NEIGHBORHOOD COALITION

NeighborhoodCoalitionSonomaCounty.com

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Toxicities of Cannabis Volatile Emissions from Cultivation

Respiratory Irritation

Toxic and Carcinogenic Levels of ß-Myrcene

(Updated from October 18, 2024 Report)

SUMMARY

Volatile emissions released from cannabis plants during cultivation are known to cause respiratory harm, including irritation of the lower and upper respiratory track. Up to 70% of workers at indoor facilities without proper protection reported respiratory irritation, half with asthma, which can be serious, even fatal. Residents, customers and workers near outdoor cultivation fields breathe these same volatile cannabis emissions and have also experienced respiratory illness. Workers, often performing strenuous activities, on or near outdoor cultivation fields have increased exposure due to increased respiratory rates. Although workers are exposed 40-60 hr/week, residents are exposed to high emission levels continuously during flowering and harvest, often 6 months/year with two growth cycles.

Furthermore, one of the dominant volatile compounds released during cultivation, the terpene ß-Myrcene, is a potent carcinogen listed on California's Proposition 65, in addition to its irritant properties. Scientific analyses confirm that people living or working near cannabis fields are exposed to high levels of ß-Myrcene, in the range that caused toxicities and ultimately cancer in laboratory animals. Children are particularly vulnerable, as they are more susceptible to all air pollutants.

Analyzing odors can be achieved with quantitative scientific methods in real time in the field (e.g., gas chromatography), thus avoiding subjective and varying determinations by human nasal receptors. Multiple studies have defined that necessary mitigation is to attenuate odors by long separation distances. Thus, **policy makers have the tools necessary to require mitigations to protect public health: either sufficiently long setbacks to prevent terpenes from leaving the cultivation parcel or only allowing indoor/greenhouse cultivation with appropriate filters, both in conjunction with quantitative terpene monitoring.**

I. TOXICITIES OF CANNABIS EMISSIONS DURING CULTIVATION

The Neighborhood Coalition[1] has confirmed that published literature shows that inhalation of cannabis emissions causes respiratory harm [1-8,41] including asthma which can lead to deaths. Children are at greater risk of respiratory harm as they are more susceptive to air pollutants [45].

People often find odors from cannabis cultivation objectionable. These emissions are not mere annoyances. They have resulted in immediate deleterious effects (including in cannabis workers) encompassing nausea, headaches, cough, eye irritation, respiratory distress and asthma, including two deaths from asthma [2-7, 17, 41]. Up to 70% of cannabis workers reported respiratory irritation with half of those reporting asthma [4]. Neighbors are exposed to these same cannabis emissions but are exposed 24/7 (168 hr/wk), thus 4 times the exposure time as workers who are only exposed 40 hr/wk. In addition, workers on adjacent agricultural lands are exposed for 40-60 hr/wk.

ß-Myrcene is also contained in Hops used in beer manufacture; it was found to be the sensitizing agent in a brewery inspector employee with respiratory hypersensitivity [8].

Real-life experience confirms that exposure to even low levels of cannabis cultivation odor causes physical illness (nausea, respiratory irritation, headache, aggravation of asthma) and prevents residents from using their yards or opening their windows. In addition to the confirmed toxicities of cannabis cultivation emissions, a dominant component in cannabis emissions, ß-Myrcene, inhaled by people living near outdoor cannabis cultivation sites can reach amounts that may be toxic and carcinogenic [3]. The risks are likely even greater for children and fetuses in utero.

ß-Myrcene is listed under Proposition 65 as a cancer-causing compound [9][2] and also showed toxic effects in rodents on liver and kidney after only 3 months [3]. Neighbors living and working near an outdoor cannabis cultivation operation are involuntarily exposed to cannabis emissions that contain this toxin and carcinogen. ß-Myrcene also contributes to formation of secondary toxic pollutants in the air including formaldehyde [10], also a carcinogen listed on Proposition 65, and formic acid, both of which cause eye irritation and nausea [11]. It also leads to formation of ground-level ozone [12, 37], also a known irritant.

Volatile cannabis emissions are greatest during flowering and harvest [13, 37]. Often cannabis harvest occurs at the same time as harvest on adjacent parcels, increasing exposure; inhalation of these emissions by farm workers also increases their health risks. Exposures are amplified when winds are

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[1] The Neighborhood Coalition advocates for sustainable, environmentally sound, and neighborhood-compatible cannabis policies in conjunction with education of the public on the health and safety impacts of cannabis use. Our lead researcher, Dr. Deborah Eppstein, consulted with experts in the pharmaceutical, cannabis analytical chemistry, drug delivery, pulmonary and public health fields, including two former Public Health Officers for California.

[2] As OEHHA wrote in its response to comments concerning listing of ß-Myrcene under Proposition 65 [36] and as per CCR Title 27, § 25703 [38] "Human cancer potency shall be derived from data on human or animal cancer potency."

blowing toward workers or residences. Anyone who smells cannabis is being exposed to ß-Myrcene by inhalation, which is a direct and rapid route for small lipophilic compounds to enter the bloodstream [14] as well as to cross the blood-brain barrier entering the brain [50, 51].

The longer the exposure and the higher the concentrations, the greater the toxicity and cancer risk to individuals. The amount of ß-Myrcene inhaled in a single season may cause liver or kidney toxicity and over several years can exceed the carcinogenic dose determined through controlled tests with animals [3]. Children are subject to greater risk due to higher exposure per body weight, increased respiratory rate, and their developing lungs and more rapidly dividing cells as they grow. During pregnancy, terpenes inhaled in cannabis emissions can cross the placenta [15, 53] and the developing fetus may be exposed to significantly greater amounts of ß-Myrcene than the mother on a relative body-weight basis.

Cannabis emission and terpenes have been shown to travel over 3000 ft in the absence of wind [16] and up to 2 miles ([42] including data from Kern County Cannabis EIR and Kram report]). Air dispersion models show very high odor levels from a 1-acre grow [Figure 3-1 in ref 42, including data from Yolo County Cannabis EIR]: at 100 ft, 30 Odor Units (~600-1500 parts per billion (ppb) ß-Myrcene [21]); at 1000 ft, 5 Odor Units (~100-250 ppb ß-Myrcene), and even at 2500 ft, 2.5 Odor Units (~50-125 ppb ß-Myrcene). This projection is for flat topography with no wind or air inversions. Depending on the proximity to the neighboring properties, size of the cultivation field, wind direction, local topography, and prevailing climatic conditions, appropriate separation distances for neighbors, farmworkers and businesses (e.g., winery tasting rooms) from outdoor cannabis fields can require thousands of feet from the cannabis operation. This problem is compounded when multiple grows are in close proximity.

Analyzing odors can be achieved with quantitative scientific methods in real time in the field (e.g., gas chromatography) [21], thus avoiding subjective and often varying determinations by human nasal receptors. Toxic/safe limits can be set using conversion from toxic levels in animal studies to equivalent human dosing levels, using methodology employed by pharmaceutical industry, toxicologists, and FDA (Appendix; [33], [Appendix C in ref 42]).

II. TOXICITY OF ß-MYRCENE

A. Animal Toxicity Testing

Toxicity testing in rodents is a normal first step before testing a new drug candidate in people. The National Toxicology Program (NTP) decided to test ß-Myrcene in animal carcinogenicity models due to its use as a flavoring food additive (albeit in very small amounts) and its structural similarity to D-limonene, another terpene that NTP had previously tested [35].

The NTP studies of ß-Myrcene showed toxicity and clear evidence of carcinogenicity in animal testing: it caused toxicity (kidney and liver) in rodents by oral gavage after 3 months and at 2 years caused liver and kidney cancers in mice and rats, respectively, at the lowest dose tested [3]. Toxicity increased in the animal studies with duration of dosing from 3 weeks to 2 years, including liver and

kidney toxicities, chronic inflammation, bone marrow and lymph node atrophy, and tissue necrosis. At the highest dose tested, all animals died within a week. At the 2-year point, liver and kidney cancers were present at the lowest dose.

A subchronic (90 day) toxicity study was done in a different strain of rats with much lower doses (in feed) to assess safety for the food additive industry [48]. Although this study was not a carcinogenicity study as that requires 2-years of dosing in two species, it is relevant that the authors also concluded the results "were indicative of target organ toxicity pattern that at higher intake levels, such as those tested in the NTP studies, produces more severe toxicity and leads to associated neoplastic lesions." The amounts of ß-Myrcene used in food additives are orders-of-magnitude lower than those neighbors are exposed to from breathing cannabis emissions (Appendix). Other effects of ß-Myrcene in animal testing include developmental [46] and reproductive toxicities [47] in rats at slightly higher doses, and sedative-hypnotic activity in mice [54].

All the animal toxicity studies were done with ß-Myrcene dosed orally. As detailed in the Appendix (p11), oral dosing results in ~3-5 times lower uptake than by inhalation [34]. Thus, due to this greater update by inhalation, equivalent toxic levels by inhalation are reached at 3-5 times lower calculated levels as compared to oral dosing (i.e., to expose the body to the same toxic amount).

Toxicity/safety studies in humans with isolated ß-Myrcene have not occurred. Thus, one must rely on animal safety/toxicity data to project safety for humans. Separate from carcinogenicity, liver and kidney toxicity concerns based on animal testing, we found two publications on effects of ß-Mrycene in people, one on cognitive impairment and one on sensitization, both of which are relevant to human safety concerns:

1) As high ß-Myrcene levels in cannabis have been associated with sedation [22], a pilot study was conducted in human volunteers tested with a driving simulator [55]. Ingestion of isolated ß-Myrcene in people showed significant impairment of driving-related skills (double-blind, placebo-controlled crossover study).

2) ß-Myrcene was determined to be the sensitizing agent in respiratory hypersensitivity in a brewery inspector [8].

Furthermore, inhalation provides a direct route to the brain via the olfactory pathway [50, 51], resulting in much higher drug levels in the brain than from oral delivery. No animal toxicity/safety studies have been conducted using the inhalation delivery route for ß-Myrcene; unknown brain toxicities may exist.

In 2015,California listed ß-Myrcene under Proposition 65 as a compound known to cause cancer, concluding that the cancers formed were relevant to human toxicity [9]. Additionally, the Food and Drug Administration (FDA) removed ß-Myrcene from its list of approved food additives in 2018 [19].[3] More recently, we and others have raised additional safety concerns from ß-Myrcene from

[3] FDA wrote that as the dose of ß-Myrcene typically used as a food additive is very low, it did not think it posed a public health risk as a food additive under those conditions of intended use. Nevertheless, because it is a proven carcinogen the agency delisted it. Some cannabis and ß-Myrcene proponents argue that because ß-Myrcene occurs naturally in certain foods, it is non-toxic. This is an incorrect assumption; natural occurrence

cannabis cultivation as ß-Myrcene is a terpene present in cultivation emissions from all varietals of cannabis, often as the dominant terpene [1, 18, 40]. ß-Myrcene is also known to be a skin and eye irritant [49].

Our research team employed two accepted methods to project "toxic" and "safe" exposure levels of ß-Myrcene for people living or working near outdoor cannabis cultivation sites, based on animal toxicity and carcinogenicity data. These are detailed in the Appendix and summarized below.

B. Calculating Projected Toxic and Safe Levels of ß-Myrcene

Method 1 (Appendix Part B):

Using well-accepted factors employed by the pharmaceutical industry to convert rodent dosing to human dosing [33], plus accounting for higher bioavailability of inhalation versus oral ingestion of cannabis compounds [34] and predicted accumulation in humans [34, 43], the calculated human-equivalent toxic dose of ß-Myrcene is reached at only 3.5 mg/day for a 15 kg person (e.g., a 33 pound 3-year-old) and at 14 mg/day for a 60 kg person (e.g., a 132 pound adult) (Appendix, Table 2). These doses were calculated as the human-equivalent to the lowest dose tested in mice, which was deemed highly carcinogenic after 2 years of exposure. Toxic effects on kidney and liver were also observed in the 3-month rodent study [3].

ß-Myrcene was carcinogenic at the lowest dose tested in the animal carcinogenicity study; no lower, non-toxic dose was determined [3; footnote 4]. Thus, one must factor in further dose reduction to estimate toxic and safe exposure levels for chronic exposure. The pharmaceutic industry and the FDA understand that even a non-toxic dose in mice or rats may be toxic in humans, and clinical trials typically start at 1/10th or less of the equivalent non-toxic dose in animals. Since no non-toxic dose was determined in the carcinogenicity study, we used 1/10 reduction to estimate a lower "toxic" level of 0.35 mg/day for a small child and 1.4 mg/day for an adult, which at 50% absorption of ß-Myrcene from air gives a projected **toxic chronic exposure** at **~5 ppb for a small child**, and **~20 ppb for an adult**. If another 1/10 factor is then used as an estimate of a "safe" level (thus 1/100 of lowest toxic dose tested), this predicts a **"safe"** exposure level of **0.5 ppb for the child and 2 ppb for the adult**.

Method 2 (Appendix Part B)

Another accepted approach for determining safe chronic human exposure levels calculates

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does not equate with safety. Many toxic compounds occur naturally, including in tobacco, sprouted potatoes, many mushroom species, kidney beans, castor beans, and some fish as well as cannabis. Many compounds with significant cellular toxicity occur naturally in plants, hence their use to treat cancer. Likewise, many foods and beverages people choose to consume are known to cause health issues including cardiovascular disease and cancer (e.g., animal fats, red meat, bacon, excess sugar, alcohol), but people consume these by choice. ß-Myrcene also is present in low levels in some foods (e.g., carrots) and beverages (e.g., beer made from hops) [22, supplement Table 2 for levels], but the levels ingested by humans are orders-of-magnitude lower than the grams inhaled from chronic exposure to cannabis odors. For example, even ignoring the much lower absorption by ingestion vs inhalation, one would need to consume 135,000 g carrots (297 pounds) or drink 1400 beers each day to equal the amount of ß-Myrcene inhaled in one day at 100 ppb ß-Myrcene [Appendix, A, p 10-11]. As the human exposure is chronic, that same 297 pounds of carrots or 1400 beers would need to be consumed each day, chronically.

Occupational Exposure Limit (OEL) of ß-Myrcene in the air using the formula OEL = POD/ (AF_c x a x S x MF x V) [42, 44]. Using the rat data, this projects a "**safe"** exposure level of **ß-Myrcene** in the air of **2.7 ppb for the child** and **10.7 ppb for the adult** (Appendix, Table 3).

III. DISCUSSION

Harm to Residents, Workers and Vulnerability of Children

People living or working near outdoor cannabis cultivation operations are involuntarily exposed to cannabis emissions including ß-Myrcene on agricultural parcels, in their yards as well as in their homes when vapors enter through windows and doors. They breathe it in 24/7, often for 3-6 months a year. Exposures can continue year after year. Chronic exposure [20] to a compound generally causes toxicity at much lower doses than observed for acute exposure. The quantity of ß-Myrcene inhaled by neighbors (which is similar for children and adults) can be calculated (Appendix Part A, Table 1) and compared to the projected toxic levels (Appendix Part B, Tables 2 and 4). As shown in Table 4B, at **100 ppb, projected toxic doses are reached for children after only 3 months exposure**, and for adults after 12 months exposure. Even at ambient levels of only 10 ppb, which is below the level of odor detection for most people [21], the amount of ß-Myrcene absorbed in 6 months per year over a 5-year period is at the projected toxic dose for children (Appendix, Table 4A) and approaches the projected toxic levels for adults by 10 years. **For outdoor cultivation sites with two harvests per year, cumulative exposure of 60 grams ß-Myrcene occurs after 10 years at 1000 ppb, which is 50 times the projected toxic dose for adults (Safety Margin 0.02), and 200 times the projected toxic dose for children (Safety Margin 0.005), respectively (Table 4C).**

Safety Margin is Toxic Dose divided by Inhaled Dose. When the Safety Margin of projected toxic dose to inhaled dose is less than 1, this means that the inhaled dose is greater than projected toxic dose; exposures even at a Safety Margin of 10:1 (i.e., inhaled dose is one-tenth of the toxic dose) can pose health risks.[4] Note that this risk is in addition to the known respiratory toxicities that occur from breathing cannabis emissions (ß-Myrcene, which is a known irritant, may be one of the compounds causing this irritation.) If exposures are even higher (e.g., due to proximity, meteorological conditions, grow area and/or several large nearby cultivation areas), the amount inhaled can reach hundreds of grams after several years.

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[4] Safety Margin and Therapeutic Index. Therapeutic Index (TI), the ratio of the toxic dose to the effective dose [23], is often well over 100 for pharmaceutical drugs, indicative of a good risk/benefit profile. It is lower for some drugs, such as morphine with a TI of 70. Drugs with lower TI often require extensive monitoring of drug levels. Risk-benefit is taken into account for drugs with lower TI. For ß-Myrcene, there is no therapeutic benefit, only risk, and we thus calculated a "**Safety Margin**" (the ratio of calculated toxic dose of ß-Myrcene over the inhaled amount absorbed); this Safety Margin is extremely low (thus toxic) by pharmaceutical safety standards, and exposure even at 100 ppb shows that the inhaled dose approaches or exceeds the calculated toxic dose after 3 or 12 months exposure (child and adult; Appendix Table 4B). As the lowest level of toxic dose was not determined in animal studies, we also performed the analysis at 1/10 the lowest dose tested. Results at that level showed likely toxicity even over 3 months of inhalation by a child at ambient levels of only 10 ppb (Tabler 4A).

These calculations show that based on ambient levels of ß-Myrcene near outdoor cannabis cultivation sites [21; 42 Fig 3-1, 1 Odor Unit = 20-50 ppb; thus for a 1 acre grow, ave is ~1050 ppb at 100 ft, ~175 ppb at 1000 ft, and ~82 ppb at 2500 ft, with higher ppb levels for larger grows], the total amount of ß-Myrcene that neighbors are involuntarily exposed to is significant (Table 1), may cause toxic effects and ultimately be carcinogenic, with even greater risk for children (Table 4) as well as infants and fetuses in utero.

Furthermore, unlike pharmaceutical drugs that take into account risk-benefit, there is **absolutely no therapeutic benefit** for neighbors or workers **from inhaling ß-Myrcene, only risk**. To put this in context, the lower limit of terpene concentration where more than 50 percent of volunteers reported odor detection is between ~20-50 ppb [21, 42 Kram report]. Levels when the odor is strong or close to cultivation fields can be orders of magnitude higher. Uncontrolled emissions from an outdoor cannabis cultivation site (or from an indoor facility if the odors are not properly filtered) can reach hundreds to thousands ppb ß-Myrcene. Exposure levels can be even higher at locations downwind of a cannabis site when flowering plants are mature. Odor masking or neutralizing agents are not effective for outdoor cannabis fields.[5]

Risk is higher for infants and children due to multiple factors including their lower weight, higher respiratory rates, immature and developing lungs, and greater physical activity as well as the fact that their cells are more rapidly dividing as they are growing. Since children are smaller than adults, the dose of ß-Myrcene in mg/kg body weight is much higher. As such, a lower total dose may be toxic (Appendix, Table 2). This is consistent with the fact that air pollution affects children [25] much more than adults. On average, a 20 kg (44 pound) 5 yr old child inhales 11,664 L/day, [25] similar to but slightly higher than adult inhalation. Yet for the 5 yr-old, pollutants are concentrated over a 3-4 times smaller body mass. Risk is further magnified for infants or with in-utero exposure of developing fetuses. Most low molecular weight and lipophilic compounds cross the placenta [15], and ß-Myrcene is both low molecular weight and very lipophilic [28]. Cannabis can cross the placenta [26, 53] and can cause low birth weight and neurocognitive deficits [27]. As the projected toxic dose level of ß-Myrcene in cannabis is calculated in mg/kg body weight, the dose experienced by the mother is magnified manyfold in the tiny fetus. Toxicity and carcinogenicity are increased by exposure over a multi-year period. As the rodent studies confirmed, longer exposure times to ß-Myrcene increased toxicity in various organs, long before full-blown cancers appeared.

This paper is not intended to address safety or health effects from ingesting or smoking cannabis. Interested readers are referred to a recent review [52] concerning therapeutic use of cannabis and cannabinoids as well as a review of health risks. The authors concluded "Evidence is insufficient for

[5] Odor "neutralizer" approaches have employed chemicals that "trap" the cannabis odor-causing molecules (but do not destroy the carcinogen) [24] and approaches using chemical oxidizing agents [24]. Although odor-neutralization may have utility when sprayed near exhaust fans along the perimeter of indoor grow facilities, it is very different for outdoor grows. For outdoor sites, the physics associated with odor emissions from a large area converging with neutralizer releases at the perimeter render contact incomplete over the larger area of the outdoor cultivation site.

the use of cannabis or cannabinoids for most medical indications." They also referenced many studies showing health harms from ingesting or inhaling cannabis including cardiovascular, pulmonary, neurocognitive and psychiatric risks and fetal risks from use in pregnancy [52, and Table 4 in 52].

IV. CONCLUSIONS AND POLICY RECOMMENDATION

This report summarizes known respiratory harms caused to workers, customers as well as residents near outdoor cannabis cultivation fields. It also addresses that ß-Myrcene, a carcinogen listed on California's Proposition 65, may reach levels in people from breathing cannabis emissions that are equivalent to those confirmed carcinogenic in animals. Children are known to be particularly vulnerable to respiratory and lung damage from air pollutants and may be exposed to toxic levels of ß-Myrcene from outdoor cannabis cultivation in as little as 3 months. Although the calculations in this report are illustrative and actual levels will vary, they show that neighboring properties are exposed to substantial levels including ß-Myrcene. Due to the known respiratory harm and carcinogenicity risk, these cannabis emissions should be regulated as a serious health threat to humans.

There is no effective way to prevent cannabis emissions from leaving cultivation site premises when grown outdoors; the only mitigation for outdoor cultivation to protect the public from exposure to these toxins is long separation distance that takes into consideration size of the cultivation area, topographic and meteorological conditions. Enclosed cannabis operations including indoor and greenhouse cultivation, drying, and processing facilities must be equipped with sufficient filtration (carbon scrubbers) to remove and prevent all emissions from leaving the structure. As topography and climate conditions affect the distance terpenes travel, quantitative monitoring (e.g., gas chromatography) should be required at parcel line to confirm no terpenes leave the cannabis operation's parcel.

Cannabis cultivation represents a public health threat to residents, customers and workers[6] on neighboring properties when it involuntarily exposes them to breathing unhealthy emissions containing toxins and carcinogens, including ß-Myrcene. Property owners must be able to safely and peacefully enjoy their businesses, yards and homes as is codified in the California Code's nuisance provisions (CCC § 3479 and HSC 41700).

[6]Cannabis workers are exposed to ß-Myrcene [34] as well as other toxins and should use protective respirators and dermal protection. Due to the recent adoption of legal commercial of cannabis in California and elsewhere, studies on health of cannabis workers have only recently been appearing in scientific publications. Publications reported dermal irritation and respiratory problems including asthma [2-7, 17], and two asthma fatalities [5, 41]. Terpenes in hops, which contains ß-Myrcene, have been reported to cause dermatitis and asthma in workers [8]. See also review of air quality impacts from cannabis cultivation facilities [18]. Farm workers on adjacent parcels are also exposed to these same toxins.

APPENDIX

Calculation of Amount of ß-Myrcene Inhaled by Humans From Outdoor Cannabis Cultivation Sites

A. Amount of ß-Myrcene Inhaled by Humans Exposed to Cannabis Odor

Facts: Avogadro's number is 6.022×10^{23} molecules/mole of compound and a mole is defined as the molecular weight in grams [29]. ß-Myrcene (abbreviated as ß-Myr in calculations) has a molecular weight of 136.23 g/mole [30]. The volume of a mole of a gas (e.g., air) at Standard Temperature and Pressure (STP) is 22.4 L [31].

Thus: the number of molecules in one Liter of air is calculated as:

$(6.022 \times 10^{23}$molecules/mole of air) x (1 mole of air/22.4 Liters) = 0.27×10^{23}moleules air per 1 Liter air

The definition of 1ppb (part per billion) of ß-Myr is 1 molecule ß-Myr per billion (10^9) molecules of air.

Thus: the **number of molecules of ß-Myr, at 1 ppb, per L of air, is:**
(1 molecule ß-Myr/10^9 molecules air) x (0.27×10^{23} molecules air/1L of air) = 0.27×10^{14} molecules ß-Myr/L air at 1 ppb.

Thus: converting to **g of ß-Myr at 1 ppb** of air:
(0.27×10^{14} molecules ß-Myr/L air) x (136.23 g ß-Myr/6.022×10^{23} molecules/mole ß-Myr) =
6.1×10^{-9} g ß-Myr/L air at 1 ppb of ß-Myr.

An average human adult breathes 11,000 L air/day. [32]. A 5-yr old child weighing 20 kg (44 pounds) breathes 11,664 L air/day [25], which is concentrated over a 3-4 fold lower body mass than in an adult.

Thus: average amount ß-Myr inhaled per day by a human adult at 1 ppb of ß-Myr if 100% absorption[7] from the lungs is [30, 32]:

(11,000 L air/day) x (6.1×10^{-9} g ß-Myr/L air) = $67{,}188 \times 10^{-9}$ g ß-Myr/day.
Multiplying by 10^3 mg/g gives $67{,}188 \times 10^{-6}$ mg ß-Myr/day,
which is **0.067 mg ß-Myr/day at 1 ppb ß-Myr (or 0.071mg for child [25]; for simplicity, we used the lower value of 0.067 mg ß-Myr/day for both adult and child calculations)**.

Over one outdoor growing per year, neighbors typically are exposed to high levels of cannabis odors for 3 months. The exposure time is doubled to 6 months per year with two growing cycles.

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[7] The actual amount of ß-Myrcene absorbed by neighbors (and workers) exposed to cannabis odors will vary depending on factors including concentration of ß-Myrcene in the odor plume, wind and weather conditions, level of exertion by the person and hence daily volume of air inhaled, and amount absorbed by the lungs. The amount absorbed by inhalation depends on relative efficiency of inhaled absorption, which at generally is 3-5X greater by inhalation vs oral ingestion for cannabinoids [34]. The calculations in Appendix Parts A and B used the more conservative estimate of 3X great bioavailability of inhaled vs oral, which show that ß-Myrcene inhaled from cannabis emissions by neighbors (and workers) can be significant and in the realm of toxic and carcinogenic levels.

Projected ß-Myrcene inhaled per day by a human (adult or child); summarized in Table 1 below:

At 10 ppb

if 100% absorption from air:

0.67 mg ß-Myr/day x 90 days = 60 mg in 3 mo, or 120 mg in 6 mo; or 1200 mg = 1.2 gm in 10 yr (at 6 mo/yr)

if 50% absorption from air:

0.336 mg ß-Myr/day x 90 days = 30.2 mg in 3 mo; or 60.4 mg in 6 mo; or 604 mg = 0.60 g in 10 yr (at 6 mo/yr)

At **100 ppb**

if 100% absorption from air:

= 6.7 mg ß-Myr/day x 90 days = 600 mg in 3 mo; or 1200 mg in 6 mo; or 12,000 mg = 12 g in 10 yr (at 6 mo/yr)

if 50% absorption from air:

= 3.36 mg ß-Myr/day x 90 days = 302 mg in 3 mo, or 604 mg in 6 mo; or 6040 mg = 6 g in 10 yrs yr (at 6 mo /yr)

At **1000 ppb**

if 100% absorption from air:

= 67 mg ß-Myr/day x 90 days = 6000 mg in 3 mo, or 12,000 mg in 6 mo; or 120,000 mg = 120 g in 10 yr (at 6 mo/yr)

if 50% absorption from air:

= 33.6 mg ß-Myr/day x 90 days= 3020 mg in 3 mo; or 6040 mg in 6 mo; or 60,400 mg =60 g in 10 yr (at 6 mo /yr)

Table 1. ß-Myrcene Dose in People Breathing Air at 10-1000 ppb ß-Myrcene

Exposure Concentration (ppb) (% absorption from air)	Daily Dose (mg)	Dose in 3 - 6 mo (g)	Cumulative Exposure in 5-10 yr (at 6 mo/yr)
10 ppb (100% uptake)	0.67 mg	0.060 - 0.120 g	0.6 - 1.2 g
10 ppb (50% uptake)	0.336 mg	0.030 - 0.060 g	0.3 - 0.6 g
100 ppb (100% uptake)	6.7 mg	0.60 - 1.200 g	6 - 12 g
100 ppb (50% uptake)	3.36 mg	0.30 - 0.60 g	3.0 – 6.0 g
1000 ppb (100% uptake)	67 mg	6.0 - 12 g	60 - 120 g
1000 ppb (50% uptake)	33.6 mg	3.0 - 6.0 g	30 - 60 g

The exposure ranges of 10-1000 ppb are based on real-time field measurements [21]. As children and adults have similar inhalation volumes, amount absorbed is comparable for children and adults.

ß-Myrcene occurs naturally in some food and beverage substances, such as hops, carrots, lemongrass, and bay leaves as well as cannabis [22], yet the levels consumed by people are orders of magnitude lower than when exposed to cannabis emissions. For example, to get the same amount of ß-Myrcene absorbed by a person in one day at 100 ppb ß-Myrcene (assuming 100% absorption; 6.7 mg/day), using the levels measured in ref [22], Suppl Table 2, and also assuming 100% absorption of ß-Myrcene from oral ingestion of food and beverages (which is a large over estimate, as it likely is in the 5-10% range as is THC oral absorption [34]), one would need to **consume 135,000 grams = 297 pounds of carrots each day** *[Calculation: 6.7 x 10^{-3} g ß-Myr inhaled / 150 x 10^{-9} g ß-Myr per g carrots, x 3 to account for higher bioavailability of inhalation vs oral = 135,000 g carrots*

each day] **or 1406 beers each day** [*Calculation: average 40 µg ß-Myr/L beer x 3.8 L/128 oz = 1.19 µg/oz beer = 14.3 µg B-Myr/12 oz beer. Comparing to the 6.7 mg/day inhaled by people exposed to 100 ppb, this is [6.7 x x 10^{-3} g ß-Myr inhaled/ 14.3 x10^{-6}g ß-Myr/beer] x 3 to account for higher bioavailability of inhalation vs oral) = 1406 beers/day].* In summary, the amount of ß-Myrcene people ingest by choice in foods and beverages in miniscule compared to the amount absorbed by inhalation of cannabis volatile emissions.

B. Calculation of Mouse and Rat Toxicity and Carcinogenicity Dose-Equivalent in Humans

Assumptions. Since human absorption and pharmacokinetic data do not exist for ß-Myrcene, we used assumptions of inhalation bioavailability, inhalation vs oral bioavailability and half-life/accumulation based on other similar molecules with like lipophilicity, as detailed below.

ß-Myrcene Is Predicted to Accumulate in People

Although it is known that ß-Myrcene does not accumulate in rats due to its short half-life and thus rapid clearance, the chemical properties of ß-Myrcene, being highly lipophilic (fat-soluble), would predict **accumulation in humans but not rats**. Although ß-Myrcene is very lipophilic, it has a short 4.5 hr half-life in rodents which lack much body fat and thus ß-Myrcene does not accumulate in rodents. (It takes 4-5 half-lives to clear a compound from the body; thus 4.5 hr/half-life x 4-5 half-lives/clearance is 18- 22.5 hr, meaning no ß-Myrcene remains in rodents from each daily dose before the next dose is given and thus there is no accumulation). However, very lipophilic compounds such as ß-Myrcene are predicted to accumulate in humans as do other lipophilic cannabis compounds (humans have more body fat). One can look at the similarly very lipophilic cannabis compound THC to extrapolate to predict levels of accumulation of ß-Myrcene that will likely occur in people. THC accumulates for 1-3 weeks in occasional users [34], and for 4-8 weeks in chronic users (vs short half-life and no accumulation in rats who have little body fat [43]). We used a conservative value of assuming only 2 weeks accumulation for ß-Myrcene in humans when calculating the equivalent human toxic dose.

METHOD 1- Calculation of Human Equivalent "Toxic" and "Safe" Dose of ß-Myrcene

The lowest dose tested in the NPR toxicity and carcinogenicity study [3] was 250 mg/kg/day, 5 days/wk in rats and mice by oral gavage; this equates to 179 mg/kg/day ß-Myrcene as the animals were dosed 5 days a week. There was dose-dependent toxicity at all doses with increasing toxicity associated with duration of dosing. Toxicities were documented for all doses and included kidney and liver degeneration, chronic inflammation, and weight loss and lethargy. Toxicity increased with duration of dosing from 23 to 70 days (over a 3-month period), culminating with kidney and liver carcinogenicity after 2 years, the longest duration evaluated (2 years is the standard carcinogenicity test). Regarding acute toxicity, all animals died early on at the highest dose tested (4g/kg), with many deaths at the intermediate doses as well. Applying well accepted factor employed by the pharmaceutical industry of 0.081 to convert from mouse dose (or 0.135 from rat) to human dose [33] (to account for the slower metabolic rate of humans and the difference in surface area to blood volume) results in a projected toxic daily dose in humans if ingested orally of 14 mg ß-Myrcene/kg/day. However, since humans will be exposed by inhalation from breathing the air containing cannabis emissions, one must account for the higher bioavailability of cannabis compounds by inhalation vs oral uptake. Compounds have much higher bioavailability when inhaled, as this provides a direct route into the blood stream, avoiding first-pass metabolism by the liver and low absorption from the gut as occurs for many compounds, including cannabinoids [34]. Literature on cannabinoids shows 3-5 fold lower bioavailability from

oral administration vs inhalation in humans [34]. The animal toxicity studies were all done by oral administration. When this much lower uptake by the oral route vs inhalation is factored in, the equivalent toxic dose calculated for humans from inhalation is then 3-5X lower than shown from oral dosing to account for the 3-5 higher uptake by inhalation. We used the more conservative figure of 3X lower; then projected toxic daily dose of ß-Myrcene in humans when inhaled is 14 divided by 3 = 4.7 mg/kg/day[8], before adjustment for accumulation. To adjust for accumulation, we conservatively assumed a 2-week accumulation in humans as compared to no accumulation in rodents as discussed on page 11. This would result in ~20-fold higher levels in humans than if it had the same 4.5 hr half-life as in rodents. Accounting for this accumulation in humans, the daily toxic dose would be projected to be ~20-fold lower, which is **0.23 mg/kg/day[8].** Finally, it should be noted that the toxic daily dose may likely be further lower as discussed above and in footnote 4, as a maximum non-toxic level (toxic threshold) was not determined in the animal tests since the lowest exposure dose was carcinogenic.

Multiplying 0.23 mg/kg/day by the weight of a human in kg gives the projected toxic inhalation dose based on the lowest dose tested and 1/10 lowest dose, on a daily basis and after 3 months (when significant toxicity was seen in animal studies) as shown in Table 2.

Table 2. "Toxic" Dose of ß-Myrcene by Inhalation in People (Method 1)*

A. Weight in kg (pounds)	**Toxic Dose**	
	lowest dose tested	1/10 lowest dose
15 kg (33 pound) child		
mg/d (ppb)	3.5 mg/d (= 52 ppb)	0.35 mg/d (= 5.2 ppb)
g in 3 months	0.31 g	0.031g
60 kg (132 pound) adult		
mg/d (ppb)	14 mg/d (= 209 ppb)	1.4 mg/d (= 21 ppb)
g in 3 months	1.24 g	0.124 g

B. Projected accumulated toxic dose after 3 months	
Adult (0.23 mg/kg/day)	1.24 g
Using 1/10 low dose	0.124g
Child (0.23 mg/kg/day)	0.31 g
Using 1/10 low dose	0.031 g

*To convert ß-Myrcene from mg/day to ppb (Appendix Part A, p8):
Air inhaled per day is 11 x 10^3L/day; 1 ppb is 6.1 x 10^{-6} mg/L air.
Thus [6.1 x 10^{-6} mg/L air/1 ppb] x [11 x 10^3L air/day] = 67.1 x 10^{-3} mg/ day = **0.0671 mg/day = 1 ppb**

[8] If the rat data are used instead of the mouse data, the conversion factor is 0.162 and calculated daily toxic human dose is 0.38 mg/kg/d, which is 8.7 ppb for the child. If the ratio of 5X inhaled/oral bioavailability is used, the human projected toxic dose is 60% (i.e., 3/5) lower than that shown.

METHOD 2. Calculation of Human Equivalent "Safe" Dose of ß-Myrcene

This method calculates an Occupational Exposure Level (OEL) using the formula [42]

$$\mathbf{OEL = POD/ (AF_c \times a \times S \times MF \times V)}$$

POD is Point of Departure, the highest non-toxic dose tested - or in this case as all doses tested were toxic, the lowest dose tested. POD is in mg/kg body weight/day, x the body weight of the affected individual. We used 15 and 60 kg, to account for a 33-pound small child and a 132-pound adult. We used a figure of 179 mg/kg/day from the rat carcinogenicity study as we did also in Method 1.

AF_c is a composite adjustment factor, accounting for interspecies difference (5), intraspecies differences (5), subchronic to chronic (1), conversion from lowest toxic dose tested to extrapolate to a non-toxic dose (10). Thus total AFc was 5 x 5 x 1 x 10 = 250.

"**a**" is a factor to account for differences in bioavailability from the dosing route used in the animal to the exposure in humans. As the rat studies were done with oral dosing (oral gavage) but humans will be inhaling myrcene from the air, the increased bioavailability for inhalation vs oral is 3-5 fold [34]. We used "a" of 3 to be on the conservative side.

S is the accumulation factor. We used a conservative estimate of accumulation over 2 weeks (detailed above on page 11) [34]), which gives ~20-fold higher levels at steady state than if no accumulation.

MF is to account for residual uncertainties; we used 1 (no residual uncertainties).

V is the volume of air inhaled during the assessed period. We assumed 24 hr exposure a day as neighbors are exposed to the contaminated air chronically, thus 11,000 L/day or 11 m^3/day for a 24 hr day [30, 32].

Using the above factors, OEL = 179 mg/kg/day x (15 or 60 kg)/ (250 x 3 x 20 x 1 x 11m^3/day) is calculated for people weighing 15 kg or 60 kg (33 or121 pounds) and shown in Table 3.

Table 3. Safe Exposure Levels (OEL) of ß-Myrcene in the Air for Humans (Method 2)[9]

Weight in kg (pounds)	mg/m^3	ppb
15 kg (33 pound) child	0.016 mg/m^3	2.7 ppb
60 kg (132 pound) adult	0.065 mg/m^3	10.7 ppb

Although the two methods use different assumptions, their results are similar. Method 2 shows safe level of ß-Myrcene in the air as 2.7 ppb for the child, whereas Method 1 shows 5 ppb or 8.7 ppb as toxic as calculated from the lowest toxic dose from the mouse or rat studies, respectively. Given that the adjustment factor in

[9] See Appendix Part A, p8. To convert ß-Myrcene from 1 mg/m^3 (= 1 mg/10^3L) to ppb myrcene:
1 mg /103 L x (L/6.1 x 10-6 mg)/1 ppb = 0.164 x 103 ppb = 164 ppb myrcene. Thus 1 mg/m^3 = 164 ppb.
(When SafeBridge used the above formula to calculate OEL [42], they failed to account for the higher bioavailability by inhalation, the increased accumulation in humans, the 24 hr daily exposure, and the smaller weight of children.)

Method 2 to extrapolate to non-toxic dose was just an estimate, and Method 1 only gave the toxic dose, this difference is not unreasonable. If the same adjustment factor of 3 is applied to Method 1, then a 'safe' level 1.7 or 2.9 ppb is obtained, thus very similar to Method 2. These numbers are consistent with the further analysis in Table 4, which suggests that 10 ppb would show toxicity. These methods all use different assumptions, but the results all suggest that chronic exposure should be limited to less than 2-3 ppb ß-Myrcene.

C. Calculation of Safety Margin (Toxic Level divided by Amount ß-Myr Inhaled)

A Safety Margin[4] (Toxic Dose divided by the Inhaled dose) of 1 means that the Toxic Dose equals the Inhaled Dose. **The higher the Safety Margin, the better the safety.** To put this in context: Pharmaceutical drugs typically have a Safety Margin (called Therapeutic Index for pharmaceuticals) of 100 or more (i.e., the toxic dose is 100 time higher than the therapeutic dose) and even a recreational drug such as cocaine has a Safety Ratio of 15:1 [23], yet cocaine use still results in overdoses and death.

As shown in Table 4, the projected Toxic Dose of ß-Myrcene over Inhaled Dose (i.e., Safety Margin) is quite low (thus very dangerous) at 100 ppb exposure after only 3 to 6 months and even at 10 ppb, it is at toxic amounts after 5 years and will be progressively worse (i.e., it decreases) for every additional year of exposure. **Sonoma County has approved granting permits with no term limits; neighbors will be exposed to ß-Myrcene for as long as they live near an outdoor cannabis operation.** Even if only at 10 ppb ß-Myrcene, often undetectable by the human nose, the Safety Margin for a child after 30 months is 1, thus at toxic levels. People are being forced to breathe a known toxin and carcinogen with no therapeutic benefit, with a projected lower Safety Margin than cocaine, that causes liver and kidney toxicity with short term exposures, can cause developmental/reproductive toxicities and can cause cancer with longer-term exposures. No pharmaceutical drug, perhaps other than a cancer therapeutic, would be approved with such a high toxicity.

Note that the above Safety Margins assume that a lower non-toxic ß-Myrcene level is known. However, since no lower non-toxic dose was determined[10], we also included a calculation using 1/10 of the lowest dose tested, projecting that this lower dose may also be toxic. When the 1/10 lower dose levels are used, the Safety Margin even at the low exposure level of 10 ppb shows that a toxic exposure is obtained after just 3 months (one growing season) for a child (Table 4A). Furthermore, as chronic exposure causes toxicity at lower dose levels than does shorter-term exposure [20], over a multi-year period, humans could suffer permanent damage to liver and kidneys long before any cancer appeared.

IN SUMMARY, the daily and cumulative amount of ß-Myrcene inhaled by people living or working near outdoor cannabis cultivation sites can be substantial and in the range of or greater than the comparable dose that was unequivocally determined to be toxic and carcinogenic in animals. Furthermore, as a non-toxic and non-carcinogenic dose was not determined in animals, the lowest toxic dose calculated for humans is projected to be lower than that shown. Taken together, one can project that chronic safe exposure levels should be no greater than 1 ppb ß-Myrcene to protect children.

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[10] The NTP toxicology and carcinogenicity study of ß-Myrcene was conducted due its structural relationship to d-limonene, its high production volume, and high level of human exposure (eg, use in food flavorings) [3]. Dr. R. S. Chhabra of NTP stated that "this was one of the very few studies in the history of the NTP where doses were missed so dramatically" [ref 3, comment by Dr. Chhabra, p14].

Table 4. Safety Margin for ß-Myrcene (Method 1)[4]

Toxic Dose of ß-Myrcene[11] Vs. Cumulative Dose Inhaled by Humans[12]

A. 10 ppb: Inhaled human dose of ß-Myrcene in g

	3 - 6 mo		5 yr (6 mo/yr)		10 yr (6 mo/yr)	
	Adult	Child	Adult	Child	Adult	Child
Cumulative inhaled dose	0.03- 0.06	0.03-0.06	0.3	0.3	0.6	0.6
Safety Margin	**41 - 21**	**10- 5**	**4.0**	**1.0**	**2.1**	**0.5**
at 0.1 low dose	**4.1 - 2.1**	**1 - 0.5**	**0.4**	**0.1**	**0.2**	**0.05**

B. 100 ppb: Inhaled human dose of ß-Myrcene in g

	3 - 6 mo		5 yr (6 mo/yr)		10 yr (6 mo/yr)	
	Adult	Child	Adult	Child	Adult	Child
Cumulative inhaled dose	0.3 – 0.6	0.3 – 0.6	3.0	3.0	6.0	6.0
Safety Margin	**4.1 – 2.1**	**1.0 – 0.5**	**0.4**	**0.1**	**0.2**	**0.05**
at 0.1 low dose	**0.4 – 0.2**	**0.1 – 0.05**	**0.04**	**0.01**	**0.02**	**0.005**

C. 1000 ppb: Inhaled human dose of ß-Myrcene in g

	3 - 6 mo		5 yr (30 mo)		10 yr (60 mo)	
	Adult	Child	Adult	Child	Adult	Child
Cumulative inhaled dose	3 – 6	3 – 6	30	30	60	60
Safety Margin	**0.4 – 0.2**	**0.1 – 0.05**	**0.04**	**0.01**	**0.02**	**0.005**
at 0.1 low dose	**0.04–0.02**	**0.01–0.005**	**0.004**	**0.001**	**0.002**	**0.0005**

[11]Calculated from mouse toxicity studies as described in Appendix, Part B, Method 1, Table 2B, using projected 3-month toxic dose (based on human equivalent to lowest dose tested in mice). As noted, oral bioavailability of cannabis compounds is 3-5 times less than by inhalation [34]; we used the more conservative figure of 3 times lower in calculating the projected human toxic dose. If the oral bioavailability is 5 times lower, then the projected toxic dose and Safety Margin would be 60% less than is shown.

[12]Dose of ß-Myrcene projected as inhaled in either 1 growing season (3 months) or 2 growing seasons (6 months) each year, at 10, 100 and 1000 ppb (See Table 1). These estimates assume 50% absorption of inhaled ß-Myrcene; the projected dose will vary depending on amount of ß-Myrcene absorbed by the lungs.

Bibliography

1. Zheng et al, A narrative review on environmental impacts of cannabis cultivation. J. Cannabis Res (3), Article 35 (2021)

2. Beckman et al, California cannabis cultivation and processing workers: A qualitative analysis of physiological exposures and health effects. Am J Ind Med Jan 66(1):75-84 (2021)

3. Natl Toxicol Program Tech Rep Ser 2010: Dec (557): 1-163 (2010).

4. Beckman et al, A pilot study of respiratory and dermal symptoms in California cannabis cultivation workers. J Agromedicine 2023; Jan 28(1):28-35.

5. Weaver et al, Fatal Occupational Asthma in Cannabis Production - Massachusetts, 2022. MMWR Morb Mortal Wkly Rep 2023 Nov 17;72(46):1257-1261 (2023)

6. Sack et al, The Emerging Spectrum of Respiratory Diseases in the US Cannabis Industry. Semin Respir Crit Care Med. 2023 Jun; 44(3): 405–414 (2023).

7. Strunk, C.D. Yes to cannabis! Just not in my backyard. An analysis of odor-based claims in the cannabis industry. American Bar Assn Brief 2020 49:32-37.

8. Newmark, F.M. Hops allergy and terpene sensitivity: An occupational disease. Ann Allergy 1978; 41(5): 311-312

9. OEHAA. Chemical Listed Effective March 27, 2015 as Known to the State of California to Cause Cancer: ß-Myrcene (2015)

10. Atkinson,R and Arey, J. Gas-phase tropospheric chemistry of biogenic volatile organic compounds: A review. Atmos. Environ.3, 37: 197-219.

11. Delikhoon et al, Characteristics and health effects of formaldehyde and acetaldehyde in an urban area in Iran. Environ. Pollut. 2018, 242: 938-951.

12. Wang et al, Potential regional air quality impacts of cannabis cultivation facilities in Denver, Colorado. Atmospheric Chemistry and Physics 2019, 19:13973-13078

13. Urso et al, Indoor monoterpene emission rates from commercial cannabis cultivation facilities in Colorado. J Waste Manag Assoc 2023 Apr;73(4):321-332 (2023)

14. Patton et al, The lungs as a portal of entry for systemic drug delivery. Proc Am Thorac Soc. 2004, 1(4):338-44 (2004)

15. Feghali et al, Pharmacokinetics of drugs in pregnancy. Semin Perinatol 2015: Nov 39(7):512-519 (2015)

16. Ortech, Inc, presentation provided to Permit Sonoma Dec13, 2019

17. Silvey et al, Occupational exposure to particulate matter and volatile organic compounds in two indoor cannabis production facilities. Annuals of Work Exposure and Health 2020, 64:715-729.

18. Davi de Ferreyro Monticelli et al, Cannabis cultivation facilities: A review of their air quality impacts from the occupational to community scale. Environ. Sci. Technol. 2022 Mar 1;56(5):2880-2896

19. U.S. Food & Drug Administration. FDA Removes 7 Synthetic Flavoring Substances from Food Additives List. Oct 5, 2018

20. Hulla et al, Toxicity, Subchronic and Chronic, Encyclopedia of Toxicilogy, 3rd edition,, p 626-633 (2014).

21. Vaporsafe, Inc. Cannabis Odor Threshold Tests. Summary Report Aug 20, 2020

22. Surendran et al, Myrcene – What are the potential health benefits of this flavouring and aroma agent? Front Nutr 2021; 8:699666

23. Wikipedia. Therapeutic Index (2024)

24. EcoSorb, OMI Industries
Cannabusters, Inc.

25. Dobric et al, Preschool Children's Inhalation Rates Estimated from Accelerometers—A Tool to Estimate Children's Exposure to Air Pollution. Aerosol and Air Quality Research; 22(8) (2022)

26. Thompson et al, Marijuana Use in Pregnancy: A Review. Obset Gynecol Surv. 2019 Jul; 74(7): 415–428 (2019)

27. Thompson et al, Marijuana Use in Pregnancy: A Review. Obset Gynecol Surv. 2019 Jul; 74(7): 415–428 (2019)

28. Cameo chemicals, ß myrcene chemical data sheet,

29. Britannica, Avogadro's number.

30. National Institute of Standards and Technology, NIST chemistry webbook SRD 69,B-Myrcene.

31. Textbook, https://testbook.com/question-answer/how-many-molecules-are-there-in-1-liter-of-any-gas--5fb7d8681de2257ca5229931#:~:text=6.022%20×%2010%2023%2022.4,0.27%20×%201023%20molecules.

32. Discovery Health, How much oxygen does a person consume in a day?

33. Nair and Jacob, A simple practical guide for dose conversion between animals and human. J basic Clin Pharm. Mar-May 2016; 7(2): 27-31 (2016)

34. Chayasirisobhon, S. Mechanisms of action and pharmacokinetics of cannabis. Perm J 2020; 25: 19.200 (2020)

35. Natl Toxicol Program Tech Rep Ser, NTP Toxicology and Carcinogenesis Studies of d-Limonene (CAS No. 5989-27-5) in F344/N Rats and B6C3F1 Mice (Gavage Studies) Natl Toxicol Program Tech Rep Ser Jan:347:1-165 (1990).

36. Office of Environmental Health Hazard assessment (OEHHA), California Environmental Protection Agency, Response to comments pertaining to the notice of intent to list B-Myrcene as causing cancer under proposition 65, March 2015.

37. Seltenrich, Odor Control in the Cannabis Industry: Lessons from the New Kid on the Block. Environ Health Perspect.2022 Jun; 130(6):062001 (2022)

38. California Code of Regulations, Quantitative risk assessment, Title 27, § 25703.

39. Canvast Supply Company, Cannabis & Hemp Growers Most Frequently Asked Questions.

40. Samburova et al, Dominant volatile organic compounds (VOCs) measured at four Cannabis growing facilities: Pilot study results. J Air Waste Manag Assoc. 2019 Nov; 69(11):1267-1276

41. California Dept of Public Health May 2024 Occupational Health Watch;
Pacheco et al. Work-related asthma in the cannabis industry; findings from California, Massachusetts, Michigan and Washington. J. Occup Environ Med. June 3; 67 (10: 862-868 (2025)

42. Sonoma County Cannabis Final Environmental Impact Review, Sept 2025.

43. Ravula et al. Pharmacokinetic and Pharmacodynamic Characterization of Tetrahydrocannabinol-Induced Cannabinoid Dependence After Chronic Passive Cannabis Smoke Exposure in Rats. Cannabis Cannabinoid Res Dec 6;4(4):240–254 (2019)

44. Sargent EV, Kirk GD. Establishing airborne exposure control limits in the pharmaceutical industry. Am Ind Hyg Assoc J. 1988 Jun;49(6):309-13 (1988)

45. California Air Resources Board : Children and air pollution

46. Delgado et al. Peri and post-natal developmental toxicity of ß-myrcene in rats. Food Chem Toxicol 33(9): 6623-628 (1993)

47. Paumgartten et al. Study of the effects of ß-myrcene on rat fertility and general reproductive performance. Braz J Med Biol Res 31(7): 955-965 (1998)

48. Bastaki et al. Absence of renal adverse effects from B-myrcene dietary administration in OECD guideline-compliant subchronic toxicity study. Food and Chemical Toxicology 120: 222-229 (2018)

49. Safety Data Sheet for B-Myrcene. Sigma-Aldrich 2024.

50. Endo et al. Evaluation of intranasal route of drug administration for brain targeting. Brain Res Bulletin October 2018: 155-170

51. Formica et al. On a highway to the brain: A review on nose-to-brain drug delivery using nanoparticles. Applied Materials Today 29 December 2022

52. Hsu et al. Therapeutic use of cannabis and cannabinoids. A Review. JAMA Nov 26, 2025

53. Zoorob et al. Cannabis use during pregnancy. Fam Pract Manag 31(4): 19-21 (2024)

54. Chen et al. ß-Myrcene as a sedative-hypnotic component from lavender essential oil in DL-4-chlorophenylalanine-induced insomnia in mice. Pharmaceuticals 17(9), 1161 (2024)

55. Johnson et al. The effects of ß-myrcene on simulated driving and divided attention: a double-blind, placebo-controlled, crossover pilot study. Cannabis 6(1) 9-19 (2023)