

Cannabinoid Profile: Cannabigerolic Acid (CBGa)

(<http://theleafonline.com/c/science/2014/08/cannabinoid-profiles-crash-course-cbga/>)

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August 5, 2014



Formula: $C_{22}H_{32}O_4$

Molecular Mass: 360.48708 g/mol

Decarboxylation Point: ????

Boiling Point: ????

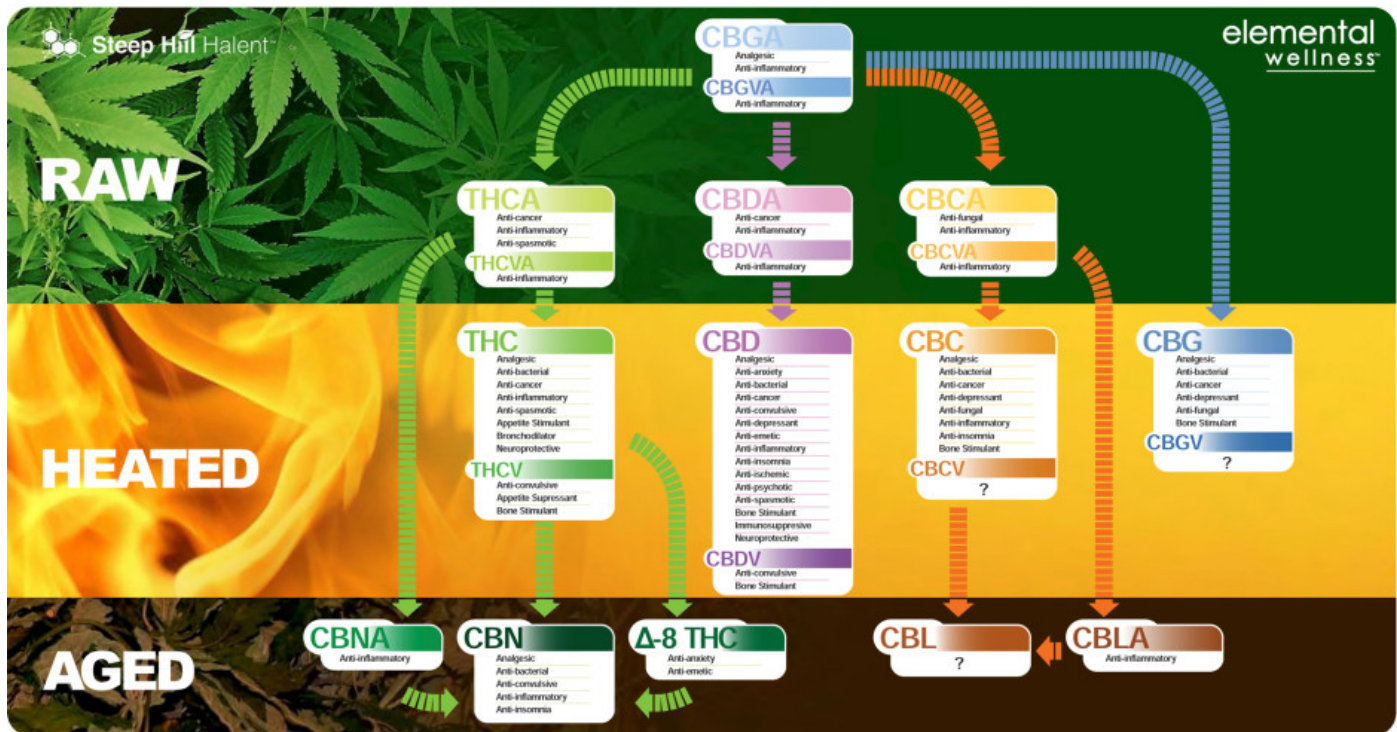
LD50 (Lethal Dose): Currently Unknown, likely similar to CBG, 300mg/kg for mice (<http://www.clearsynth.com/docs/MSD-CS-N-01916.pdf>) (Compare to Nicotine: for mice – 3mg/kg (<http://www.news-medical.net/health/Nicotine-Toxicology.aspx>), for humans – 40–60 mg/kg (http://www.oregon.gov/pharmacy/Imports/Marijuana/StaffReview/ReschedulingCannabis-NOTES_3-10.pdf)),



Cannabigerolic acid (CBGa) (<http://pubchem.ncbi.nlm.nih.gov/summary/summary.cgi?cid=6449999>) is formed when geranyl pyrophosphate (http://en.wikipedia.org/wiki/Geranyl_pyrophosphate) combines with olivetolic acid (<http://en.wikipedia.org/wiki/Olivetol>) within the cannabis plant. It is thanks to CBGa that all other medicinal effects of cannabis are possible. Cannabigerolic acid (CBGa) can be thought of as the stem cell cannabinoid, which becomes THCa (<http://theleafonline.com/c/science/2014/07/cannabinoid-profile-crash-course-thca/>)/THC (<http://theleafonline.com/c/science/2014/06/cannabinoid-profiles-crash-course-thc/>), CBDa (<http://theleafonline.com/c/science/2014/07/cannabinoid-profile-crash-course-cbda/>)/CBD (<http://theleafonline.com/c/science/2014/06/cannabinoid-profiles-crash-course-cbd/>), CBCa/CBC (<http://theleafonline.com/c/science/2014/07/cannabinoid-profile-crash-course-cbc/>), and CBG (<http://theleafonline.com/c/science/2014/07/cannabinoid-profile-crash-course-cbg/>). It does this through different types of biosynthesis (<http://en.wikipedia.org/wiki/Tetrahydrocannabinol#Biosynthesis>), where chemicals combine to form new compounds, examples being the THC biosynthase (http://en.wikipedia.org/wiki/THC_acid_synthase) and the CBD biosynthase (http://en.wikipedia.org/wiki/Cannabidiolic_acid_synthase). Hemp strains of cannabis have higher amounts of CBG due to a recessive trait (<http://link.springer.com/article/10.1007/s10681-005-1164-8#page-2>), which may imply higher amounts of CBGa present in those strains as well.

UNDERSTANDING MEDICAL CANNABIS

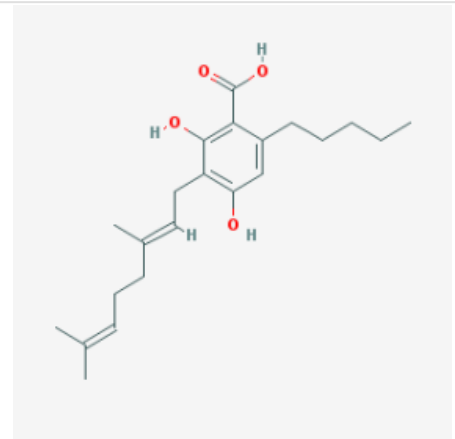
Cannabinoids and Their Therapeutic Effects



Halent 2011

Therapeutic Uses

- Analgesic** – Relieves pain.
- Antibacterial** – Slows bacterial growth.
- Anti-inflammatory** – Reduces inflammation systemically.
- Anti-Proliferative** – Inhibits cancer cell growth through apoptosis.



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Halent 2011 – Cannabinoid and Terpenoid Chart

Currently Being Studied For

Cannabinoid Biosynthesis: Nearly all current research on CBGA focuses solely on its role in the biosynthesis of other cannabinoids. Virtually no money is going to study its analgesic, antibacterial, anti-inflammatory, and anti-proliferative properties.

We learned in 2005 (<http://pcp.oxfordjournals.org/content/46/9/1578.full>) that the enzyme controlling the conversion of CBGA into THCa and further THC is held within the trichomes of the plant. This makes sense, as the trichomes have long been known to be the home of THC. Sirikantaramas did a follow up study (<http://www.ncbi.nlm.nih.gov/pubmed/17669365>) on his 2005 research which showed they could grow THCa in a laboratory (<http://www.sciencedirect.com/science/article/pii/S0006291X07015665>) using a yeast culture as a host. If you want to know more about the THCa synthase, which is the first biosynthase to see any major study, you can look at this 2009 literature review (<http://www.ddtjournal.com/getabstract.php?id=218>) profiling it. It wasn't until 2014 that any of this research turned back to focus on CBGA again, when Alaoui et Al (2014) (<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3916817/>) identified how and where CBGA binding happened, then explored how it was converted into THCa. Their research could be key to better understanding how THCa.

Cancer: While there are no current studies being done on CBGA for its abilities to help with cancer, it has been shown to be an anti-proliferative just like CBG, THC, and CBD. CBGA encourages apoptosis (http://www.digitalarchives.wa.gov/GovernorGregoire/priorities/healthcare/petition/exhibit_b.pdf), which is also known as *programmed cell death*. Defective apoptosis is believed to be a major reason for the formation and progression of cancer (<http://mct.aacrjournals.org/content/2/6/573.abstract>), so oncologists are naturally eager to find new ways to stimulate that bodily response. Cannabinoids appear to stimulate apoptosis in previously unknown ways, posing a novel way to mitigate and potentially cure cancer. While this much is known about CBGA, more research should be done.

***Note:** Decarboxylation (<http://en.wikipedia.org/wiki/Decarboxylation>) – A chemical reaction that removes a carboxyl group and releases CO₂, often triggered by heat.

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10. A Crash Course in CBGa [this post]



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