

# Cancer risk and legalisation of access to cannabis in the USA: overview of the evidence



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Cannabis in the USA is transitioning from a nationwide illegal status to liberalisation for medicinal or recreational use across different jurisdictions. As the acceptability and accessibility of cannabis continue to grow, updated knowledge on the cancer risk from recreational cannabis use is necessary to inform recommendations by public health organisations, policy makers, and clinical practitioners. We reviewed the evidence to date. Our umbrella review of current global epidemiological evidence reveals that links between cannabis exposure and cancer risk are more suggestive than conclusive. The cancer type most closely linked to cannabis use is non-seminoma testicular cancer. However, evidence is emerging of an increased risk of other types of cancer (eg, lung squamous cell carcinoma, head and neck squamous cell carcinoma, and oral, breast, liver, cervical, laryngeal, pancreatic, thyroid, and childhood cancer), underscoring the potential importance of incorporating prevention and cessation of cannabis use in cancer prevention efforts. Our review also identified the need for replication of previous studies for additional epidemiological investigations that use rigorous study designs, and data collection protocols free from the biases of major confounders, misclassification, and measurement error in assessing cannabis exposure. Research on the long-term health and economic consequences of all cannabis products (both medical and recreational) are also needed. Currently, the insufficient evidence on the health risks of cannabis use reduces the ability of policy makers, health-care professionals, and individuals to make informed decisions about cannabis use and could expose the public to a potentially serious health risk.

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## Introduction

Since the 1970s, cannabis in the USA has transitioned from nationwide illegal status to liberalisation for medicinal or recreational use. As of August, 2024, medical cannabis laws were in effect in 39 states, three territories, and the District of Columbia, and recreational cannabis laws in 24 states and the District of Columbia.<sup>1</sup> In 2021, 35·4% of Americans aged 18–25 years, and 17·2% of those aged 26 years or older reported using cannabis in the past year;<sup>2</sup> in 2022, the prevalence of cannabis use among 8th graders (aged 13–14 years) was 8·3%, 10th graders (aged 15–16 years) was 19·5%, and 12th graders (aged 17–18 years) was 30·7%.<sup>3</sup> Evidence for the effect of recreational cannabis laws on changes in cannabis use is mixed: most studies have reported no association among youth (ie, people aged 12–20 years) whereas others found increases among youth and adults.<sup>4</sup> Much less is known about the long-term effects of full legalisation of cannabis use. As cannabis products become readily available in the market at affordable prices and in more potent forms, and as the social acceptability of adult cannabis use increases, the prevalence and duration of lifetime exposure is likely to increase.

The growing acceptability and accessibility of cannabis use in the absence of national-level regulations and product standards have raised public health concerns. Marijuana smoke or aerosol contains many of the same carcinogenic compounds and particulates found in tobacco smoke.<sup>5</sup> It is suspected that cannabis could increase cancer risk, but cannabis smoke contains cannabinoids, some of which have anti-inflammatory properties that could potentially moderate carcinogenic effects of cannabis smoke.<sup>6</sup> Other concerns include

contamination of cannabis with chemicals (eg, insecticides or fungicides) that might pose health risks,<sup>7</sup> increases in paediatric ingestion of cannabis edibles,<sup>4</sup> an increase (associated with recreational cannabis laws) in alcohol, cannabis, and e-cigarette use among adolescents and young adults,<sup>4</sup> adverse cardiovascular and pulmonary effects of vaping cannabis,<sup>8,9</sup> associations between cannabis use and neuropsychiatric disorders,<sup>8</sup> and increasing incidences of overdose and toxicity.<sup>8</sup>

Public health organisations are wary of an expanding cannabis market while the regulatory environment is still developing. In a policy statement in October, 2020, the American Public Health Association called for an evidence-based public health approach to regulating cannabis markets.<sup>10</sup> The American Medical Association is taking a cautious approach by opposing cannabis legalisation for adult recreational use until sufficient research is available on the consequences of consumption.<sup>11</sup> The current public statement of the American Cancer Society is limited to the medicinal use of cannabis: it recommends use of cannabinoid drugs approved by the US Food and Drug Administration at the discretion of the physician and patient; the American Cancer Society has not yet taken a position on the use of cannabis-derived drugs not approved by the Food and Drug Administration or recreational cannabis.<sup>12</sup> The American Cancer Society Cancer Action Network opposes the smoking or vaping of cannabis in public places due to health hazards that carcinogens in cannabis smoke might pose.<sup>12</sup>

In 2017, the Committee on the Health Effects of Marijuana of the US National Academies of Sciences, Engineering, and Medicine (NASEM) published the most comprehensive review of evidence to date on the health effects of cannabis.<sup>13</sup> The report found modest evidence

**Panel: Summary of the US National Academies of Sciences, Engineering, and Medicine's 2017 report<sup>13</sup> conclusions on associations of cannabis smoking and cancer incidence**

- There is moderate evidence of no statistical association between cannabis smoking and the incidence of lung cancer
- There is moderate evidence of no statistical association between cannabis use and the incidence of head and neck cancers, including upper aerodigestive tract, oral cavity, and nasopharyngeal cancers and head and neck squamous cell carcinoma
- There is little evidence of a statistical association between current, frequent, or chronic cannabis smoking and non-seminoma testicular germ cell tumours
- There is no or insufficient evidence to support or refute a statistical association between cannabis smoking and the incidence of oesophageal cancer
- There is no or insufficient evidence to support or refute a statistical association between cannabis use and the incidence of prostate cancer, cervical cancer, malignant gliomas, non-Hodgkin lymphoma, penile cancer, anal cancer, Kaposi's sarcoma, or bladder cancer
- There is no or insufficient evidence to support or refute a statistical association between parental cannabis use and subsequent risk of developing acute myeloid leukaemia or acute non-lymphocytic leukaemia, acute lymphocytic leukaemia, rhabdomyosarcoma, astrocytoma, or neuroblastoma in offspring

that cannabis use was associated with one subtype of testicular cancer and minimal evidence that parental cannabis use during pregnancy is associated with greater cancer risk in children (panel). Although the primary use of cannabis in 2014 in the USA was for recreational purposes (53·4% reporting recreational only, 10·5% reporting medicinal only, and 36·1% reporting both),<sup>14</sup> evidence for short-term and long-term health effects of recreational cannabis use, including cancer risk, remained inconclusive in the report.

Currently, there are more studies and reviews of the therapeutic effects of medical cannabis than on the health effects of recreational use. These studies have mostly addressed whether cannabis or cannabinoids are effective in managing cancer-related pain and treatment-related symptoms.<sup>15</sup> Updated knowledge on cancer risk of recreational cannabis use is necessary to inform the decisions of public health organisations, policy makers, clinical practitioners, and the public.

### **Cancer risk and cannabis exposure: review of the evidence**

We did a rapid umbrella review of global epidemiological evidence on the cancer risk of cannabis use to provide an update and synthesis of review papers and primary studies published since January, 2017, the release date of

the (NASEM) report (which itself was based on a comprehensive and systematic review of evidence up to December, 2016). For this reason, we selected papers in peer-reviewed journals from Jan 1, 2017, to June 30, 2024, that were identified in searches on PubMed and Web of Science for systematic reviews, scoping reviews, narrative reviews, umbrella reviews and meta-analyses, and primary articles, and from citation searching of the aforementioned. These papers were supplemented with primary research studies published since January, 2017, that were not covered in reviews. Because our review also examined reviews published since January, 2017, and because those reviews incorporated primary research studies published before January, 2017 there is some overlap in studies included in those reviews and in the NASEM report.

We synthesised evidence in two steps. First, we summarised findings from the review articles by cancer sites. Second, we added findings for corresponding cancer sites from primary research studies published from January, 2017 to June, 2024, but that were not included in those review articles. We assessed whether the additional evidence from these primary research studies supported or did not support conclusions of the reviews.

The following terms were used in our search: "recreational" AND ["cannabis" OR "marijuana"] AND "cancer risk". As in the NASEM review, we used the term cannabis to refer to a broad class of organic products derived from the cannabis sativa plant that include cannabinoids (active chemical compounds, such as tetrahydrocannabinol [THC], cannabiol, cannabichromene, cannabigerol, and cannabidiol), marijuana (cannabis plant or derivative products that exceed the 0·3% delta-9 THC concentration legal limit), and hemp (cannabis plant with not more than 0·3% delta-9 THC concentration).

We restricted our search to recreational use as opposed to both medical and recreational cannabis use because an initial search that included the medical cannabis literature yielded results focused primarily on the therapeutic benefits of cannabis use in cancer treatment (eg, relief of pain and chemotherapy-induced nausea and palliative oncology and use of cannabinoids as analgesics, antiemetics, sedatives, and antitumour agents). These studies were in preclinical or clinical settings and either involved animal models or patients with pre-existing mental and physical health conditions, including cancer. Because comorbid conditions among cannabis users with cancer can confound associations between cannabis use and cancer outcomes, our literature search excluded the terms medical, medicinal, or therapeutic. Our search strategy is consistent with the approach followed in the NASEM report, which reviewed the cancer risk of cannabis separately from therapeutic effects. We recognise that if the cancer risk from cannabis exposure is real then cannabis use will increase cancer risk regardless of reasons for use and that it would be important for

users of cannabis products for medical reasons to be informed about their risks.

Our search initially retrieved 386 titles and abstracts. After excluding editorials, commentaries, case reports, and grey (non-peer-reviewed) literature, we selected 75 reviews and primary research studies on cancer outcomes linked to cannabis use for full-text review. After full-text review, we excluded 40 articles that did not include the exposure (eg, cannabis use), outcome (eg, cancer endpoints), or study design (eg, clinical and observational research on humans) of interest. The final selection consisted of 35 studies (16 reviews and 19 primary research articles) that focused on cancer incidence or risk linked to cannabis exposure (appendix pp 3–16). All 19 primary research articles were observational studies (four case-control, a cross-sectional, three cohort, and 11 ecological studies). Details of the studies are presented in the appendix (pp 10–16).

Based on our umbrella review, we conclude that links between cannabis exposure and cancer risk are more suggestive than definitive. Moreover, reviews published between January, 2017, and June, 2024, together with evidence from studies not covered in reviews, are consistent in indicating insufficient evidence that cannabis is a proven risk factor for cancer (appendix pp 3–16). The cancer type most closely linked with cannabis use was non-seminoma testicular cancer; however, there is emerging evidence for an increased risk for other types of cancer (eg, lung squamous cell carcinoma, head and neck squamous cell carcinoma, and oral, breast, liver, cervical, laryngeal, pancreatic, thyroid, and childhood cancer). This emerging evidence underscores the urgency of monitoring the association between cannabis use and cancer and the potential importance of cannabis use prevention and cessation in cancer prevention.

### Evidence gaps and directions for future research

We observed that several ecological studies (published between 2021 and 2023) based on longitudinal data from the USA and Europe used inverse probability of treatment weights and relevant diagnostics (eg, minimum expected value for the association—ie, e-Value) to suggest an independent association between cannabis use and cancer risk.<sup>16–21</sup> Data used in these studies include those from the Surveillance Epidemiology and End Results databank of the US Centers for Disease Control and Prevention and the US National Cancer Institute, the European Cancer Information System, and European national cancer registries.

Findings from these ecological studies should be interpreted with caution for two reasons. First, any observational study is potentially subject to confounding and high e-Values do not necessarily exclude the possibility of confounding, which can bias the estimated relationship between exposure and outcome. To minimise confounding bias, it is necessary to identify confounders based on a causal model (eg, directed acyclic

graphs, modified disjunctive cause criterion, or target trial emulation),<sup>22</sup> measure the confounder or proxy variable, and control for the confounders in a stratified or multivariable regression analysis. It is also important to measure the confounder accurately, as measurement error in the confounder variable could introduce residual confounding. Second, causal inference from grouped data can be prone to ecological fallacy; group-level data showing that states or countries with high cannabis prevalence have high cancer rates do not imply that individual cannabis users are more likely to develop cancer.

More large-scale, population-based, well designed cohort studies with well defined exposures that are free from measured and unmeasured confounding are needed to corroborate these results with causal inference. However, prospective studies with large cohorts of cannabis users that can compare cancer risk among different routes of administration and different frequencies and durations of regular cannabis use are challenging and will require years of follow-up. In the interim, well designed and analysed case-control studies could fill the knowledge gap. Research studies should also prioritise specific types of cancer (eg, testicular, oral, respiratory, and childhood cancers) in view of the emerging evidence suggesting increased risk of these cancers associated with cannabis exposure.

Our review also found that virtually all research investigated cannabis exposure primarily through smoked cannabis.<sup>23</sup> Yet there are several new categories of cannabis products available that have remained outside the purview of product-specific research (eg, e-cigarettes used to inhale cannabis extracts, edibles, concentrates, topicals, flowers, pre-rolls, beverages, capsules, tinctures, and sprays).<sup>24</sup> Research is needed to investigate these largely unknown but potential risks from emerging cannabis products because the route of exposure to cannabis could be an important factor in its potential carcinogenicity. One study found that smoking cannabis joints (ie, tobacco-free cannabis wrapped in rolling paper) led to an increase in N-acetyl-S-(2-cyanoethyl)-L-cysteine, a carcinogenic compound, whereas vaping or oral consumption of cannabis products did not.<sup>25</sup> As legalisation of recreational cannabis continues, the evolving variety of cannabis-derived products, cannabinoid composition, and modes of intake complicate the assessment of cannabis exposure and reduce the comparability of conclusions about the cancer risk of cannabis exposure. There is thus a need for a risk assessment of the association between potentially hazardous compounds from novel cannabis products and cancer and adverse non-cancer health risks.

Based on our review, we also conclude that many of the earliest epidemiological studies had several methodological limitations that could have led to null or mixed results. First, underassessment of cannabis exposure might have occurred due to under-reporting because of illegality of cannabis at the time of the survey.

See Online for appendix

### Search strategy and selection criteria

We searched PubMed and Web of Science for systematic reviews, scoping reviews, narrative reviews, umbrella reviews and meta-analyses, and primary articles from Jan 1, 2017, to June 30, 2024, with the search terms “recreational” AND [“cannabis” OR “marijuana”] AND “cancer risk”. We excluded editorials, commentaries, case reports, and grey literature. We excluded studies that did not include the exposure (eg, cannabis use), outcome (eg, cancer endpoints), or study design (eg, clinical and observational research in humans) of interest.

Second, most epidemiological studies were unable to determine amounts of cannabis intake and dose–response relationships because detailed information about history and measures of intensity of cannabis exposure (eg, quantity, frequency, and duration) were not assessed.

Third, although tobacco and alcohol are among the known risk factors for cancer, dual use of cannabis with tobacco or alcohol could represent an even greater cancer risk. Current literature based on individual-level data does not address dual use of cannabis and other substances and many studies do not adjust for confounding by these risk factors. This knowledge gap makes the effects of cannabis difficult to unravel. Moreover, tobacco is often mixed with cannabis through different routes of administration, which could make the adjustment for tobacco use as a confounder intractable, unless data are collected on whether tobacco is mixed with or used separately from cannabis.<sup>26</sup> In a rapidly evolving marketplace offering a wide array of conventional tobacco, novel tobacco, and cannabis products, it is increasingly important to identify how individual-level substitution and co-use of these products and renormalisation of smoking can potentially affect cancer risk at the population level and across different sociodemographic subgroups.<sup>27</sup> This kind of evidence is essential to address the public health concerns associated with cannabis legalisation in an integrated framework of tobacco control, cannabis regulation, and cancer prevention and treatment. To that end, an updated NASEM report is in progress that is expected to provide insight on the use or co-use of other substances alongside cannabis (including alcohol and tobacco), adverse cancer outcomes, and interactions with cancer treatments, along with broader public health implications of the changes in the cannabis policy landscape.<sup>28</sup>

Fourth, the cannabis–cancer connection might be heterogeneous across cancer types. The absence of data on other relevant exposures, and the long incubation period of many cancer types, makes it difficult to connect cannabis exposure to cancer outcomes without long observation periods allowing follow-up of youth and young adults into older adulthood.

Fifth, we observed that studies were characterised by small sample sizes of people who used only cannabis, small numbers of people who had heavy and chronic cannabis intake, low amounts of cannabis exposure in study participants, and recall bias and misclassification from self-reports. Some of these circumstances could change with increasing legalisation of recreational use across states. Currently, the evidence base in the USA is small due to the classification of cannabis as a schedule I controlled substance under federal law; this has resulted in little federal funding for clinical research. However, an increasing number of states are allowing cannabis cultivation, sale, distribution, possession, and use under medical and recreational cannabis laws. A federal cannabis legalisation bill to reschedule cannabis from a schedule I to schedule III controlled substance category is also in progress.<sup>29</sup> These legislative shifts could increase the scope of federal and state funding for clinical research and facilitate larger studies with more participants who vary in intensity and modalities of cannabis consumption.

Finally, we found that the measures of cannabis exposure available from population-level surveys rarely distinguish between medical and recreational uses. For the surveillance of the health effects of cannabis use, public health surveys should be required to include questions to identify the purpose of cannabis use.

### Conclusion

The cancer risk of cannabis exposure continues to be understudied. Our review reinforces the call for epidemiological investigations and replication of studies that use rigorous study design, protocols, and methods. High-quality evidence is essential to resolve contradictory findings and to develop informed policies and health recommendations free from biases and measurement errors. Future research should also examine the long-term health and economic consequences of emerging cannabis products. Understanding these aspects could provide a comprehensive view of the implications of cannabis legalisation in the broader public health context. Until then, the insufficient evidence on the health risks of all cannabis use, both recreational and medicinal, reduces the ability of policy makers, health-care professionals, and individuals to make informed decisions about cannabis use and exposes the public to a potentially serious health risk.

### Contributors

NN and JLW led the conceptualisation, methodology, and writing of the original draft. EO and MMA assisted with methodology and data validation. AJ, FI, PPC, and JLW contributed to writing the original draft and reviewing and editing the subsequent drafts. All authors contributed equally to revisions and review of the revised manuscript for resubmission.

### Declaration of interests

We declare no competing interests.

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