

Marijuana and Psychosis: The Effects of Adolescent Abuse of Marijuana and other Drugs in a Group of Forensic Psychiatric Patients

Michael H Stone, MD*

Department of Clinical Psychiatry, Columbia College of Physicians & Surgeons; Attending Psychiatrist: Mid-Hudson Forensic Psychiatric Hospital, New York *Corresponding address: Michael H Stone, Columbia University College of Physicians and Surgeons Psychiatry, United States Tel: 1-917-750-6627; Fax: 1-212-799-7163; E-mail: michaelhstonemd@gmail.com

Received date: December 20, 2014, Accepted date: February 04, 2015, Published date: February 11, 2015

Copyright: © 2015 Michael H Stone. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Introduction

The use of cannabis, in the form of marijuana and related products, has been on the increase in the US and elsewhere in recent years; a significant proportion of this increase stems from use by adolescents of age 17 or less [1]. Abuse of other drugs, such as Ecstasy (methylenedioxy methamphetamine), PCP (phencyclidine), cocaine, opiates and various hallucinogenic compounds), has also been on the rise, especially in young persons. Some have considered cannabis as a "gateway" to abuse of these other drugs [2], though such an evolution is by no means inevitable, since there are other users of cannabis who do not expand their range to include some of the other compounds.

When used heavily by persons under eighteen, marijuana can precipitate psychotic reactions, with effects resembling the "positive symptoms" of schizophrenia (i.e., delusions and hallucinations). The same is true following use of the "hallucinogenic" drugs, such as psilocybin and mescaline [3]. The psychomimetic effects of cannabis, to which adolescents are especially prone [4-6], are even more common in those with a traumatic childhood [7]. Persistent, as opposed to occasional, cannabis use, along with early onset (age 14 or under), heightened the risk of psychosis - including one with an early onset [8,9]. The presenting symptoms in a psychosis induced by cannabis (or by the other "hallucinogenic" drugs) are often not easily distinguished at the clinical level - from the symptoms of a primary psychotic disorder [10]. It may be difficult to disentangle the "natural" (genetic) from the drug-induced psychoses [11], though as Caton suggests if psychotic symptoms persist during a period of drugabstinence in excess of four weeks, it is likely that the psychosis is not related solely to the drug, and is therefore primary". The task of differentiation becomes more difficult, nevertheless, when confronting a young adolescent who was using cannabis heavily, since the signs of a primary (schizophrenic or bipolar) psychosis may not as of yet become apparent. The current generation of young cannabis users may be at greater risk than was so previously, owing to the generally much higher tetrahydrocannabinol [THC] content in the marijuana preparations currently in use [12,13]. Adolescents with a strong family history of psychosis may be especially vulnerable to a "cannabis-psychosis" [14], given that there does not appear to be a clinical syndrome linked specifically, and reliably, to cannabis. Confronted with a young cannabis-user, the absence of the "negative" signs of schizophrenia (e.g., blunting of affect) or of a formal thought-disorder may point with better specificity to a drug-related, rather than to an "authentic" schizophrenia. The two conditions can at times co-occur, given that many young persons with a incipient schizophrenia may "selfmedicate" with marijuana and/or other drugs - such as amphetamines, by way of alleviating their mental distress [15-17]. Irrespective of genetic vulnerability to psychosis, the task of assessing the role of cannabis in a young user - is the possibility of (a) the known

concomitant use of other psychotomimetic drugs, and (b) the use – about which the smoker had no fore-knowledge - of such compounds via "joints" that were laced with PCP or cocaine.

Regarding the genetic vulnerability to (a schizophrenic or bipolar) psychosis, the clinician's task is complicated by the difficulty in guessing at the strength of such predisposition in the adolescent [18-20]. Cannabis may, for example, present little or only modest risk to those with low vulnerability, but when used by those with high vulnerability – may provoke a psychotic reaction even when smoked in seemingly trivial amounts. The present study was prompted by the need to disentangle as best one can the primary versus secondary psychoses among young persons who abuse marijuana -- in concert with, or in the absence of, unrelated psychomimetic compounds. The need has become all the more urgent, in view of the increasing percentage of young persons who resort to such drugs – marijuana being very much at the head of the pack.

Marijuana use among forensic patients

The present study was carried out at the Mid-Hudson Forensic Psychiatric Hospital, located sixty miles northwest of New York City. The hospital population (approx. 300) constitutes a concentrated sample of persons who have abused marijuana and other drugs, since two-thirds of all the patients have acknowledged having used drugs in the past. There are two main groups of patients, all of whom, having committed an offense, were remanded to our forensic hospital. One group had been advised by attorney, when first arrested, to take a plea of NGRI: "not guilty by reason of mental disease or defect." A second group, though also considered mentally ill, declined when first arrested to take NGRI plea as suggested by their attorney. This group was admitted for treatment to restore legal competency, permitting them to stand trial eventually for their offense.

The present study is based on a sub-sample of 109 patients, all from the NGRI group, whom the author has evaluated over the past two years regarding diagnosis and drug abuse. Apart from one patient who was admitted with a diagnosis of "Borderline Personality Disorder," all the others were considered to suffer from one or another psychosis when first seen at the hospital.

The two major questions the study sought to answer were: (1) to what extent could the admission-diagnoses be substantiated? and (2) what role, if any, could drug abuse be said to have played in their psychosis? As a corollary to the 2nd question: in how many cases could the psychosis be convincingly viewed as primary, as opposed to brought about through drug abuse?

Diagnosis upon admission

The diagnosis recorded for each patient upon admission often reflected impressions recorded previously, when the patient was first arrested and examined by a psychiatrist, often by two psychiatrists working on behalf of the court, shortly after the patient was admitted to a conventional hospital for an injury or an acute condition, or else – incarcerated at a local jail. The major diagnoses (corresponding to DSM's "Axis-I") recorded initially at Mid-Hudson are presented in Table 1.

Original (Admission) Diagnosis	N	Additional Information
Schizophrenia [SZ]	59	Drug-abuse mentioned as a secondary diagnosis in 9 cases
Schizoaffective Psychosis [SzAff]	19	Drug-abuse mentioned as a secondary diagnosis in 4 cases
Uncertain: SZ or SzAff	2	
Bipolar/ Manic Type	9	Polysubstance Abuse mentioned as a secondary diagnosis in 1 case
Schizophreniform Psychosis	2	
Drug-Induced Psychosis	6	Secondary to alcohol (2); to marijuana (2); to cocaine (1); unspecified (1)
Other forms of psychosis	12	Psychotic depression (2); due to brain trauma (2); Fronto-temporal dementia (1); encephalomalacia with delusional disorder (1); post-partum psychosis (1);
		Mental retardation (1); Mood disorder "not otherwise specified" (1); Psychosis "not otherwise specified" (2); Borderline Personality Disorder with pedophilia

 Table 1: Admission-Diagnoses for the Forensic Patients

As can be seen from Table 1, a "schizophrenia-spectrum" disorder (if we include here: schizophrenia, schizoaffective disorder, and the two cases that were "uncertain" as to one or the other of these conditions) represented the "default" position, diagnostically - embracing 70 (64%) of the 109 patients. Another 9 (8%) of the patients were considered "bipolar" manic-depressives. Although the term schizophreniform is often used for conditions resembling schizophrenia, but whose etiology relates to drug abuse (such as LSD) or to some form of braindamage, drugs were not mentioned in the admission diagnosis of the two patients in this category. In the remaining cases: "Other Diagnoses": alcohol, cocaine, and marijuana were mentioned in three of those labeled either "Psychosis Not Otherwise Specified [NOS] or "Mood Disorder NOS." Drug abuse, otherwise said, was seldom given pride of place in the hierarchy of contributing factors that underlay the psychoses for which (in addition to their original offenses) they had been remanded to our hospital in the first place. Though substanceabuse was included in the Axis-I diagnoses in about a fifth of the patients, in only six was this viewed as the causative agent in the ensuing psychosis.

Patterns of drug abuse

Since 97 of the 109 (89%) of our patients had been admitted because of a violent offense (chiefly murder, attempted murder, assault, arson, robbery, and rape), many have been retained over long periods of time (in 24 instances: upwards of 15 years), information about the patterns of drug abuse and about the particular substances used is often embedded in records going back many years. Sufficient attention was paid, however, to the specific substances that one can create a list of the drugs used. Between information from the records and questions posed to the patients in the here and now (concerning the names of the drugs and the ages when first used), one can create a list of which patients had used which drugs, along with an estimate of the age of first usage of the drugs in question. Marijuana was by far the commonly abused drug (74 instances). In thirty of the patients (28%) there was no record of their ever abusing drugs or even alcohol to any significant extant. In this latter group there were, for example, 4 patients who had tried marijuana once or a few times in their life, and another 4 who had spoken of drinking "a few beers" or "rarely" - such as to suggest that marijuana and alcohol had played no role in their overall condition, let alone in the offense that had led to their hospitalization. Table 2 focuses on marijuana and on the age of first known use.

Age at first marijuana use	Number	Percent of those under 18
7 to 12	10	15
13	12	18
14	11	17
15	19	29
16 to 17	8	12
teen years (precise age uncertain)	6	9
age 18 or older	8	

Note: Six of the patients acknowledged using marijuana before age 18, but were not able to be more precise. Eight of the patients used marijuana for the first time at age 18 or older: they constituted 10.8% of the 74 who had used marijuana. Conversely, 89.2% of those who used marijuana began their use at ages 17 or younger.

Table 2: Marijuana Use in the Forensic Patients: Age at First Use

From inspection of Table 2 it can be seen that of the 74 patients who had used marijuana, the age of first use could be determined for 68; for the remaining 6 it was not clear whether first use was before or after age 17. As for the 68 with a known first age, only 8 began smoking marijuana at 18 or older. The other 90% were "early users" (below age 18. Among the 66 who began below age 18, fully 64% were concentrated within the 13 to 15 age range, 15 being the commonest age at first use. Eight of the patients had tried their first marijuana smoking even before puberty. Next to marijuana, cocaine was the most commonly abused drug. Sixty-four of the patients had used cocaine. But there was only one patient who used cocaine to the exclusion of all other drugs or alcohol. When the age at first cocaine-use could be determined, the range spanned the years from 13 to 30, but the average was shifted toward the older adolescent years (17) compared with first marijuana-use, where the average was 15. There were only two cocaine-using patients who had not also used marijuana. Only one patient spoke of having used cocaine (at 18) before he first tried marijuana (at 19); three others claimed to have tried both during the same year: 13 in one instance; 15 in the other two. Otherwise, marijuana use preceded cocaine use.

Page 2 of 8

Among the marijuana users there were hardly any who restricted their substance abuse to marijuana alone: there were just four such patients. Some alcohol use being almost universal in our culture, only six of the marijuana users refrained from alcohol (apart from an occasional beer) in addition to avoiding all the "street" drugs. In another eight, some episodes of problem-drinking accompanied their marijuana habit. There were ten patients who used only alcohol; eight – to the level of "problem drinkers" or confirmed alcoholics, and two in whom alcohol-use had been negligible.

Diagnostically, seven of the eight patients who abused marijuana and (to a lesser extent) alcohol - were classified originally as schizophrenic; one was regarded as having a "psychosis not otherwise specified." The four patients who abused only marijuana were, in two instances, considered "bipolar" when first evaluated; one, "schizophrenic," and one "schizoaffective." Almost a third of the patients (N = 33, or 30%) also used, at one time or another, one or more of the "exotic" drugs, such as PCP, Ecstasy, LSD, ketamine, mescaline, or psilocybin - or a stimulant drug like amphetamine or methamphetamine. Every one of these patients had also started with marijuana, use of which had preceded the use of the exotic drugs except in one patient who reportedly used marijuana along with PCP, alcohol and cocaine, all at age 13. Usually these 33 patients had tried only one of the exotic drugs, though in 7 cases there had been use of two (such a PCP and LSD) or even three (PCP, Ecstasy, and mescaline). In order of frequency, PCP had been used by 15 patients; heroin or other opiates by 12; Ecstasy by 11; amphetamines by 10; LSD by 7;

psilocybin or other hallucinogenic "mushrooms" by 4; glue-sniffing by 2; and barbiturates, benzodiazepines, ketamine, and anabolic steroids – each by just one patient. In no case was one of these "exotic" drugs the only drug a patient had used. Most of these drugs had been used only on a few occasions, though there was one patient who (besides his frequent use of marijuana) used heroin on a daily basis; several others used heroin or oxycontin with some regularity.

The most common pattern of drug use was that of marijuana, cocaine, and alcohol (14 examples). Other varieties of three different drugs were also common. But 28 of the patients abused still greater varieties: 4 different drugs (12 patients); 5 drugs (10 patients); 6 drugs (2 patients), 7 drugs (2 patients) and 8 different drugs: 1 patient.

Diagnosis following re-examination

After the patients had been in the hospital for varying lengths of time (always in excess of a year), they were reevaluated and reexamined by the author, and in some instances, also by other attending forensic psychiatrists at the hospital.

The 109 patients could be divided into two main groups: (a) those who had never abused drugs (N = 30). Of these, 23 had never even used alcohol, and of the remaining nine, only one had used alcohol fairly heavily in his late teens. Among these 30 patients, the admitting diagnosis and that after re-examination were the same in 23; some modification was felt indicated in the other seven. This situation is spelled out in Table 3.

Original Diagnosis	Re-Diagnosis	N	Re-Diagnosis was changed	N
Schizophrenia	same	14		
"			Birth hypoxia; febrile convulsions	1
"			Psychosis NOS; Sz –features	1
"			Delusional Disorder	1
MDP - Bipolar	same	2	Schizoaffective; bipolar features	1
Schizoaffective Disorder	same	2		0
Delusional disorder	same	1		0
Post-partum psychosis	same	1		0
Post-surgical dementia	same	1		0
Traumatic brain injury (car acid.)	same	1		0
Schizophreniform	same	1		0
Psychosis NOS		0	Depressive Psychosis with paranoid features	1
AD/HD, Psychosis NOS		0	Antisocial Personality, AD/HD	1
ADD, Antisocial Personality, Mild Mental Retardation		0	Bipolar MDP; Antisocial Personality Disorder	1

Note: Among the patients who did not abuse drugs, the diagnoses made by the psychiatrists at the time of arrest, especially those of the classic "genetic" psychoses: schizophrenia, bipolar disorder, and schizoaffective psychosis – were seen as valid when the patients were re-examined at the forensic hospital. In a few instances, an initially vague diagnosis (e.g., "Psychosis Not Otherwise Specified/NOS") was felt to warrant a more specific diagnosis.

Table 3: Diagnosis after Re-examination in the Patients Who Did Not Abuse Drugs

In the larger group of patients in whom drug abuse had been a major factor in their mental illness, the diagnosis upon re-examination often differed from the original. In some, the psychosis for which they were hospitalized appeared secondary to heavy drug abuse (usually of

Page 4 of 8

marijuana in combination with other drugs; in some instances, of marijuana alone). These were the "drug-induced" psychoses. In others, there had been signs of either mental illness preceding drug abuse, or else a strong family history of either schizophrenia or manicdepression. These were the "drug-aggravated" psychoses (akin to the "primary" psychoses alluded to above).

Thirty-one of the patients were re-diagnosed as belonging to the "drug-induced" category. The original and re-examined diagnoses are given in Table 4.

Original Diagnoses	Re-Diagnosis	N	Diagnosis was Changed to:	N
Schizophrenia			Psychosis NOS, secondary to marijuana	9
Schizophrenia			Psychosis NOS	1
Schizophrenia			Psychosis NOS secondary to drugs	6
Schizophrenia			Psychosis secondary to alcohol, drugs and Antisocial Personality	2
Schizoaffective psychosis			Psychosis NOS secondary to (a) marijuana, (b) drugs, (c) drugs & Antisocial Personality, (d) drugs, (e) marijuana & alcohol	5
Schizoaffective psychosis; subst.abuse	same	1		
Schizoaffective psychosis, bipolar			Psychosis NOS secondary to marijuana; Psychopathy	1
Bipolar Disorder			Psychosis NOS secondary to marijuana	2
Schizophrenia or Schizoaffective ?			Psychosis NOS secondary to marijuana & cocaine	1
Psychosis secondary to marijuana	same	1		
Psychosis secondary to marijuana & cocaine	same	1		
Schizophreniform disorder			Schizophreniform psychosis secondary to marijuana	1
Total		3	Total	28

Note: In the 31 patients whose psychosis, upon re-examination at the hospital, appeared to be drug-induced, only three had been diagnosed in this manner when first evaluated by court-appointed psychiatrists shortly after their arrest.

Table 4: Psychoses That Were Considered Drug-Induced.

In this group it was rare for the original and re-examined diagnoses to remain in agreement. There were only two patients whom the first evaluator (usually in a prison or conventional hospital setting) regarded as psychotic because of drug abuse (marijuana in one instance; marijuana and cocaine, in the other).

Thirty-nine of the patients were considered to have suffered from a psychosis that may have been primary, but which had been aggravated in intensity by substance abuse. The latter was most often

"polysubstance abuse," involving two or more drugs (in addition to alcohol, which was often used in excess, alongside the "street" drugs – of which marijuana was the most common). Polysubstance abuse was rarely accorded pride of place in the original diagnosis as the major factor in the psychosis for which the patient was hospitalized; this was the case only in five instances. The breakdown of original and revised diagnoses for this group is given in Table 5.

Original Diagnosis	Secondary Diagnosis	N	Revised Diagnoses	
Schizophrenia (19)	(none)	7	Psychosis NOS, Substance Abuse	
	(none)	1	Delusional disorder, Subst. Abuse	
	(none)	1	Sz, epilepsy, marijuana abuse	
	(none)	1	Sz, marijuana abuse	
	Capgras' Syndrome	1	Sz, marijuana abuse, head injury	
	"adolescent onset"	4	Sz, Substance abuse	
	Polysubstance abuse	1	(same)	
	Polysubstance abuse	1	Delusional disorder, polysubstance abuse	

Page 5 of 8

	Polysubstance abuse	1	Bipolar Disorder, polysubstance abuse
	Mild Mental Retardation ("MMR")	1	Psychosis NOS, marijuana abuse. Head injury, MMR
Sz versus Sz-Affective? (1)	(none)	1	Schizoaffective Disorder, bipolar type; marijuana abuse
Schizoaffective Disorder (8)	(none)	2	Schizoaffective disorder, marijuana abuse
	(none)	2	Psychosis NOS, secondary to drug abuse
	(none)	1	Psychosis NOS; drug abuse; head injury
	Bipolar type	1	Schizoaffective disorder; drug abuse; head injury
	Polysubstance abuse	1	Schizophreniform disorder; marijuana abuse
	Alcohol abuse	1	Psychosis NOS, with Sz-Aff traits; alcohol encephalopathy
Other (11) (Various, including Bipolar disorder, mood disorder, Alcohol psychosis, Psychosis NOS, Polysubstance abuse with ASPD)			Alcohol-induced psychosis (2); Psychosis due to cocaine (1) Bipolar Disorder & substance abuse (3); ASPD & substance abuse (1); Psychosis NOS, ? Sz. Substance abuse (1); Organic Brain Syndrome, ASPD, Substance abuse (1); Depression psychopathy, substance abuse (1); Frontal lobe damage 8 marijuana abuse (1)
Total (39)			

Note: In this group patients had early histories that pointed to pre-existing mental illness, or else had a strong family history of psychotic disorders. Their subsequent drug-abuse appeared to aggravate an underlying or incipient psychosis.

Table 5: Psychoses that were considered Drug-Aggravated

There remained, in addition to the three groups mentioned above, ten patients who abused drugs, but for whom information about their early background or about the specifics of their drug habits was not sufficient to place them in either the "drug-induced" or the "drugaggravated" categories.

Patterns of violence

The offenses that led to the arrest and hospitalization of the patients, all of whom had earlier taken a plea of "not guilty by reason of mental disease or defect," were almost invariably of a violent nature. Murder, attempted murder, and assault accounted, collectively, for 67 of 98 offenses in the three main groups (no-drug, drug-induced, drug-exaggerated). The frequencies for the different types of crime were fairly comparable across the groups, apart from the larger number of murders and attempted murders in the "no-drug" patients than in the "drug-induced" patients (Fisher's Exact Test, two-tailed: P = 0.0033). The findings are summarized in Table 6

Type of Offense	No Drug Abuse	Drug- Induced	Drug- Exaggerated
Murder/Attempted Murder	19	9	20
Assault	3	9	7
Rape	1	2	2
Arson	2	4	4
Other Violent Crimes (viz., kidnap, robbery)	1	4	3
Total:Violent Crimes	26	28	36
Non-Violent Crimes	4	1	3

% Violent Crimes	26/30 (87%)	28/29 (97%0)	36/39 (92%)
------------------	-------------	-----------------	-------------

Table 6: Patterns of Violence. Violent and Non-Violent Offenses in the Three Major Groups

Discussion

The current study was carried out in the hope of shedding some light on the thorny issue of the relationship between marijuana use and psychosis. Of the 109 forensic patients studied, the effect of drug-abuse upon their mental state was vague in 9 cases, and not such as could be fitted into the three major categories: drugs-not-a-factor, drug-induced, or drug-aggravated. Of the 100 remaining, the figures were 30, 31 and 39 respectively; that is, close to a third in each group – though there were more in the drug-aggravated group.

As would be expected in a forensic hospital population, the proportion of the patients with a significant drug-problem was considerably greater (at least 70%) than what one would anticipate in the general population. Nora Volkow, the director of the National Institute for Drug Abuse [NIDA] has shown that 12% of people aged twelve or older used marijuana in the prior year [21]. In the present study 9 of the patients had used marijuana starting at ages seven to eleven; the majority (71) had begun using marijuana in the ages 12 through 15; only four stated that their first exposure came at age 20 or more. Most of the patients came from poor or working-class background; ethnicity was Caucasian in 42%, African-American in 40%, Hispanic in 13%, the remaining 5% - Asian. They did not constitute a "representative sample" of the general population, but because drug abuse was so marked among them – some meaningful data arise from the emerging statistics.

In the present series, marijuana was almost always the first illicit drug to be used. There were five patients who acknowledged using marijuana and cocaine in the same adolescent year. There were three who used cocaine shortly before they first tried marijuana, but only one patient who used cocaine but never used marijuana. These findings may help resolve the issue concerning marijuana as a "gateway drug" to the use of other drugs Boffey [22], in a recent editorial to the New York Times allowed as how early and heavy cannabis use may be associated with a subsequent drop in IQ, but disagreed with the idea that it can serve as a gateway drug. I suspect his impression was based on samples drawn from the general population of teenagers, the majority of whom come from better socioeconomic backgrounds than the Mid-Hudson patients, and who have a much lower percentage of significant psychiatric disorders. But in our forensic population (as may be true in economically disadvantaged young persons in general) marijuana did appear indeed as a gateway to other and to harder drugs. Many of our patients, for example, grew up in a milieu where many of their age-mates (or somewhat older adolescents) were using a multiplicity of drugs: Ecstasy, Angel-Dust, amphetamines, opiates, "shrooms" (psilocybin mushroom with hallucinogenic properties), LSD - in addition to marijuana and alcohol - with the result that there was a strong temptation to "supplement" one's use of marijuana (in its various forms, including hashish, ganja, etc) with drugs of a different sort. Twenty-seven of our patients had tried four to eight different drugs (including glue-sniffing, barbiturates, and methamphetamine); the majority had tried three - the most common combination being marijuana, alcohol and cocaine. Recently Drs. Eric and Denise Kandel [23] have reported on research showing that almost nine out of ten cocaine users had smoked cigarettes before trying cocaine, whereas only 3.4% had used cocaine before they began to smoke cigarettes. From their studies in mice, those primed with nicotine first showed enhanced locomotor sensitization, compared with those exposed to cocaine alone. Their view was that smoking cigarettes may well potentiate the use of another dangerous drug (cocaine), such that the gateway model and common liability model are complementary. Their study focused on nicotine rather than cannabis, but did lend support to certain compounds serving as gateways to use of other drugs that can exert deleterious effects on the brain. The patients in our series may best be understood as a sub-group of persons, aged 18 and younger when first using the drug, in whom cannabis did serve as a gateway to often "stronger" drugs. Our patients also had often been exposed to another risk factor - in the form of child abuse (physical, sexual, verbal) - which can predispose to early drug abuse, the two factors then acting synergistically to both psychiatric illness and to violent behavior - of the sort that eventually led to their being remanded to a forensic psychiatric facility [24,25].

In the study there were 31 patients whose psychosis was, on reexamination, considered drug-induced. The original diagnosis was usually "schizophrenia" or "parnanoid schizophrenia" (19 cases). Six had been diagnosed "schizoaffective" on admission; the remainder: "bipolar" (2), "depressive psychosis" (1), "delusional disorder" (1), "schizophreniform psychosis" (1), and "drug-induced psychosis secondary to marijuana" (1).

Typical of this drug-induced group was a male patient who started using marijuana at 14, progressing to seven "joints" a day. He became paranoid, and while living in a group-home, began to suspect his roommate was trying to poison him. He attacked the man, enucleating his left eye. There was no family history of mental illness. He was athletic in school and did not show signs of emotional disturbance prior to his use of marijuana. After he was remanded to the forensic

hospital (where he was diagnosed "paranoid schizophrenia" initially), he no longer had access to marijuana or other psychotomimetic substances. He was treated with antipsychotic medication, and rapidly regained rational thought, and no longer shows signs or symptoms of a schizophrenic (or other) psychosis.

The drug-aggravated group consisted of patients who either showed signs of mental disturbance before they began to abuse marijuana or other drugs – or who had close relatives with an established psychotic condition. In some cases both these attributes were present.

An example is that of a man who at 19 had stabbed his brother in an attempt to kill him (the brother survived). He had begun to abuse marijuana heavily since age fourteen. He used no other drugs apart from occasional alcohol. He had become progressively more paranoid several months before the assault, and heard voices urging him to kill his brother – as though his brother were the "Devil." After his arrest, he was remanded to the forensic hospital, where he was diagnosed with "paranoid schizophrenia." He came from a well-to-do family in which his paternal grandfather and two maternal aunts had been treated for bipolar manic-depression. The patient was treated with valproic acid [Depakote^{*}] to which he responded well, becoming stable, rational, and free of psychotic symptoms. He was re-diagnosed with a bipolar disorder that had been aggravated by early drug abuse (cannabis). The original diagnosis of paranoid schizophrenia was apparently predicated upon his "positive signs": auditory hallucinations and delusory ideation. He had not shown the negative signs of schizophrenia. Another example is that of a man who at thirty stabbed a woman to death, believing she was the "AntiChrist." At twelve he had tried to kill his step-mother. That was shortly after he had begun to use marijuana, to which other drugs (cocaine and psilocybin) were added when he was fifteen. Even after being sent to the forensic hospital and treated with antipsychotic medications, he still harbored religious delusions, such that his original diagnosis of paranoid schizophrenia still appeared valid. He has several first-degree relatives - some with schizophrenia; others with bipolar disorder.

From the diagnoses given by the first evaluators of our patients, it is clear that the term schizophrenia had become a "rubber stamp" applied to almost the entire group. In retrospect it appears that the presence of "positive signs" - delusions, especially of a paranoid sort, and hallucinations (whether auditory, visual, or both) - clinched the diagnosis for those evaluators. Very few of the patients showed signs of a formal thought disorder of the more bizarre type, suggestive of a primarily cognitive psychosis for which schizophrenia would indeed be the most compelling diagnosis. By bizarre, I refer to comments like: "There's a radio in my back tooth that's broadcasting that I'm a faggot," or "The guy in the TV is pulling all the thoughts out of my head." Whereas the majority of patients (perhaps 90%) diagnosed as schizophrenic in the past showed many of these more bizarre peculiarities, had the negative signs as well, and in many instances, close relatives with schizophrenic conditions, and could thus be considered genuine examples of the disorder, this is no longer the case. Beginning around 1962 marijuana use - and abuse - became much more common (very few adolescents had tried it in the pre-1962 years; if they abused a drug at all, it was usually alcohol) [26]. What had hitherto seemed largely a unitary, genetic-based, psychosis, schizophrenia has more and more come to be recognized as a syndrome made up of the "classic" (genetic-based) cases, but admixed not just with the rare brain tumors and endocrine abnormalities that sometimes led to a schizophrenia-like condition; instead, admixed with increasingly large numbers of drug-induced cases [27]. Marijuana is

J Child Adolesc Behav, an open access journal ISSN: 2375-4494

high on the list of these drug induced schizophrenias – that might be better called schizophreniform psychoses. Many of the drug-induced psychoses in the present study were of this sort: most mimicked classic schizophrenia; a few resembled delusional disorder or bipolar disorder. In some instances, however, the marijuana (with or without concomitant use of other drugs) may have unleashed a true schizophrenia earlier than it would have manifested itself, had not the adolescent abused the drugs [28,29]. Since drug abuse usually begins around the time of puberty, it is not easy to determine whether a particular adolescent drug-abuser who appears schizophrenic at say, sixteen, was destined to have shown signs of the classic psychosis at twenty, but for the early drug abuse. Deterioration in cognitive function, even short of psychosis, was common in adolescents abusing cannabis before fifteen [30].

Attention has been drawn to the heightened risk for psychosis following cannabis use - in persons with first-degree relatives who suffer from psychotic disorders [31]. In patients already considered psychosis-prone - or psychotic prior to first use of cannabis (as in our drug-aggravated group), cannabis may have a dual effect. The initial effect may be mood-enhancing. But this may be followed shortly by psychosis-inducing effects (viz., increased levels of hallucinatory experiences) [32]. This was often noted in our drug-aggravated group: patients mentioned that they enjoyed smoking marijuana because it made them feel "mellow," but the next day or so - they began to feel paranoid (and in that state, committed the violent offense that led to their arrest and forensic hospitalization). The hypothesis concerning self-medication with cannabis in psychosis-prone persons becomes relevant here. In some adolescents, for example, their early signs of psychosis may - upon cannabis abuse a year or so later - provoke a more florid psychosis (either schizophreniform, or else a premature awakening of a genuine schizophrenia). But their already fragile mental state may have led to a craving for cannabis and its effects (the "mellowing" especially) [33]. This would constitute the "vicious circle" of pre-existing psychosis leading to craving for psychosis-inducing drugs - leading then to a worsening of the underlying psychosis [32]. As alluded to earlier, bipolar psychoses may also have their clinical onset advanced via cannabis abuse in adolescence, such that a bipolar disorder instead of becoming clinically recognizable in one's early 20s, is already apparent in mid-adolescence, thanks to the abuse of cannabis (and often - other drugs as well) [11].

In summary, the patients at the Mid-Hudson Forensic Psychiatric Hospital who accepted a plea of "not guilty by reason of mental disease or defect" constituted a sub-group in which the effects of prior abuse of marijuana and other drugs was facilitated owing to (a) the high proportion (70%) of patients who had used such drugs, usually before age 18, and (b) the length of stay. Because the latter varied between one year at the least – to several decades – there was ample time to assess the evolution of the clinical picture over time, in those who had a history of drug abuse.

The drug-induced group had a history of heavy drug abuse during mid-adolescence or earlier, but no family history of psychosis and no clear signs of mental illness prior to their first experience with marijuana (which was almost always the first drug tried). Their psychosis had cleared up either in a jail or in another hospital. In the drug-aggravated group either the psychosis persisted many months or even years past the cessation of drug-use, and/or there had been a strong family history of psychosis, along with some signs of mental illness prior to the onset of drug abuse. In the two-thirds of the entire patient-group in whom drug-abuse had been a factor, marijuana had almost always been the first drug tried, but almost never the only drug eventually abused. This lent support to the hypothesis of marijuana as a "gateway" drug in this population – partly because the disturbing emotions experienced during adolescence fostered experimentation with several drugs as attempts at "self-medication"; partly because many of the patients grew up, while adolescents, in a milieu that encouraged the use of a wide variety of mind-altering drugs.

Several limitations affected the assessment of the different subgroups. Some of the patients were poor historians vis-à-vis the ages at which they had begun to use various drugs, or the age when the use of a particular drug, such as marijuana, could be considered "heavy." Some patients had grown up in families with an absent or otherwise unknown father, such that thorough background information regarding family history of mental illness was not obtainable. In about a tenth of the group there had been a history of marijuana or other drug abuse - but the abuse appeared to be quite moderate and not linked closely in time to the offense that had precipitated their original arrest and confinement in a psychiatric facility. These patients constituted an "uncertain" sub-group that could not be placed accurately either in the drug-induced or aggravated subgroups. In the main, however, abuse of marijuana and other drugs played an important role in the predisposition to violent and other offenses in this group of forensic patients.

The present study, though based on a forensic population, does point to the potential dangers of marijuana in those who begin to use it in adolescence. This has important public health implications. Because the major psychoses (schizophrenia and bipolar disorder) tend not to manifest themselves till late adolescence or the early 20s, it will not be easy to forecast which adolescents are the ones with genetic vulnerability to the psychoses, and who, if we had such foreknowledge, should be warned strongly against the use of marijuana (and to the other "street-drugs" which young marijuana-users are prone to indulge in). But these adolescents constitute the at-risk subgroup of persons particularly likely to develop the more serious side-effects, such as the "amotivational syndrome," or tendencies to paranoid ideation and to violence - as were so common in those whose behaviors led to their committing the acts that had forensic consequences. In contrast to alcoholic beverages, whose production is easier to standardize and whose consumption is easier to control by appropriate legally-imposed age limits, marijuana is easier to grow in ways less readily controlled by law. Yet it is now becoming apparent that marijuana-use (especially if heavy) in adolescents is associated with lower volume in the orbitofrontal cortex (which is not fully myelinated until the early 20s) a region associated with social decision-making [34]. The present study also suggests that very heavy use of the currently high-THC contentmarijuana, even by itself, can precipitate a schizophrenia-like syndrome in genetically non-vulnerable adolescents, or can lead to a genetic psychosis cropping up years earlier and more severe in nature than would have been the case, absent the abuse of marijuana [35,36]. It is beyond the scope of this paper to suggest how medical and governmental authorities can make the public more aware of the potential dangers of the drug in persons under age 18. Further studies are now needed, in order to assess which sub-populations of young persons might be especially vulnerable to the socially undesirable, and at times, dangerous effects of early use of marijuana.

References

 Curtis L, Rey-Bellet P, Merlo MC (2006) [Cannabis and psychosis]. Rev Med Suisse 2: 2099-2100.

- Degenhardt L, Coffey C, Carlin JB, Swift W, Moore E, et al. (2010) Outcomes of occasional cannabis use in adolescence: 10-year follow-up study in Victoria, Australia. Br J Psychiatry 196: 290-295.
- 3. Vollenweider FX, Geyer MA (2001) A systems model of altered consciousness: integrating natural and drug-induced psychoses. Brain Res Bull 56: 495-507.
- 4. Konings M, Henquet C, Maharajh HD, Hutchinson G, Van Os J (2008) Early exposure to cannabis and risk for psychosis in young adolescents in Trinidad. Acta Psychiatr Scand 118: 209-213.
- Rubino T, Zamberletti E, Parolaro D (2012) Adolescent exposure to cannabis as a risk factor for psychiatric disorders. J Psychopharmacol 26: 177-188.
- Large M, Sharma S, Compton MT, Slade T, Nielssen O (2011) Cannabis use and earlier onset of psychosis: a systematic meta-analysis. Arch Gen Psychiatry 68: 555-561.
- Houston JE, Murphy J, Shevlin M, Adamson G (2011) Cannabis use and psychosis: re-visiting the role of childhood trauma. Psychol Med 41: 2339-2348.
- 8. Schimmelmann BG, Conus P, Cotton SM, Kupferschmid S, McGorry PD, et al. (2011) Prevalence and impact of cannabis use disorders in adolescents with early onset first episode psychosis. Eur Psychiatry.
- 9. Schimmelmann BG, Conus P, Cotton SM, Kupferschmid S, Karow A, et al. (2011) Cannabis use disorder and age at onset of psychosis--a study in first-episode patients. Schizophr Res 129: 52-56.
- 10. Caton CLM (2011) The need for close monitoring of early psychosis and co-occurring substance misuse. The Psychiatrist 35: 241-243.
- Lagerberg TV, Sundet K, Aminoff SR, Berg AO, Ringen PA, et al. (2011) Excessive cannabis use is associated with earlier age at onset in bipolar disorder. Eur Arch Psychiatry Clin Neurosci 261: 397-405.
- 12. Robayo L (2011) Genetically modified marijuana problem growing in Colombia.
- Morgan CJ, Gardener C, Schafer G, Swan S, Demarchi C1, et al. (2012) Sub-chronic impact of cannabinoids in street cannabis on cognition, psychotic-like symptoms and psychological well-being. Psychol Med 42: 391-400.
- 14. Hall W and Degenhardt L (2006) Is there a specific "cannabis psychosis"? In D Castle & R Murray (Eds.): Marijuana and Madness. New York: Cambridge Univ Press pp: 101-118.
- 15. Arseneault L, Cannon M, Witton J and Murray R (2006) Cannabis as a potential causal factor in schizophrenia. In D Castle & R Murray (Eds.), Marijuana and Madness. New York: Cambridge Univ Press pp: 101-118.
- Linzen D, Peters B and de Haan L (2006) Cannabis use and the course of schizophrenia. In D Castle & R Murray (Eds.): Marijuana and Madness. New York: Cambridge Univ Press pp: 119-126.
- 17. Sara GE, Burgess PM, Malhi GS, Whiteford HA, Hall WC (2014) The impact of cannabis and stimulant disorders on diagnostic stability in psychosis. J Clin Psychiatry 75: 349-356.
- 18. Matthysse SW, Kidd KK (1976) Estimating the genetic contribution to schizophrenia. Am J Psychiatry 133: 185-191.

 Leff J, Vaughn C (1980) The interaction of life events and relatives' expressed emotion in schizophrenia and depressive neurosis. Br J Psychiatry 136: 146-153.

Page 8 of 8

- Rosenthal D (1975) The spectrum concept in schizophrenic and manicdepressive disorders. In Freedman DX (Edn.), Biology of the Major Psychoses. New York: Raven Press pp: 19-25.
- 21. Watts V (2014) Psychiatric News.
- 22. Boffey P (2014) What science says about marijuana. New York Times: July 31, editorial.
- 23. Levin A (2014) Evidence backs gateway hypothesis in drug addiction. Psychiatric News, November 7th.
- 24. Konings M, Stefanis N, Kuepper R, de Graaf R, Have MT, et al. (2011) Replication of two independent population-based samples that childhood maltreatment and cannabis use synergistically impact on psychosis risk. Psychol Med 16: 1-11.
- 25. Hinch B (2014) Adult implication of childhood maltreatment. Psychiatric Times August pp: 10-12.
- 26. Stone MH (1990) The Fate of Borderlines. New York: Guilford Press
- 27. Paparelli A, Di Forti M, Morrison PD, Murray RM (2011) Drug-induced psychosis: how to avoid star gazing in schizophrenia research by looking at more obvious sources of light. Front Behav Neurosci 5: 1.
- Compton MT, Broussard B, Ramsay CE, Stewart T (2011) Pre-illness cannabis use and the early course of nonaffective psychotic disorders: associations with premorbid functioning, the prodrome, and mode of onset of psychosis. Schizophr Res 126: 71-76.
- Foti DJ, Kotov R, Guey LT, Bromet EJ (2010) Cannabis use and the course of schizophrenia: 10-year follow-up after first hospitalization. Am J Psychiatry 167: 987-993.
- Fontes MA, Bolla KI, Cunha PJ, Almeida PP, Jungerman F, et al. (2011) Cannabis use before age 15 and subsequent executive functioning. Br J Psychiatry 198: 442-447.
- Martinotti G, Di lorio G, Tedeschi D, De Berardis D, Niolu C, et al. (2011) Prevalence and intensity of basic symptoms among cannabis users: an observational study. Am J Drug Alcohol Abuse 37: 111-116.
- Henquet C, van Os J, Kuepper R, Delespaul P, Smits M, et al. (2010) Psychosis reactivity to cannabis use in daily life: an experience sampling study. Br J Psychiatry 196: 447-453.
- 33. Kuepper R, Oorschot M, Myin-Germeys I, Smits M, van Os J, et al. (2013) Is psychotic disorder associated with increased levels of craving for cannabis? An Experience Sampling study. Acta Psychiatr Scand 128: 448-456.
- 34. Tedesco L (2014) How marijuana really affects the brain.
- Cohen M, Solowij N, Carr V (2008) Cannabis, cannabinoids and schizophrenia: integration of the evidence. Aust N Z J Psychiatry 42: 357-368.
- DeLisi LE (2008) The effect of cannabis on the brain: can it cause brain anomalies that lead to increased risk for schizophrenia? Curr Opin Psychiatry 21: 140-150.