Therapeutic Properties of Cannabigerol (CBG)

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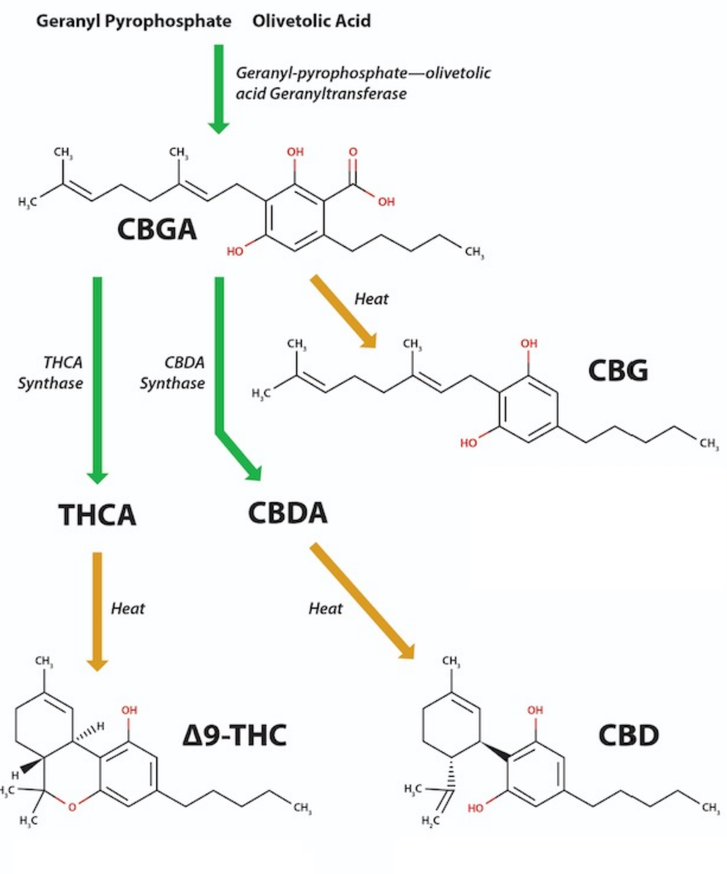
Introduction

Cannabigerol (CBG), is the precursor molecule to most cannabinoids including delta 9-tetrahydrocannabinol (THC) and cannabidiol (CBD). CBG displays potency at distinct non-cannabinoid receptors: alpha-2 adrenoceptors, PPARα/γ, and serotonin 5-HT_{1A} receptor. We propose CBG as a novel therapeutic in the cannabinoid class of drugs with potential indications of hypertension, pain, inflammation, and psychiatric disorders (substance abuse, ADHD, etc.)



The Cannabis plant produces CBG-Acid from geranyl diphosphate and olivetolic acid. CBGA is converted to the acid form of many common cannabinoids through specific enzymatic reactions. CBGA is converted to CBG by heat. Figure from Nachnani et al. JPET Feb. 2021

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Methods

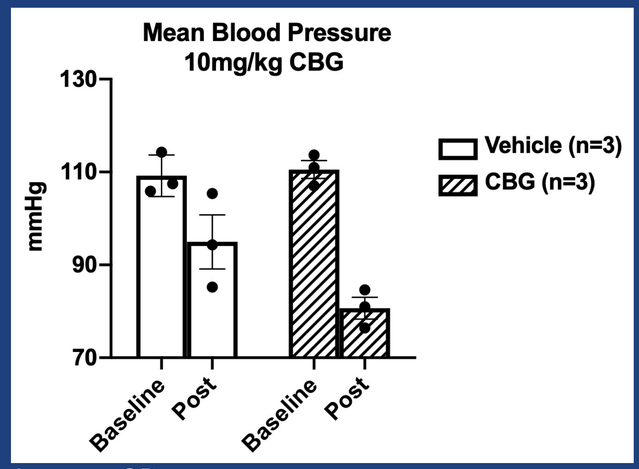
Neuropathic pain:

Mice with chronic neuropathic pain induced by cisplatin received i.p. Vehicle, 5, 10, or 20 mg/kg CBG (n=2,5,5,5, respectively) and had pain sensitivity measured using the von Frey test.

Radiotelemetry Blood Pressure Recordings:

Radiotelemetry probes, fitted to conscious, freely-moving mice, measured mean blood pressure (mmHg) before and after i.p. injection of vehicle (n=3) or 10 mg/kg CBG (n=3).

10mg/kg CBG may acutely lower blood pressure

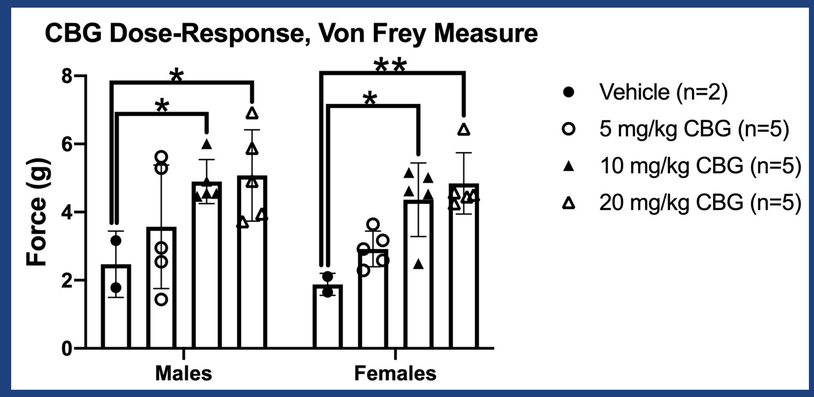


Error bars are SD

Discussion

Non-significant trend:10mg/kg CBG lowered mean blood pressure compared to vehicle. Presumably due to alpha-2 agonism, this warrants further investigation into cardiovascular effects of CBG as taken acutely and chronically in varying doses.

Increasing CBG administration reduces cisplatin-induced neuropathic pain



*Error bars are SD; *: p<.05; **: p<.01 as measured by 2-way ANOVA*

CBG i.p. injections at 10mg/kg and 20mg/kg provided dose-dependent relief of cisplatin-induced neuropathic pain sensitivity as measured in mice.

Discussion

CBG may be a useful pain-relieving agent for certain types of pain. This is potentially due to analgesic effects of alpha-2 agonists, the most common clinical example being clonidine.

Conclusion

Novel *Cannabis* compounds show promise as new pharmacotherapeutics, but most still do not yet have thorough characterization. CBG provides new opportunities and hazards as its popularity increases among cannabinoid supplement users. Our findings suggest that CBG has unique pharmacological potential for pain and blood pressure regulation, presumably due to its agonist activity at alpha-2 adrenoceptors. Translational and clinical research must be prioritized to reduce harm from pharmacological interactions and investigate CBG's ability to modulate diseases as a therapeutic.