Marijuana and the Lung
Evolving Understandings

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KEYWORDS

• Marijuana • Cannabis smoking • Chronic obstructive pulmonary disease
• Lung cancer

KEY POINTS

- Marijuana can cause bronchitis and may have some impacts on pulmonary physiology, but, even when smoked, does not cause COPD.
- Data with respect to lung cancer and marijuana are suboptimal, but a summary of current collective data argues against causality.
- Marijuana use for recreational purposes should be discouraged in teens.

Very few drugs, if any, have such a tangled history as a medicine. In fact, prejudice, superstition, emotionalism, and even ideology have managed to lead cannabis to ups and downs concerning both its therapeutic properties and its toxicological and dependence-inducing effects.

—E. A. Carlini[1]

INTRODUCTION

Marijuana is the second most widely smoked substance after tobacco.[2] Globally, in 2019 an estimated 200 million persons aged 15 to 64 years (4% of the adult population) used cannabis products.[2,3] The usage is increasing. The overall number of people who used cannabis in the past year is estimated to have increased by nearly 18% over the past decade (2010–2019), greater than the 10% increase in the global population over the same period.[2,3] In the United States, the past-year and past-month prevalences of cannabis use among the adult population have increased by 60% and

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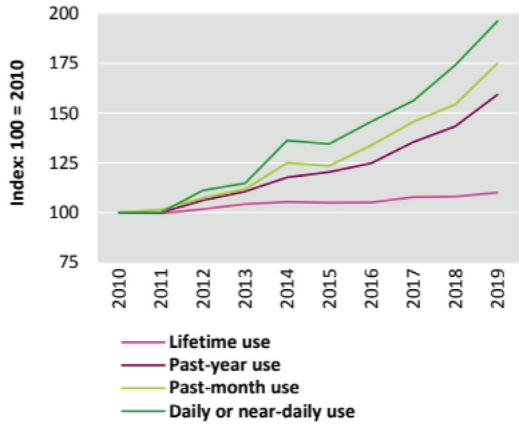

Fig. 1. Trends in cannabis use among the population aged 18 years and older, United States, 2010 to 2019 (*Data from* United States, substance abuse and mental health services administration (no permission required).)

75%, respectively; the prevalence of daily or near-daily use has doubled over the period 2010 to 2019[3] (**Fig. 1**).

Marijuana has been subjected to fascinating cross-currents of medicine, politics, prejudice, and economics. Marijuana has been and is being used medicinally while at the same time being labeled by some as highly dangerous. We know of no other drug/product that is simultaneously legal (some state laws) and illegal (federal law). Categorization of marijuana by some societies as "illicit" has limited both transparency and research, and bias pervades existing literature, with flagrant contradictions not supported by data. The goal of this review is to cover current understanding about physiologic impact, applications, and potential injury related to the use of marijuana with the hope of minimizing bias by relying on the limited but growing body of available data. This review concentrates on pulmonary implications but includes important contextual information.

Biology of Cannabis

The taxonomy of marijuana is confusing and somewhat vague. There are 2 prevalent subspecies of the Cannabaceae (sativa and indica) and 4 varieties.[4] These 2 are often hybridized and vary with respect to characteristics.[4] Usage ranges from utilitarian (rope, with the plant commonly named *hemp*) to medicinal/psychoactive. The medicinal applications are produced from the dried and crumbled leaves and flowering tops of the cannabis plant and are produced and consumed in almost every country. By contrast, cannabis resin, the concentrated extract of cannabis flowers and plants, is produced mainly in a few countries in North Africa, the Middle East, and Southwest Asia.[2,4] Hash oil is a cannabis product that can be extracted from any part of the plant. An increasing number of *cannabis* extracts is being produced from the dried flowering tops and leaves of the female plant, expanding the options for ingestion and inhalation of the active components.[2,4]

The main psychoactive ingredient in marijuana is delta-9-tetrahydrocannabinol (Δ9-THC); however, more than 120 compounds (cannabinoids) have been identified within the cannabis plant with cannabidiol (CBD) being the principal cannabinoid.[4–6] Δ9-THC is used for both recreational and medical purposes, whereas the applications of other CBDs are primarily medical.[4–6] CB1 and CB2 receptors, which bind both Δ9-THC and cannabinoids, were discovered in 1990 and 1993, respectively, followed by the

discovery of endogenous ligands that attach to those receptors.[5,6] This endocannabinoid system (our understanding of which is still in its infancy) is widely distributed throughout the body, with cannabinoid receptors suspected to be the most numerous single receptor type in the human brain.[5,6]

Marijuana History

Members of the Cannabaceae family have been used by humans for millennia[5–8]; they have been used by multiple civilizations. Uses have ranged from utilitarian to medicinal to religious. In India, medical and religious usage of cannabis probably began around 1000 years BC.[5,7,8] Medicinal applications have ranged from analgesic to anti-inflammatory to tranquilizer to anticonvulsant. Cannabis was introduced into Western medicine during the nineteenth century when W. B. O'Shaughnessy, an Irish physician working in Calcutta in the 1830s, wrote a paper extolling cannabis titled, "Indian Hemp. Traditional Eastern Medicine met Western."[7,8] By the mid-nineteenth century, the medical use of cannabis had received official legitimacy—it was listed in the United States and British Pharmacopeia, and cannabis extracts or tinctures were used extensively by physicians in the Western world. The availability and usage of medical marijuana reached a climax in the last decade of that century.[7,8]

In the United States, marijuana was used medicinally as a bronchodilator and analgesic until 1941, when it was dropped from the US Pharmacopeia.[1,7,8] The Controlled Substances Act, passed in 1970, classified marijuana as a schedule I drug, defined by the Drug Enforcement Administration as follows: "Schedule I drugs, substances, or chemicals are defined as drugs with no currently accepted medical use and a high potential for abuse. Schedule I drugs are the most dangerous of all drugs of all the drug schedules with potentially severe psychological or physical dependence" (http://www.justice.gov/dea/druginfo/ds.shtml). This definition is inaccurate, although it persists to this day.[1,7,8] The labeling was political, not medical. In fact, in the 1930s the American Medical Association lobbied Congress against rendering marijuana federally illegal.[8]

Marijuana and the Lung

The most common route of acquisition of marijuana compounds is via smoke inhalation. Smoke is usually inhaled from combusted compacted and rolled leaves (a "joint"), analogous to a cigarette, or from a water pipe ("bong").[4,7] Most studies quantify exposure to cannabis smoke in "joint-years"; 1 joint-year is equivalent to 1 joint smoked daily for a year. When marijuana is smoked, THC is absorbed into the bloodstream via the lungs.[5,9] Marijuana smoke contains a complex mixture of chemicals that has significant overlap with tobacco smoke.[10,11] In addition, the authors' colleagues have demonstrated that cigarette smoke particles and marijuana smoke particles are similar in both size and distribution when studied with an electrical aerosol analyzer.[12] Given these commonalities, the widespread and increasing use of marijuana has raised concerns that it may cause short- and long-term respiratory complications including bronchitis, pneumothorax/pneumomediastinum, and chronic lung diseases such as chronic obstructive pulmonary disease (COPD), interstitial lung disease, and lung cancers. For this review of respiratory implications of marijuana use, inhalation will be the route of absorption in the cited studies unless specifically stated otherwise.

EFFECTS OF SHORT-TERM AND LONG-TERM MARIJUANA SMOKING ON LUNG FUNCTION

Acute Physiologic Impact

Acutely, cannabis is a bronchodilator; it was listed as such in early pharmacopeias from the mid-1800s until its reclassification as a schedule I product in 1941.[1,5,7,8]

Formal studies postdated this usage, with publications in the 1970s by Tashkin and colleagues[13] and Vachon and colleagues.[14] Tashkin and colleagues[13] demonstrated that after smoking marijuana, there was an immediate increase in specific airway conductance, which peaked at 15 minutes after smoking and remained elevated at 60 minutes; this finding was also demonstrated for ingestion; after ingestion of 10, 15, and 20 mg Δ^9-THC subjects' specific airway conductance increased significantly when compared with placebo. Bronchodilation reached peak levels 3 hours after ingestion, and airway conductance remained elevated for 4 to 6 hours.[13] Tashkin and colleagues[15,16] subsequently reported a similar bronchodilator effect of cannabis in patients with mild asthma and in patients with methacholine- and exercise-induced bronchospasm.

Long-Term Usage and Lung Function

Given that cigarette smoke and marijuana smoke share many components,[10–12] one would have expected long-term marijuana to cause spirometric impairment. Multiple cross-sectional and longitudinal studies have evaluated the long-term effects of marijuana on lung function and compared lung function in marijuana smokers with that of nonsmokers, tobacco smokers, and smokers of marijuana and tobacco[17–30] (**Table 1**). Most of the studies have several limitations such as difficulty in obtaining accurate information for an illicit drug, quantifying its use, and small proportions of marijuana-only smokers (and even smaller numbers of very heavy users). Another possible source of error is a large variation in volume of marijuana smoked; inclusion criteria for the data that are available vary widely, from 50 joints over a lifetime to more than 40 joint-years (>10,000 joints in lifetime). One can, nevertheless, reach some conclusions that seem to be supported by the literature. Key studies are listed in **Table 1**, and key findings are summarized in the following sections.

Long-Term Usage: Marijuana Alone

Results have been conflicting (see **Table 1**). After an early study suggested that marijuana caused a decrease in forced vital capacity (FVC), 2 meta-analyses (with far greater numbers of subjects) came to the opposite conclusion—that marijuana does not cause spirometric impairment.[31,32] One large study found statistically significant nonlinearity for marijuana smokers, with increases of both forced expiratory volume in the first second of expiration (FEV_1) and FVC at low levels of exposure (up to 7 joint-years of lifetime exposure) followed by decreases in FEV_1 at higher levels of exposure.[27] However, the more recent larger cohort studies have shown that the FEV_1 is not impacted.[28,29] Some studies, including the largest cohort study by Kempker and colleagues[28] have demonstrated a decrease in the FEV_1/FVC ratio.[26,27,29,30] This decrease was caused not by a decrease in FEV_1, but by a significant ($P = .0001$) increase in FVC_1 (of note, there is no known disease process that increases the FVC; tobacco reduces the FEV_1/FVC ratio, but by causing a decrease in FEV_1). Every study that looked at diffusing capacity has found no significant impact from marijuana (in never-tobacco-smokers). These collective data suggest that marijuana may have some impact on lung function, but that if it does the nature of those changes differs distinctly from those caused by tobacco.

Long-Term Marijuana Usage Coupled with Cigarette Use

The results of a comparative study by Aldington and colleagues[24] suggest that persons who consume both cannabis and tobacco have a pattern of response similar to tobacco-only users, although, surprisingly, it may be mildly attenuated. Tan and colleagues[25] reported that concomitant usage was associated with increased risk

Table 1
Effects of the use of marijuana on lung function measures in comparison with nonsmoking subjects with adjustment for tobacco use in those who have smoked both marijuana and tobacco

Study/Year/Design	Cohort Size	Age, years	Marijuana Use Average	FEV_1	FVC	FEV_1/FVC	TLC	D_{LCO}
Tashkin et al,[17] USA 1980 Cross-sectional	148	24.1	3 d per week (several times per d) for more than 2–5 y	↔	↔	NR	↔	↔
Tashkin et al,[18] USA 1987 Cross-sectional	446	25–49	54.4 MJ joint years	↔	↔	↔	↔	↔
Bloom et al,[19] USA 1987 Cross-sectional	990	15–40	58.2 MJ joint years	↔	NR	↓	NR	NR
Sherrill et al,[20] USA 1991 Longitudinal	1802	15–60	5–5.5 MJ cigarettes per week	↓	NR	↓	NR	NR
Tashkin et al,[21] USA 1997 Longitudinal	394	25–49	>3.5 MJ joints per day	↔	NR	NR	NR	NR
Taylor et al,[22] 2000 NZ Longitudinal	900	Birth–26	230 times in the past year	NR	NR	↓	NR	NR
Moore et al,[23] 2005 USA Cross-sectional	6728	20–59	10.2 d in last month	NR	NR	↔	NR	NR
Aldington et al,[24] 2007 NZ Cross-sectional	339	18–75	Mean 54.2 MJ joint years	↔	NR	↔	↔	↔
Tan et al,[25] 2009 USA Longitudinal	856	56.3	Lifetime median number of MJ joints: 80.5–208	↑	↑	↔	NR	NR

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Table 1
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Study/Year/Design	Cohort Size	Age, years	Marijuana Use Average	FEV_1	FVC	FEV_1/FVC	TLC	D_{LCO}
Hancox et al,[26] 2010 NZ Longitudinal	967	Birth-32	≤1 MJ joint-year 461/967 >1 MJ joint-year 222/967	↔	↑	↔	↑	↔
Pletcher et al,[27] 2012 USA Longitudinal	5115	18–30	2–3 times of use in past 30 d	↑	↑	↔	NR	NR
Kempker et al,[28] 2015 USA Cross-sectional	7716	18–59	15.8 MJ joint-years 12 d of use/month	↔	↑	↓	NR	NR
Morris et al,[29] 2018 USA Cross-sectional	2304	40–80	30.15 MJ joint years	↑	↑	↔	NR	NR
Hancox et al,[30] 2022 NZ Longitudinal	881	Birth–45	≤5 MJ joint-year 433/881 >5 MJ joint-year 107/881	↔	↑	↓	↑	↓

Abbreviations: ↑, Increase; ↓, decrease; ↔, no association; D_{LCO}, diffusing capacity for carbon monoxide;FEV_1, forced expiratory volume; FVC, forced vital capacity; FEV_1/FVC, ratio; ; MJ, marijuana; NR, not reported; NZ, New Zealand; TLC, total lung capacity; RV, residual volume.

(compared with tobacco use alone) of COPD (odds ratio [OR], 2.90; 95% confidence interval [CI], 1.53–5.51) when the lifetime dose of marijuana exceeded 50 marijuana cigarettes, suggesting synergy between marijuana and tobacco. The findings of Morris and colleagues[29] are consistent with mild attenuation of injury when marijuana is added to tobacco. When these data are combined, it is reasonable to conclude that tobacco is the dominant cause of injury in those who use both products, with an unclear contribution from marijuana.

Clearly there are some contradictory data, some of which suggest that marijuana may cause some lung injury and some of which suggests that marijuana has a protective effect on lung function even in smokers. When changes have been documented, they are not consistent with the pathophysiology of COPD. Given the collected data, it is now reasonable to make 2 conclusions: (1) marijuana in the short term is a bronchodilator and (2) the cumulative effects of long-term marijuana use are minor, and smoking marijuana does not cause COPD. That marijuana smoke contains somewhat counterbalancing injurious and beneficial components is a plausible construct. In contrast to the sometimes contradictory results for marijuana, all cited studies (see **Table 1**) that followed cohorts of tobacco-only smokers consistently showed the expected pattern of lung injury.

RESPIRATORY SYMPTOMS IN MARIJUANA SMOKERS

Associations between marijuana smoking (alone or in combination with tobacco) and respiratory symptoms have been systematically examined in 11 cross-sectional observational studies[18–20,22–25,29,33–35] (**Table 2**). Despite its documented bronchodilation, marijuana smoking is consistently associated with wheeze, cough, and increased sputum production. This apparent contradiction may be related to anatomic considerations. Respiratory symptoms in marijuana smokers are likely due to inflammatory changes in large airway mucosa. Fligiel and colleagues[36] demonstrated large airway inflammation on endobronchial biopsy specimens of habitual marijuana smokers. The histopathologic abnormalities reported were goblet cell hyperplasia, loss of ciliated epithelial cells, and intraepithelial and subepithelial inflammation; the investigators concluded that smoking of marijuana alone caused at least as extensive histopathologic abnormalities in the tracheobronchial mucosa as tobacco alone, including metaplastic changes and nuclear alterations that could be premalignant. Another study of healthy (relatively asymptomatic) habitual marijuana smokers correlated visual changes from bronchoscopy with biopsies.[37] Marijuana smokers (compared with nonsmokers) had significantly higher bronchitis index scores (based on central airway erythema, edema, and airway secretions). These visual changes were confirmed on bronchial biopsies.[37] Cessation does lead to symptom resolution implying normalization of histopathology, but there are no studies involving biopsies in prior users.[33]

The collective data suggest that chronic marijuana smoking is often associated with large airway inflammation, which can be symptomatic. There is no good evidence for the distal airway/alveolar injury that leads to COPD. The dichotomous areas of involvement may explain the contradictions noted earlier.

MARIJUANA AND LUNG CANCER

Moir and colleagues[11] extensively examined the compositions of both mainstream and side-stream smoke from marijuana and tobacco cigarettes and showed many qualitative similarities and some quantitative differences. In addition, the histopathologic and immunohistologic evidence in marijuana users, including bronchial squamous metaplasia and overexpression of molecular markers of pretumor

Table 2
Effects of the use of marijuana on respiratory symptoms when compared with nonsmoking subjects or with adjustment for tobacco use

Study/y/Design	Cohort Size	Age, Years	Marijuana Use Average	Dyspnea	Wheeze	Cough	Sputum
Tashkin et al,[18] 1987 USA Cross-sectional	446	25–49	54.4 MJ joint-years	↔	↑	↑	↑
Bloom et al,[19] 1987 USA Cross-sectional	990	15–40	58.2 MJ joint-years	↔	↑	↔	↑
Sherrill et al,[20] 1991 USA Longitudinal	1802	15–60	5–5.5 MJ cigarettes per week	↔	↑	↑	↑
Taylor et al,[22] 2000 NZ Longitudinal	900	Birth–26	230 times in the past year	↑	↑	↑	↑
Moore et al,[23] 2005 USA Cross-sectional	6728	20–59	10.2 d in last month	↔	↑	↑	↑
Aldington et al,[24] 2007 NZ Cross-sectional	339	18–75	Mean 54.2 MJ joint years	NR	↑	↑	↑
Tan et al,[25] 2009 USA Longitudinal	856	56.3	Lifetime median number of MJ joints: 80.5–208	↑	↑	↔	NR

Tashkin et al,[21] 2012 USA Longitudinal	299	33.4	30 MJ joint-years	
Macleod et al,[34] 2015 UK Cross-sectional	500	37	Men: 104.5 MJ joint-years Women: 53.2 MJ joint-years	
Hancox et al,[30] 2015 NZ Longitudinal	943	Birth-38	≤1 MJ joint-year 99 >1 MJ joint-year 146	
Morris et al,[29] 2018 USA Cross-sectional	2304	40–80	30.15 MJ joint-years	

Abbreviations: ↑, Increase; ↓, decrease; ↔, no association; MJ, marijuana; NR: Not reported; NZ, New Zealand.

progression, are consistent with a lung cancer risk from marijuana.[36,38] Taking this evidence a step further, Maertens and colleagues[39] used a toxicogenomics approach and used murine lung epithelial cells to compare and contrast the toxicologic molecular pathways affected by marijuana smoke condensate (MSC) and tobacco smoke condensate (TSC). Both TSC and MSC exposure were associated with the expression of genes involved in xenobiotic metabolism, oxidative stress, inflammation, and DNA damage response. It is interesting that the MSC was more potent than TSC in dose-response analyses for most common pathways. The data clearly demonstrate pathways affected by MSC that are similar to those for TSC. This study strengthens the link supporting the biological plausibility of marijuana smoking as a risk factor for the development of lung cancer.

Given the aforementioned data, it is surprising that the collective clinical data do not prove an association between marijuana and lung cancer. The current epidemiologic evidence with respect to marijuana smoking and lung cancer is conflicting and sparse with the net balance to date against causation. A pooled analysis of few older case-control studies from Tunisia, Morocco, and Algeria did find that the OR for lung cancer was increased for cannabis smoker after adjusting for lifetime tobacco pack-years (OR, 2.4; 95% CI, 1.6–3.8), raising concerns of oncogenicity.[40] Another population-based Swedish cohort study with 40-year follow-up found a 2-fold risk of lung cancer (CI, 1.08–4.14) in the 831 subjects who reported heavy cannabis use (defined by lifetime use of more than 50 times for their study).[41] A more recent study by Zhang and colleagues[42] contradicts the aforementioned studies. The investigators pooled data on 2159 lung cancer cases and 2985 controls from 6 case-control studies in the United States, Canada, United Kingdom, and New Zealand within the International Lung Cancer Consortium. The overall pooled OR for habitual marijuana smokers versus nonhabitual or never users was 0.96 (95% CI, 0.66–1.38) after adjusting for sociodemographic factors, tobacco smoking status, and pack-years.[42] Another case-control study including 611 lung cancers found no association between use of marijuana and lung cancers (with ORs <1 regardless of intensity of marijuana usage).[43] A recent meta-analysis by Ghasemiesfe and colleagues[44] concluded that evidence of any association between marijuana use and incident lung cancer was insufficient due to confounders.

To summarize, one would expect marijuana to be associated with an increased risk for lung cancer. Older studies corroborated that impression, with more recent studies negating it. Once again, there is a suggestion that marijuana smoke may contain both injurious and beneficial substances. None of these conclusions should be considered to be final; studies have been subject to confounding by concomitant use of tobacco, small sample sizes, young age of participants, and, most important, underreporting.[40–44] It has been proved that marijuana has far less toxicity than does tobacco, but it is reasonable to suggest caution against regular heavy marijuana use.

EMPHYSEMATOUS BULLAE, BAROTRAUMA (PNEUMOTHORAX), AND INTERSTITIAL LUNG DISEASE IN MARIJUANA SMOKERS?

In 2007, Beshay and colleagues[45] reported an unusual increase in the number of young patients at their surgical emergency unit in Switzerland who presented with pneumothorax and history of marijuana smoking. The investigators analyzed and reported their observations as the first and largest case series of spontaneous pneumothorax in heavy marijuana smokers. The radiographic findings on chest computed tomography showed large apical bullae, up to 12 cm in size, which were not present in controls (patients with pneumothorax and no history of marijuana smoking) over the same interval (2002–2004). In another review, 36 case reports of apical bullous lung

disease attributable to heavy cannabis smoking have been reported in young adults.[46] It is evident that marijuana smokers tend to have deeper inhalations and hold their breath for up to 4 times longer than cigarette smokers, sometimes accompanied by Valsalva maneuvers, which may predispose them to barotrauma.[47] It had been postulated that this smoking technique (rather than cannabis itself) is responsible for cases of spontaneous pneumothorax and bullous lung disease reported in young marijuana smokers.[46,47] Contradicting the aforementioned reports, the recent study by Morris and colleagues[29] showed that marijuana smokers had a decrease in emphysema and no evidence for bullous disease, bringing into doubt any association of marijuana with bullae and pneumothorax.

Fligiel and colleagues[48] reported interstitial fibrosis in 24 rhesus monkeys inhaling marijuana on autopsy. No studies have been found showing such effects in humans. Interestingly, in a case report of interstitial disease in a marijuana smoker, biopsies demonstrated particulate matter consistent with talc pneumoconiosis, implicating talcum adulteration and not the marijuana itself.[49]

MEDICINAL USE AND NONPULMONARY ADVERSE EFFECTS OF MARIJUANA

A detailed discussion of nonpulmonary adverse effects and of medicinal use of marijuana is beyond the scope of this review. However, the authors briefly touch on some of them because they are all germane to the issue of marijuana use. There have been substantial changes to the cannabis policy landscape in the twenty-first century; in the last 2 decades almost all states and the District of Columbia have legalized cannabis in some form for the treatment of medical conditions. Marijuana and other cannabinoids (including synthetic cannabinoids) are modestly effective in symptom palliation in patients with cancer; one of the earliest recognized indications for cannabinoids was chemotherapy-induced nausea and vomiting.[50] However, the first milestone was reached in 2018 when the US Food and Drug Administration approved a cannabinoid derived from marijuana to treat severe forms of childhood epilepsy.[51] The available literature suggests that it is also effective in alleviating pain related to cancer, especially neuropathic pain.[50,52] In fact, the comprehensive 2017 consensus report on cannabis compiled by the National Academies of Sciences states that there is conclusive or substantial evidence that cannabis is effective for treating chronic pain in adults, as an antiemetic in chemotherapy-induced nausea/vomiting, and in improving patient-reported spasticity in multiple sclerosis.[53]

No evidence that recreational cannabis use improves general health has been found. The main acute adverse effects for some marijuana users include tachycardia, anxiety, and panic, especially in occasional or naive users.[53,54] Marijuana can be considered to be addictive, and is especially so when individuals start using it in their teens.[48] Marijuana smoking can lead to impairment of cognition, coordination, and judgment and can result in automobile accidents.[55,56] A systematic review of high-quality studies by Asbridge and colleagues[57] concluded that acute cannabis consumption nearly doubles the risk of a collision resulting in serious injury or death. (Ironically, marijuana is less toxic than alcohol, the dominant legal cause of impaired cognition and automobile accidents.) Data suggesting long-term harmful effects of cannabis on neuropsychological function in the developing brain of adolescents are emerging and caution strongly against recreational use in teens.[54,55]

SUMMARY

Marijuana has been used throughout the world for thousands of years. The vilification of marijuana was a relatively recent political—not medical—intervention, which has

negatively impacted our capacity to use and study its components. Although confounded by several factors, knowledge does continue to evolve. The delineation of the prevalence of endocannabinoid receptors has led to a better mechanistic understanding of its capacity to affect us. It is known that different components help (or may help) in the treatment of epilepsy, nausea, anorexia, chronic pain, depression, and possibly cancer.

There are 2 caveats with respect to the use of marijuana. First, it contains mind-altering substances. Any use of marijuana needs to be tempered by knowledge of contexts in which this can cause harm. Second, apart perhaps from focused medicinal uses, marijuana presents a danger to the developing brain, with increased risk of addiction and possibly of cognitive impairment.

The literature on inhaled marijuana and the lung, the primary topic of this review, is somewhat contradictory, but some principles have emerged. That marijuana smoke contains both injurious and protective substances is a reasonable conclusion given what is known today. Marijuana can cause large airway inflammation, but it is also a bronchodilator and does not seem to cause small airway injury. Marijuana does not cause COPD. The collective literature suggests that marijuana does not cause lung cancer. (The authors would argue that any conclusions with respect to the heavy habitual marijuana user should be tentative, because the data are too sparse for this subpopulation.) The data support continued evaluation of the potential benefits of marijuana and its components and of alternative means of acquisition that bypass airway inflammation. Finally, the data on marijuana contrast starkly with the consistent demonstration of injury from tobacco, the greatest legalized killer in the world today. Any possible toxicity of marijuana pales in comparison.

CLINICS CARE POINTS

- Marijuana smoking can cause respiratory symptoms but does not cause COPD.
- Marijuana and tobacco smoke share many components, but a summary of current collective data argues against marijuana and lung cancer causality
- Marijuana use for recreational purposes should be strongly discouraged in teens.

DISCLOSURE

There are no financial conflicts to disclose for all the authors.

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