Review Article

Cannabis — its clinical effects

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Summary

The range of clinical, intoxicating, psychological and psychiatri­c effects of cannabis (‘daggga’) are reviewed. Controversial sub­jects, such as the entities of toxic cannabis psychosis and the cannabis amotivational syndrome, are discussed.

Cannabis sativa (‘daggga’) affects the central nervous system (CNS) in a variety of ways although its biochemical modes of action are unknown. There is some evidence to suggest that Δ-9-tetra-hydrocannabinol (Δ-9-THC), the most psychically active constituent, effects serotonin metabolism in the brain by increasing serotonin release from reserpine-sensitised sites by causing a shift from the bound to the free intraneuronal pool of serotonin and by causing an increased rate of serotonin synthesis from serotonin precursors.1-3

There is also evidence to suggest that cannabinoids interact with other psycho-active drugs and can potentiate the effects of alcohol, caffeine, emetamines and barbituates in man.4 This phenomenon could be a potentiation in the CNS but in non-chronic users of cannabis is more probably mediated by the microsomal oxidase enzyme system in the liver.

Clinical effects

The typical immediate physical effects of cannabis use include dry mouth and throat, tachycardia, postural hypotension, con­junctival vessel injection that causes red eyes and leaves the pupils unaffected, and mild initial bronchoconstriction followed by bronchodilatation. Tachycardia is probably the most reliable index of physiological response to cannabis.6,7

The major intoxication effects of cannabis include mood elevation and a feeling of well-being and perceptual and sensory distortions. Both external senses and internal stimuli are enhanced and experienced as more intense and meaningful. Time and distance perception are distorted. Libido and appetite may be enhanced and short-term memory and judgement may be impaired. These effects usually last for up to 8 hours.8-12

These central changes may be reflected in alterations in electrical activity.13,14 The electro-encephalographic signs of cannabis use in man are not clear cut. Early reports described a reduction in α-wave activity with an increase β-activity for up to 6 days after cannabis use,15,16 while others have demonstrated an increase in α-wave and a reduction in β-wave activity.17,18 Some studies demonstrated a dose-response relationship with altered electro-encephalographic patterns and suggested that a tissue tolerance to cannabis develops, since chronic users demonstrated these changes only with increasing doses of cannabis.19,20 Hollister et al.17 have suggested that these changes are nonspecific effects caused by relaxation and setting.

To date there is no convincing evidence that cannabis use causes brain damage, despite earlier reports of cerebral atrophy in cannabis users.21,22

References


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Cannabis may exert anti-epileptic, anti-emetic and analgesic effects that have therapeutic implications. Cannabidiol has been associated with anti-epileptic properties; the anti-emetic and analgesic effects are associated with δ9-TIC, which may also reduce intra-ocular pressure and has been used to treat wide-angle glaucoma. Its effects on reproductive function are uncertain. Reduced testosterone levels, oligospermia, impotence and gynaecomastia have all been reported. No sound evidence from human studies exists which shows teratogenic effects. Respiratory function can be affected by bronchodilation, but bronchitis, sinusitis and obstructive airways disease may follow chronic irritation and deposition of tar so that its use in asthma has not been recommended.

Adverse psychological effects

Anxiety reactions may occur with acute cannabis intoxication as a ‘bad trip’ experience. Reactions may vary from mild restlessness to more severe states with depersonalisation, de-realisation, a sense of loss of control, fear of dying, panic reactions and paranoid ideas. These adverse reactions may last for a few hours to a few days. Flashbacks from cannabis abuse have been reported but these are infrequent when compared with other hallucinogens. The flashbacks are usually associated with ‘good trips’.

A mild withdrawal state from long-term use has been described. It consists of irritability, restlessness, anorexia, insomnia, nausea, vomiting, diarrhoea and sweating. This reaction is based on the tolerance and mild physical dependence that develops. It is not thought to be severe enough to lead to drug-seeking behaviour. Other studies have failed to demonstrate tolerance and withdrawal phenomena.

Psychotic reactions

A wide variety of psychotic phenomena have been attributed to cannabis use. These include delusional thinking, paranoid ideas, paranoid psychotic reactions, visual and auditory hallucinations, acute brain syndrome, toxic psychosis, transient schizophrenic reaction and schizophrenic psychosis. This consists of irritability, restlessness, anorexia, insomnia, nausea, vomiting, diarrhoea and sweating. The picture observed in this study was very similar to that described in an earlier study of cannabis-related mania.

A number of studies have demonstrated that chronic cannabis use, as well as other socio-economic and family factors, may be associated with an increased incidence of use of ‘harder’ drugs such as cocaine, amphetamines, opiates, narcotics and lysergic acid diethylamide (LSD). Very few people who habitually use hard drugs do not or have not used cannabis at the same time or in the past. An association between chronic cannabis use and alcoholism has also been found.

Amotivation syndrome

An amotivation syndrome has been postulated as a long-term consequence of chronic cannabis use. It is thought to consist of diminished drive, volition and ambition, a loss of motivation, apathy, inertia, self-neglect and a lack of concern about the future.

A Canadian Commission of Inquiry reviewed the evidence for the amotivation syndrome in the late 1960s. It found that retention rates were high in foreign, particularly oriental, countries and that most of the studies were methodologically flawed with results that were not generalisable. Most studies failed to take premorbid personality and sociocultural factors into account. Subsequent studies challenged the earlier reports on an amotivation syndrome and argued that a cannabis-induced amotivation syndrome probably does not exist.

Some authors have ascribed the observed features of the amotivation syndrome to personality factors that predated cannabis use, as well as other socio-economic and family background factors. Other studies have suggested that, to the contrary, chronic cannabis use leads to no differences in productive work output and may in fact be taken to promote motivation among people performing fatigue and monotonous work.

Data on the amotivation syndrome drawn from university student populations also suggest that drop-out rates are not causally associated with cannabis use but rather with background factors, such as closeness to parents, sociopolitical alienation, multiple drug use, parental education level, value orientations and pre-cannabis educational ambitions.

Some studies have reported a productivity decline as well as neuro-psychological and personality changes in chronic cannabis users when tested in controlled experimental settings; these findings have not been confirmed by other researchers.

An increased incidence of personality disorders has been reported, particularly the antisocial spectrum, often with a greater incidence of criminal records among chronic cannabis users compared with matched samples of non-cannabis users. This finding has been proposed as an alternate explanation for the higher drop-out rates and lower levels of social achievement noted among chronic cannabis users.

It is likely that the features indicative of the putative amotivation syndrome are due to either pre-existing personality or sociocultural factors. There is also a much higher risk that chronic cannabis users may be using other illegal drugs and that the amotivational symptoms may be related to this.

Cannabis use and other drugs

A number of studies have demonstrated that chronic cannabis use is associated with an increased incidence of use of ‘harder’ drugs such as cocaine, amphetamines, opiates, narcotics and lysergic acid diethylamide (LSD). Very few people who habitually use hard drugs do not or have not used cannabis at the same time or in the past. An association between chronic cannabis use and alcoholism has also been found.
Conclusion

Research with Cannabis sativa has been difficult to interpret. Probably the major single problem in this regard has been that low potency cannabis has neither been demonstrated to induce organic brain disease nor prolonged psychotic reactions. This contrasts markedly with the clinical picture that is seen in areas such as the RSA where high potency cannabis appears to induce more serious reactions. The differences may be caused by varying composition of ∆9-THC and other metabolites. The full impact of widespread cannabis use in the RSA in terms of psychiatric morbidity, road and industrial accidents and other indices of violence, still has to be elucidated.

REFERENCES