). Acute cannabis (marijuana) intoxication does not produce severe agitation, extreme vital sign abnormalities (eg, hyperthermia, marked tachycardia, or hypertension), or significant altered mental status in adolescents and adults. Also, the duration of intoxication is typically shorter than for other recreational drugs. Thus, investigation for other intoxicants is indicated if symptoms are prolonged, or if other marked physiologic abnormalities exist such as hyperthermia, acidosis, significant rhabdomyolysis, or end-organ toxicity.

Synthetic cannabinoids (eg, Spice or K2) cause findings that are very similar to cannabis intoxication but are more frequently associated with more pronounced sympathomimetic effects, aggressive behavior and agitation, dystonia, and seizures. A urine drug screen for cannabinoids will typically be negative after synthetic cannabinoid use. (See "[Synthetic cannabinoids: Acute intoxication](#)", section on 'Clinical manifestations'.)

Cannabis use may exacerbate preexisting mental illness (eg, psychosis, anxiety, or depression). Thus, clinicians should ask about cannabis use in patients who display worsening of known psychiatric disease.

MANAGEMENT — The management of cannabis (marijuana) intoxication consists of supportive care. Because of the differences in toxic manifestations the management differs significantly by age.

Children — Children with cannabis (marijuana) exposure are much more likely to demonstrate severe or life-threatening toxicity consisting of excessive and purposeless motor activity (hyperkinesia) or deep coma. Consultation with a regional poison control center and a medical toxicologist is encouraged for all symptomatic exposures. (See '[Additional resources](#)' below.)

Central nervous system depression — Severe central nervous system (CNS) depression from marijuana exposure is unique to the pediatric population and can present with profound depression, lethargy, and coma.

Treatment is supportive and consists of the following measures:

- Maintain airway, breathing, and circulation. Patients with lethargy and coma should receive supplemental oxygen, assessment and support of airway and breathing, and vascular access. Patients with apnea or at risk for aspiration should undergo rapid sequence endotracheal intubation and receive assisted ventilation ([table 2](#)). (See "[Emergency endotracheal intubation in children](#)", section on 'Indications' and "[Rapid sequence intubation \(RSI\) outside the operating room in children: Approach](#)", section on 'Indications'.)
- Measure rapid blood glucose to exclude hypoglycemia.
- Administer [naloxone](#) to patients presenting with features of opioid intoxication. Naloxone will **not** reverse coma due to cannabis toxicity.

The duration of coma is typically one to two days [[45,46,58,59](#)]. Full recovery is expected.

Seizures — Seizures have rarely been described after cannabis intoxication in children and may be associated with coingestants (eg, cocaine) [[44,46,48,59](#)]. Initial treatment of toxin-associated seizures consists of benzodiazepines (eg, [lorazepam](#) or [midazolam](#)). If seizures persist despite multiple doses of benzodiazepines, then treatment for status epilepticus caused by toxins, as described in the table, is warranted ([table 3](#)). (See "[Management of convulsive status epilepticus in children](#)".)

Dysphoria — Dysphoria is not a common presentation in pediatric marijuana exposure. However, if symptoms of marked anxiety or agitation develop, benzodiazepines (eg, [lorazepam](#)) are frequently effective

and have a low adverse effect profile.

Adolescents and adults

Mild intoxication — Mild intoxication with dysphoria can be a common presentation in either naïve or chronic marijuana users after ingestion or inhalation of a high-potency product such as an edible or concentrate. Most patients can be managed with a dimly lit room, reassurance, and decreased stimulation. Short-acting benzodiazepines (eg, [lorazepam](#)) can be helpful in controlling symptoms of anxiety and have a low side effect profile.

Severe intoxication — Severe physiologic effects are rare after cannabis use and their presence should prompt the clinician to consider coingestion of other recreational drugs including cocaine, amphetamines, and phencyclidine or coexisting mental illness. (See '[Adolescents and adults](#)' above.)

Marked agitation or combativeness not responsive to reassurance and benzodiazepines may necessitate the use of other medications, depending upon the cause, and is rarely encountered with intoxication from cannabis alone. The approach to sedation of the acutely agitated or violent adult is discussed in detail separately. (See '[Assessment and emergency management of the acutely agitated or violent adult](#)', section on '[Chemical sedation](#)'.)

Chest pain — Chest pain in association with cannabis use should be managed according to etiology as follows:

- **Acute coronary syndrome** – Substernal squeezing chest pain suggestive of myocardial ischemia or infarction may occur rarely in association with cannabis use [[50,51,64,65](#)]. Patients complaining of chest pain suggestive of coronary insufficiency should be evaluated for acute coronary syndrome and treated accordingly. (See '[Initial evaluation and management of suspected acute coronary syndrome \(myocardial infarction, unstable angina\) in the emergency department](#)', section on '[Clinical presentation](#)' and '[Initial evaluation and management of suspected acute coronary syndrome \(myocardial infarction, unstable angina\) in the emergency department](#)', section on '[Management](#)'.)
- **Pneumothorax or pneumomediastinum** – Inhalation and breathholding during cannabis use may cause a pneumothorax or pneumomediastinum with sharp, pleuritic chest pain and subcutaneous crepitus. Management of a pneumothorax depends upon its size and includes oxygen administration and, if necessary, evacuation with needle decompression or chest tube insertion. (See '[Secondary spontaneous pneumothorax in adults](#)', section on '[Initial management](#)'.)

No specific treatment is necessary for uncomplicated pneumomediastinum. (See '[Spontaneous pneumomediastinum in children and adolescents](#)', section on '[Treatment](#)'.)

- **Asthma exacerbation** – Cannabis use may cause chest tightness with bronchospasm and wheezing. Standard therapy for status asthmaticus should be provided. (See '[Management of acute exacerbations of asthma in adults](#)', section on '[Algorithms for assessment and treatment](#)'.)

Gastrointestinal decontamination — We suggest that patients who ingest cannabis (marijuana) **not** undergo gastrointestinal decontamination with activated charcoal (AC). After ingestion, most symptoms are delayed up to three hours, which limits the efficacy of AC. Also, the clinical effects of cannabis ingestion are often limited and good outcomes occur with supportive care alone. In addition, in children, clinical toxicity may include rapid onset of altered mental status or vomiting, which may raise the risk of aspiration if AC is administered.

There is no role for gastrointestinal decontamination after toxicity caused by inhaled cannabis.

Cannabis hyperemesis syndrome — Cannabis hyperemesis syndrome is typically seen with chronic marijuana use but can be seen with acute or acute on chronic use. Patients may complain of abdominal pain, vomiting, or nausea that is typically relieved by hot showers. Acute treatment consists of symptomatic care including intravenous fluid hydration, antiemetics (eg, [ondansetron](#)), and benzodiazepines [85,86]. Cessation of marijuana use is also recommended.

Limited observational evidence (case reports and case series) also suggests that topical [capsaicin](#) cream (supplied in concentrations of 0.025 to 0.1 percent) applied once in a thin film over the abdomen may improve acute severe abdominal pain and emesis in patients not responsive to [ondansetron](#) or benzodiazepines [87-90]. Evidence is lacking to determine if capsaicin cream has a role for the treatment of chronic symptoms.

In addition, case reports have documented the successful use of [haloperidol](#) to abort severe episodes of hyperemesis not responsive to fluid hydration and administration of antiemetics, and benzodiazepines [91,92]. In one instance, hospital admission was avoided after administration of 5 mg of haloperidol intravenously. However, more evidence is needed to evaluate the safety and efficacy of this therapy including the indications, dose, and route of administration. (See "[Cyclic vomiting syndrome](#)", section on '[Chronic cannabis use](#)'.)

DISPOSITION — Disposition is determined by several factors including patient age, social circumstances, duration of toxicity, and type of symptoms as follows:

- **Children** – The duration of symptoms after acute marijuana exposure in children can vary from four to 48 hours depending upon the dose ingested [7,8]. Patients with persistent vomiting, altered mental status, or excessive, purposeless motor activity (hyperkinesia) warrant hospital admission.

Patients who remain asymptomatic or become asymptomatic following exploratory ingestion of legally acquired cannabis products may be discharged after a brief observation period (eg, four to six hours after ingestion).

Ingestion of illicit marijuana or intentional exposure of a child warrants involvement of a child abuse team, when possible, and should be reported to child protection services. (See "[Child abuse: Social and medicolegal issues](#)", section on '[Reporting suspected abuse](#)'.)

- **Adolescents and adults** – Most symptoms after acute marijuana use in adults and adolescents resolve within a few hours and will **not** require hospital admission.

Hospital admission may rarely be needed for prolonged delirium or agitation requiring repeated doses of benzodiazepines or antipsychotics. These patients should also be screened for substance use disorder, mood disorders, and, if needed, undergo psychiatric consultation and appropriate referrals to substance-use treatment programs. (See "[Treatment of cannabis use disorder](#)".)

The disposition for patients with complications of marijuana use depends upon the degree of illness and response to therapy. Patients with proven myocardial infarction or pneumothorax requiring chest tube thoracostomy warrant hospital admission to an appropriate level of care.

ADDITIONAL RESOURCES — Regional poison control centers in the United States are available at all times for consultation on patients who are critically ill, require admission, or have clinical pictures that are unclear (1-800-222-1222). In addition, some hospitals have clinical and/or medical toxicologists available for bedside consultation and/or inpatient care. Whenever available, these are invaluable resources to help in the diagnosis and management of ingestions or overdoses. The World Health Organization provides a listing of international poison centers at its [website](#).

The Partnership at Drugfree.org maintains a [drug guide](#) for 40 commonly abused drugs including common slang terms.

SOCIETY GUIDELINE LINKS — Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "[Society guideline links: Cannabis use disorder and withdrawal](#)" and "[Society guideline links: General measures for acute poisoning treatment](#)" and "[Society guideline links: Poisoning prevention](#)".)

SUMMARY AND RECOMMENDATIONS

- Serious cannabis intoxication is rare in adolescents and adults. Exploratory ingestions of marijuana products have been described in young children and are more frequent in regions with decriminalization or legalization of cannabis use. Less commonly, intentional exposure of children by caretakers, including encouragement of cannabis inhalation to promote sleepiness and to decrease activity, has been reported. (See '[Epidemiology](#)' above and '[Children](#)' above.)
- In young children, ingestion of cannabis (marijuana) may cause life-threatening coma with apnea or depressed respirations. Seizures have also been reported. Other features following limited pediatric exposures include behavioral changes, lethargy, and physiologic effects of intoxication as seen in adolescents and adults. (See '[Children](#)' above.)
- Children may warrant testing for other potential causes of altered mental status depending upon whether the exposure is known and based upon specific physical findings. (See '[Other ancillary studies](#)' above and '[Differential diagnosis](#)' above.)
- The findings of cannabis intoxication in adolescents and adults include tachycardia, blood pressure changes (hypertension, or in the elderly, orthostatic hypotension), conjunctival injection, dry mouth, increased appetite, nystagmus, ataxia, slurred speech, euphoria, perceptual changes, and psychomotor impairment. (See '[Adolescents and adults](#)' above.)
- Chest pain in adolescents and adults who use cannabis recreationally may arise from a pneumothorax, exacerbation of underlying pulmonary disease such as asthma, or, rarely, myocardial ischemia or infarction. (See '[Adolescents and adults](#)' above.)
- In adolescents and adults, no specific testing is necessary. Investigation for other intoxicants may be indicated if symptoms are prolonged, or if other marked physiologic abnormalities exist such as hyperthermia, acidosis, significant rhabdomyolysis, or end-organ toxicity. (See '[Adolescents and adults](#)' above.)
- Regardless of age, acute cannabis intoxication is a clinical diagnosis. Urine drug screens can be helpful in confirming the diagnosis in young children because any positive result identifies acute exposure. Urine drug screens are less helpful in adolescents and adults and are not routinely needed for diagnosis or management. (See '[Diagnosis](#)' above and '[Drug testing for cannabinoids](#)' above.)
- Management of young children with ingestion of cannabis (marijuana) consists of exclusion of hypoglycemia in patients with altered mental status and supportive care of coma. Excessive muscle activity (hyperkinesia) should be initially treated with benzodiazepines (eg, [diazepam](#) or [lorazepam](#)). (See '[Children](#)' above.)
- Most adolescents and adults presenting for treatment of acute cannabis intoxication have mild intoxication with dysphoria that can be managed with a dimly lit room, reassurance, decreased stimulation, and, in some patients, benzodiazepines (eg, oral [lorazepam](#)). (See '[Mild intoxication](#)' above.)

- Chest pain in association with cannabis use should be managed according to the underlying etiology (eg, acute coronary syndrome, pneumothorax, or asthma exacerbation). (See '[Chest pain](#)' above.)
- We suggest that patients who ingest cannabis (marijuana), either unintentionally or for recreational use, **not** undergo gastrointestinal decontamination with activated charcoal (AC) (**Grade 2C**). (See '[Gastrointestinal decontamination](#)' above.)

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Science News

from research organizations

Developing a roadside test for marijuana intoxication isn't as easy as it sounds

Date: January 25, 2018

Source: Cell Press

Summary: As marijuana legalization gains momentum in the United States, researchers worry about keeping the public safe, particularly on the roads. Recent studies have identified new biomarkers that can be used to estimate a person's recent cannabinoid intake. But, using those markers to judge cognitive and behavioral impairment is complex, say toxicologists.

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FULL STORY

As the movement to legalize marijuana in the United States gains momentum, researchers worry about keeping the public safe, particularly on the roads. Recent studies in which marijuana users took controlled doses of cannabis in the lab have identified new biomarkers that can be used to estimate a person's recent cannabinoid intake. But, using those markers to judge cognitive and behavioral impairment is complex, say toxicologists in a commentary published on January 25 in a special issue of the journal *Trends in Molecular Medicine* on biomarkers of substance abuse.

"There is no one blood or oral fluid concentration that can differentiate impaired and not impaired," says Marilyn Huestis, who spent over 20 years leading cannabinoid-related research projects at the National Institute on Drug Abuse. "It's not like we need to say, 'Oh, let's do some more research and give you an answer.' We already know. We've done the research."

Alcohol can impair a user more than cannabis, and indeed, the risk of an accident while driving increases in proportion with blood alcohol concentrations. But pot is different: many variables can affect how impaired someone is at any given concentration of Δ^9 -tetrahydrocannabinol (THC), the primary psychoactive agent in cannabinoids. Whether it is inhaled or consumed, or whether the user titrates their own dose, can affect the level of impairment. And pairing cannabis with alcohol makes the high higher, and the alcohol buzz last longer.

Another problem is that THC quickly leaves the bloodstream. Previous research by Huestis has shown that while an occasional user is impaired for 6 to 8 hours, blood THC concentrations can be effectively zero after 2.5 hours. And on average in the United States, it takes from 1.4-4 hours after a crash or traffic stop to administer a blood test. "If someone is driving impaired, by the time you get their blood sample, you've lost 90% or more of the drug. So, we have to change what we do at the roadside," says Huestis.

Long-term daily cannabis users, like those who use marijuana for medical reasons, also present a challenge for developing roadside protocols. THC accumulates in the tissues of the body and then slowly releases over time, meaning that chronic users can test positive for cannabis even after 30 days of abstinence. Psychomotor impairment can be observed three weeks after the last dose. "You want people to be taking medicinal cannabinoids and now you know that their driving is going to be impacted," says Huestis. "So how do you handle that problem?"

Huestis, like most researchers, doesn't support a legal driving limit for cannabis like the one in place for blood alcohol concentrations. Instead, she advocates for well-trained police officers who can identify the behavioral signs of impairment and less invasive biological marker tests, which could be immediately performed at the roadside to confirm the presence of a cannabinoid. To that end, recent research has identified new blood and urine markers, and tests using breath and saliva markers are being developed.

The implications go beyond driving. These new markers and tests could also be used to assist in treating drug dependence, in determining appropriate therapeutic levels of medical marijuana, and for monitoring women who want to stop using cannabinoids during pregnancy.

Huestis, who also owns a toxicology consulting company with her co-author, Michael Smith, isn't opposed to legalization. But she does want to make sure that marijuana's status as a legal drug and a medicine doesn't make us complacent. "Cannabis probably is less dangerous to use than alcohol," she says. "There's less morbidity and mortality associated with it, but there's still a lot of problems. And we as a public are not recognizing this."

The Intramural Research Program of the National Institute on Drug Abuse, and the National Institutes of Health funded this research.

Story Source:

Materials provided by **Cell Press**. *Note: Content may be edited for style and length.*

Journal Reference:

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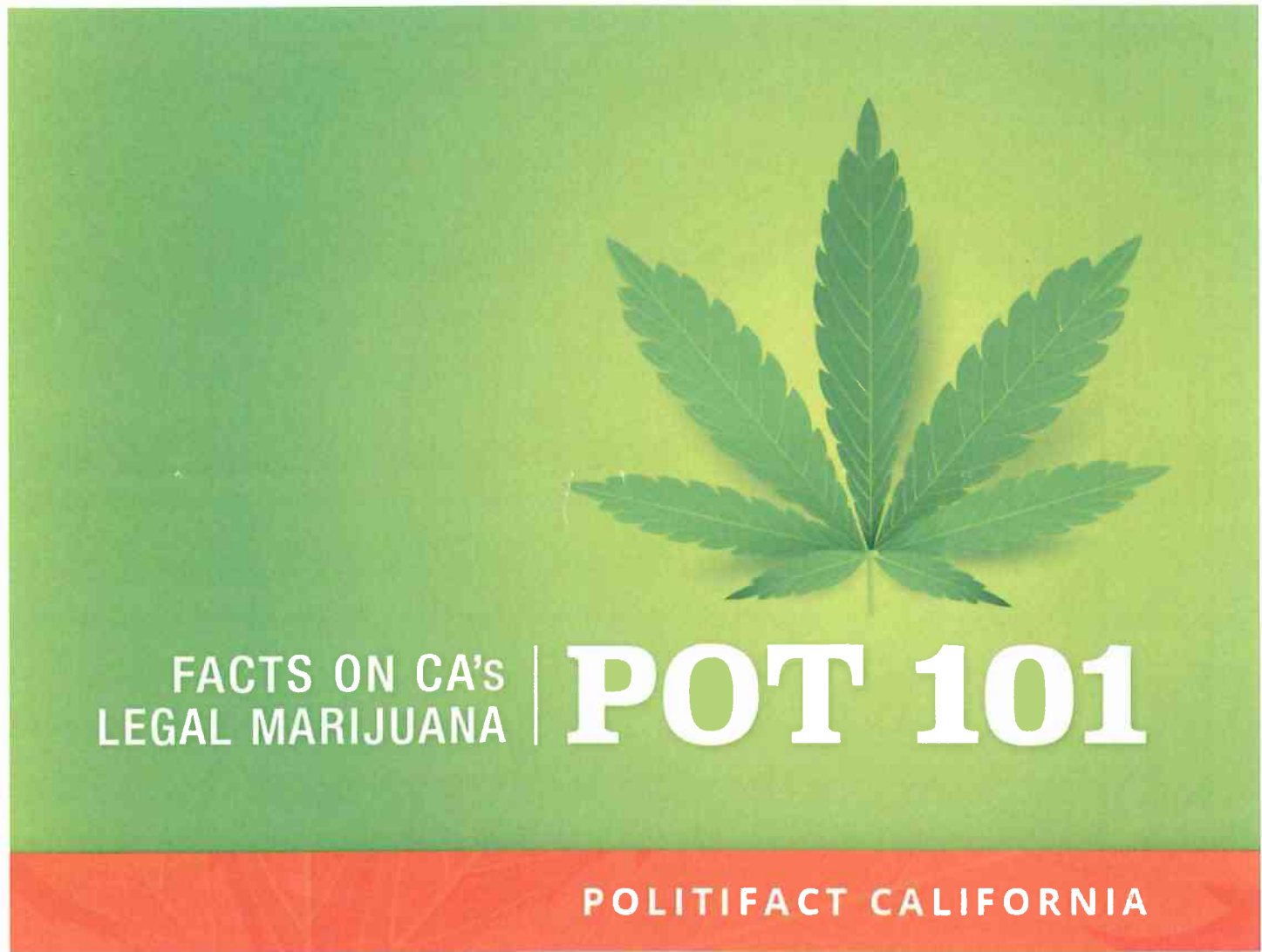
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Recreational Marijuana Legalization: Do More Youth Use or Do Youth Use More?



Pot 101: Facts you should know about California's legal marijuana

By *Chris Nichols* on Friday, January 5th, 2018 at 1:50 p.m.



California's recreational pot law doesn't offer the complete freedom to buy, smoke or transport weed anywhere you please.

Recreational marijuana sales became legal in California on Jan. 1, 2018, marking a major milestone for the state's cannabis industry and its many customers.

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Proposition 64, the voter-approved [law](#) that governs recreational pot in the Golden State, however, doesn't offer the complete freedom to buy, grow and smoke marijuana just anywhere you please.

In reality, the rules are fairly strict. And they are consequential for employers and employees and anyone who wants to sell, purchase, consume or transport cannabis in the state.

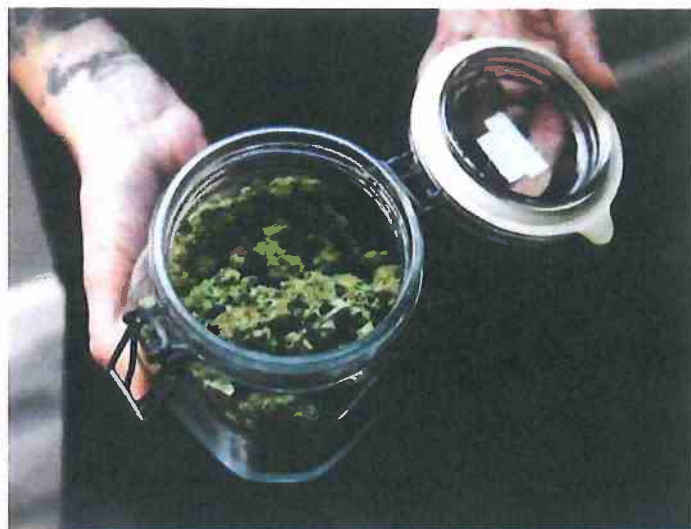
Cloud over legal weed?

There's also the complication that marijuana remains illegal federally.

That point was made clear this week when U.S. Attorney General Jeff Sessions [rescinded](#) an Obama administration memo that recommended a hands-off approach to marijuana prosecution in states that have legalized pot. The move was seen as possibly paving the way for a federal crackdown on marijuana, though it's uncertain whether that will happen.

President Trump, during his campaign for the White House, pledged to leave legalization up to the states, a [topic we previously examined](#).

Below are some of the other key facts about what's legal and what's not -- at least under state law -- in California's new cannabis landscape. It's not a complete guide but we plan to update it as more questions arise.



Consumers can purchase recreational marijuana at licensed dispensaries in California, but can't consume it on public property. Andrew Nixon / Capital Public Radio



A customer purchases marijuana at Harborside marijuana dispensary, Monday, Jan. 1, 2018, in Oakland, Calif. Starting New Year's Day, recreational marijuana can be sold legally in California. (AP Photo/Mathew Sumner)



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What are the rules for buying recreational marijuana?

The law permits adults 21 and up to buy 1 ounce of cannabis per day, which is enough material to fill **a few dozen joints**. You can alternately purchase up to 8 grams of cannabis concentrates found in marijuana edibles such as candies, brownies and breakfast bars.

The number of edibles you can legally possess depends on the product itself. A can of cannabis butter, for example, contains a larger amount of concentrates than a single edible, so concentrate-heavy products could put the carrier over the legal limit.

No matter the kind of cannabis you want, you'll need to visit a state-licensed recreational dispensary. The agency that regulates marijuana, the California Bureau of Cannabis Control, has a complete and updated list of licensed dispensaries on its [website](#).

You'll need a valid ID or driver's license. And you'll also likely need cash as most dispensaries don't accept checks or credit cards.

Finally, don't expect to see any 24-hour pot shops: The law makes it illegal to sell between 10 p.m. and 6 a.m.



Different types of marijuana sit on display at Harborside marijuana dispensary, Monday, Jan. 1, 2018, in Oakland, Calif. Starting New Year's Day, recreational marijuana can be

sold legally in California. (AP Photo/Mathew Sumner)

How much will legal weed cost?

Buying legal recreational pot won't be cheap, at least not initially.

The cannabis culture website greenstate.com estimated legal marijuana buyers in California "will pay about eight dollars more" for their usual \$50 eighth of an ounce of "top-shelf flowers," under the new law. That cost is far more than on the black market, causing some marijuana [advocates to worry](#) customers will turn to underground sales.

Licensed dispensary costs include a new statewide 15 percent tax on all recreational and medical cannabis products plus additional local taxes and fees. In Oakland, for example, the added [local tax is 10 percent](#) for recreational pot and 5 percent for medical cannabis.

California consumers, however, could see prices reduced by as much as half over the next year if market patterns follow those in Washington state and Colorado, both of which earlier legalized recreational pot. That analysis came from cannabis analytics firm Headset and was [reported](#) on by MarketWatch.com early this year.

Where can you legally smoke or consume recreational pot? Short answer: Not in public

To stay within the law, you'll need to consume cannabis on private property. "You cannot consume, smoke, eat, or vape cannabis in public places," according to the state Department of Public Health's [website](#).

Smoking marijuana where tobacco is prohibited is also illegal, unless there is a local ordinance expressly allowing its usage. This includes school campuses, restaurants, bars, public parks and hospitals -- essentially any public building.

Don't expect to legally light up on campus: The University of California and California State University systems say they don't plan to change their policies barring marijuana, according to a recent Sacramento Bee [article](#).

No smoking pot while driving or riding in car

Prop 64 has [specific language](#) empowering employers to tailor their drug policy according to their wishes.

Employers that contract with the federal government may see a prohibition of use as well, since those employers will defer to federal law that still classifies marijuana as an illegal drug.


Since publication, we've added responses to several reader-generated questions below:


Can I take marijuana with me on a plane?

It is not legal to transport marijuana of any amount by plane, even if it's medicinal. That includes trips within state borders and to other states that have legalized marijuana. The Transportation Security Administration, or TSA, defers to federal law, which still classifies marijuana as a Schedule I illegal drug.

Although possession while attempting to travel by plane is strictly illegal, the [TSA website](#) says that "security officers do not search for marijuana or other drugs." A [TSA spokesman told the New York Times](#) in April 2017 that screeners look for things that can take down an airplane, not marijuana. But that is not an assurance by any means. While the TSA dogs may be sniffing around your luggage for explosives, the Customs and Border Protection dogs are searching for illegal substances, marijuana included. This one is pretty cut and dry - if you attempt to fly with marijuana, you are breaking the law.

How long after ingesting marijuana am I allowed to drive?

You are free to hit the road once you are no longer impaired. It is illegal to drive under the influence of any substance, marijuana included. There is not yet a standard way of measuring marijuana impairment, although researchers [are racing to develop technology](#) that can measure the drug's levels, much like a breathalyzer test authorities use for alcohol. Unlike the  .08 blood alcohol content measurement, there is [no established threshold](#) for THC levels in California that would automatically result in a DUI.

 THC, or [tetrahydrocannabinol](#), is the chemical compound in cannabis responsible for a euphoric high.

The California Office of Traffic Safety, or OTS, warns that even small doses of THC can slow a driver's reaction time and the ability to make decisions. [According to OTS](#), marijuana effects are strongest within the first hour of consumption and those who drive within that time



About this article

Researchers: [Chris Nichols](#), [Kathryn Palmer](#)

Names in this article: [Gavin Newsom](#), [Jeff Sessions](#), [Donald Trump](#)

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Legalizing Recreational Marijuana

Kody Kershner



A Protester makes known her argument at a rally. Photo courtesy of Wikimedia Commons

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For centuries marijuana has been a large problem in the world causing a lot of crime and leading to drug trafficking across the

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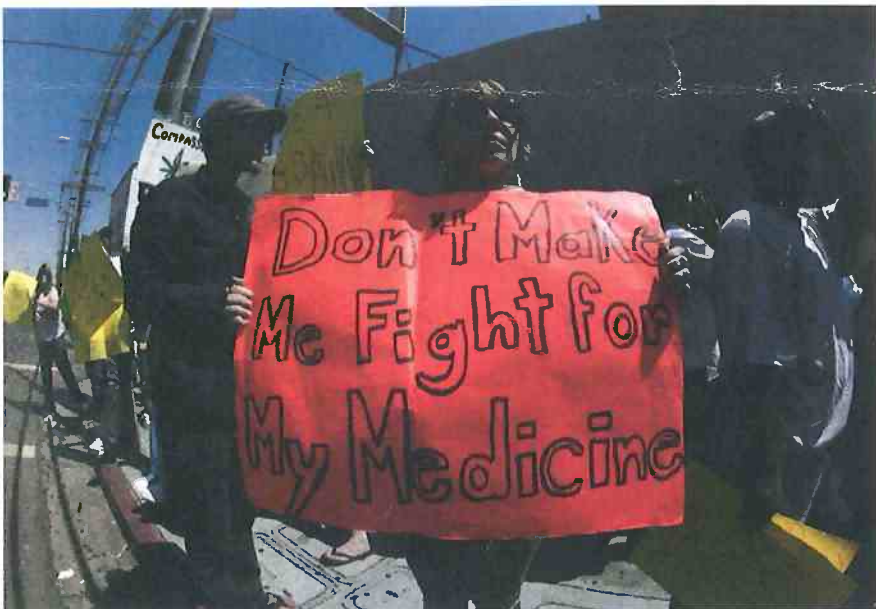
(<http://www.schoolnewspersonline.com/get-started>)

some users. Stopping the use of the substance cold turkey can cause withdrawal symptoms like irritability and anxiety, but the same could be said about cigarette smokers. More studies will need to be done to determine if marijuana is really as addictive as the opponents of cannabis legalization claim.

* Another downfall of the use is that users have decreased mental health. Users suffer from restricted blood flow to the brain, memory loss, and increased likeliness of depression. Cannabis alters your perception; like alcohol, this could lead to impaired driving. Opponents also claim that this could lead to an increase in harder crimes like robbery and violence because of your altered judgment. This drug is also considered a gateway drug, once a person tries marijuana they are more likely to want to try another drug to get that "better high".

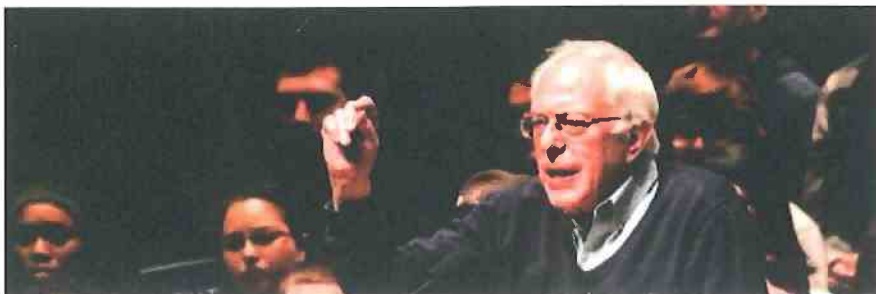
This issue will only be a more prominent

discussion in the world and will lead to more bills being passed and more meetings.



(<https://khqtoday.com/wp-content/uploads/2018/05/DEA.jpg>)

A Protester makes known her argument at a rally. Photo courtesy of Wikimedia Commons



SMOKEFREE IS SMOKEFREE

Nobody should have to breathe secondhand marijuana smoke at work, in public, or where they live.

Smoke is smoke — regardless of the device or description. Secondhand marijuana smoke contains hundreds of chemicals — just like secondhand tobacco smoke. Many of the chemicals in secondhand marijuana smoke are **toxic and contain hazardous fine particles that pose a significant health risk to non-smokers.**

- More laws legalizing marijuana = increased exposure to secondhand marijuana smoke.
- Employees and patrons protected by current smokefree laws may have their health put at risk by exposure to marijuana smoke. Marijuana smoking should not be allowed in smokefree spaces.
- The commercialized marijuana industry looks and sounds a lot like Big Tobacco. Together they are working to circumvent progress on smokefree air.
- The vast majority of the population are non-smokers. Smokefree means smokefree — no cigarettes and cigars, e-cigarette use, or marijuana use.

SECONDHAND MARIJUANA SMOKE

contains many of the same **CANCER-CAUSING SUBSTANCES** and **TOXIC CHEMICALS** as secondhand tobacco smoke, including:



3 times the amount of **ammonia**



significant levels of **mercury, lead, formaldehyde, benzene, hydrogen cyanide, & toluene.**

PROTECT HEALTH

Protect workers and the public from exposure to secondhand smoke by prohibiting marijuana smoking in all workplaces and enclosed public places.



Currently, approximately **266 municipalities** and **11 states** specifically restrict marijuana use in smokefree spaces in some manner. **Protect smokefree workplace laws — include marijuana in your policy!**

For more information about marijuana and smokefree laws, visit

 www.no-smoke.org



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