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History of cannabis use is associated with altered gait

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ABSTRACT

Background: Despite evidence that cannabinoid receptors are located in movement-related brain regions (e.g., basal ganglia, cerebral cortex, and cerebellum), and that chronic cannabis use is associated with structural and functional brain changes, little is known about the long-term effect of cannabis use on human movement. The aim of the current study was to investigate balance and walking gait in adults with a history of cannabis use. We hypothesised that cannabis use is associated with subtle changes in gait and balance that are insufficient in magnitude for detection in a clinical setting.

Methods: Cannabis users (n = 22, 24 ± 6 years) and non-drug using controls (n = 22, 25 ± 8 years) completed screening tests, a gait and balance test (with a motion capture system and in-built force platforms), and a clinical neurological examination of movement.

Results: Compared to controls, cannabis users exhibited significantly greater peak angular velocity of the knee (396 ± 30 versus 426 ± 50°/second, P = 0.039), greater peak elbow flexion (53 ± 12 versus 57 ± 7°, P = 0.038) and elbow range of motion (33 ± 13 versus 36 ± 10°, P = 0.044), and reduced shoulder flexion (41 ± 19 versus 26 ± 16°, P = 0.007) during walking gait. However, balance and neurological parameters did not significantly differ between the groups.

Conclusions: The results suggest that history of cannabis use is associated with long-lasting changes in open-chain elements of walking gait, but the magnitude of change is not clinically detectable. Further research is required to investigate if the subtle gait changes observed in this population become more apparent with aging and increased cannabis use.

1. Introduction

Approximately 3.9% of the world's adult population have used cannabis, with the Oceania region having the highest prevalence of use (United Nations Office on Drugs and Crime, 2014). In Australia, 35% of individuals aged 14 years and over have tried cannabis at least once, with the estimated age of cannabis initiation at 16.7 years (Australian Institute of Health and Welfare, 2014).

The major psychoactive component of cannabis, Δ^9-tetrahydrocannabinol (THC), binds to cannabinoid-type 1 (CB1) receptors that are widely distributed throughout the central nervous system (Hirvonen et al., 2012). High densities of CB1 receptors are found in the hippocampus, amygdala, and movement-related brain regions, including the basal ganglia, cerebellum, and cerebral cortex (Oliviero et al., 2012). CB1 receptors are primarily located on the presynaptic membrane (Hirvonen et al., 2012) and activation can inhibit neurotransmitter release (Schlicker and Kathmann, 2001).

Cannabis intoxication results in *acute* motor deficits, including changes in balance (Ramaekers et al., 2006). An acute concentration-dependent disturbance in balance has been observed, with increasing levels of THC resulting in increased body sway (Kiplinger et al., 1971; Zuurman et al., 2008), possibly due to activation of CB1 receptors in movement-related brain regions. However, it is not known if cannabis use is associated with *long-lasting* disturbances in functional movement tasks such as balance and walking gait.

There are several lines of evidence to suggest that cannabis use may

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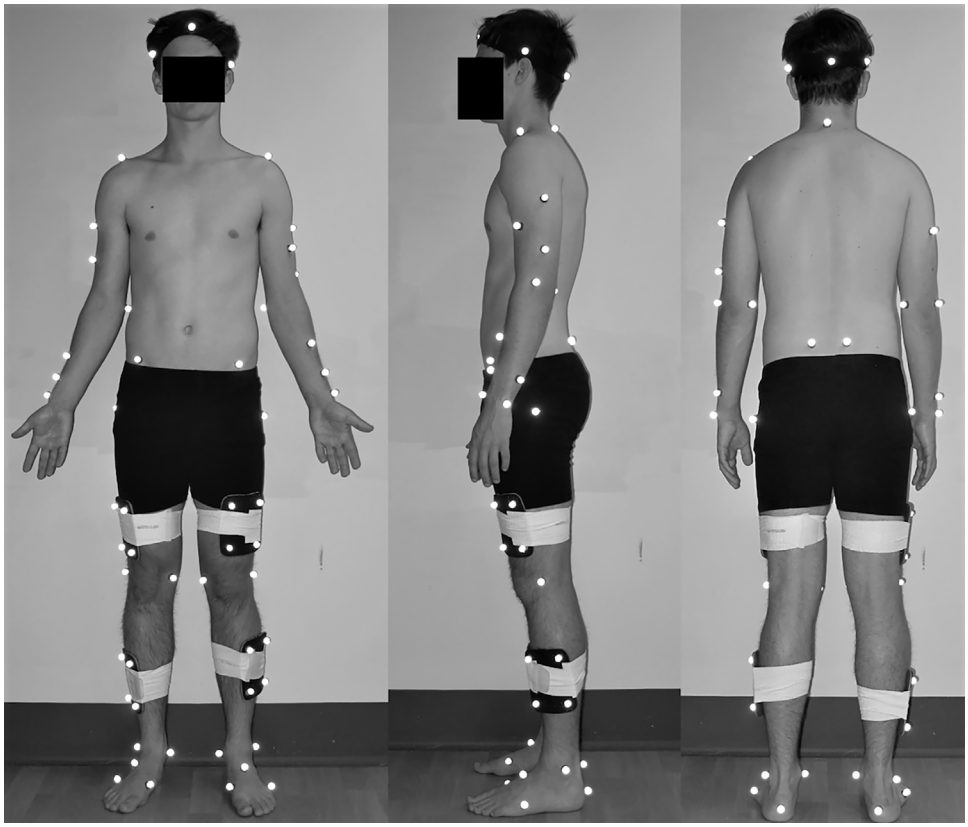

Fig. 1. Positions of the reflective markers for assessment of gait and balance. Markers were placed bilaterally on the head of the first and fifth metatarsal bones, dorsal aspect of the second metatarsal, posterior aspect of the calcaneous, medial and lateral malleoli, medial and lateral femoral epicondyles, greater trochanter, anterior inferior iliac spine, posterior inferior iliac spine, acromion process, spinous process of the 7th cervical vertebra, medial and lateral humeral epicondyles, and ulna and radial styloids. Non-collinear clusters of markers were placed on the forearm and upper arm (n = 3 per cluster) and the lower leg and thigh (n = 4 per cluster).

have a long-lasting effect on both gait and balance. There is a high density of CB1 receptors in movement-related brain regions (Takahashi and Linden, 2000) and within the dorsal and ventral horns of the spinal cord (Ong and Mackie, 1999). Animal studies suggest that cortical neurons exhibit a dose-dependent widening of the synaptic cleft and development of nuclear inclusion bodies in response to THC administration (Harper et al., 1977; Heath et al., 1980). Chronic cannabis use in humans is also associated with decreased white matter density in the left parietal lobe (Matochik et al., 2005) and long-term changes in cognitive functions (e.g., memory and executive functioning) (Solowij and Pesa, 2011). Therefore, it is conceivable that functional changes in the motor system may occur.

The aim of the current study was to investigate gait and balance in individuals with a history of cannabis use. We hypothesised that cannabis users would exhibit subtle features of ataxic gait and increased postural sway during quiet standing compared to non-drug users. The hypotheses were based on: (i) subjective observations of gait and balance abnormalities in individuals dependent on alcohol and either cannabis, stimulants, and/or opiates (Fein et al., 2012); and (ii) pathological and pathophysiological changes observed in the brains of cannabis users (Harper et al., 1977; Heath et al., 1980; Matochik et al., 2005). It was also hypothesised that disturbances in gait and balance observed in a laboratory setting would be too small to detect in a clinical setting. This hypothesis was based on the lack of reports of clinical movement dysfunction in cannabis users. The results of the current study advance knowledge of the long-lasting consequences of cannabis use in humans.

2. Materials and methods

2.1. Subjects and screening

Two groups of adults aged 18–49 years participated in the study: 22 subjects with no history of illicit drug use ('control group') and 22 subjects with a history of cannabis use (> 6 occasions) but no history of illicit stimulant or opioid use ('cannabis group'). All experimental procedures were approved by the Southern Adelaide Clinical Human Research Ethics Committee and the University of South Australia Human Research Ethics Committee. The experimental procedures were conducted in accordance with the Declaration of Helsinki and written informed consent was obtained from each subject prior to participation.

Each subject underwent a series of screening tests prior to participation. The full details of the screening procedure have been published previously (Pearson-Dennett et al., 2014). It included provision of a urine sample for drug screening (PSCupA-6MBAU, US Diagnostics Inc., Huntsville, Alabama, USA), a brief medical history questionnaire, an in-house drug history questionnaire, and a neuropsychological assessment of four cognitive domains. New learning was assessed with Logical Memory I and II (Wechsler, 1987), executive functioning was assessed with Verbal Trails (Grigsby and Kaye, 1995) and Verbal Fluency (Benton and Hamsher, 1983; Grigsby and Kaye, 1995), and attention and working memory were assessed with Digit Span forwards and backwards (Wechsler, 1981), respectively. Performance on each test was compared to published normative data matched for age and years of education. Recent symptoms of depression were also assessed with the Beck Depression Inventory-II (BDI-II) (Beck et al., 1996).

Exclusion criteria included history of neurological damage and/or neurological illness prior to first use of cannabis, current use of medications that affect movement (e.g., antipsychotics), self-reported history of major orthopaedic injury or surgery, and positive urine drug test for amphetamine, methamphetamine, MDMA (3,4-methylenedioxymethamphetamine or 'ecstasy'), cocaine, benzodiazepines, and/or opioids. Subjects who tested positive for cannabis were allowed to participate if self-reported use was greater than 12 h prior to experimentation. This exemption was due to THC remaining in body fat for up to 80 days after last use (Verstraete, 2004). Subjects were also excluded if poor performance was observed on more than two of the cognitive domains tested. Poor performance was defined as greater than two standard deviations below the mean of published normative data for Logical Memory I and II (Mittenberg et al., 1992), Verbal Fluency

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