Cannabis oil

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Cannabis oil is a resinous substance extracted from *Cannabis sativa* or *Cannabis indica*. Obtained by separating the resins from cannabis flowers using isopropyl alcohol, cannabis oil is increasingly being sought after for its purported anticancer activity.

PREPARATIONS: Cannabis oil can be taken orally, inhaled by vaporisation, applied topically or formulated into suppositories. High quality oil has a high percentage of active constituents, and is dependent on the cannabis species, quality of raw plant material, and the extraction procedure.

COMMON NAMES: Marijuana oil, hash oil, pot oil, CBD oil, Rick Simpson Oil (RSO).

LATIN NAME: *Cannabis sativa and C. indica* belong to the Cannabaceae family.

ACTIVE CONSTITUENTS: The cannabis plant contains over 500 compounds including flavonoids, terpenoids and cannabinoids. Cannabinoids are the active compounds, of which delta-9-tetrahydrocannabinol (THC), the main psychoactive compound, and cannabidiol (CBD), which counteracts the psychoactive effect of THC, are produced in the highest concentration.

MEDICAL CLAIMS: There is a growing interest, as well as numerous unsubstantiated claims, that cannabis and cannabinoids (especially in the high doses found in oil extraction) may kill cancer cells. It is claimed that this is through action on cannabinoid (CB1 and CB2) receptors and results in direct induction of apoptosis, direct inhibition of tumour growth, and inhibition of tumour angiogenesis and metastasis.

EVIDENCE: An early clinical trial whose primary end point was to determine the safety of highly purified THC administered intracranially in patients with glioblastoma multiforme, showed possible antiproliferative actions on tumour cells. However, owing to obvious legal reasons, virtually all research has been *in vitro* or in animal models. Thus far, the best laboratory results have come from using a combination of highly purified THC and CBD, which have shown to exert anti-proliferative, pro-apoptotic, anti-migratory and anti-invasive actions in a variety of cancers. Conversely, evidence suggests that cannabinoids may encourage cancer cell growth, or have different effects depending on the receptors present on the cancer cell. Resistance to cannabinoids is also likely. More human trials are needed to understand exact mechanisms and signalling by which cannabinoids function.

ADVERSE EFFECTS: Cannabinoids are considered to have a favourable safety profile. The lethal dose in animal studies is estimated to be several grams per kilogram. Most reported adverse effects in trials were fatigue and dizziness. Tachycardia, hypotension, decreased gastrointestinal motility, and muscle relaxation may occur as well as CNS effects (stimulation and depressing); however tolerance to unwanted effects develops quickly. Synthetic cannabinoids are advised not to be used in patients with a history of cardiovascular or psychotic disorder, children under 18 years, women who are pregnant and/or breastfeeding, and women and men who are planning a family.

DRUG INTERACTIONS: Drug-drug and drug-disease interactions are largely unknown due to few human trials available. There may be an

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Summary message

Legal reasons have meant that few scientific clinical studies have been done on medical cannabis. Initial *in vitro* and animal studies show promise in using high quality cannabinoids, eg cannabis oil in a variety of cancers. However, different cannabinoids appear to have different effects and more clinical studies are needed to explore antitumoural effects. While considered to have a good safety profile, due to the uncontrolled production of cannabis in various preparations, vastly different concentrations in products make it difficult to predict pharmacological responses. Little information is available on clinically significant interactions but should be used with caution with CNS depressants and drugs that increase heart rate.

additive effect when used concomitantly with medication that affects heart rate (eg anticholinergics and tricyclic antidepressants) and alertness (benzodiazepines, opiates, alcohol). Studies show cannabinoids may induce CYP1A2, CYP2C and CYP3A, and CBD can inactivate CYP3A4, however it is unknown whether this is clinically significant. A known interaction with dronabinol is ritonavir.

Key references

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