Published in final edited form as:

Cogn Neuropsychiatry. 2011 March ; 16(2): 158–173. doi:10.1080/13546805.2010.524494.

Specificity of verbal learning impairment and recovery in a marijuana-dependent male: the effects of sustained marijuana abstinence

Nehal P. Vadhan, Wilfred G. van Gorp, and Frances R. Levin

Columbia University and the New York State Psychiatric Institute 1051 Riverside Drive New York, NY 10032

Abstract

We present the case of a young adult in treatment for marijuana dependence, with recurrent depression and a history of possible TBI, complaining of concentration, memory and initiation problems. Testing at treatment baseline revealed performance that was generally in the High Average range on measures of reaction time and attention, with a selective impairment in verbal learning (Borderline to Extremely Low range). Following 8 weeks of abstinence from marijuana, his verbal learning recovered to expected levels (High Average range), with signs of improved learning strategy, efficiency, rate, and capacity. However, his reaction time and attention showed minimal evidence of change. This finding is consistent with the literature that demonstrates that marijuana-associated neurocognitive impairments may be specific to verbal learning and may remit with abstinence. The clinical implications of these case findings and recommendations for neuropsychological assessment of marijuana abusers are discussed.

Introduction

An extensive body of empirical literature indicates that regular marijuana smokers exhibit mild-to-moderate impairment on tests of verbal memory (Grant et al., 2003; Pope et al., 1995). However, the clinical neuropsychology literature is limited with respect to the specific components of verbal learning and memory that may be impacted by marijuana use. We present the case of a young adult in treatment for marijuana dependence, complaining of depression, and concentration, memory and initiation problems. He was assessed with clinical measures of verbal memory, attention and reaction time during a period of regular marijuana smoking and again following extended abstinence from marijuana. Some personal and historical details, including his initials, have been changed or omitted to protect his confidentiality.

Identifying Information

The patient (MF) is a single male in his mid-thirties, who smoked marijuana daily and was enrolled in a clinical trial of treatment for marijuana dependence with comorbid depression. He was a full time graduate student and worked part-time. He had never been in substance abuse treatment before, but sought professional assistance because of the accumulation of negative psychosocial and emotional consequences from long-term marijuana use (e.g., “It’s affecting my personal and social life”, “It’s getting to me psychologically – anger and mood swings” and “My memory is bad”).

History

MF began smoking marijuana and drinking alcohol at age 16, with alcohol use peaking at age 17 (3 days/wk; ¼ bottle liquor/occasion) and marijuana use peaking at age 19 (>10 “blunts”¹ daily). Two notable consequences of his substance abuse at this time were a motorcycle accident with loss of consciousness, while intoxicated on marijuana, and an arrest for public intoxication and fighting. Two periods of lengthy substance abstinence were reported, the first at age 21 (15 months), and the second within the past year (6 months), with only infrequent alcohol use. At intake, MF reported that he had recently relapsed and been smoking 1-2 blunts nightly, with his alcohol use remaining recreational.

MF also reported a history of recurrent depression, with two past episodes each lasting a year or longer, and the current episode in its fourth month at intake. Two of these episodes, including the current one, occurred in the context of marijuana use and were triggered by relationship breakups. MF's current episode consisted of daily depressed mood, anhedonia, decreased appetite, psychomotor agitation, impaired concentration, and irritability, as well as anxiety. His depression was of a moderate-to-severe level at intake (Hamilton Depression Scale [HAM-D] = 20; Beck Depression Inventory - Second Edition [BDI-II] = 39; Hamilton Anxiety Scale [HAM-A] = 16 (Beck et al., 1996; Hamilton, 1959, 1960). He attributed his depressive symptoms to marijuana smoking, stating that while marijuana intoxication provided him acute relief from dysphoria, its overall effect was to decrease his mood.

The patient's recollection of his motorcycle accident was somewhat vague, and should be interpreted with caution. He reported that at age 20, while intoxicated from marijuana, he crashed his speeding motorcycle after hitting a pothole in the road. The back of his head struck the pavement, and despite wearing a helmet, he lost consciousness for 5-10 min. He was treated for an inverted vertebra, bruised ribs, and some peripheral nerve damage, but denied experiencing any traumatic brain injury (TBI) or requiring inpatient rehabilitation. However, he became significantly depressed and remained so after physical recovery, even leaving his job because of apathy. He also reported that his concentration became much poorer following the accident, despite a long abstinence from marijuana, but denied other cognitive changes.

Psychiatric Diagnoses and Cognitive Complaints

Axis I psychiatric diagnoses were derived at intake from the Structured Clinical Interview for the DSM-IV (SCID-I; First et al., 1995) as well as an independent psychiatric evaluation. MF received diagnoses of current Marijuana Dependence (severe) and Major Depressive Disorder (recurrent)², and lifetime Alcohol Dependence. Physical examination revealed a healthy individual, including no abnormal findings on basic neurological evaluation or laboratory tests.

At intake, MF reported cognitive problems in the domains of attention, learning and memory (e.g., “I have weak concentration and memory” and “trouble remembering things”), which he attributed to regular marijuana use. For example, he reported often being distracted in class and during conversation, and having to ask people to repeat things. He also reported difficulty remembering material while studying without extensive repetition. In addition, he stated that his initiation and time management were impaired. For example, he had difficulty getting started on projects and assignments, and completing them efficiently after starting

¹A “blunt” is a cigar filled with marijuana, generally equivalent to three typical marijuana cigarettes or “joints.”

²The patient and clinical staff judged the patient's current depressive episode to be marijuana-related. However, marijuana is not recognized by DSM-IV as a substance that can induce depression; hence the primary mood disorder diagnosis.

them. He reported attending the gym inconsistently, despite being somewhat preoccupied with his appearance and physical health.

The current case study reports on MF's longitudinal neuropsychological functioning, with the baseline evaluation conducted during a period of heavy marijuana smoking, and the follow-up evaluation following extended abstinence from marijuana.

Treatment protocol

The treatment protocol consisted of pharmacological treatment (Venflaxine/placebo) and cognitive-behavioral psychotherapy (Relapse Prevention Therapy; Carroll, 1992). Although medication assignment was initially double-blind, MF's blind was broken 5 weeks into treatment for psychiatric reasons (see below). This revealed that he had been in the placebo group, and had not been receiving active pharmacological treatment.

Behavioral observations

MF was seen for weekly psychotherapy sessions by the first author. He was typically well-dressed, appeared his age, and was always oriented to person, place and time. The patient was polite and personable, and rapport was achieved quickly. He was engaged in therapy, supported by his perfect attendance and infrequent lateness, and seemed particularly motivated to achieve marijuana abstinence. Notably, he was able to identify actual and hypothetical situations (e.g., parties) that would imperil his goal to become and stay marijuana-free, and was often able to produce adaptive strategies to address these situations.

However, MF initially exhibited weak concentration and memory, as he often needed statements and questions to be repeated or written down, and he often forgot to complete or bring in his therapy "homework" assignments. He also had a tendency towards circumstantial speech, and exhibited cognitive distortions such as "all or nothing" thinking.

Findings

Figure 1 shows the co-occurrence of marijuana use frequency (left Y-axis), assessed by the timeline follow-back method (Sobell & Sobell, 1992), and depressive symptoms (right Y-axis), assessed by the HAM-D, for 12 weeks of clinical contact. At the time of baseline testing (week 0), both his marijuana use frequency and depressive severity were still elevated, though decreased from intake (see Fig 1).

The testing battery consisted of the computerized California Computerized Assessment Package (CalCAP; Miller et al., 1991) and the examiner -administered California Verbal Learning Test - Second Edition (CVLT-II; Delis et al., 1987), which was given by a trained research assistant. The battery was designed to be sensitive to neurocognitive impairments related to marijuana use, but was brief and fixed due to the research nature of the testing. The examiner reported that MF was engaged in testing and appeared to put forth full effort during both sessions. Results from both testing sessions are presented in Table 1.

Baseline testing

As seen in the upper left side of Table 1, MF's baseline CalCAP scores indicated strong psychomotor functioning in terms of reaction time and attention. His basic and complex reaction time (Simple, Choice and Sequential Reaction Time) ranged from the High Average to Superior range. His selective attention (Visual Selective Attention) measured in the Superior range, and when elements of cognitive flexibility and visual scanning were added, he performed in the High Average range (Response Reversal/Visual Scanning). His visual

perception (Form Discrimination) was also in the High Average range. The one exception to this profile was his attention to lexical categories (Lexical Discrimination), which fell in the Average range, and was considered weak relative to the rest of his abilities. Thus, his attention and reaction time at baseline were generally strong and commensurate with his level of education.

However, his performance on the CVLT-II was noticeably weaker than on the CalCAP (Table 1, lower left side). His overall learning of the list (Trials 1-5) was in the Borderline range, with initial learning in the Extremely Low range (Trial 1, List B), and learning after five trials only in the Low Average range (Trial 5). Additionally, the consistency of words remembered was poor (Consistency), implying a lack of strategy to his learning. Correspondingly, his recall remained in the Low Average range following a 20-min delay (Long Delay Free Recall), with application of cues aiding only marginally (Long Delay Cued Recall), and he was no better at recognizing the words he had been presented (Recognition Hits). He also made a considerable number of intrusions during the test. This performance was considerably weaker than would be expected for a man of his education and performance in other neurocognitive domains, but is consistent with his complaints of memory problems.

Although MF was a daily marijuana smoker at intake, he achieved marijuana abstinence by week 4 and maintained it throughout treatment, with his use and nonuse confirmed by urine toxicology tests. As such, he had been abstinent for 8 weeks by follow-up testing. Figure 1 shows that MF's depression ratings decreased from a high of 32 (severe) in week -1 to a low of 5 by week 5 (WNL). Formal mood ratings were not taken after this point since he entered open treatment, but his self-report and clinical observations indicated that was largely free of depressive symptoms up to and including follow-up testing. Further, it can be seen from Figure 1 that MF's depressive symptoms began to decrease one week after his marijuana use began to decrease, reflecting the substance-associated nature of his depression. HAM-A anxiety ratings were taken at weeks -1 and 3 (not shown) and exhibited a mild decrease as well (16→12).

Follow-up testing

MF exhibited a similar pattern of performance on the CalCAP at follow-up, compared to baseline (Table 1, upper right side). His scores again ranged from the High Average to Superior range, and a good degree of test-retest consistency was seen across subtests. However, his primary scores on the CVLT-II (Table 1, lower right side) were now significantly stronger than baseline, generally ranging from Low Average to High Average. Initial learning was still low (Trial 1, List B), but he improved his learning by the 5th trial to the High Average range, increasing his total learning (Trials 1-5) from the Borderline range to the Average range. His rate (Slope) and consistency of learning also improved from baseline. Consequently, his delayed recall was in the High Average range, and his recognition was perfect. He made fewer intrusions than baseline, although his repetitions did increase. In general, though, his performance improved markedly from baseline. Importantly, the increases seen on the CVLT-II indices far exceeded the average test-retest improvement of healthy adults on this test (Woods et al., 2006), suggesting an improvement in learning function over-and-above a potential practice effect.

Finally, his scores on CalCAP simple reaction time, which was assessed three times during each CalCAP administration, showed mild increases in each session. This suggests that cognitive fatigue or performance amotivation were not factors during either testing session. After the follow-up evaluation, the patient was given feedback on his evaluations, and the relationship of marijuana abstinence to improved neurocognitive function was emphasized.

The patient reported greater ease in learning academic material, consistent with his performance improvement, and indicated that the results provided him with further motivation to remain abstinent.

Hypomania

In week 5 of treatment, MF presented to the clinic with abnormally bright affect, pressured and tangential speech, and reported irritability. He reported sudden increases in energy and goal-directed activity, such as involvement with his apartment building association, and initiation of home-improvement projects. He also reported increased sexual dreaming and activity, including a sexual encounter with an ex-girlfriend that he met by chance. This syndrome only lasted for several days, and thus he did not require hospitalization, but a similar episode did reoccur several weeks later. Aside from a lack of impaired sleep, these episodes resembled hypomania, although he had denied any history of such symptoms. Medication side effects were ruled out after he was found to have been in the placebo condition of the study, and he was moved into open treatment consisting only of psychotherapy and psychiatric monitoring. His insight was poor, only acknowledging that he had had an “edgy couple of weeks”, and he declined referrals for bipolar treatment. Signs of hypomania were not observed for the rest of treatment.

Discussion

This was a case presentation of a young adult who was in treatment for marijuana dependence and complaining of memory problems and depression. Long episodes of regular marijuana use, recurrent depression, and a potential TBI were noted in his history. Testing at treatment baseline revealed strong performance on tests of reaction time and attention (CalCAP) in the High Average range, with a selective performance impairment on a test of verbal memory (CVLT-II), with many scores in the Extremely Low to Low Average range. The patient succeeded in achieving marijuana abstinence early in treatment, and correspondingly, his verbal memory test scores increased after 10 weeks to the High Average range. However, his reaction time and attention test scores remained stable. Thus, aspects of his memory function recovered to expected levels when measured again after extended abstinence from marijuana.

It is well-documented that regular marijuana users' performance on tests of verbal memory can be impaired, relative to healthy controls, even when they are not intoxicated (e.g., Pope et al., 2001; Solowij & Battisti, 2008). Further, the level of impairment may be related to the recency and amount of marijuana last smoked (Pope et al., 2001), as well as the lifetime duration of marijuana smoking (Solowij et al., 2002). The current findings highlight the potential specificity of marijuana-related cognitive impairment, since verbal learning was impaired, but attention and reaction time were unaffected. This is consistent with a meta-analysis of 15 neurocognitive studies (Grant et al., 2003), which found evidence for learning and memory impairment in marijuana smokers (but not other domains), albeit to a mild degree. Likewise, MF's recovery of verbal learning function during extended abstinence is consistent with a well-controlled longitudinal study of neurocognitive function in young daily marijuana smokers (Pope et al., 2001). Finally, his baseline impairment and recovery are consistent with his self-report of how marijuana affects his learning and memory ability.

A closer analysis of MF's CVLT-II recall data reveals several interesting patterns about the components of verbal memory that were affected by marijuana abstinence. Although his CVLT-II scores were in impaired ranges at baseline, he was generally able to reproduce the same number of words during delayed recall as he did during Trial 5 learning (Table 1). This suggests that marijuana use impacted his ability to learn new information, but not

necessarily his ability to recall, at a later time, what information he had learned. His trial-by-trial learning on both evaluations is shown in Figure 2.

This data show that his learning slope increased from baseline to follow-up. For example, at baseline, he produced 2 words on Trial 1 and 9 words on Trial 5, an increase of 7 words. However, at follow-up, he increased his number of words from Trial 1 to Trial 5 by 10 words. This indicates an improvement not only in total words learned, which might simply reflect a practice effect, but in learning rate, which is evidence of improved learning function. Also, the typical healthy adult has been found to improve his/her total learning on the CVLT-II by only about 8 words after a 4-week retest (Woods et al., 2006). MF improved by 20 words after a 10-week retest, and this occurred despite him continuing to smoke marijuana in the week after baseline testing. This further argues against a practice effect as the sole explanation for his improvement on the CVLT-II. Signs of executive dysfunction (deficiencies in word categorization, learning strategy and efficiency) that were apparent in his baseline CVLT-II performance were reduced in the follow-up exam. Thus, learning strategy, efficiency, capacity and rate were all improved as a function of marijuana abstinence, and recall/recognition scores correspondingly increased.

Regarding other potential contributing factors to his baseline verbal learning impairment, the most obvious risk factor was potential brain injury resulting from the impact to his head during the motorcycle accident. On the one hand, he reported that he was wearing a helmet at the time of the accident, had only lost consciousness for a short time and only required half a day of hospitalization. Further, a basic medical evaluation at treatment baseline (15 years later) indicated no gross neurological impairment, nor physical scarring on his head. Finally, neurocognitive testing with the CalCAP revealed generally strong attentional functioning in nonverbal domains. These observations suggest minimal long-term neuropsychological consequences of his accident.

On the other hand, he reported that he had experienced problems with concentration following the accident, and although no formal measures were taken at that time, subtle signs of attentional impairment on the CVLT-III were seen on the current evaluation. This consisted of his performance on Trial 1 and List B, which remained impaired at follow-up. Combined with his relative and stable weakness on the CalCAP Lexical Discrimination subtest, this may be a residual sign of an accident-related attentional impairment that is verbally based. The accident also resulted in his first serious depressive episode, and therefore may have triggered his recurrent mood disorder.

So the evidence with regard to TBI is mixed, although the possibility that he suffered a TBI with significant and long-lasting neuropsychiatric consequences cannot be discounted. Another classic risk factor was his period of alcohol dependence, yet the reported frequency, amount and duration of his past drinking seems too low to have resulted in lasting neurocognitive impairment. Finally, his learning deficit at baseline was too circumscribed, and improved too rapidly, to be primarily the result of either past TBI or alcohol dependence. This finding also makes clear that differentiating between TBI- and substance abuse-related neurocognitive effects can be difficult (e.g., Iverson et al., 2005; Lange et al., 2008) without repeated evaluation during extended abstinence.

The interaction between his marijuana use, depression and learning impairment is also difficult to disentangle, since MF's depressive symptoms reduced on a similar time course as his marijuana use frequency. In another case study (Payne, 2000) of a young adult male marijuana smoker (RB) who was also educated and intelligent (NART estimated IQ = 118-125), depression was concluded to be a more prominent factor in the patient's neurocognitive impairment than either marijuana use or history of TBI. In that study, the

baseline neuropsychological evaluation was conducted two years after a verified right-frontal TBI, and after one month of reported abstinence from daily marijuana smoking.

This evaluation revealed mild-to-moderate depression (BDI = 20), impaired verbal learning (AMIPB³ Trials 1-5, <10th percentile) and decreased psychomotor speed (Digit-Symbol, 9th percentile, AMIPB Information Processing, 25th - 50th percentile). By 17 weeks, RB had resumed marijuana smoking, but his depressive symptoms had been successfully treated by cognitive-behavioral therapy. A follow-up evaluation at that time revealed marked increases in these functions (75th - 90th percentile), as well as intellectual functioning. In contrast to MF, RB's learning and psychomotor performance was impaired during a period of mild depression, but marijuana abstinence, and was significantly improved during a period of remission from depression, but daily marijuana smoking.

However, some differences exist between these two patients that may explain the seemingly disparate findings. The amount of marijuana that RB resumed smoking prior to follow-up examination was about half of the amount that MF reported during his baseline evaluation. Further, RB had been smoking marijuana for about 10 less years than MF, and was able to stop smoking marijuana on his own, which suggests a less severe dependence on marijuana than MF.

RB also exhibited weak psychomotor performance at baseline, which would be consistent with depression-related neurocognitive effects (Hill et al., 2004; Smith et al., 2006), whereas MF's psychomotor performance was quite strong. Recent empirical studies have shown minimal evidence for verbal learning deficits in depressed individuals who are young (Castaneda et al., 2008), including null findings in those who have recurrent depression (Smith et al., 2006; Wang et al., 2006) and are mildly-to-moderately depressed (Wang et al., 2006). However, depression may be associated with verbal learning impairment specifically for those patients in the post-acute phase (i.e., within 2 years) of recovery from TBI (Keiski et al., 2007), as was RB. In terms of verbal learning impairment, these clinical and research findings point towards a greater role for marijuana use for MF, and a greater role for depression for RB (Payne, 2000). Taken together, these two case studies point to the plasticity of neurocognitive functioning vis-à-vis state-related factors, and highlight the importance of measuring and weighing the relative severity of various neurocognitive risk factors.

An interesting characteristic of MF's mood disorder was the emergence of hypomania a month into marijuana abstinence, which has diagnostic and treatment implications. First, it is possible that marijuana acted as an anti-manic agent, masking the presence of bipolar disorder in MF. Second, in addition to decreasing his marijuana use and receiving psychotherapy, his mood improvement may have been related to the cyclical nature of his mood disorder. In any case, since MF had been free of hypomanic signs for several weeks or longer surrounding each neuropsychological evaluation, hypomania was not likely to have impacted test performance in the acute sense.

There are several limitations to this clinical case report, primarily due to the research context in which the data was collected. First, the neurocognitive battery was brief, fixed and mostly computerized. As such, several important domains of neurocognitive function (e.g., intellectual and executive function) and testing behavior were not assessed, or were assessed indirectly. Second, MF's baseline testing was not conducted at the peak of his marijuana use, perhaps limiting marijuana's neurocognitive impact. Third, no medical records were available concerning his motorcycle accident, so its neurological impact was unclear. For

³AMIPB = Adult Memory and Information Processing Battery (Coughlan & Hollows, 1985)

these reasons, it is difficult to get a full picture of marijuana's effects on MF's neurocognitive functioning, at least as far as his baseline assessment goes.

However, the research context also added a layer of strength to the evaluation process. It provided a framework for longitudinal study, where data on both marijuana use and depression were collected repeatedly via formal psychometric instruments, and reported marijuana use and nonuse were verified biologically. Multiple clinical observations of the patient by a number of clinicians and staff informed the case formulation considerably.

In conclusion, this case report demonstrated the specificity of verbal learning impairment in a regular marijuana smoker on a neuropsychological test, which appeared to be impacting his functioning in academic and social realms. Despite a history of classic neuropsychological risk factors like possible TBI and alcohol dependence, the patient's verbal learning function improved considerably upon extended abstinence from marijuana, highlighting the temporary nature of such impairments in these individuals. This case report also demonstrated the clinical utility of neuropsychological evaluations in primary marijuana abusers that complain of cognitive difficulty (see Miller, 1985, for comprehensive recommendations on clinical neuropsychological evaluation of substance abusers). The various pathways by which marijuana may disrupt neurocognitive function include intoxication (Vadhan et al., 2009), withdrawal (Haney, 2005) and impaired sleep (Schierenbeck et al., 2008), so careful assessment of frequency, amount and recency of marijuana smoking is required. If learning and memory deficits do not resolve with extended abstinence, other etiological factors should be considered. Finally, while cessation of marijuana smoking may lead to decreases in some psychiatric symptoms, it may lead to increases in others. Therefore, marijuana-dependent individuals with a psychiatric history may continue to require clinical monitoring even several weeks or months into abstinence.

Acknowledgments

This research was supported by NIDA grant R01 DA015451-05 to Dr. Levin.

The authors gratefully acknowledge Patrick Roebke and Eliezer Pickholtz for their assistance in preparation of this report.

References

- Beck, AT.; Steer, R.; Brown, G. BDI-II Beck Depression Inventory Manual. 2nd ed.. Harcourt Brace & Company; San Antonio, TX: 1996.
- Carroll, KM. Clinician's guide to cocaine addiction: Theory, research, and treatment.. In: Kosten, Thomas R.; Kleber, Herbert D., editors. Psychotherapy for cocaine abuse: Approaches, evidence, and conceptual models. 1992. (1992)
- Castaneda AE, Tuulio-Henriksson A, Marttunen M, Suvisaari J, Lönnqvist J. A review on cognitive impairments in depressive and anxiety disorders with a focus on young adults. *Journal of Affective Disorders*. 2008; 106(1-2):1–27. [PubMed: 17707915]
- Coughlan, AK.; Hollows, SE. The Adult Memory and Information Processing Battery: Test Manual. Psychology Department, St. James University Hospital; Leeds, U.K.: 1985.
- Delis, DC.; Kramer, JH.; Kaplan, E.; Ober, BA. California Verbal Learning Test--Adult Version. Psychological Corporation; San Antonio, TX: 1987.
- First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders-Patient Edition (SCID-I/P, version 2.0). Biometrics Research Department, New York State Psychiatric Institute; New York: 1995.
- Grant I, Gonzalez R, Carey CL, Natarajan L, Wolfson T. Non-acute (residual) neurocognitive effects of cannabis use: a meta-analytic study. *Journal of the International Neuropsychological Society*. 2003; 9(5):679–689. [PubMed: 12901774]

- Hamilton M. The assessment of anxiety states by rating. *British Journal of Medical Psychology*. 1959; 32(1):50–55. [PubMed: 13638508]
- Hamilton M. A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*. 1960; 23:56–61.
- Haney M. The marijuana withdrawal syndrome: diagnosis and treatment. *Current Psychiatry Reports*. 2005; 7(5):360–366. [PubMed: 16216154]
- Hill SK, Keshavan MS, Thase ME, Sweeney JA. Neuropsychological Dysfunction in Antipsychotic-Naive First-Episode Unipolar Psychotic Depression. *American Journal of Psychiatry*. 2004; 161(6):996–1003. [PubMed: 15169687]
- Iverson GL, Lange RT, Franzen MD. Effects of mild traumatic brain injury cannot be differentiated from substance abuse. *Brain Injury*. 2005; 19(1):11–18. [PubMed: 15762097]
- Keiski MA, Shore DL, Hamilton JM. The Role of Depression in Verbal Memory Following Traumatic Brain Injury. *The Clinical Neuropsychologist*. 2007; 21(5):744–761. [PubMed: 17676541]
- Lange RT, Iverson GL, Franzen MD. Comparability of neuropsychological test profiles in patients with chronic substance abuse and mild traumatic brain injury. *The Clinical Neuropsychologist*. 2008; 22(2):209–227. [PubMed: 17853134]
- Miller EN, Satz P, Visscher B. Computerized and conventional neuropsychological assessment of HIV-1-infected homosexual men.[see comment]. *Neurology*. 1991; 41(10):1608–1616. [PubMed: 1922803]
- Miller L. Neuropsychological assessment of substance abusers: review and recommendations. *Journal of Substance Abuse Treatment*. 1985; 2(1):5–17. [PubMed: 3900432]
- Payne HC. Traumatic brain injury, depression and cannabis use--assessing their effects on a cognitive performance. *Brain Injury*. 2000; 14(5):479–489. [PubMed: 10834343]
- Pope HG Jr, Gruber AJ, Hudson JI, Huestis MA, Yurgelun-Todd D. Neuropsychological performance in long-term cannabis users. *Archives of General Psychiatry*. 2001; 58(10):909–915. [PubMed: 11576028]
- Pope HG Jr, Gruber AJ, Yurgelun-Todd D. The residual neuropsychological effects of cannabis: the current status of research. *Drug & Alcohol Dependence*. 1995; 38(1):25–34. [PubMed: 7648994]
- Schierenbeck T, Riemann D, Berger M, Hornyak M. Effect of illicit recreational drugs upon sleep: Cocaine, ecstasy and marijuana. *Sleep Medicine Reviews*. 2008; 12(5):381–389. [PubMed: 18313952]
- Smith DJ, Muir WJ, Blackwood DHR. Neurocognitive impairment in euthymic young adults with bipolar spectrum disorder and recurrent major depressive disorder. *Bipolar Disorders*. 2006; 8(1):40–46. [PubMed: 16411979]
- Sobell, LC.; Sobell, MB. Timeline follow-back: a technique for assessing self-reported alcohol consumption.. In: Litten, RZ.; Allen, AJ., editors. *Measuring Alcohol Consumption*. Humana Press; Totowa, New Jersey: 1992. p. 41-72.
- Solowij N, Battisti R. The chronic effects of cannabis on memory in humans: a review. *Current Drug Abuse Reviews*. 2008; 1(1):81–98. [PubMed: 19630708]
- Solowij N, Stephens RS, Roffman RA, Babor T, Kadden R, Miller M, et al. Cognitive functioning of long-term heavy cannabis users seeking treatment.[see comment][erratum appears in JAMA2002 Apr 3;287(13):1651]. *Journal of the American Medical Association*. 2002; 287(9):1123–1131. [PubMed: 11879109]
- Vadhan NP, Serper MR, Haney M. Acute effects of smoked marijuana on working memory: implications for schizophrenia? *Primary Psychiatry*. 2009a; 16(4):51–59. [PubMed: 20351792]
- Wang CE, Halvorsen M, Sundet K, Steffensen AL, Holte A, Waterloo K. Verbal memory performance of mildly to moderately depressed outpatient younger adults. *Journal of Affective Disorders*. 2006; 92(2-3):283–286. [PubMed: 16533537]
- Woods SP, Delis DC, Scott JC, Kramer JH, Holdnack JA. The California Verbal Learning Test - second edition: Test-retest reliability, practice effects, and reliable change indices for the standard and alternate forms. *Archives of Clinical Neuropsychology*. 2006; 21(5):413–420. [PubMed: 16843636]

Figure 1. Marijuana use frequency (blunts/wk; left Y-axis) and depression scores (HAM-D total; right Y-axis) over time.

Figure 2. CVLT-II learning. Number of words learned on Trials 1-5 at baseline evaluation (open circles) and follow-up evaluation (filled circles).

TABLE 1
Baseline and follow-up neurocognitive scores on the California Computerized Assessment Package

Testing session	Baseline			Follow-up			Change
	SS (z)	Percentile	Range	SS (z)	Percentile	Range	
California Computerized Assessment Package							
Simple reaction time 1	1.21	89	High average	1.25	89	High average	
Simple reaction time 1 (nondominant)	1.49	93	Superior	0.81	79	High average	↓
Simple reaction time 2	1.25	89	High average	1.04	85	High average	
Simple reaction time 3	1.67	95	Superior	1.41	92	Superior	
Choice reaction time	1.44	93	Superior	1.51	93	Superior	
Sequential reaction time	2.00	98	Very superior	1.24	89	High average	↓ ↓
Lexical discrimination	0.32	63	Average	0.47	68	Average	
Visual selective attention	1.87	97	Superior	1.56	94	Superior	
Response reversal/visual scanning	1.07	86	High average	1.13	87	High average	
Form discrimination	0.95	83	High average	1.24	89	High average	

Notes: SS = Standard score; Scores less than average range are in bold; ↑ = increased performance, ↓ = decreased performance; number of arrows represents the magnitude of change.

TABLE 2

Baseline and follow-up neurocognitive scores on the California Verbal Learning Test – Second Edition

Testing session	Baseline			Follow-up			Change
	Raw	Percentile	Range	Raw	Percentile	Range	
Trial 1	2	2	Extremely low	5	16	Low average	↑↑
Trial 5	10	16	Low average	15	84	High average	↑↑
Trials 1–5	34	7	Borderline	51	54	Average	↑↑
Slope (Trials 1–5)	1.9	84	High average	2.4	98	Very superior	↑↑
Consistency	46	<1	Extremely low	86	50	Average	↑↑↑
List B	3	7	Borderline	3	7	Borderline	
Short delay free recall	11	31	Average	13	69	Average	
Short delay cued recall	10	16	Low average	15	84	High average	↑↑
Long delay free recall	10	16	Low average	15	84	High average	↑↑
Long delay cued recall	10	31	Average	15	84	High average	↑
Recognition hits	14	16	Low average	16	69	Average	↑
False positives	1	50	Average	0	84	High average	↑
Repetitions	4	50	Average	9	7	Borderline	↓
Intrusions	7	16	Low average	1	69	Average	↑

Notes: Scores less than average range are in bold; ↑ = increased performance, ↓ = decreased performance; number of arrows represents the magnitude of change.