

SCIENTIFIC & TECHNICAL CRITIQUE · OPEN-SOURCE INTELLIGENCE ASSESSMENT

Rick Simpson Oil: A Pharmacological, Toxicological, and Open-Source Intelligence Assessment of an Unstandardized Cannabis Extract

Standardization failures, residual-solvent risk, oral pharmacokinetics, clinical evidence, and the misinformation ecosystem

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Abstract

Rick Simpson Oil (RSO), as prepared at home from widely shared recipes, is a crude, solvent-derived cannabis concentrate promoted since the early 2000s as a treatment for cancer and other serious disease. This memorandum tests RSO against the basic requirements of a medicine—identity, potency, purity, dose accuracy, and evidence of efficacy—and examines the online ecosystem that sustains it. Laboratory replication of Simpson-style extraction found petroleum hydrocarbon residues in naphtha and petroleum-ether extracts, and showed that home heating either leaves cannabinoids largely in their acidic form or destroys terpenes. Lipophilic contaminants present in the plant can be carried into the concentrate. The popular protocol (60 g over 90 days, escalating to about 1 g per day) implies daily THC intakes tens of times larger than Canada's 10 mg per-unit limit for ingestible extracts, taken orally, where absorption is slow and erratic and the liver produces the psychoactive metabolite 11-OH-THC. Antitumour activity of cannabinoids in cells and animals is real, but it has not translated into demonstrated antitumour benefit in humans: human data are limited to a pilot study and a small phase 1b trial, with a phase II trial under way, and none of these used RSO. Licensed RSO-labelled products in regulated markets address potency and contamination, but not this evidence gap. Meanwhile, in one study, the most-shared false “cannabis cures cancer” story drew more than one hundred times the engagement of the most-shared accurate article. Standardized cannabinoid medicines and tested, dose-capped legal products are safer alternatives.

Keywords: Rick Simpson Oil, RSO, pharmaceutical standardization, residual solvents, ICH Q3C, decarboxylation, 11-hydroxy- Δ^9 -THC, CYP2C9, health misinformation, OSINT.

AI Disclosure: This memorandum was drafted with Gemini and revised with Claude (Anthropic) under the analytical direction of Marie-Soleil Seshat Landry (CEO, LandryIndustries.ca & MarieLandrySpyShop.com). AI tools can generate errors, so every reference was checked against publisher, registry, or government sources on 13 October 2026, as logged in Appendix A.

Competing Interests: The author is CEO of LandryIndustries.ca and MarieLandrySpyShop.com, which publish this memorandum.

1. INTRODUCTION

Rick Simpson Oil has a reputation problem, and its most fundamental part is standardization: RSO is unstandardized. It is not a consistent product. It is not a medicine in any meaningful regulatory sense. It is a crude recipe—one that produces widely variable chemical profiles depending on starting flower, solvent choice, heating, and storage.

Yet it continues to be marketed, shared, and promoted as a treatment for cancer and a range of other serious conditions [1, 2].

Conflating a crude botanical extraction with a targeted pharmaceutical is bad science and dangerous clinical practice.

Each factual claim in this memorandum was checked against primary literature, product labels, and regulatory documents retrieved on 13 October 2026, and every number in the text is traceable to the numbered references.

1.1 Scope

This memorandum concerns RSO as it is usually encountered: made at home or sold informally from widely shared recipes, without testing or labelling. It does not claim that every product sold under the RSO name is unstandardized. In regulated markets, licensed producers sell RSO-labelled extracts with measured potency—for example, a 1 g RSO oil listed by the Ontario Cannabis Store at 62.5–70.5% THC in a dose-metering applicator [3], and RSO capsules listed at 8.5–10 mg THC each [4]. Products like these answer the identity, potency, and contamination objections in Sections 3 and 4. They do not answer the evidence objection in Section 5: a tested oil is still not a proven cancer treatment, and a 1 g-per-day protocol with such an oil still delivers several hundred milligrams of THC per day.

2. WHAT RSO ACTUALLY IS

RSO takes its name from Rick Simpson, described in popular accounts as a Canadian engineer and cannabis activist [2], who reports that, after a basal-cell skin cancer was diagnosed in 2003, he applied a homemade cannabis concentrate to the site and credited the oil with healing it [1, 2]. The method he popularized soaks dried cannabis in a solvent—originally naphtha, later 99% isopropyl alcohol or ethanol—filters the liquid, and boils off the solvent to leave a thick, dark resin [1, 2, 5]. Widely circulated versions start with about 1 lb (≈ 450 g) of dried cannabis and 8–9 L of solvent to yield roughly 60 g of oil [1], a biomass-to-oil mass ratio of about 7.5:1.

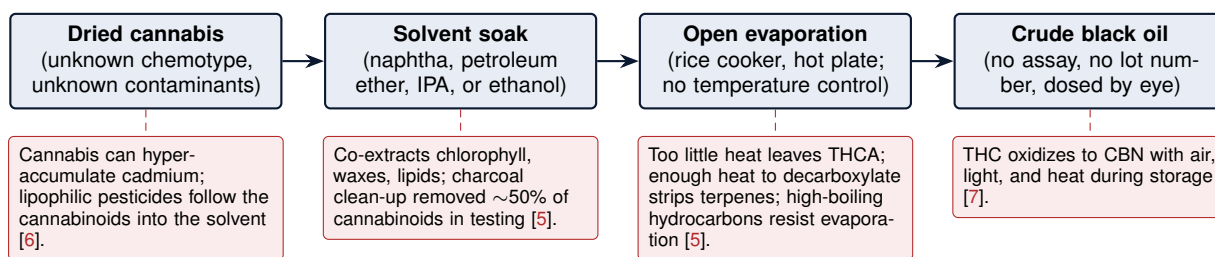


Figure 1. The RSO process and its uncontrolled failure points. Each step adds variability that the end user cannot see or measure.

There is no single chemical entity called “RSO”. Its composition depends on chemotype, solvent, contact time, evaporation temperature, and storage. Three plausible batches illustrate the range:

- **Batch A (well heated):** largely decarboxylated and THC-dominant—but aroma compounds are gone. In laboratory replication, 30 minutes at 145 °C fully decarboxylated THCA, CBGA, and CBCA while leaving only traces of most major terpenes [5].
- **Batch B (gently heated):** mostly acidic cannabinoids. In the same study, most low-heat extracts held only 5–10% of their total THC in the neutral, psychoactive form (33% for the naphtha extract) [5]. The oil is far weaker than its “total THC” suggests—until the next batch is not.
- **Batch C (aged or solvent-laden):** THC partly converted to cannabinol (CBN); in cannabis samples stored at room temperature, nearly all THC degraded to CBN over four years, faster with light [7]. Naphtha and petroleum-ether extracts also retained measurable petroleum hydrocarbons [5].

These extracts share a name, not a defined composition.

3. PRIMARY LIMITATION: FAILURE OF PHARMACEUTICAL STANDARDIZATION

Modern pharmacotherapy relies on chemical reproducibility. A 10 mg capsule of an active pharmaceutical ingredient (API) contains 10 mg of that molecule within specified tolerance limits. It has documented absorption, characterized pharmacokinetics, minimal batch-to-batch variance, and specified limits for impurities such as residual solvents [8, 9].

Homemade, unregulated RSO fails every one of these criteria (Table 1); licensed RSO-labelled products meet some of them (Section 1.1).

Table 1. Homemade RSO versus a standardized cannabinoid medicine.

Parameter	Homemade crude extract (RSO)	Standardized medicine or licensed product
Potency	Unknown without a lab assay; depends on flower, solvent, and heat [5]	Labelled strength, e.g. dronabinol 2.5, 5, or 10 mg capsules [10]
Dose unit	“Half a grain of rice”, escalating to ~1 g of oil per day [1]	Calibrated unit; Canada caps ingestible extracts at 10 mg THC per unit or per dispenser activation [11]
Residual solvents	Untested; petroleum hydrocarbon residues documented in naphtha and petroleum-ether extracts [5]	Limits set by ICH Q3C and USP <467> [8, 9]
Stability	THC → CBN with air, light, and heat [7]	Stability-tested shelf life and storage conditions
Contaminants	Whatever the plant carried: pesticides, metals, mycotoxins [6, 12]	Mandatory pesticide testing in Canada; contaminant limits per lot [13]
Pharmacokinetics	Oral bioavailability ~4–12%; peaks at 1–2 h, sometimes 4–6 h [14]	Characterized and stated in the product label [10]

3.1 Unmeasured Dosing

The canonical RSO regimen starts with a bead half the size of a grain of rice every eight hours, doubling stepwise until the user reaches about 1 g of oil per day, which is maintained until 60 g has been consumed over 90 days [1]. The user knows neither the mass of a “rice-grain” bead of viscous resin nor the oil’s THC content. Table 2 shows what that uncertainty means in milligrams.

Table 2. Illustrative arithmetic: THC delivered per dose (mg) for assumed bead masses and oil potencies. These are worked examples with assumed inputs, not measurements.

Oil taken	30% THC oil	50% THC oil	70% THC oil
10 mg bead	3	5	7
20 mg bead	6	10	14
40 mg bead	12	20	28
1 g/day maintenance [1]	300	500	700

Note. Reference points: Canadian cap of 10 mg THC per ingestible unit [11]; largest dronabinol capsule 10 mg [10].

A doubling of bead size doubles the dose, and the maintenance phase targets daily THC intakes 30 to 70 times the Canadian per-unit limit, without any of the controls that limit is meant to provide.

3.2 Decarboxylation Imprecision

THCA becomes Δ^9 -THC only when heated, and the rate depends on both temperature and time [15]. Kitchen methods cannot hit a target: in laboratory replication, a 5-minute boiling-water bath did not significantly change the acid-to-neutral ratio, while 30 minutes at 145 °C converted the acids fully but destroyed most terpenes [5]. The authors concluded that decarboxylation without substantial terpene loss was not feasible with simple equipment. Extended heat, air, and light then convert THC to CBN by first-order kinetics [7]. CBN is often marketed as sedating, but published evidence for that claim is thin [16].

3.3 Carry-Over of Agricultural Contaminants

Cannabis can hyperaccumulate metals such as cadmium, and many pesticides used in cultivation are lipophilic: they dissolve in extraction solvents and can be concentrated in extracts [6]. Moulds that produce aflatoxins and fumonisins are a recognized hazard of poorly dried or stored cannabis [6, 12]. For a non-volatile contaminant that is fully co-extracted, mass balance sets the concentration factor at up to the biomass-to-oil ratio—roughly 7.5-fold for the popular recipe. The increase is proportional, not exponential, but for an immunocompromised cancer patient it is still an avoidable exposure.

4. TECHNICAL DEEP-DIVE: SOLVENTS, ANALYTICAL TESTING, AND PHARMACOKINETICS

4.1 Solvents and Their Toxicological Classification

Simpson-style protocols have used naphtha, petroleum ether, and isopropyl alcohol [1, 2, 5]. Note that isopropyl alcohol is a *polar* solvent; naphtha and petroleum ether are non-polar hydrocarbon mixtures. Table 3 sets out how the international residual-solvent guideline treats each.

Table 3. Extraction solvents under ICH Q3C(R9) (Step 4, 24 January 2024) [8], with findings from laboratory replication [5].

Solvent	ICH Q3C status and limit	Evidence and hazards
Naphtha (petroleum distillate mixture)	Not classified as a single solvent. Possible constituents: benzene (Class 1, 2 ppm), hexane (Class 2, 290 ppm), toluene (Class 2, 890 ppm)	Significant petroleum hydrocarbon residues by GC and NMR; higher-boiling fractions resisted evaporation; hexane and benzene are potentially neurotoxic; flammable [5]
Petroleum ether	Listed among solvents with no adequate toxicological data (Table 4); use must be justified	Residues found, as with naphtha [5]
Isopropyl alcohol (2-propanol)	Class 3 (low toxic potential); $\leq 5,000$ ppm (50 mg/day) acceptable without justification	Highly flammable during open evaporation [1]
Ethanol	Class 3; $\leq 5,000$ ppm	Efficient extractant; the authors' preferred options were ethanol or, for home use, olive oil [5]

The core problem with naphtha is not its label but its behaviour in a resin. When Romano and Hazekamp replicated Simpson-style extraction, the hydrocarbon residue left in the naphtha extract was of the same order as its total terpene content, and a patient-made sample gave equivalent results [5]. Without vacuum equipment and analytical verification, there is no way to know when a sticky resin is “purged”.

4.2 The Analytical Gap

Regulated cannabis products in Canada are tested to the standard of their product class: THC and CBD content must be accurately tested and labelled, testing for 96 pesticide active ingredients is mandatory, and contaminant limits for mycotoxins, elemental impurities, microbes, and residual solvents draw on pharmacopoeial standards [13]. Kitchen-made RSO receives no analytical verification at all (Table 4).

Table 4. What gets measured, and what does not.

Quality attribute	Regulated product (Canada / pharmaceutical)	Homemade RSO
Cannabinoid identity and potency	Assayed and labelled per lot [13]	Not measured
Residual solvents	Limits per ICH Q3C / USP <467> [8, 9]	Not measured
Pesticides	96 active ingredients, mandatory [13]	Not measured
Metals, mycotoxins, microbes	Pharmacopoeia-based limits [13]	Not measured
Dose per unit	≤ 10 mg THC per unit or activation [11]	“Grain of rice”

4.3 Oral Pharmacokinetics and 11-OH-THC

Swallowed THC is absorbed slowly and incompletely. In controlled studies, oral bioavailability was about 6% (range 4–12%) from a THC-containing cookie and 7% (2–14%) for dronabinol in oil, compared with roughly 10–35% for inhalation; peak plasma levels usually occur 1–2 hours after dosing but can be delayed to 4–6 hours, sometimes with multiple peaks [14].

THC is hydroxylated mainly by CYP2C9 and CYP3A4 [10, 17]. Oral dosing produces much more 11-hydroxy-THC (11-OH-THC) than inhalation: after oral dosing, plasma 11-OH-THC can equal or exceed THC itself, whereas after smoking THC peaks about 20-fold higher than its metabolite [14]. 11-OH-THC is psychoactive in its own right—roughly equipotent to THC in human intravenous studies and three to seven times more potent in some animal tests [14].

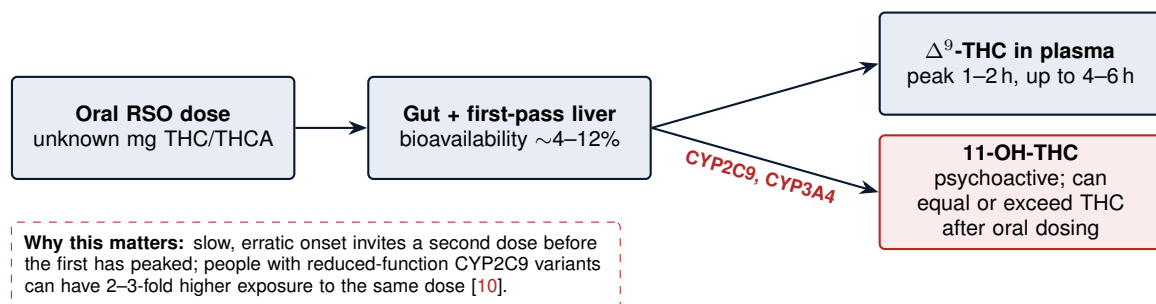


Figure 2. Simplified oral pathway for THC, with values from Grotenhermen [14] and the dronabinol label [10].

The real-world pattern fits the pharmacology. In a Colorado emergency department study, edible cannabis accounted for 10.7% of cannabis-attributable visits in 2014–2016 while making up only 0.32% of THC sold, and edible exposures more often involved acute psychiatric symptoms (18.0% vs 10.9%), intoxication (48% vs 28%), and cardiovascular symptoms (8.0% vs 3.1%) than inhaled exposures [18].

5. THE UNSUPPORTED CANCER-CURE NARRATIVE

The main driver of RSO's popularity is the claim that it cures cancer. That claim rests on a misreading of preclinical research.

Preclinical studies have shown that Δ^9 -THC, CBD, and other cannabinoids can induce apoptosis, inhibit tumour angiogenesis, and suppress proliferation in specific cancer cell lines and animal models [19–21]. The picture is not uniformly favourable: the US National Cancer Institute also summarizes studies in immunocompetent mice in which THC caused immunosuppression and enhanced tumour growth [22].

Many compounds—including common solvents and disinfectants—kill cancer cells in culture. Turning that into clinical benefit requires clearing hurdles that a dish never presents:

- reaching an effective concentration in the tumour without unacceptable systemic toxicity;
- acting across heterogeneous tumour cell populations and their microenvironment;
- crossing delivery barriers such as the blood–brain barrier and dense tumour stroma.

The human evidence is thin. Health Canada's professional monograph (2018 edition; later trials are described below) notes that, at that time, only one limited clinical study had been performed, in recurrent glioblastoma, and that intratumoural injection of high-dose THC did not improve survival beyond conventional chemotherapy [23, 24]. Since then, a phase 1b trial randomized 21 patients with recurrent glioblastoma to nabiximols or placebo alongside temozolomide (after 6 open-label patients). One-year survival was 83% versus 44%, but six-month progression-free survival was 33% in both arms, and the authors presented the result as a reason for a properly powered trial, not as proof of efficacy [25]. That trial, ARISTOCRAT, is a phase II study planned to randomize 234 patients 2:1 with overall survival as the primary endpoint [26]; registry listings in 2026 still describe it as recruiting. The NCI summary likewise describes clinical trials of medicinal cannabis as limited [22].

None of these studies used RSO; they used pharmaceutical THC or nabiximols. No phase III trial has shown that any cannabis preparation cures or controls cancer as an antitumour treatment. The approved cancer-related uses of cannabinoid medicines are for symptoms, such as chemotherapy-induced nausea and vomiting [10, 27].

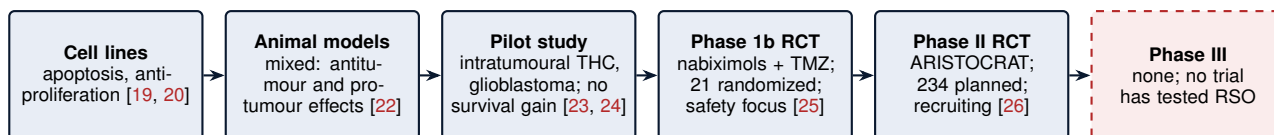


Figure 3. The evidence ladder for cannabinoids as cancer treatment. RSO has no rung of its own above anecdote.

The clinical danger is delay. In a National Cancer Database analysis of patients diagnosed from 2004 to 2013, 280 patients who chose alternative medicine alone were compared with 560 who received conventional treatment; the alternative-medicine group had a greater risk of death (hazard ratio 2.50, 95% CI 1.88–3.27), which persisted for breast, lung, and colorectal cancer [28]. A companion analysis of 258 patients who used complementary medicine alongside conventional treatment found that they were more likely to decline radiotherapy and chemotherapy and had a higher risk of death [29]. A patient who postpones surgery, chemotherapy, or immunotherapy for a crude-oil protocol is trading proven benefit for anecdote.

6. THE RSO INFORMATION ECOSYSTEM: AN OSINT VIEW

RSO persists less because of its chemistry than because of how claims about it travel. Open-source evidence shows a consistent pattern.

Engagement asymmetry. In one study of social-media engagement, the most-engaged false story claiming cannabis cures cancer drew about 4.26 million engagements, against about 0.036 million for the most-engaged accurate article debunking the claim—roughly 118 to 1 [30]. That ratio describes the specific articles studied and is illustrative, not a general rule; the direction, however, matches the broader findings below. Cancer organizations rarely posted on the subject, and their posts averaged 98 Facebook engagements against 452,050 for false news (Figure 4).

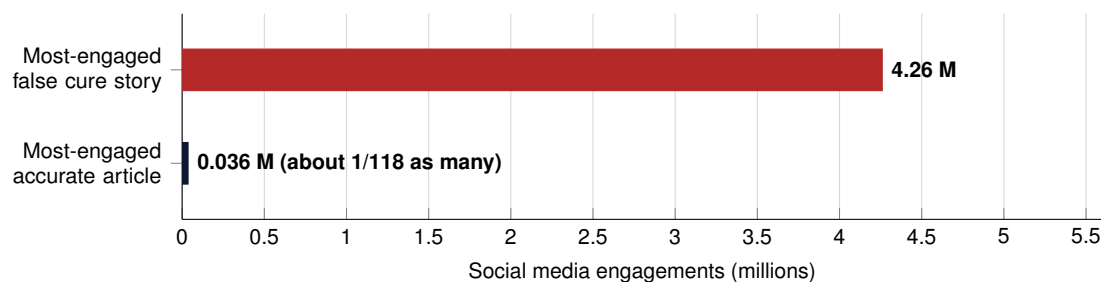


Figure 4. Engagement for the most-shared false “cannabis cures cancer” story versus the most-shared accurate article, from Shi et al. [30]. Single-study figures, shown for illustration.

Harmful content outperforms. Among 200 popular social-media articles about the four most common cancers, expert reviewers judged 32.5% to contain misinformation and 30.5% to contain harmful information; harmful articles drew significantly more engagement than safe ones (median 2,300 vs 1,500) [31].

Crowdfunding. An analysis of GoFundMe campaigns raising money for CBD as a cancer treatment documented the hype and misinformation that circulate when patients fund unproven cannabinoid therapy through their own networks [32].

Enforcement record. In November 2017 the US FDA warned four companies marketing more than 25 cannabis-derived products with claims such as “combats tumor and cancer cells” [33]. FDA product testing also exposed label failures: one product labelled 1,500 mg CBD tested at 5 mg/g, and a tincture labelled 200 mg CBD had none detected [34]. In December 2020 the US FTC’s “Operation CBDeceit” targeted six sellers whose unsupported claims included treating cancer; the orders bar such claims without scientific evidence [35].

OSINT red flags for analysts, clinicians, and patients

- Claims that a product cures, shrinks, or “eliminates” tumours.
- Dosing in rice grains, drops, or syringe marks instead of milligrams of THC.
- No lot-specific certificate of analysis covering cannabinoids, residual solvents, pesticides, metals, and microbes.
- Evidence consisting of testimonials, crowdfunding stories, or “protocols” rather than trials.
- Any advice to delay, reduce, or abandon oncology care.

7. ADVERSE EFFECTS AND CLINICAL COMPLICATIONS

Even when RSO is used only for symptom control—pain, appetite, or nausea—its lack of standardization creates real clinical risks. The warnings below come from the label of a pure, precisely dosed THC product; they apply with more force to an unmeasured one.

- **Psychiatric effects:** dronabinol “has been reported to exacerbate mania, depression, or schizophrenia,” can cause cognitive impairment and altered mental state, and elderly patients may be more sensitive [10]. Edible exposures more often bring acute psychiatric symptoms to the emergency department than inhaled ones [18].
- **Cardiovascular effects:** THC can cause hypotension, hypertension, syncope, or tachycardia, and patients with cardiac disorders may be at higher risk [10]. The American Heart Association concluded that cannabis has few cardiovascular benefits and many cardiovascular concerns, with risks that may depend on the route of delivery [36].
- **Drug interactions:** THC is metabolized mainly by CYP2C9 and CYP3A4, and CBD mainly by CYP2C19 and CYP3A4 [17]. Inhibitors such as fluconazole, amiodarone, ketoconazole, clarithromycin, and ritonavir can raise THC exposure [10]. CBD raises levels of clobazam’s active metabolite and can elevate liver enzymes, especially with valproate [37]. A systematic review judged interaction risk low for most cannabis use while noting that specific human data are lacking [17]; at hundreds of milligrams per day, in patients taking chemotherapy, anticoagulants, anticonvulsants, or immunosuppressants, that reassurance does not transfer.
- **Tolerance:** tolerance develops to most behavioural and somatic effects, presumably through CB₁ receptor downregulation and desensitization; in rats, five days of THC reduced receptor binding by 20–60% in various brain regions [14]. Escalating doses buy diminishing returns.

8. MODERN ALTERNATIVES: STANDARDIZED CANNABINOID THERAPEUTICS

Patients who want what cannabinoids can genuinely offer do not need kitchen extraction. Regulated medicines and legal products provide known content and characterized risks (Table 5).

Table 5. Standardized and regulated alternatives, with indications as stated by the regulator or label.

Product	Composition	Approved use and controls
Dronabinol (Marinol)	Synthetic Δ^9 -THC; 2.5, 5, or 10 mg capsules	US FDA: anorexia with weight loss in AIDS; chemotherapy-induced nausea and vomiting unresponsive to conventional antiemetics [10]
Nabilone (Cesamet)	Synthetic cannabinoid; 0.25, 0.5, or 1 mg capsules	Canada: severe nausea and vomiting associated with cancer therapy [27]
Nabiximols (Sativex)	Standardized botanical extract; 2.7 mg THC and 2.5 mg CBD per 100 μ L buccal spray	Canada: adjunctive treatment of spasticity in multiple sclerosis in patients not adequately helped by other therapy (product monograph revised 2019) [38]
Purified CBD (Epidiolex)	100 mg/mL oral solution	US FDA: seizures of Lennox–Gastaut syndrome, Dravet syndrome, or tuberous sclerosis complex, age 1 year and older [37]
Licensed ingestible extracts (Canada), including tested RSO-labelled oils and capsules [3, 4]	Labelled THC/CBD; lot-tested	≤ 10 mg THC per unit or activation; $\leq 1,000$ mg per container; mandatory pesticide testing [11, 13]

These options are not risk-free: the psychiatric, cardiovascular, and interaction warnings above still apply. The difference is that the dose is known, the contents are verified, and the risks are characterized.

9. LIMITATIONS

- **Design.** This is a narrative critique, not a systematic review or meta-analysis. Sources were identified through targeted searches rather than a pre-registered search strategy, so relevant studies may have been missed.
- **No primary laboratory work.** The author did not test any RSO samples. Findings on residual solvents and on the trade-off between decarboxylation and terpene loss rest largely on a single laboratory replication [5].

Decarboxylation kinetics [15] and storage degradation [7] have independent support, but residual-solvent data for home-made RSO have had limited independent replication.

- **Protocol sources.** The RSO origin story and dosing protocol are taken from popular secondary accounts [1, 2], not from a primary protocol document.
- **Currency of clinical evidence.** Clinical summaries reflect sources available on 13 October 2026, including Health Canada's 2018 professional monograph [23]. The ARISTOCRAT phase II trial [26] may report after this date.
- **Illustrative figures.** Social-media engagement figures come from specific study samples [30, 31], and the dose values in Table 2 are arithmetic illustrations, not measurements.
- **Regulatory change.** Product listings, labels, and regulatory limits change; those cited here are as published on the dates given in the references.
- **Review status.** Every claim was checked against its cited source, but the memorandum has not yet been externally peer reviewed by specialists in pharmacology, toxicology, or oncology.

10. DEMAND PRECISION IN MEDICINE

Rick Simpson Oil emerged from an era of prohibition, no analytical access, and deep institutional distrust. It introduced many people to the therapeutic potential of the cannabis plant, but its continued promotion as a medical treatment is no longer defensible.

Homemade, unregulated RSO is an unstandardized, crude extraction method dosed by grains of rice, without batch-to-batch consistency, at risk of solvent and agricultural contamination, and backed by testimonials rather than trials.

Patients who choose cannabinoids for symptom relief should use a product with known, labelled content, start low and go slow [14], and tell their oncology team. Real healthcare demands consistency, analytical verification, precise dosing, and rigorous safety controls. When it comes to managing serious illness, consistency is not optional—it is the baseline requirement.

This document is intended for educational and analytical purposes only and does not constitute medical advice, diagnosis, or treatment.

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APPENDIX A. SOURCE VERIFICATION LOG

All references were retrieved and checked on 13 October 2026 (America/Moncton). Each entry was confirmed through at least one of the following channels.

- **Read at source (regulatory, label, guideline, and product-listing documents):** [3, 4, 8–11, 13, 22, 23, 27, 34, 35, 37, 38].
- **Confirmed against publisher, university repository, or bibliographic index records, with content checked:** [5–7, 12, 14, 15, 17, 19–21, 24–26, 30, 31].
- **Title, authors, journal, year, and DOI confirmed; volume, issue, or page range not displayed by accessible sources and therefore omitted rather than guessed:** [16, 18, 28, 32, 36]. For [29], volume and pages were confirmed from the authors' publication list and the DOI from Cancer Research UK's report of the study.
- **Confirmed through secondary reporting of a primary document:** [33].
- **Journalistic sources, used only for the RSO origin story and protocol as popularly described:** [1, 2].