## **SLEEP & CANNABIS USE**

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Chronic neuropathic pain affects 1%–2% of the adult population and can often be resistant to conventional pharmacologic treatment. A single dose of 25 mg of 9.4% THC inhaled three times daily for five days decreased the intensity of pain, improved sleep and was tolerated well. Previous research also identified a decrease in opioid overdose deaths in US states with legalized medical cannabis. A potential explanation of this phenomenon may be a possible substitution effect of medical cannabis for opioids. Among the participants that responded with regular opioid use, over 3/4 indicated that they had decreased their opioid use since commencing medical cannabis.

Approximately two-thirds of participants reduced their use of anti-anxiety, migraine, and sleep medications following initiation of medical cannabis.



The two most prevalent mental health disorders in Iraq and Afghanistan veterans are post-traumatic stress disorder (PTSD) and major depressive disorder (MDD). Sleep motives significantly mediated the correlation between PTSD and MDD with cannabis use. Previous studies suggest that people with PTSD often use cannabis to aid in coping with their condition. Sleep improvement also appears to be a primary motivator for coping-oriented cannabis use. The frequency of cannabis use was increased among those with high PTSD scores when used for sleep-promoting purposes in comparison to those with low PTSD scores or those who did not use cannabis for sleep-promoting purposes.



Innovative research analyzing OSA and cannabinoids suggest that synthetic cannabinoids such as nabilone and dronabinol may have short-term benefit for sleep apnea based on their regulatory effects on serotonin-mediated apneas. Dronabinol is not approved by the Food and Drug Administration (FDA) for treatment of OSA. Medical cannabis and synthetic extracts other than dronabinol have not been studied in patients with OSA therefore, it is the position of the American Academy of Sleep Medicine (AASM) that medical cannabis and/or its synthetic extracts should not be used for the treatment of OSA due to unreliable delivery methods and insufficient evidence surrounding effectiveness, tolerability, and safety.