## REVIEW

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## Criminal Responsibility and Cannabis Use: Psychiatric Review and Proposed Guidelines

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ABSTRACT: An evaluation of the criminal responsibility of an offender who has consumed cannabis necessitates knowledge of the effect of the product on the offender's mental state at the time of the alleged offense. However, the effects induced by cannabis are numerous and the forensic psychiatrist should base his diagnosis and his evaluation on facts which are as objective as possible. A selective literature review, using the computerized databases Medline, Psychlit and Embase, has been carried out to aid evaluation from a forensic psychiatry point of view. Biological means of cannabis detection, and the difficulties associated with using them to understand the clinical effect that the product has on any one user, are shown. Eight major categories which can be used in the domain of forensic psychiatry are detailed in this review: Acute usual effects, acute adverse effects, mood disturbance, acute toxic confusion, acute psychotic reaction, chronic paranoid psychosis, amotivational syndrome or other long term effects, and flashbacks. For each of these categories the effects of cannabis intoxication on cognitive and volitional capacities are analyzed, and guidelines for the evaluation of criminal responsibility are proposed.

**KEYWORDS:** forensic science, cannabis, criminal responsibility, intoxication, ability

Cannabis is currently one of the most widely consumed drugs in western countries and throughout the world (1). As is the case with other substances, criminal acts can occur when offenders are under the influence of cannabis. Therefore, the question of the influence of this product on the level of criminal responsibility can be posed during judgement. This happens frequently since cannabis is becoming more commonplace in society; certain countries have decriminalized its use and other countries will probably follow suit in the coming years (2).

In western countries, the laws dealing with the links between mental disorders and criminal responsibility vary, especially the differences between those adopted by continental European countries and those with an Anglo-Saxon legal tradition. Yet, the point shared by these differing national laws is the fact that they all consider that certain mental disorders can lead to diminished criminal responsibility or criminal non-responsibility.

The aim of this review is to determine, from a medical point of

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view, whether certain forms of cannabis intoxication lead to the alteration of mental functioning to the extent that the criminal responsibility of the offender can be said to be diminished. We do not deal with the question of whether the intoxication is voluntary or involuntary. Equally, the objective is not to determine whether the effect of cannabis can be accepted as a legal argument for obtaining a sentence of diminished responsibility.

The research that we performed in the different computerized references (Medline, Psyclit, Embase) showed that no studies exist concerning the relationship between cannabis use and criminal responsibility, although there are many publications on this subject dealing with other substances, especially alcohol and cocaine.

We selected studies devoted to subjects which have a bearing on the question from a forensic psychiatry point of view in order to determine the biological and clinical effects brought on by exposure to cannabis. By basing our work on such data, we were able to come up with a set of guidelines for the evaluation of responsibility according to the different mental states that an offender under the influence of cannabis may, at the time of the alleged crime, be experiencing.

## Sources, Pharmacodynamics, and Detection

#### The Cannabis Plant

The nature of the substance consumed by the offender can play a role in the effects induced by the cannabis. Different preparations contain different concentrations of cannabis. They are all derived from the female Cannabis Sativa plant, but there are also many subspecies and, in the last twenty years, the potency of cannabis products has greatly increased (3).

Marijuana is prepared from the dried flowering tops and leaves, hashish consists of dried cannabis resin and compressed flowers, and hash oil is the preparation extracted from hashish by distillation.

According to a study carried out by the British Medical Association (4), there are more than 60 different cannabinoids and over 400 active components identified in different samples of cannabis.

The most active ingredient is delta-9-tetrahydrocannabinol (THC). The concentration of THC varies considerably in different plants and preparations. THC content is usually 0.5-5.0% in marijuana, 2-20% in hashish, and 15-50% in oil. Street hashish usually contains around 1% THC, Lebanese hashish contains 5-10% THC, and certain varieties such as Sinsemilla, Skunkweed, and Netherweed can have a THC content of up to 20% (5).

Cannabis may be smoked in a "joint," with tobacco, or in a water pipe. If the marijuana or the hashish is eaten, the psychoactive effects appear more slowly.

## **Pharmacodynamics of Cannabinoids**

Isbell et al. reported in 1967 (6) a dose-related psychomimetic effect and an idiosyncratic reaction experienced by some subjects at low dose. The THC binds to specific central cannabinoid receptors, abbreviated as CB1, which are widely distributed in brain regions involved in motor coordination, pain perception, memory, cognition, and reward (7). The involvement of central cannabinoid receptors in motor activity is suggested by the way that they are found in great numbers in the output nuclei of the basal ganglia (8). The discovery of several endogenous ligands that bind to the CB1 receptors suggests that an endogenous cannabinoid neurochemical system exists. There is evidence that endogenous cannabinoid transmission plays a role in the manipulation of other transmitter systems within the basal ganglia by inhibiting glutamate release, increasing gamma-aminobutyric acid (GABA) turnover, and affecting dopaminergic uptake. Some recent studies on animals suggest that a mutual interaction exists between cannabinoid and opioid systems in the reinforcing properties of these compounds (9).

Thus, biological evidence exists regarding the many, and extremely varied, effects of cannabis on the brain. The fact that different systems are directly or indirectly affected by THC explains why this substance can engender very different symptoms but does not explain why these symptoms vary according to individuals, doses, and the moment of the absorption of the drug into the body.

## Detection in Blood

It is generally accepted that mental effects begin to arise from a level of THC of 8–10 ng/ml in blood (10). However, conjunctival injection can be seen from 5ng/ml. Psychological effects are perceptible within seconds and fully apparent a few minutes after the beginning of smoking.

It is useful to know the serum THC level because the mental effects are in part dose-dependent (6). It is not yet possible to give a precise estimation of the relationship between the consumption of a product, even if the characteristics of the cannabis-product are known, and the serum THC concentration in the period after this consumption. The THC received by smoking cannabis varies from between 20% and 50% and only 10–25% of available THC enters the circulation (3).

Therefore, a blood sample taken from the offender just after the criminal act is necessary, which is rarely possible. If this blood sample can be performed, blood levels of THC could be useful for evaluating mental impairment if the method and time of cannabis use is known. However, immediately after smoking, plasma levels are high while effects are low; whereas at a later stage, the situation is reversed. The exact relationship between serum cannabinoid levels and behavioral effects is not known with any certainty (7).

Initial metabolism takes place in the lungs and liver to 11-hydroxy-THC. This metabolite crosses the blood-brain barrier more readily than THC and is absolutely as active as the latter but its half-life is shorter and its plasma concentration very low. In the liver 11-hydroxy-THC is converted into many inactive metabolites, including 11-nor-carboxy-THC (THC-COOH), the most abundant metabolite in plasma and urine (7).

After consumption by smoking, the level of THC in blood attains its peak after 7–8 min. and then quickly dissipates. A peak of 11hydroxy-THC can be detected just after smoking, but at a lower level. After a few minutes the THC-COOH increases and remains stable for a long period (11). The maximum mental effect does not occur during the peaks of THC, but when the THC and the THC-COOH are at equivalent levels, i.e., a few minutes later (7).

Huestis et al. (12) established that after a single inhalation of smoke from a 1.75% THC marijuana cigarette the THC rate in blood is 7 ng/ml. Simply inhaling from a cigarette with 1 g of cannabis containing 3.55% of THC causes a serum THC level of 18ng/ml. For a whole cigarette the level will depend on the product used and the way that it is smoked but most studies show that the peak serum THC levels will exceed 100ng/ml (13).

Following oral consumption the increase of the serum THC level is less intense, is spread over a period of 4–6 h, and is accompanied by a higher level of 11 hydroxy-THC (14).

### Detection in Urine

Since THC is very lipophilic, it quickly exits the blood to enter and fix itself to the lipid-rich tissues, which is why it heads for the brain. The retention time in these tissues is especially long, which explains why the effects of cannabis are so prolonged and also the phenomenon of flashbacks (15). Furthermore, an enterohepatic cycle and a renal reabsorption exist, which makes the elimination of the cannabinoids very slow. The elimination half-life of THC is approximately 7 days (16).

The most abundant compound in the urine is THC-COOH. This is the compound that is usually measured. Since the THC is stored in the organism and in the elimination process, its presence in a positive test cannot give a precise indication of the time that the cannabis was consumed. THC-COOH can be detected in urine up to 3 weeks after cessation of use (17).

## Other Detection Methods

Detection of THC in saliva, sweat, and tears is a good indicator of recent consumption of cannabis (18). Furthermore, these methods have the advantage of being non-invasive, easy to perform, and currently possible in the form of a rapid test (19). A study by Menkes (20) showed that it is possible to make a correlation between the salivary THC level and the mental effects of the intoxication.

Detection of cannabinoids in strands of hair is also possible but this method is limited in that it only reveals an overall picture of the extent of use by a subject and cannot prove a specific moment of consumption at a specific time (21,22).

## **Clinical Assessment**

It is not possible to determine the mental state of an offender by basing one's judgement solely on the nature and the quantity of cannabis consumed prior to an act, nor on the results of a blood test performed just after the act was perpetrated. Furthermore, such information is not usually readily available or absolutely reliable. For this reason, a clinical evaluation is indispensable.

To perform such an evaluation the forensic psychiatrist cannot rely on the statements made by the accused. He/she should also consider statements provided by witnesses who can describe their views on the state and the behavior of the offender. Table 1 is a summary of the subjective characteristics that a person intoxicated by cannabis may have, the signs that may be noted by witnesses, and the affecting factors which should be taken into consideration in the diagnostic process.

Category	Predisposing Factors	Self-reported Troubles	Disorders Observable by Witnesses		
Acute usual effects	None	Euphoria Sensation of fragmentation or special efficiency of thought Heightened or altered sensory perceptions Short-term memory deficits Alteration of time perception	Infectious laughter Talkativeness Impaired motor skills Impaired attention Delayed reaction Conjunctival injection Increased appetite		
Acute adverse reaction	Naive user Anxious tendencies Large dose	Acute anxiety Fear of dying Sense of loss of control Depersonalization Derealization Visual or auditive hallucinations or illusions, without losing awareness of reality Paranoid ideas	Dry mouth Panic reaction Restlessness Auto or heteroagressivity		
Mood disturbance	Circumstances of use After a psychotic episode	Mild and transient dysphoric reaction	Sadness, tearfulness Social withdrawal Excitation		
Acute toxic confusion	Naive user Very large dose	Apprehension, suspiciousness Confusion Memory impairment Depersonalization, derealization Hallucinations	Disorientation Alteration of concentration Disturbed speech and language Disorganized behavior Inappropriate reactions		
Acute psychotic reaction	Previous history of psychosis or psychotic reaction after drug use	Hypomania Delusion Hallucinations	Delirious conviction Passive listening attitude Agitation Behavior out of touch with reality		
Chronic paranoid psychosis	Psychosis Large dose Chronic use	Persistent problem of keeping in touch with reality Depersonalization Delirium, hallucinations Periods of mood disorder	Changeable periods in terms of being in and out of touch with reality and with social relationships		
Amotivational syndrome and other long term effects	Chronic and heavy use	Impairment of short term memory No interest in anything	Social withdrawal Apathy, lethargy Impairment of concentration		
Flashback	None	Acute, transient, and unexpected reactions	Unexpected behavioral disorders		

#### TABLE 1—Categorization of cannabis-induced mental effects.

## Acute Usual Effects

The acute usual effects of cannabis are well known (23). These psychic effects are dose-related and can arise even after consumption of weak doses (2mg) of THC (24).

The sign that is most usually described by a cannabis user following a moderate intoxication is a feeling of euphoria and relaxation. An awareness of alteration in thought processes is also frequently reported, in terms of "fragmentation" (25) or "particular efficiency" (26).

Short-term memory and attention can be impaired, but unlike the amnesia of alcoholic intoxication, all individuals can recall their experiences under the influence of cannabis.

Subjects reported alterations in their perceptions of time and an intensification of ordinary sensory experiences, such as seeing colors more brightly or hearing music more intensely.

In a study by Keeler et al. in 1971 (27), 50% of subjects described alterations in visual perception, such as objects looking twisted out of shape.

Characteristics noted by observers of those under the influence of cannabis are impaired attention, infectious laughter, and talkativeness. Impaired motor skills and delayed reaction may also be apparent. These problems in skill-related activity may potentially impair driving a motor vehicle or operating machinery (23). Among the somatic effects, conjunctival injection is almost always present and tachycardia arises in 20–50% of cases as well as blood pressure instability. Equally, a dry mouth and an increased appetite can be noticed.

For these acute effects, Ghodse (28) demonstrated that under laboratory conditions cannabis has a similar effect on different individuals and that its effects are dose-dependant. However Jones (29) highlighted that the subjective experience of intoxication depends on mental "set" and environment. Therefore, in a forensic context, the dose of cannabis should be taken into consideration when evaluating the psychic state of the person at the moment that he/she committed the criminal act, but there is no simple or direct relationship between the dose and the mental state.

## Acute Adverse Reactions

An adverse reaction might be characterized as unexpected, distressing, or unpleasant. It is more likely to occur following consumption of high doses of THC (more than 20 mg of THC) and in users with a tendency towards anxiety. It also depends on the user's experience and expectations (30).

The symptom that is overwhelmingly reported in the subjects who have had an adverse reaction is acute anxiety (25,31), with a sense of loss of control and a fear of dying. Depersonalization, transient hallucinations, mystical thinking, and suspiciousness can also be experienced (33) but should be distinguished from true psychotic symptoms.

Witnesses may observe restlessness or panic reactions. Serious behavioral problems revealing a loss of perception of reality can occur but are usually short-lived. Adverse reactions often end rapidly and spontaneously.

### Mood Disturbances

Transient and self-limiting mood disturbance is a common and frequent cannabis effect. The acute usual symptoms, especially the euphoric feeling, are often followed by depressive symptoms (25). Depressive periods have also been described after psychotic problems induced by cannabis consumption (33).

Depression attributed to cannabis use is usually characterized as mild and transient. Subjects speak of a feeling of sadness, a loss of interest and of feelings of affection and pleasure. Suicidal thoughts are rare except in combination with people who have morbid tendencies or depressive disorders. Such disturbances are not always evident to those in the user's entourage. Witnesses have been known, however, to describe periods of withdrawal, tearfulness, and irritability. Dysphoric reactions seem to be more frequent in first-time users (3).

## Acute Toxic Confusion

It is well known that a toxic psychosis can follow ingestion of cannabis (34–36). Isbell et al. (6) showed that such acute toxic confusion is most likely to occur following consumption of very large doses of THC (more than 150 mg) or first time use. According to Chaudry et al. (37) oral administration of cannabis can increase the possibility of occurrence of these states.

Typical symptoms described by patients are apprehension, suspiciousness, memory impairment, confusion, depersonalization, derealization, and hallucinations. Observers often notice disorientation, impaired concentration, disturbed speech and language, disorganized behavior, and inappropriate reactions.

These troubles are transient and self-limiting.

#### Acute Psychotic Reaction

An acute psychosis in clear consciousness may follow cannabis use (38). The relationship between this type of disturbance and a pre-existing psychopathology is difficult to establish. However acute psychotic reactions do not depend on the dose absorbed but are exacerbated by the existence of antecedents of psychosis or psychotic reactions caused by drug use (39).

The patient may describe all the symptoms of substance-induced psychotic disorder. The patient's delirium is often emphatic and hallucinations are also frequently described. Persecutory delusions are the most common type. Mood disorders often present themselves in the form of hypomanic problems accompanied by an acceleration of megalomanic thoughts and ideas or, more rarely, by a feeling of depression (38).

In the absence of treatment, and in the case of an examination having been carried out soon after the criminal act, the subject could still be under the influence of the psychotic disturbance. Should this not be the case, witnesses can aid in the diagnostic process if they describe the significant disorders of mental functioning.

To an observer the person may appear perplexed, disheveled, or eccentric. Speech may be accelerated or incoherent. The psychomotor activity is often disturbed and reveals itself by behavior that is out of touch with reality, sometimes hyperactivity or, more rarely, by an apathetic immobility. The speech reveals delirious conviction and mood problems (40).

An acute psychosis in clear consciousness is self-limited to within days or weeks but may recur after renewed exposure to cannabis.

## Chronic Paranoid Psychosis

The links between schizophrenia and cannabis abuse are complex. Cannabis may precipitate schizophrenia both in its initial presentation and in leading to relapse. A certain number of studies clearly show that there are more schizophrenic subjects within the group of cannabis consumers than within the general population (41) and that, out of the schizophrenic patients, those who use cannabis heighten their risk of succumbing to serious decompensations (42). Some authors claim that there is a specific "cannabis psychosis," characterized by prominent hypomanic features and paranoia, but different studies tend to show that this concept does not correspond to a clinical entity (43) or is not a useful diagnosis (44).

The subjective symptoms that a user may describe are those of a schizophrenia-like psychosis: persistent loss of the sense of reality, depersonalization, paranoid delusion, hallucinations, and possible periods of mood disorder. The user's family, friends, or other observers usually notice significant signs of mental perturbation. These signs are numerous and we will simply state the principal symptoms. Behavioral disorders are persistent and show the person to be out of touch with real and actual events. Speech reveals the delirium of the subject more or less directly but the verbal expression is bizarre, obscure, and sometimes incomprehensible. A perplexed attitude is also common. Periods of passivity bordering on, or attaining, the catatonic can be described, as well as violent outbursts without any objective cause. Social aptitudes are always lastingly affected.

To establish the precise role of cannabis in the development of the psychosis can be of high importance for the forensic psychiatrist because many jurisdictions, especially in the United States, exclude mental disorders caused initially by voluntary intoxication as a legitimate cause of insanity. The forensic psychiatrist should be careful because the authors who have studied this subject have not presented any special clinical findings related to the chronic psychoses induced by cannabis consumption (45).

The factors, which exacerbate persistent psychotic disorders in relation to cannabis use, are a medical history of psychosis, consumption of extremely large doses of cannabis, and chronic use of this substance (15).

#### Amotivational Syndrome and other Long Term Effects

Some scholars have shown that long-term cannabis abuse can result in a state of chronic apathy. It was Smith (46) who was first to describe this disorder as "the loss of the desire to work or compete" and to name it "amotivational syndrome." Chronic cannabis consumers can suffer from a general loss of interest and severe impairment of concentration and short-term memory. The subjects are described as apathetic, lethargic, and as having frequent periods of confusion. A study by Tennant and Groesbeck (47) shows that the disorders are reversible four to six weeks after discontinuation of cannabis use.

It is routinely admitted that "amotivational syndrome" is a subacute encephalopathy that arises after very prolonged use and consumption of large doses, such as 50 to 600 g of cannabis per month (23). This syndrome has been compared to cognitive disorders caused by long-term use described by some authors, notably Solowij et al. (48). These disorders manifest themselves by a difficulty in focusing attention and filtering out irrelevant information. Leon-Carrion (49) reported, after having carried out a study in controlled conditions, that long-term heavy cannabis users showed significant differences in their capacity to compromise, formulate adequate judgements and in their capacity to communicate. These cognitive disorders can be important to recognize within a forensic context but they are often relatively imperceptible and of moderate intensity. To be able to objectify them it is necessary to carry out psychometric tests on the offender.

## Flashbacks

It is acknowledged that flashbacks can occur, but that they are nevertheless rarely provoked by cannabis use alone (15). This phenomenon would be caused by the release of cannabis accumulated in fatty tissues. The subjects can also display all the acute symptoms already described.

This diagnosis is always difficult to prove. It is rare that a blood sample can be taken during the instance of the flashback or even just after. Furthermore, if such a sample is performed the results are difficult to analyze since no precise information exists regarding the serum THC levels during flashbacks. The diagnosis is usually solely clinical (50). Therefore, any details that witnesses can provide are very important if the subject describes acute disorders and has not consumed cannabis just before the act.

The diagnosis of a flashback remains difficult to prove and thus use in a forensic evaluation.

# Relationship between Cannabis Use and Criminal Responsibility

To evaluate the level of criminal responsibility, the forensic psychiatrist must, after having categorized the cannabis effects, analyze the link that could exist between the mental disorder and the criminal act committed. To do this, he/she must follow the standards provided by the law. In Western countries, these standards differ slightly in character from one state to another, but their bases are similar. The jurisdictions usually exclude voluntary intoxication as a legitimate cause of insanity, although substance-induced troubles may support a diminished responsibility defense. In Europe and the United States most of the laws and jurisprudence concerning criminal responsibility distinguish cognitive ability on one hand and volitional ability on the other. The cognitive standard is the most widely-used because it is the only one cited in the M' Naghten rule, which is the base of English jurisdiction and which is employed, with slight modifications, in 25 American states, the district of Columbia, and federal jurisdictions (51–53). But most of the Criminal Codes in continental Europe and the American Law Institute rule, which is used by 21 American states, mention the ability to understand the repercussions of one's acts and the ability to control one's behavior (51–58).

In this way, we have proposed guidelines (Table 2) for the evaluation of the criminal responsibility of an offender who has committed a criminal act while under the influence of cannabis by analyzing the effect of the mental state on both cognitive and volitional ability.

## Acute Usual Effects

The acute usual effects of cannabis use do not diminish the faculty to understand the illegality of an act. Experts who have studied these effects have concluded that they do not affect the subject's conscience (45) nor do they cause major cognitive disorders (23,59). In other words, these effects do not normally cause the user to lose his/her ability to distinguish right from wrong.

In contrast, the euphoria or certain deficits of attention or memory can, in certain cases, lead to a slight alteration in the ability to make decisions. The subject could misinterpret the consequences of his/her act (26) or commit involuntary errors in terms of time-related judgements (60,61). Motor activity can be altered (62). These problems are dose-dependent (63).

Thus if, at the moment of the crime, the accused showed evidence of the usual effects of cannabis consumption, his/her degree of criminal responsibility can be considered either as normal or as slightly diminished.

## Acute Adverse Reactions

Acute adverse reactions can alter a subject's capacity to understand the criminal aspect of his/her acts due to changes in perception, derealization, or paranoid troubles (45,32).

	TABLE 2-	-Proposed	l guidelines fo	r criminal	responsibility	assessment in c	ase of c	cannabis-induced	mental d	lisease at th	e time of	the offens	se.
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Category	Cognitive Ability	Volitional Ability (If Applicable)	Criminal Responsibility
Acute usual effects	Normal	Normal to slightly impaired	Full responsibility to Slightly diminished responsibility
Acute adverse reaction	Normal to moderately impaired	Slightly to moderately impaired	Slightly diminished responsibility to Moderately diminished responsibility
Mood disturbance	Normal	Normal to slightly impaired	Full responsibility to Slightly diminished responsibility
Acute toxic confusion	Moderately to severely impaired	Moderately impaired to completely absent	Moderately diminished responsibility to Highly diminished responsibility
Acute psychotic reaction	Severely impaired to completely absent	Completely absent	Highly diminished responsibility
Chronic paranoid psychosis	Slightly to severely impaired	Slightly to severely impaired	Slightly diminished responsibility to Highly diminished responsibility
Amotivational syndrome and other long term effects	Normal	Normal to slightly impaired	Full responsibility to Slightly diminished responsibility
Flashback	According to the relapse of symptoms	According to the relapse of symptoms	Full responsibility to Highly diminished responsibility

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Paranoid thoughts or a panic reaction can affect the capacity to voluntarily control one's acts. However, these disorders are usually of brief duration and they never take over the subject's mental functioning totally. Real hallucinations are rare and when they do occur they are of moderate intensity and do not engender a total loss of self-control (15,32).

If the subject was experiencing acute adverse reactions at the moment of committing the criminal act his/her responsibility can be evaluated as slightly or moderately diminished. Nevertheless, highly diminished responsibility cannot be linked with such a mental state since the effects do not cause a total loss of touch with reality (25).

#### Mood Disturbances

Mood disturbances that are directly induced by cannabis use are not psychotic and are of moderate intensity (15). Therefore, they do not hinder the cognitive faculty to be aware of the illegal nature of an act. In contrast, it is recognized that, in certain cases, depressive disorders can slightly alter the ability to control one's behavior and acts since they increase the tendency towards irritability and impulsiveness (64,65).

In some situations mood disturbances can therefore be seen as justification for a judgement of slightly diminished criminal responsibility.

## Acute Toxic Psychosis

This type of reaction following cannabis use causes the subject to lose touch with reality and experience feelings of confusion, hallucinatory disorders, and memory problems (25,26). However, the intensity of acute toxic confusion is variable and can cause medium to extremely serious damage to cognitive ability (6). Therefore, the ability to judge right from wrong can range from average to nonexistent.

The decision-making and control functions in relation to the act are also damaged by confusion and psychotic disturbances such as mental automatism, depersonalization, or derealization (38).

The reactions to acute toxic psychosis can justify judgements of moderately to highly diminished criminal responsibility.

## Acute Psychotic Reaction

Subjects' reactions to acute functional psychosis do usually display confusion (15) but their thoughts are totally delirious and they suffer from hallucinations. An analysis of responsibility is thus the same as that for the psychosis of acute schizophrenia (66,67), except that the acute psychotic reaction usually results from voluntary intoxication. Cognitive faculties are largely altered by the paranoid delusions and hallucinations, and faculties that permit awareness of one's behavior are affected by the mood disorders and psychotic disturbances. An assessment of highly diminished responsibility is usually reached.

## Chronic Paranoid Psychosis

The forensic psychiatrist cannot with any certainty distinguish the symptoms of a chronic psychosis, which is induced or at least encouraged by cannabis consumption, from other chronic psychotic disorders. Recent studies (68) agree on the fact that diagnosis of chronic psychosis should not systematically lead to a judgement of irresponsibility. Chronic psychotic states tend to have a fluctuating development and current treatments help to bring about periods of stabilization. During periods of acute decompensation, an acute psychotic disturbance can lead to a total loss of the ability to appreciate the illegality of the act and an inability to make decisions (69).

However, during periods of stabilization these faculties are only partially diminished (70). Depending on the period, chronic psychoses linked to cannabis usage can lead to a judgement of moderately to highly diminished criminal responsibility.

## Amotivational Syndrome and Other Long Term Effects

When an amotivational syndrome or another long-term effect can be demonstrated, the psychiatrist should take this into consideration in his expert's report. Yet, as a general rule, disorders of this type do not impair the ability to recognize right from wrong. They are more likely to cause cognitive problems and lack of motivation (46). At worst they can lead to a slight reduction in the ability to act in line with one's volition (47).

In the case of amotivational syndrome or other long-term effects, criminal responsibility can be considered as slightly diminished or normal.

## Flashbacks

If it can be proved that the accused was experiencing a flashback at the moment that the act was committed, then the same responsibility judgement as proposed for acute usual effects, adverse reactions or psychotic reactions would be appropriate for this phenomenon. The major difficulty is being able to prove that a flashback occurred.

## Conclusions

If the phenomena of cannabis intoxication are well documented, their use in a forensic context remains little known and complex. Given the extensive diversity of mental states to which cannabis consumption can contribute, and the fact that these states can also be dose-related and, on occasions, completely unforeseeable, the forensic psychiatrist should always exercise great caution. The equally large variety of cannabis products currently sold legally or illegally makes an evaluation of the states induced even more difficult.

We have proposed a set of guidelines to appraise the degree of responsibility according to the supposed psychic state of an offender, at the time of the crime, following cannabis consumption. The mental states induced can be very different, and the degrees of responsibility possible are extremely variable, spanning from highly diminished responsibility to total responsibility.

These guidelines must not be considered as fixed entities, nor do they aim to automatically associate a diagnosis with a particular degree of responsibility. The evaluation of criminal responsibility must always take account of the individual's history and specific situation.

Our review purposely centers on the specific aspects of cannabis but, in reality, in forensic situations it is rare to encounter states of pure cannabis intoxication and the psychiatrist regularly has to analyze the possible psychic effects of several psychotropic substances combined.

An awareness of the different substances, both old and new, and their psychic effects, whether absorbed alone or as a cocktail, represents a permanent challenge for the forensic psychiatrist.

## References

- Farrell M, Howes S, Taylor C, Lewis G, Jenkins R, Bebbington P, et al. Substance misuse and psychiatric comorbidity: an overview of the OPCS National Psychiatric Morbidity Survey. Addict Behav 1998;23:909–18.
- 2. Farrell M, Ritson B. Cannabis and health. Br J Psychiatry 2001;178:98.
- Ashton CH. Pharmacology and effects of cannabis; a brief review. Br J Psychiatry 2001;178:101–6.
- British Medical Association. The misuse of drugs. London: Harwood Academic; 1997.
- 5. Hall W, Solowij N. Adverse effects of cannabis. Lancet 1998;352: 1611-6.
- Isbell H, Gorodetzsky CW, Jasinski D, Claussen V, Spulak FV, Korte F. Effects of delta-9-trans-tetrahydrocannabidiol in man. Psychopharmacologica 1967;11:184–8.
- Adams IB, Martin BR. Cannabis: pharmacology and toxicology in animals and humans. Addiction 1996;91:1585–614.
- Muller-Vahl KR, Kolbe H, Schneider U, Emrich HM. Cannabis in movement disorders, Forsch Komplementarmed 1999;6:23–7.
- Ambrosio E, Martin S, Garcia-Lecumberri C, Crespo JA. The neurobiology of cannabinoid dependence: sex differences and potential interactions between cannabinoid and opioid systems. Life Sci 1999;65: 687–94.
- Ohlsson A, Lindgren LE, Wahlen A. Plasma delta-9-tetrahydrocannabinol concentrations and clinical effects after oral and intravenous administration and smoking. Clin Pharmacol Ther 1980;28:409–16.
- Agurell S, Halldin M, Lindgren JE, Ohlsson A, Widman M, Gillespie et al. Pharmacokinetics and metabolism of delta-tetrahydrocannabinol and other cannabinoids with emphasis on man. Pharmacol Rev 1986, 38:21–43.
- 12. Huestis MA, Henningfield JE, Cone EJ. Blood cannabinoids: I. Absorbtion of THC and formation of 11-OH-THC and THCCOOH during and after smoking marijuana. J Anal Toxicol 1992;16:276–82.
- Perez-Reyes M, Owens SM, Di Guiseppi S. The clinical pharmacology and dynamics of marijuana cigarette smoking. J Clin Pharmacol 1981;21:201–7.
- Cones EJ, Hestis MA. Relating blood concentrations of tetrahydrocannabinol and metabolites to pharmacologic effects and time of marijuana usage. Ther Drug Monit 1993;15:527–32.
- Thomas H. Psychiatric symptoms in cannabis users. Br J Psychiatry 1993;163:141–9.
- Maykut MO. Health consequences of acute and chronic marijuana use. Prog Neuropsychopharmacol Biol Psychiatry 1985;9:209–38.
- Ellis GM, Mann MA, Judson BA, Schram NT, Tashian A. Excretion patterns of cannabinoid metabolites after use in a group of chronic users. Clin Pharmacol Ther 1985;38:572–8.
- Kintz P, Cirimele V, Ludes B. Detection of cannabis in oral fluid (saliva) and forehead wipes (sweat) from impaired drivers. J Anal Toxicol. 2000;24:557–61.
- Mura P, Kintz P, Papet Y, Ruesch G, Piriou A. Evaluation de six tests rapides pour la recherche de cannabis dans la sueur, la salive et les larmes. Acta Clin Belg Suppl. 1999;1:35–8.
- Menkes DB, Howard RC, Spears GF, Cairns ER. Salivary THC following cannabis smoking correlates with subjective intoxication and heart rate. Psychopharmacology (Berl) 1991;103:277–9.
- CirimeleV, Sachs H, Kintz P, Mangin P. Testing human hair for cannabis. Rapid screening procedure for the simultaneous identification of delta-9-tetrahydrocannabinol, cannabinol, and cannabidiol. J Anal Toxicol 1996;20:13–6.
- Moeller MR. Hair analysis as evidence in forensic cases. Ther Drug Monit. 1996;18:444–9.
- Castle DJ, Ames FR. Cannabis and the brain. Aust N Z J Psychiatry 1996;30:179–83.
- Martin BR. Cellular effects of cannabinoids. Pharmacol Rev 1986;38: 45–74.
- Ames F. A clinical and metabolic study of acute intoxication with Cannabis sativa and its role in the model psychoses. J Mental Sci 1958;104:972–99.
- Chopra G, Smith J. Psychotic reactions following cannabis use in East Indians. Arch Gen Psychiatry 1974;23:193–8.
- Keeler M, Ewind J, Rouse B. Hallucunogenic effects of marijuana as currently used. Am J Psychiatry 1971;128:213–6.
- 28. Ghodse H. Cannabis psychosis. Br J Addict 1986;81:473-8.
- Jones RT. Marijuana-induced "high": influence of expectation, setting, and previous drug experience. Pharmacol Rev 1971;23:359–69.

- Zinberg NE. Drug, set and setting. New Haven: Yale University Press; 1984.
- Bialos DS. Adverse marijuana reactions: a critical examination of the literature with selected case material. Am J Psychiatry 1970;130:309–11.
- 32. Tart CT. Marijuana intoxication: common experiences. Nature 1970;226:701-4.
- Palsson A, Thulin SO, Tunving K. Cannabis psychosis in South Sweden. Acta Psychiatr Scand 1982;66:311–21.
- Tunving K. Psychiatric effects of cannabis use. Acta Psychiatr Scand 1985;72:209–17.
- Negrete JC. What's happened to the cannabis debate? Br J Addict 1988; 83:359–72.
- Johnson BA. Psychopharmacological effects of cannabis. Br J Hosp Med 1990;43:114–22.
- Chaudry HR, Moss HB, Bashir A, Suliman T. Cannabis psychosis following bhang ingestion. Br J Addict 1991;86:1075–81.
- Rottanburg D, Robins AH, Ben-Arie O, Teggin A, Elk R. Cannabis-associated psychosis with hypomanic features. Lancet 1982;ii:1364–6.
- Johns A. Psychiatric effects of cannabis. Br J Psychiatry 2001;178: 116–22.
- ThacoreV, Shukla SRP. Cannabis psychosis and paranoid psyzophrenia. Arch Gen Psychiatr 1976;33:383–6.
- 41. Andreasson S, Allebeck P, Engstrom A, Rydberg U. Cannabis and schizophrenia: a longitudinal study of Swedish conscripts. Lancet 1987; ii:1483–6.
- Linszen DH, Dingemans PM, Lenior ME. Cannabis abuse and the course of recent–onset schizophrenic disorders. Arch Gen Psychiatry 1994;51: 273–9.
- Thornicroft G. Cannabis and psychosis—is there epidemiological evidence for an association? Br J Psychiatry 1990;157:25–33.
- Littlewood R. Community initiated research—a study of psychiatrists' conceptualisations of "cannabis psychosis". Psychiatr Bull R Coll Psychiatr 1988;157:25–33.
- 45. Hall W, Solowij N, Lemon J. The health and psychological consequences of cannabis use. National Drug Strategy Monograph Series N°25. Canberra: Australian Government Publishing Service; 1994.
- Smith DE. Acute and chronic toxicity of marijuana. J Psychedelic Drugs 1968;2:37–47.
- Tennant FS, Groesbeck CJ. Psychiatric effects of hashish. Arch Gen Psychiatr 1972;27,133–6.
- Solowij N, Michie PT, Fox AM. The differential impairments of selective attention due to frequency and duration of cannabis use. Biol Psychiatr 1995;152:213–9.
- Leon-Carrion J. Mental performance in long term heavy cannabis use: a preliminary report. Psychol Reports 1990;67:947–52.
- Solomons K, Neppe VM. Cannabis-its clinical effects. S Afr Med J 1989;76:102–4.
- Steadman HJ, McGreevy MA, Morrissey JP, Callahan LA, Robbins PC, Cirincione C. Before and after Hinckley: evaluating insanity defense reform. New York: Guildford Press; 1993.
- Wettstein RM, Mulvey EP, Rogers R. A prospective comparison of four insanity defense standards. Am J Psychiatry 1991;148:21–7.
- Green CM, Naismith LJ, Menzies RD. Criminal responsibility and mental disorder in Britain and North America: a comparative study. Med Sci Law 1991;31:45–54.
- Gunn J, Taylor PJ. Forensic psychiatry, clinical legal, and ethical issues. Oxford: Butterword-Heinemann Ltd; 1993.
- Krober HL, Lau S. Bad or mad? Personality disorders and legal responsibility—the German situation. Behav Sci Law 2000;18:679–90.
- Mackay of the Clashfern Lord. Mental responsibility. Med Sc Law 1998; 38:281–2.
- Van Kalmthout A. Intoxication and criminal responsibility in Dutch criminal law. Eur Addict Res 1998;4:102–6.
- Foyer J. Contemporary trends of the law of responsibility. Bull Acad Natl Med 1996;180:2105–15.
- Hollister LE. Actions of various marijuana derivatives in man. Pharmacol Rev 1971;23:349–57.
- Meyer RE, Pillard R, Shapiro L, Mirim SM. Administration of marijuana to heavy and casual marijuana users. Am J Psychiatr 1971;128:198– 204.
- Renault P, Schuster C, Freedman D, Sikic B, de Mello DN. Repeat administration of marijuana smoke to humans. Arch Gen Psychiatry 1974; 31:95–102.
- Chait LD, Pierri J. Effects of smoked marijuana on human performance: a critical review. In: Murphy A, Bartke, editors. Marijuana/Cannabi-

noids: neurobiology and neurophysiology. Boca Raton: CRC Press; 1992,387-423.

- Melges FT, Tinklenberg JR, Deardorff CM, Davies NH, Anderson RE, Owen CA. Temporal disorganization and delusional-like ideation. Processes induced by hashish and alcohol. Arch Gen Psychiatry 1974;30: 855–61.
- Schopp RF. Depression, the insanity defense, and civil commitment: foundations in autonomy and responsibility. Int J Law Psychiatr 1989; 12:81–98.
- Frank C, Harrer G. Forensic psychiatry problems in depression. Wien Klin Wochenschr 1985; 97:196–201.
- Nedopil N. Violence of psychotic patients: how much responsibility can be attributed? Int J Law Psychiatry 1997;20:243–7.
- 67. Gross G, Huber G. Competence and responsibility in schizophrenia. Jpn J Psychiatry Neurol 1994;48:25–32.

- Slovenko R. The mental disability requirement in the insanity defense. Behav Sci Law 1999;17:165–80.
- Bannatyne LA, Gacono CB, Greene RL. Differential patterns of responding among three groups of chronic, psychotic, forensic outpatients. J Clin Psychol 1999;55:1553–65.
- Morse SJ. Craziness and criminal responsibility. Behav Sci Law 1999; 17:147–64.

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