Marijuana: Pro and Con

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Without a doubt, the great debate about marijuana will continue. Meantime, here are two nurses’ findings from their survey of evidence—some accepted, some highly controversial—about the drug’s effects.

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We have found no reported proof of major, permanent detrimental effects from the smoking of marijuana in small amounts by rational people. Yet it has been linked with everything from irreversible brain damage to marked relief of pain. Why is the literature so contradictory?

In 1967, a high priority program was inaugurated by the Department of Health, Education, and Welfare to study the implications of marijuana use. Since then, the public has been bombarded with reports of innumerable studies, many of them biased at the least, misleading at worst.

Part of the confusion is due to the great difficulty in placing drug use, especially of new substances, in realistic perspective. When coffee was introduced in the Western Hemisphere from the Near East, penalties as extreme as death were exacted for its use(1).

To help dispel the confusion, we offer, first, the facts about marijuana that are generally accepted and, second, a sampling of the evidence related to its alleged effects, good and bad.

Experts agree that no chemical compound with major pharmacological action is truly safe, that the beneficial and toxic effects of any drug are intimately related to the amount that enters the body, and that many blanks still exist in our knowledge about marijuana. Nevertheless, “in developing rational social policy, scientific evidence of a health hazard must be balanced against social and economic costs incurred by possible alternative strategies for discouraging drug abuse”(2).

Approximately 14 percent of Americans older than 12 (about 28 million people) have smoked marijuana at least once. Of these, about 50 percent no longer use it. Of the 50 percent who do, over one quarter use marijuana more than once a week, and one out of five uses it daily(3). By 1972, “substantial numbers of younger physicians, medical students, nurses, lawyers, and teachers” were included among the older adults who were beginning to experiment with marijuana(4).

Marijuana is obtained from an herb-like plant commonly known as Indian hemp. In 1753 Linnaeus named this hemp Cannabis sativa, and thus one of the oldest sources of psychoactive substances (cannabinoids) entered botanical literature. The plants have been used as a source of hemp fiber and seed oil, and as an intoxicant. Native to central temperate Asia, they are now common to all parts of the world. In the United States, “marijuana” refers to any part of the plant that evokes somatic or psychic changes in humans. “Bhang,” “ganja,” “charas,” and “hashish,” names used in other countries, refer to substances derived from specific parts of the plant(4).

At first, delta-9-tetrahydrocannabinol (delta-9-THC) was believed to be the only active ingredient of Cannabis sativa. More recent studies suggest that delta-9-THC and its hydroxylated metabolites are all active in varying degrees and together account for marijuana’s observed effects.

Cigarettes made from marijuana grown in the U.S. usually contain 2.5 mg to 5 mg of delta-9-THC(4). Mari-
juana can be absorbed orally in tea or tablet form (onset of action: 1/2 to 1 hour; duration, 3 to 5 hours); by pulmonary inhalation (onset of action: minutes; duration, 2 to 3 hours); or parenterally. Smoking is by far the most common way to take the drug, and it is three times more potent by inhalation than by mouth(4).

Because marijuana is so rapidly effective and because of the low content of delta-9-THC in U.S. marijuana, most smokers can regulate their dose by puffing slowly to avoid the excess dosage that causes unpleasant effects(4).

Marijuana has a half-life longer than 24 hours. This prolonged retention is attributed to the fat solubility and protein binding of the compound. A study by Lemberg and others showed that nonusers excreted 67 percent and chronic users excreted 71 percent of a total dose of delta-9-THC in 7 to 10 days; 41-45 percent in feces, 22-31 percent in urine(5). This drug crosses the placental barrier(4).

Subjective effects vary widely, depending on personality, dose, route, and environment. Ordinarily, smoking one marijuana cigarette (2.5-5 mg. delta-9-THC) increases a person's sense of well-being and relaxation and, if he is alone, induces sleepiness(4). Larger doses may produce varied hallucinations. In one study of 100 regular users, smoking marijuana was consistently reported to be a pleasant experience even though a majority "occasionally" had some unpleasant effects(6).

Tolerance has been demonstrated in animals(7) but not in man(8). Marijuana, as used at present by young Americans, is believed not to produce tolerance or sensitivity to its effects(9).

Other effects include increased pulse rate (the larger the dose, the faster the rate), conjunctival reddening, dry mouth and throat, and occasional nausea, vomiting, and dizziness. There are no consistent changes in respiratory rate or deep tendon reflexes(4). Subjects consistently report marked increases in appetite. Experimental research, however, shows no significant alterations in glucose levels from smoking marijuana, but impairment of glucose tolerance occurs when delta-9-THC is given in high (6 mg.) I.V.
doses(10). No proven lethal overdoses have been reported(11).

Beyond these generally accepted facts about the effects of marijuana on health lie many questions. Here is some of the controversial evidence.

How Does Marijuana Affect the Brain?

The evidence is mixed. Lewis et al. found no evidence that marijuana produced excitatory action on the central nervous system(12). Other investigators report opposite results. Both Schwinn and Hill noted an increase in the critical flicker fusion (CFF) threshold(13,14). (CFF is the smallest number of successive light flashes per second that produce a perception of steady light.) CFF is a sensitive measure of a drug's effect on CNS excitability. Alcohol and barbiturates decrease CFF; amphetamines increase it.

Low's comparison of the effects of delta-9-THC and a placebo, and Koppell's comparison of delta-9-THC with alcohol and another showed that delta-9-THC selectively enhanced contingent negative variation (CNV) amplitude(15,16). This measurement is associated with attention-related behavior. CNV is increased when attention is increased. Alcohol depresses the CNV.

Many studies indicate that marijuana slows the learning process, impairs memory, decreases concept formation, decreases tactile discrimination, impairs motor function, and produces "state dependent" or "dissociated" learning(17-23). (Dissociated learning is a phenomenon wherein certain behavior learned in particular drug states recurs more readily when the same drug is reintroduced). The larger the dose of marijuana, the more impairment of all these mental processes.

Studies by Campbell and Heath have provoked sharp controversy about the possibility of irreversible brain damage due to marijuana. From England, Campbell published two studies in 1971. The first study reported cerebral atrophy in 10 young "cannabis users"(24). This finding has been challenged, notably by Kolodny, who said:

In the 10 cases reported [by Campbell] all 10 men had used LSD—many of them over 20 times—as well as canna-

bis, and 8 of the 10 had used amphetamines. One subject had a previous history of convulsions, four had significant head injuries, and a number had used sedatives, barbiturates, heroin, or morphine. On the basis of these facts, speculative connection between cannabis use and brain damage is highly suspect(25).

Campbell's other study(26) reported such an incredibly high incidence of electroencephalographic changes that it is generally considered misleading. In the past, only gross pathology has produced such changes.

Heath reported distinct brain-wave changes in the septal region of six rhesus monkeys after their exposure to marijuana smoke daily for months(27). (The septal region is associated with pleasure and levels of awareness.) Heath also reported EEG changes in one man exposed to marijuana smoke(28). He stated that these changes were much greater than those induced by "inert" marijuana, tobacco, alcohol, or methamphetamine.

Heath's study of "man" is questionable because it dealt with only one subject, whom Heath described as having a severe character disorder and a three-year history of drug abuse. Julius Axelrod, who won a Nobel prize in 1970 for two studies, one of them on the effects of drugs on the brain, testified before a congressional committee on the studies of monkeys:

The doses he [Heath] has given for the acute effect, for example, would be equivalent to smoking 100 marijuana cigarettes. . . . And the amount he has given for the chronic effect represents smoking 30 marijuana cigarettes three times a day for a period of six months . . . the amounts used are so large that one wonders whether it's [the resultant brain damage] due to the large toxic amounts Dr. Heath has given(25).

Other evidence against brain damage exists. Grant and others gave neurological examinations to 58 medical students, 29 marijuana smokers and 29 nonsmokers. The investigators concluded that "moderate social usage of marijuana in this stable group does not
result in sufficient impairment of function that it can be detected by the most sensitive neuropsychological instruments now available"(29).

A well-controlled study, funded by the National Institute of Mental Health, found no brain damage in 30 Jamaicans who smoked ganja, much stronger than U.S. marijuana, for an average of 17.5 years(25,30).

Maugh, from his review of marijuana studies, concluded that the possible connection between heavy, chronic use and brain damage probably will remain controversial for some time and that the adverse effects of cannabis are manifested in only a fraction of susceptible users. He suggests studies of larger populations, and says, "there seems to be enough evidence suggesting the possibility of brain damage that discretion would require avoiding the risk"(31).

Does Marijuana Affect Ability to Drive?

Several studies indicate that marijuana impairs driving ability. Rafaelsen and others demonstrated that both cannabis and alcohol increase the time required to brake and-to start, and that alcohol increased while cannabis decreased the number of gear changes(32). Driving is a divided-attention task, and Casswell and Marks found a significant decrement in marijuana smokers' ability to perform divided-attention tasks in the laboratory(33). Klonoff found a decrease in driving skills in city traffic among smokers of both low- and high-THC-content marijuana cigarettes(34).

These researchers agree that these effects on driving are not uniform, in that the driving ability is affected not only by dose but by the subjects' motivation and ability to compensate.

The Medical Letter on Drugs and Therapeutics advises that "driving while under the influence of marijuana can be dangerous"(35).

Does Marijuana Affect Male Potency?

Recently Kolodny et al. reported that plasma testosterone levels were significantly lower, though within normal limits, in a group of twenty 18- to 28-year-old frequent marijuana smokers than in a control group. Oligospermia (<30 million sperm/ml of semen) occurred in six subjects, and two complained of impotence(36).

Mendelson and Meyer of Harvard were unable to confirm these results with 27 young male subjects under carefully controlled conditions(37). However, Cohen at the University of California at Los Angeles found evidence to support Kolodny(2,30).

This research poses interesting questions. Will smoking marijuana inhibit puberty in preadolescent boys? If pregnant women smoke marijuana will this inhibit the sexual development of their male fetuses? No such effects have been reported, but smoking at least during the first trimester of pregnancy seems advisable(36).

Marijuana use does not appear to inhibit the sex life of adult males. Christie at UCLA found a positive correlation between frequency of marijuana use and frequency of sexual activity among males(30). Perhaps this is due to the fact that androgen is the more important determinant of sexual desire. Also, Heath reports that EEG changes during marijuana intoxication correlate with those during orgasm(28).

Does Marijuana Damage Chromosomes?

This is question of great concern because so many people smoke marijuana during their reproductive years. Studies on animals and man have yielded contradictory results.

In 1968 Persaud and Ellington reported cannabis resin to be teratogenic in the rat but not in the mouse(38). They thus raised the question of a species difference in teratogenic response. However, in 1973 Mantilla-Plata et al. stated that giving delta-9-THC to pregnant mice produced an increase in fetal deaths, a decrease in body size, and an increase in the number of offspring with cleft palate(39).

Pace and others conducted a series of five experiments on rats, using various doses of delta-8- and delta-9-THC during different periods of fetal organ development(40). Low doses did not affect the number of offspring, average birth weight, number of offspring to survive weaning, or weaning weights. Nor did low doses produce significant incidence of gross structural defects or motor impairment in the offspring.

Higher doses, on the other hand, decreased the litter size and increased the incidence of neonatal mortality. However, the latter was explained by the development of agalactia (loss of milk production) in the mothers given marijuana. No chromosome abnormalities or breaks were noted in the offspring. No defects were apparent in the second or third generation of rats except for some stunting in second generation offspring. Nevertheless, the decreased litter size and decreased number of females littering probably indicate an adverse effect of high dose delta-9-THC on rat fertility(39). Agalactia was another adverse effect.

Results in man vary also. Gilmour et al., in 1971, found no significant chromosome abnormalities in light users but noted an increase in chromosome breakage among a few who were taking other drugs as well. These investigators could not identify which drug, if any, caused the aberrations(41). In 1972 Stenchever reported no increase in the incidence of chromosome breaks or gaps in cultures of human leukocytes exposed to delta-9-THC(42). Nichols reported like results in 1974(43).

However, Stenchever reported contradictory results in 1974. He found an average of 3.4 cells per 100 with breaks in 49 marijuana users as compared to only 1 or 2 cells per 100 with breaks in a control group of 20 nonusers(44). The significance of these results is not clear. One suggested explanation is that the delta-9-THC used in most experiments may not be the teratogenic agent—marijuana is, of course, a composite of several agents. Obviously more research is needed to confirm or disprove this explanation and to confirm or disprove chromosome damage.

Does Marijuana Affect Immunity?

Nahas has raised the possibility that marijuana affects cell-mediated immunity in rodents and in man(45, 46). In the human study, Nahas grew T-lymphocytes from marijuana smokers in laboratory cultures, then challenged
the T-cells with foreign substances. He interpreted his results as suggesting a depressed cellular immune response in vitro of chronic marijuana smokers. This response, Nahas reported, resembled that seen in some cancer, uraemic, and immunologically suppressed transplant patients. He speculated that an impairment of DNA synthesis due to marijuana smoking might be responsible, and recommended further studies.

At UCLA, Silverstein and Lesher studied the immune response of 22 chronic marijuana smokers by sensitizing them with dinitrochlorobenzene (DNCB)(25). This chemical agent usually stimulates an immune reaction in normal adults(47). All subjects showed a strong response to even small doses of DNCB. The investigators concluded, Brecher reports, that "there is no clinical or epidemiologic evidence to suggest that chronic marijuana users might be more prone to the development of neoplastic or infectious processes"(25).

In experiments with mice, Harris demonstrated that THC given by mouth,"has little if any effect on DNA synthesis in brain, testis, spleen, or bone marrow tissue in mice, but does depress DNA synthesis by as much as 75 percent in two types of transplanted malignant tumors"(48). These results, Harris believes, suggest a possible therapeutic use for THC.

Marijuana and the Lung

Tashkin et al. investigated the acute pulmonary effects of smoked marijuana and oral delta-9-THC on 32 healthy experienced marijuana smokers. The specific airway conductance, a measure of airway caliber, increased immediately after subjects smoked marijuana that contained 1 or 2 percent delta-9-THC. Conductance decreased, on the other hand, after tobacco smoking and after deep-breathing maneuvers that simulated marijuana smoking. Inhaling 1250 mg. of isoproterenol (Isuprel) increased specific airway conductance to less than 60 percent of the peak observed after subjects smoked 2 percent marijuana. Oral marijuana yielded similar results. Smoked marijuana and oral delta-9-THC dilated airways up to one hour and six hours respectively(49).

These investigators then studied the acute effects of smoked marijuana and oral delta-9-THC on asthmatic subjects. The results showed that, although the maximum mean change in specific airway conductance after smoking 2 percent marijuana was less than after inhaling Isuprel, the bronchodilator effect of marijuana was more sustained, lasting as long as two hours. The bronchodilator effect of oral marijuana lasted four hours(50).

Vachon and others reported the single-dose effect of marijuana smoke on 17 normal subjects with previous marijuana smoking experience. Nine received high-concentration delta-9-THC; eight, low-concentration.

In the high-dose group, airway resistance decreased 38 percent, heart rate rose 28 percent, functional residual capacity did not change, and specific airway conductance increased 44 percent. The low-dose group experienced no increase in heart rate but showed significant, though lesser, changes in airways dynamics.

In both groups, the carbon dioxide sensitivity, measured by rebreathing, did not change. The researchers concluded that "marijuana smoke, unlike cigarette smoke, causes bronchodilation rather than bronchoconstriction and, unlike opioids, does not cause central respiratory depression"(51).

Leuchtenberger and others studied the effects of marijuana and tobacco smoke on human lung explants. Their work suggested that exposing human lung explants to fresh smoke from marijuana or Kentucky Standard tobacco cigarettes evoked abnormalities in DNA synthesis and cell mitosis and growth, and altered DNA and chromosomal complement. "The finding that these changes were observed very early and that they persisted for prolonged periods after exposure indicates," Leuchtenberger stated, "that these alterations are not lethal to the cells"(52,53).

Lung damage also has been reported by Tennant, who performed bronchial biopsies on 30 young soldiers in Europe who smoked more than 25 Gm. of hashish per month, a large amount by U.S. standards. Hashish is the resinous exudate of the top of female plants that contains most of the active ingredients. The biopsies revealed many lesions characteristic of early cancer(48). Two other studies that Maugh cites have reported an increased likelihood of bronchitis and emphysema in people who smoke large amounts of marijuana for long periods(48).

The critical point is whether the lung is damaged by marijuana itself or by smoke inhalation in general. Brecher sees "no reason to doubt that marijuana smoke like tobacco smoke and other kinds of smoke may damage human
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lung cells” and adds that “the extent of damage is probably more closely related to the amount of smoke inhaled than to the type of smoke”(25).

Has Marijuana a Therapeutic Potential? Cannabis has been used as a medicine for a long time in the south of Africa, South America, Turkey, Egypt, and parts of Asia. Its medical use in the Western world peaked between 1840 and 1900, then declined as synthetic hypnotics and analgesics were introduced. The Tax Act of 1937 restricted its use, and marijuana was removed from the U.S. Pharmacopoeia and National Formulary in 1941.

Our review of some current literature suggests that marijuana may have some potential as a medically useful substance in the following categories.

Analgesia Many historical accounts described cannabis as an analgesic. Recent reports, however, imply that this probably is not true for cannabis used alone. One study of human subjects showed that marijuana increased sensitivity to painful and nonpainful stimuli and reduced tolerance for pain(54).

Appetite stimulation Physicians in the 1800’s used marijuana as an appetite stimulant. Although Nahas states there is little recent information to confirm this effect(55), subjective reports and some studies state that marijuana has a strong effect on the appetite, sometimes causing the “blind munchies.”

Anti-epileptic A report was written in 1949 on the anticonvulsant activity of two THC congeners in five institutionalized epileptic children who had severe grand mal epilepsy that was inadequately controlled by phenobarbital and diphenylhydantoin (Dilantin) or mephenytoin (Mesantoin). The THC congeners worked as well or better than these drugs in the five children(11).

Recent reports (1973) show that diphenylhydantoin and THC compounds have a similar qualitative spectrum of anticonvulsant activity(56). Another study has confirmed these data, showing that, by dose, the cannabis compounds were more active than either diphenylhydantoin or primidone (Mysoline)(57).

Antibiosis Czechoslovakian researchers found that the nonpsychoactive, cannabinol fraction of the cannabis plant is a potent gram-positive antibiotic. It can be stabilized in a medical base but used only for external infections at the present. Some researchers believe this is one proven therapeutic effect.(55,30).

Reduction of intraocular pressure In 1971, Hepler et al. reported a substantial decrease in intraocular pressure in youthful subjects after smoking marijuana(58). This was supported further by evidence showing the reduction of intraocular pressure due to vasoconstriction, which decreases blood pressure and blood flow in both aqueous outflow channels and in the ciliary body(59). The treatment of glaucoma may, therefore, be a promising use for marijuana.

Bronchodilation The potential use of marijuana in treating asthma already has been discussed.

Psychotherapeutic aid Marijuana may have implications for psychotherapy. Primary suggestibility in subjects was enhanced to a degree comparable with the state produced by hypnosis in the same subjects(30). Marijuana may promote associational fluidity(60). Despite its ability to elevate mood, delta-9-THC did not produce an antidepressant effect in Kotin’s study of eight severely depressed patients(61).

Anesthesia A century ago dentists used marijuana as a topical anesthetic(11). Two recent studies on animals, combining marijuana with either cyclopropane or halothane, showed that less of both anesthetics was needed(62,63). Smith gave marijuana intravenously, studying its anesthetic or preanesthetic potential on human subjects. The physiological effects noted were marked sedation with minimal respiratory depression, decreased salivation, modest bronchodilation, and an increased cardiac out-put(64). The preliminary research results suggest that marijuana may be a useful adjunct to anesthesia.

Immunotherapy adjunct As discussed previously, marijuana may have a potential for use in decreasing the rejection of organ transplants or in slowing the growth of tumors.

Anti-emesis Sallan and others studied cancer patients who had severe nausea and vomiting as a result of chemotherapy. The usual anti-emetics were not effective. When these patients were given oral delta-9-THC, their nausea and vomiting were reduced and they experienced the pleasant side effects of better appetite and elevated mood(65). Various studies have been done in an attempt to categorize marijuana users in terms of personality and motivation. The rationale for these studies is that potential users, once identified, could be controlled. So far, these attempts have been unsuccessful, probably because users include persons from all walks of life. Low says “cannabis may allow the individual to exist at a phylogenetically old mammalian level, literally within the emotional brain itself.”
free from the conflict which is constant between the old sensory and feeling-oriented system and the newly imposed 'civilization brain'..." (15).

To put it simply, we believe that people smoke marijuana for enjoyment and relaxation. Like any substance, including food and alcohol, marijuana used to excess causes unpleasant and harmful side effects.

References


2. Ibid.

3. Ibid., p. 5.


19. DARLEY, C.F., AND OTHERS. The nature of store-