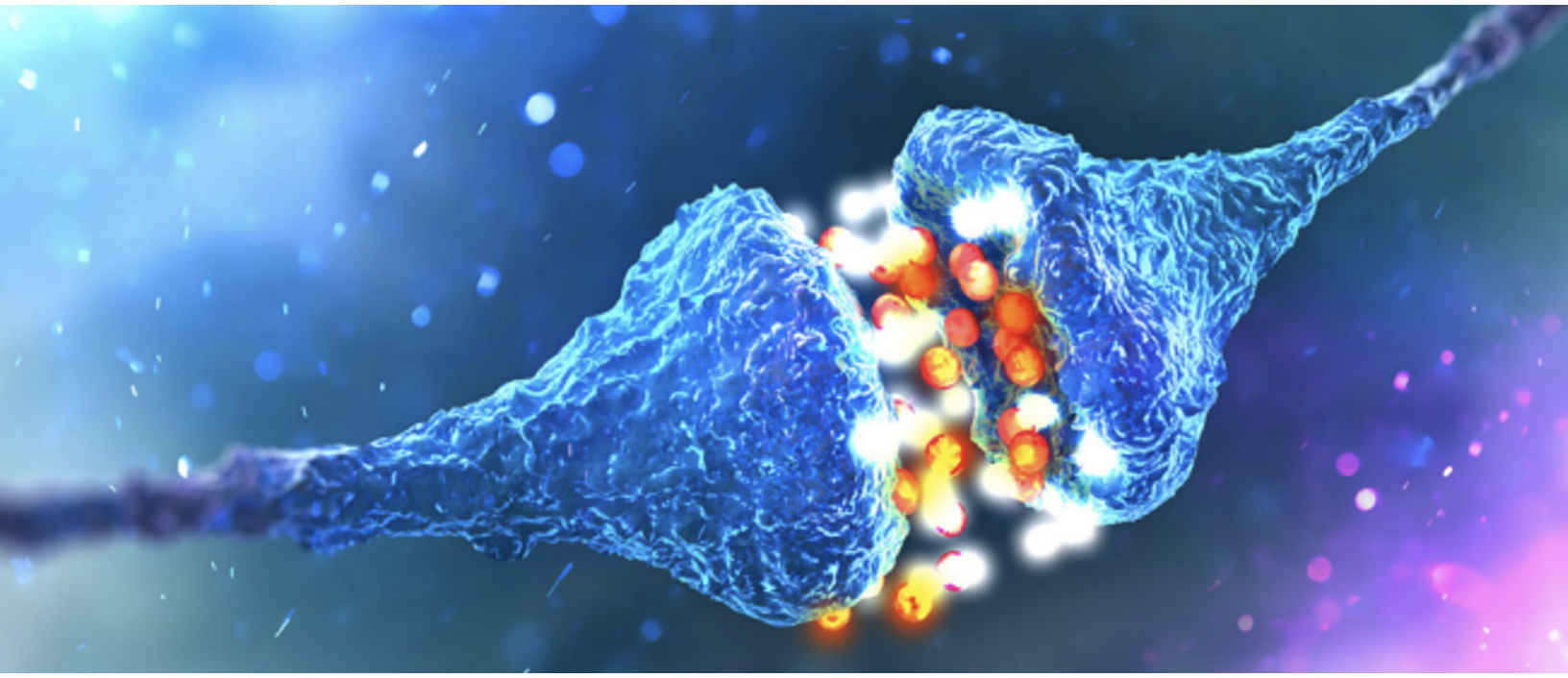
BLAIR MEDICAL GROUP

White Paper

Optimizing Metabolic Balance with Bio-molecules BCP and OEA

A Science-based Perspective on Natural Appetite Regulation and Fat Metabolism



Latest Research on the Metabolic Impacts of Two Combined "Super Metabolizers" - September 2025

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Optimizing Metabolic Balance with BCP and OEA

A Science-based Perspective on Natural Appetite Regulation and Fat Metabolism

Abstract

Metabolic balance is foundational to human health; influencing energy levels, fat storage, appetite, and long-term wellness in many ways. While diet, exercise, and lifestyle choices remain key contributors, natural bioactive compounds like **Beta-Caryophyllene (BCP)** and **Oleoylethanolamide (OEA)** are emerging as powerful tools for metabolic support.[1,11]

These naturally occurring molecules provide a stimulant-free, research-backed approach to optimizing metabolic function, regulating appetite, enhancing fat metabolism, and reducing inflammation without the harsh side effects associated with synthetic interventions. This white paper presents the separate merits of both BCP and OEA in metabolic regulation; also exploring the *synergistic benefits* derived from combining both compounds in a single, naturopathic blend. Detailed diagrams and content footnotes can be found in the Appendix section located at the end of this publication.

Introduction:

Metabolism is the process by which the body converts food into energy. Metabolic disorders or conditions occur when this process is disrupted, such as when there are too many or too few hormones involved in metabolism, or when organs like the pancreas or liver do not function properly. Disruptions to this important process may trigger disease.

Metabolic disorders are complex and multifactorial, often involving dysregulated metabolism, chronic inflammation, and oxidative stress. They encompass a broad range of conditions characterized by abnormal processing of nutrients by the body. These disorders, including obesity, type 2 diabetes, and Metabolically Associated Fatty Liver Disease (MAFLD) (formerly called non-alcoholic fatty liver disease (NAFLD)), are on the rise globally and pose a significant health burden. Metabolic syndrome, also known as syndrome X or insulin resistance syndrome, is a group of conditions that often occur together and can increase the risk of serious health conditions such as **diabetes, stroke**, and **heart disease.**

Current treatment options often focus on managing symptoms rather than addressing underlying causes and *pathophysiological mechanisms*. Natural compounds like beta-caryophyllene and OEA offer a potential alternative—or a complementary therapy due to their multifaceted biological activities.

Understanding Metabolic Disorders

Metabolic disorders arise when the body's systems for processing fats and carbohydrates become imbalanced—often due to lifestyle factors like dietary habits, and physical inactivity that can lead to chronic inflammation, or insulin resistance. This imbalance contributes to a cascade of unhealthy conditions.

When cells become less responsive to insulin (hormone responsible for regulating blood sugar) due to inflammation or oxidative stress damage, the liver works harder to function and is eventually impaired. These factors are all interconnected, creating a vicious cycle that impacts metabolic health over time.[2]

Common Metabolic Disorders

- **Obesity:** Excessive fat accumulation that increases risks for cardiovascular, musculoskeletal, liver, and behavioral health issues.[3]
- **Type 2 Diabetes Mellitus (T2DM):** A condition of insulin resistance and pancreatic beta-cell dysfunction leading to elevated blood sugar and complications like neuropathy, vision loss, and kidney damage.[4]
- **Dyslipidemia:** Characterized by abnormal lipid profiles—elevated LDL, low HDL, and high triglycerides—this condition is a key driver of atherosclerosis.[5]
- **Metabolic Syndrome:** A cluster of factors—including high blood pressure, abdominal obesity, and abnormal lipid levels—that significantly increase the risk of cardiovascular disease and type 2 diabetes.[6]
- **MASLD (metabolic dysfunction-associated steatotic liver disease)** is a group of liver diseases that happens when your body stores lots of fat in your liver. Over time, this fat in your liver can cause inflammation in your liver. Until recently, MASLD was known as nonalcoholic fatty liver disease (NAFLD.)[7]

Together, BCP and OEA target several of these metabolic dysfunctions through mechanisms rooted in **inflammation control**, **hormonal regulation**, and **fat metabolism**.

The Relationship Between the Endocannabinoid System and Metabolic Health

Understanding the Endocannabinoid System (ECS). The ECS is a sophisticated cell-signaling network present in all vertebrates, including humans. Discovered in 1988, it was named after the cannabis plant because *phytocannabinoids* such as THC and CBD led to its discovery. The term "endocannabinoid" itself reflects this origin—*endo* meaning "within the body" and *cannabinoid* referring to plant-derived compounds. It's important to understand that **the ECS is not solely activated by THC and CBD.** Stress management, diet, exercise, and dietary cannabinoids such as BCP can be used to balance the ECS. This system is an integral piece of the human organism and is composed of three core components:

- **Endocannabinoids** – Naturally produced messenger molecules like anandamide and 2-arachidonoylglycerol (2-AG) that help regulate internal processes.
- **Cannabinoid Receptors** – CB1 receptors are primarily found in the brain and nervous system, while CB2 receptors are found mainly in the immune system and peripheral tissues. These receptors act like locks, activated or inhibited by molecular "keys" to trigger or suppress physiological responses. **BCP acts solely on the CB2 receptor, a non-psychotropic channel.**
- **Enzymes** – These are responsible for breaking down endocannabinoids after they have carried out their function, ensuring the system resets and avoids overstimulation.[8]

The Endocannabinoid System is one 12 major bodily systems and impacts physiological effects throughout the human body.

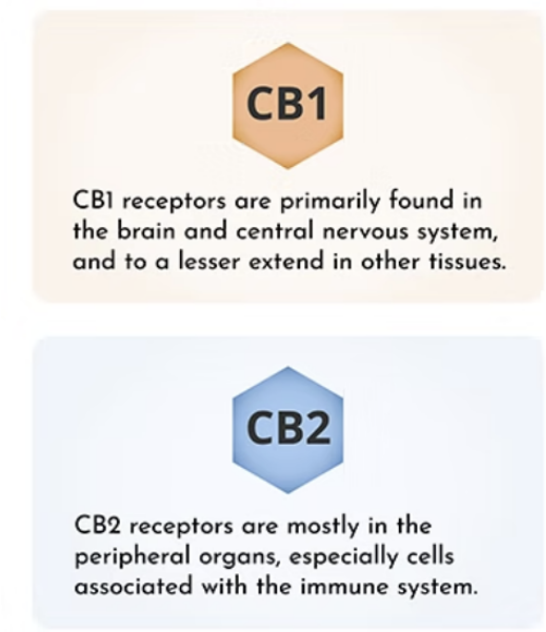
The ECS plays a vital role in maintaining **homeostasis**, or internal balance. It helps regulate mood, memory, pain perception, immune function, stress recovery, and *appetite and metabolism*.

One of its most critical functions is to manage the body's stress response—activating when threats arise and helping the body return to equilibrium afterward. It is increasingly recognized as a *master regulator of wellness* across many body systems.[9]

> "The endocannabinoid system is possibly the most important physiologic system involved in establishing and maintaining human health."
>
> Dr. Dustin Sulak

Key Metabolic Benefits of BCP

Beta-Caryophyllene (BCP) is a plant-derived compound classified as both a terpene and a dietary cannabinoid, and is found in cloves, black pepper, rosemary, oregano, hops, and hemp. BCP selectively binds to **CB2 receptors** in the endocannabinoid system, influencing inflammation, immune function, and metabolic processes. Unlike THC, *BCP does not have psychoactive effects or come from cannabis,* making it an attractive alternative for those seeking natural metabolic support. Importantly, BCP does not react with any other medications.[10]



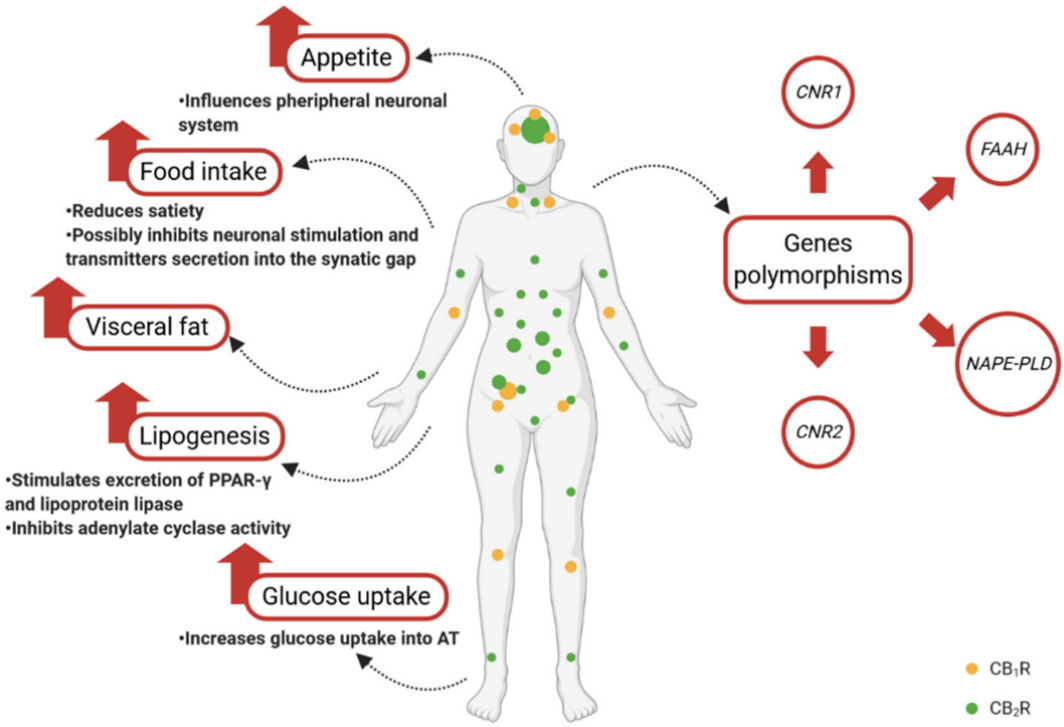
Research suggests that BCP has major therapeutic potential for managing metabolic disorders via the ECS. BCP's interaction with **CB2 receptors** has been linked to reductions in pro-inflammatory cytokines, which are often elevated in metabolic disorders. Research suggests that BCP supplementation can improve glucose tolerance and lipid profiles, thereby reducing the risk of insulin resistance and type 2 diabetes. Its ability to modulate the gut microbiome further supports its role in metabolic homeostasis, countering dyslipidemia and hypertension.

BCP also interacts with hormone regulators of metabolism and appetite. In a study of obesity induced asthma, BCP was found to stimulate the release of GLP-1 (glucagon-like peptide), without the side effects of the current GLP-1 drugs in the market. In a head-to-head study with a leading prescription, BCP showed 3 times the weight changes in a 4-week time frame. In another human study, BCP decreased serum levels of

orexin-A (an appetite-regulating hormone) and caused a 50% reduction in food addiction behaviors, according to the Yale Food Addiction Scale Score.

Through interacting with the receptors of the endocannabinoid system (ECS), BCP improves insulin sensitivity and supports lipid metabolism while reducing inflammation-related metabolic dysfunction. These support mechanisms make it a valuable tool for weight management and metabolic balance.

Endocannabinoid System Pathways in Appetite, Fat Storage, and Glucose Regulations



This graphic illustrates how activation of cannabinoid receptors (CB_1R in orange, CB_2R in green) influences key metabolic processes. Genetic polymorphisms in *CNR1, CNR2, FAAH,* and *NAPE-PLD* affect receptor activity, which in turn regulates appetite, food intake, visceral fat accumulation, lipogenesis, and glucose uptake. These pathways highlight the role of the endocannabinoid system in energy balance and metabolic health.

The net effect of these processes is better control over appetite, body fat, and blood sugar. In practical terms, a well-balanced endocannabinoid system can help support healthier weight management, improved energy use, and reduced risk of metabolic problems such as insulin resistance.

Metabolic and Anti-Inflammatory Effects of Beta-Caryophyllene.

Beta-caryophyllene (BCP) has been shown to play a significant role in metabolic regulation through several mechanisms:

Metabolic Health: Some studies suggest a link between the ECS and obesity, diabetes, and metabolic syndrome.

- **Enhances Metabolic Hormones**: By stimulating glucose uptake in muscle cells and reducing glucagon secretion from the pancreas, BCP upregulates *glucagon-like peptide-1 (GLP-1)* and *adiponectin*, two key hormones involved in glucose regulation, insulin sensitivity, and fat metabolism.

- **Improves Mitochondrial Function**: It increases mitochondrial biogenesis and the expression of mitochondrial uncoupling protein 1 (UCP-1), which supports thermogenesis and promotes energy expenditure.

- **Reduces Oxidative Stress**: BCP lowers reactive oxygen species (ROS), helping protect mitochondria from oxidative damage.

- **Promotes Browning of White Fat**: It facilitates the conversion of energy-storing white adipose tissue (eWAT) into energy-burning brown-like adipose tissue (BAT), contributing to increased caloric burn and potential weight loss

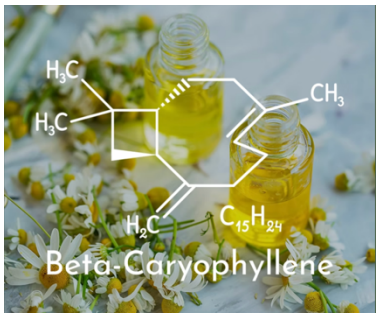
In addition to its metabolic effects, BCP exhibits strong anti-inflammatory and anti-allergic properties:

- **Inflammatory modulation:** BCP demonstrates modulation through the CB2 receptors NLRP3 and NF-KB signaling pathways, potentially mitigating chronic low-grade inflammation associated with metabolic disorders.

- **Activates the Adiponectin and Nrf2/HO-1 Pathways**: These pathways are associated with increased production of anti-inflammatory cytokines and enhanced cellular defense mechanisms.

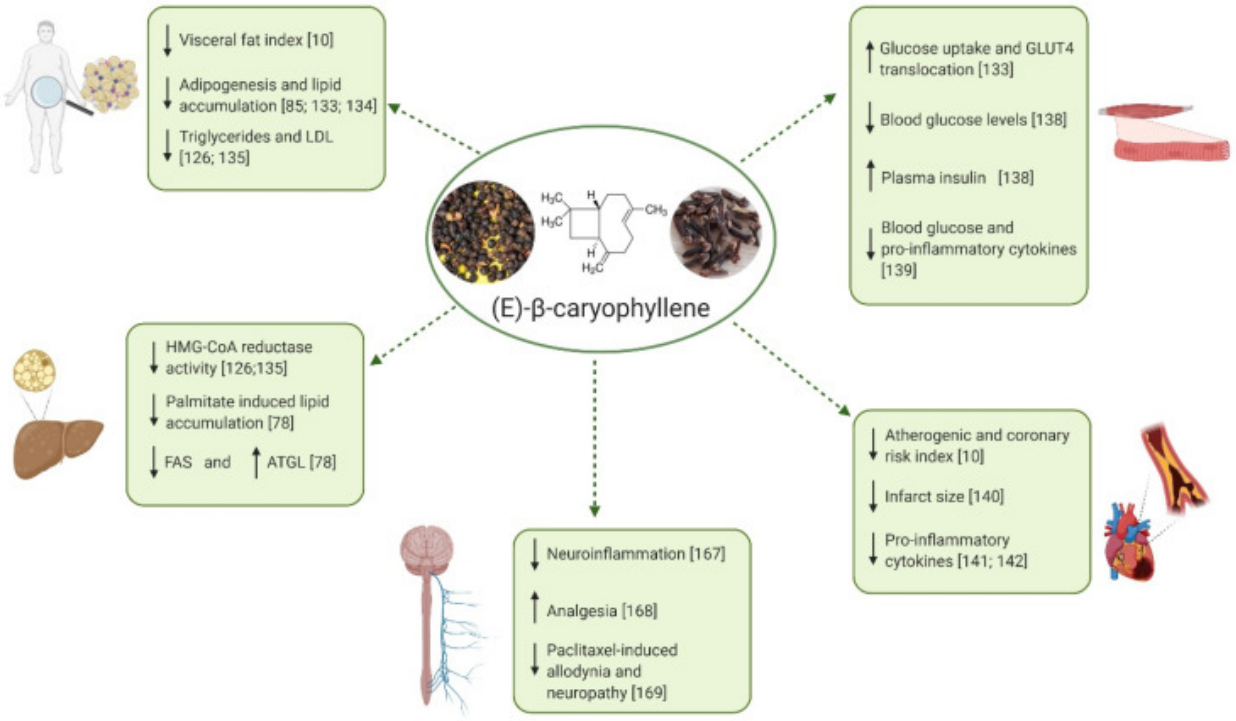
- **Suppresses Pseudo-Allergic Reactions**: BCP has been shown to inhibit mast cell degranulation, reduce histamine release, and prevent anaphylaxis-like responses.
  Together, these mechanisms highlight BCP's dual action: supporting metabolic balance while offering broad-spectrum anti-inflammatory protection—making it a compelling compound for managing weight, inflammation, and metabolic health.

Research Studies on the Therapeutic Potential of Beta-Caryophyllene



Research on BCP and metabolic disorders is still in its early stages. Preclinical studies using cell and animal models have shown promising results. For example, BCP administration improved insulin sensitivity, reduced inflammation, and prevented fatty liver disease in animal models. However, human clinical trials are limited. More research is needed to determine the optimal dosage, long-term safety, and efficacy of BCP in managing metabolic disorders in humans.

Scientific research exploring the therapeutic potential of beta-caryophyllene in managing metabolic disorders has yielded promising results, highlighting the compound's ability to regulate key metabolic pathways and improve metabolic health. Studies have shown that beta-caryophyllene can influence adipogenesis, the process by which fat cells develop and accumulate in the body, thereby potentially reducing obesity-related complications.



This diagram illustrates the biological effects of (E)-β-caryophyllene, a dietary sesquiterpene. The schematic highlights its role in regulating lipid metabolism, glucose balance, neuroinflammation, and cardiovascular risk through multiple pathways.

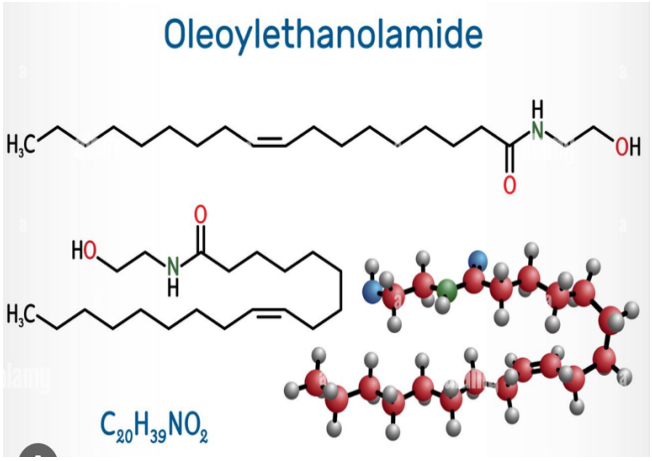
BCP **Summary:**

- **Reduces Inflammatory Cytokines** Through CB2 activation, BCP lowers levels of pro-inflammatory cytokines commonly elevated in metabolic disorders.
- **Improves Glucose and Lipid Profiles** Studies show that BCP enhances insulin sensitivity and glucose tolerance.
- **Modulates the Gut Microbiome** BCP influences microbiota populations linked to blood pressure regulation and dyslipidemia.
- **Stimulates GLP-1 Secretion** GLP-1 is a satiety and insulin-stimulating hormone. In preclinical models, BCP triggered its release without the adverse effects seen in current GLP-1 drugs.
- **Suppresses Appetite and Food Addiction** BCP was found to reduce orexin-A levels and halve food addiction behaviors in human trials.

In comparative trials, *BCP delivered three times the weight reduction over a four-week period versus a leading prescription medication.[4] This underscores its potential as a* ***natural, effective intervention*** *for weight management.*

OEA: A Potent Appetite Regulator

Oleoylethanolamide (OEA) is a naturally occurring food substance derived from olives. It is a biological lipid molecule produced in the small intestine in response to dietary fat intake. It interacts with PPAR-alpha receptors, playing a crucial role in energy homeostasis and appetite control. It is believed to play a key role in inhibiting food-seeking behaviors, insulin resistance, and how the brain processes food noise.



OEA levels naturally rise after meals, signaling satiety to the brain and reducing the urge to overeat. Additionally, OEA enhances fat oxidation, encouraging the body to use stored fat for energy.

OEA is a well-documented anorexigenic compound that influences hypothalamic pathways governing appetite regulation. By increasing PPAR-α activation, OEA enhances mitochondrial fatty acid oxidation and reduces appetite cravings in both preclinical and human studies.

Additionally, OEA's role in modulating gut hormone secretion further reinforces its impact on sustained weight management and metabolic optimization. Collectively, numerous studies in animal models have revealed the considerable anti-obesity effects, following OEA administration.

OEA suppresses food intake by releasing hypothalamic neuropeptides involved in appetite such as oxytocin and the activation of the GPR-119 gene resulting in the production of GLP-1 hormone. In one human study, 60 obese individuals took 2 x 125-mg OEA capsules for 8 weeks. Weight, body mass index, waist circumference, and fat percent decreased significantly at the end of the study. Hunger, the desire to eat, and cravings for sweet foods also decreased, and fullness increased significantly by the end of study in the intervention group.

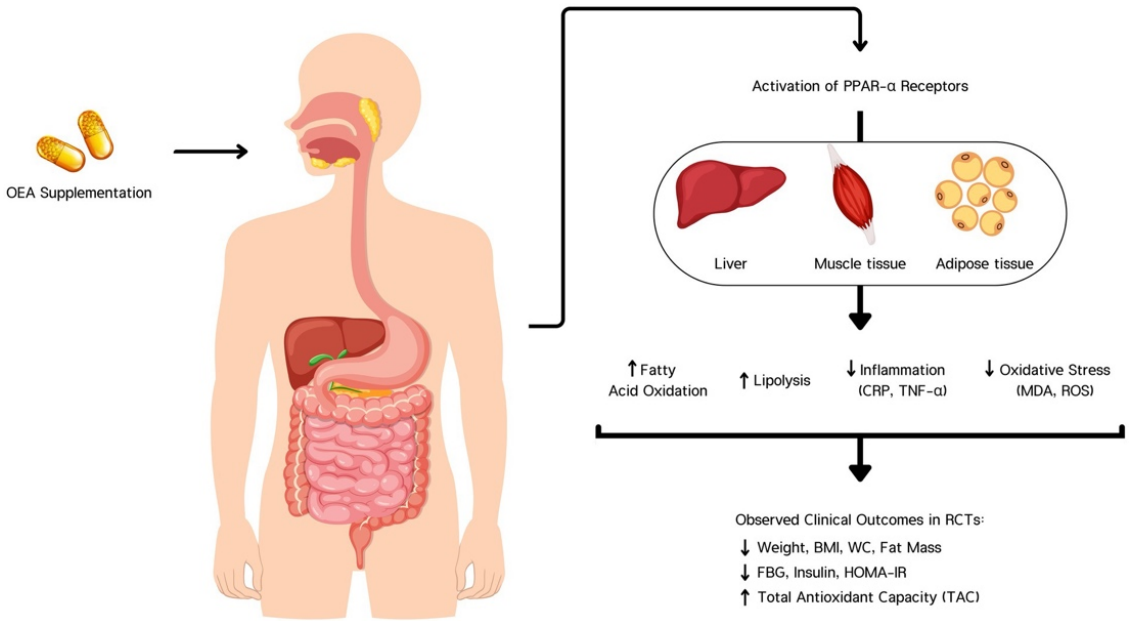
There is also growing evidence from these studies indicating the beneficial effects of OEA on MASLD (NAFLD) risk factors. This dual mechanism — reducing hunger while increasing fat metabolism — makes OEA a promising compound for weight management and metabolic health.

Key Benefits of OEA:

- **Suppresses Appetite** OEA increases satiety after meals and reduces food cravings.
- **Boosts Fat Oxidation** It triggers the body to burn stored fat more efficiently through mitochondrial activation.
- **Regulates Gut Hormones** OEA increases GLP-1 and activates the GPR-119 gene, enhancing appetite control and digestive efficiency.
- **Supports NAFLD Risk Reduction** Emerging research links OEA to improvements in non-alcoholic fatty liver disease markers.

In a double-blind clinical trial, obese participants who took OEA for 8 weeks saw reductions in body weight, BMI, waist circumference, and hunger levels.

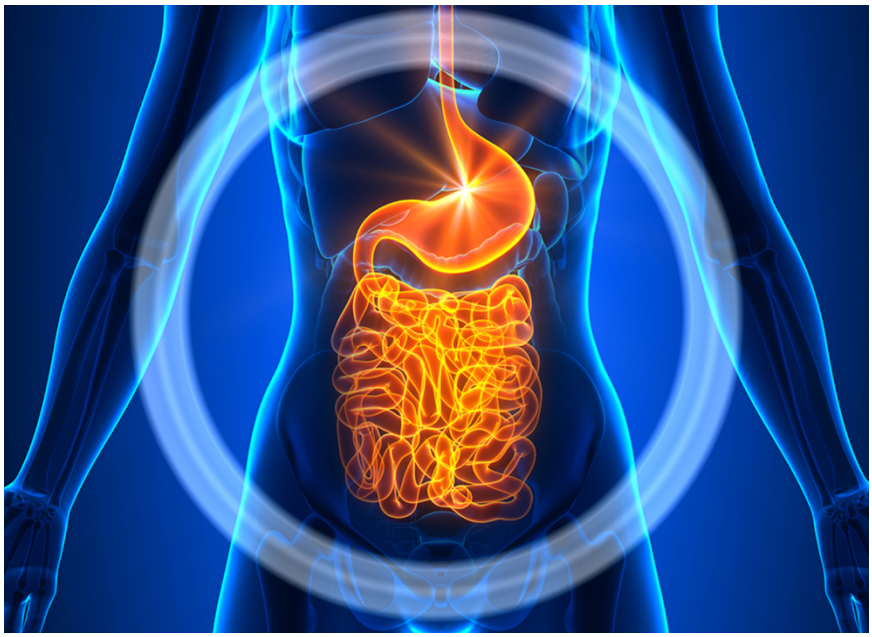
OEA ACTION CYCLE



This is a schematic representation of the metabolic effects of oleoylethanolamide (OEA) supplementation. Following ingestion, OEA activates PPAR-α receptors in liver, muscle, and adipose tissue, leading to increased fatty acid oxidation and lipolysis, reduced inflammation and oxidative stress, and clinically observed improvements in weight, insulin resistance, and antioxidant capacity.

Advanced Insights

Oleoylethanolamide (OEA) continues to emerge as a potent metabolic regulator with multifaceted physiological actions that *extend well beyond* appetite suppression and fat oxidation. Here's a deeper dive into underappreciated mechanisms and clinical nuances



Gut Microbiome Modulation & Intestinal Homeostasis

While OEA is well known for its metabolic effects, recent preclinical work suggests it may also reshape the gut microbiota. In mice, sub-chronic OEA administration did not significantly alter species richness but did shift microbiota composition—reducing Firmicutes and enriching Bacteroidetes, indicating a lean-associated microbial profile.[11] These findings hint at a potential microbiome-mediated pathway for some of OEA's systemic benefits, particularly regarding energy homeostasis and gut-immune interactions.

GPR119 Activation Beyond PPAR-α

Most of OEA's functionality has been linked to PPAR-α signaling. Yet it also serves as an endogenous ligand for **GPR119**, a G protein-coupled receptor primarily localized in the pancreas and gut.[12] Activating GPR119 improves incretin hormone (e.g., GLP-1) secretion and reduces food intake, providing an additional axis through which OEA supports metabolic balance.

Cardiometabolic Biomarkers & Lipid Regulation

Meta-analyses and recent randomized controlled trials have revealed significant effects of OEA supplementation on a broad panel of health markers.

Benefits include reductions in C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), malondialdehyde (MDA), fasting glucose, insulin, HOMA-IR, weight, BMI, waist circumference, fat mass, and fat percentage.[13] While cholesterol levels (LDL, HDL, total) and HbA1c often remained unchanged, OEA consistently delivered improvements in triglyceride levels and insulin sensitivity. One randomized, placebo-controlled trial observed a significant drop in triglycerides in obese participants after eight weeks of **125 mg OEA taken twice daily**—but without notable changes in fasting blood sugar or broader dietary habits.[14] Another trial pairing

250 mg OEA with calorie restriction over 12 weeks in non-alcoholic fatty liver disease (NAFLD) patients not only reduced triglycerides but also suppressed inflammatory markers such as NF-κB and IL-6 while doubling IL-10 levels.[15]

Anti-Inflammatory & Antioxidant Signaling

OEA doesn't just signal satiety—it actively promotes systemic resilience. Supplementation has been shown to lower pro-inflammatory cytokines (e.g., IL-6, TNF-α, NF-κB) and bolster antioxidant defenses across multiple models. These effects are especially promising in managing chronic low-grade inflammation, a hallmark of metabolic disorders such as obesity and MASLD (NAFLD).[16]

In a double-blind clinical trial, obese participants who took OEA for 8 weeks saw reductions in body weight, BMI, waist circumference, and hunger levels.[17]

Summary Table: Highlighting Novel OEA Actions

Microbiome Modulation	Shifts microbial composition toward lean-associated profile
GPR119 Activation	Supports GLP-1 release & glucose regulation
Metabolic Biomarkers	Strong effects on CRP, TGs, insulin sensitivity, body composition
Anti-Inflammatory Effects	Reduces key inflammatory markers, enhances antioxidant capacity

OEA: In Conclusion

OEA's benefits extend **well beyond appetite suppression**. Its ability to modulate gut microbiota, activate GPR119, improve cardiometabolic markers, reduce inflammation, protect neurons, and even support longevity demonstrates its versatility as a metabolic and cellular modulator.

Taken together, these findings position OEA as more than just a signaling molecule—it functions as a broad regulator of whole-body balance. By influencing pathways that connect the gut, brain, and metabolic tissues, OEA helps synchronize nutrient sensing with energy use, while also reinforcing protective systems against oxidative stress and chronic inflammation. This integrative profile suggests that OEA may play an important role not only in addressing current metabolic challenges, but also in promoting resilience and healthy aging over the long term.

The Synergy of BCP and OEA

While β-caryophyllene (BCP) and oleoylethanolamide (OEA) each have well-documented individual benefits, emerging evidence suggests that their concurrent use may deliver *multiplicative* effects—particularly for metabolic regulation, appetite control, and inflammation management. The synergy arises from the fact that each molecule acts on **distinct but complementary receptor systems**, converging on shared downstream pathways that affect energy balance, lipid metabolism, and whole-body homeostasis.

1. Distinct Entry Points, Shared Goals

- **BCP** acts as a *dietary cannabinoid*, selectively binding to CB_2 receptors in the peripheral endocannabinoid system (ECS). This engagement triggers anti-inflammatory cascades, promotes lipid mobilization, and helps restore insulin sensitivity in tissues without producing CB_1-mediated psychoactive effects.

- **OEA**, on the other hand, does not interact with cannabinoid receptors directly. Instead, it binds to *peroxisome proliferator-activated receptor-alpha* (PPAR-α), a nuclear receptor that governs the transcription of genes involved in fatty acid transport, β-oxidation, and satiety signaling.

Because these entry points are separate, the two molecules can operate in **parallel**, influencing different control nodes in the body's metabolic network without competing for receptor sites.

2. Converging on Metabolic and Appetite Regulation
BCP's ECS-mediated anti-inflammatory action helps correct low-grade systemic inflammation, a known driver of leptin and insulin resistance. OEA's PPAR-α activation amplifies fatty acid oxidation and improves nutrient partitioning, while also stimulating **oxytocinergic and GLP-1-mediated satiety pathways**. Together, they produce a one-two effect:

- BCP removes the *brakes* of metabolic inflammation.
- OEA steps on the *gas pedal* of fat utilization and appetite suppression.

This dual effect supports both **energy expenditure** and **caloric moderation**—two goals often at odds in standard dietary interventions.

3. Reinforcing Gut–Brain Communication
The gut–brain axis is another area where these compounds complement each other. BCP's ECS engagement may stabilize intestinal barrier function and reduce inflammatory cytokine leakage into circulation. OEA, produced naturally in the small intestine after fat ingestion, amplifies vagus nerve signaling to the hypothalamus, producing a clear "stop eating" command. In concert, the two molecules may produce a *cleaner satiety signal*—less distorted by inflammation, more consistent in timing.

Synergy Model Table

Pathway	**BCP Mechanism**	**OEA Mechanism**	**Synergy Outcome**
Inflammation Control	CB_2-mediated suppression of cytokine release	Indirect via reduced adipocyte stress	Lower baseline inflammation improves metabolic signaling
Fat Utilization	Improves insulin sensitivity → better fuel selection	PPAR-α activation → increased β-oxidation	More efficient shift toward fat as primary fuel
Satiety	Reduces ECS overactivity linked to compulsive eating	Activates PPAR-α, GLP-1, oxytocin pathways	Stronger, cleaner satiety response
Gut Health	Enhances barrier function, microbiome stability	Promotes lipid sensing in small intestine	Better gut–brain hormonal communication

A Natural Alternative

Many synthetic weight-loss and appetite-suppressant drugs act on a single pathway, such as amphetamine-like stimulation of norepinephrine release or GLP-1 receptor agonism. While potent, such approaches often carry a significant side-effect burden, ranging from cardiovascular strain to gastrointestinal distress. BCP and OEA, in contrast, are **endogenous or diet-derived molecules** that the body already recognizes and metabolizes safely:

- **BCP** is a common terpene in black pepper, cloves, and cannabis, classified as *Generally Recognized as Safe* (GRAS) by the FDA.

- **OEA** is a naturally occurring lipid mediator synthesized in the gut or supplemented from dietary oleic acid (olive oil being a prime source).

Their combined mechanism does not force a single metabolic function; instead, it engages **multiple functional harmonies**, producing cumulative changes without overtaxing any single pathway.

Enhancing Energy and Gut Health The metabolic improvements of BCP and OEA are not limited to weight control—they extend to **energy production efficiency** and **gut-brain immune axis optimization**.

- **Energy Efficiency:** OEA's upregulation of mitochondrial fatty acid transport proteins (e.g., CPT-1) allows a greater proportion of energy to come from β-oxidation, sparing glycogen and stabilizing blood glucose. BCP's anti-inflammatory profile reduces mitochondrial oxidative stress, making ATP production more efficient.

- **Gut Health:** BCP's ECS-mediated microbiome modulation may favor beneficial bacteria linked to lean phenotypes. OEA's signaling encourages coordinated digestive motility and secretion patterns, which can improve nutrient assimilation without promoting overeating.

Conclusion: A Balanced, Sustainable Approach

The complementary nature of BCP and OEA presents a **scientifically credible basis** for their combined formulation in metabolic support supplements. By addressing inflammation, energy utilization, satiety, and gut–brain communication through separate yet converging pathways, this pairing may deliver more reliable, sustained results than either compound alone.

Emerging as a promising combination with significant potential for managing metabolic disorders through interactions with the ECS, **NEW proprietary blended capsules** have been designed by a team of physicians to take full advantage of the molecular synergy. Addressing the root causes of complex conditions in a holistic approach and bridging the gap between dietary support and targeted metabolic intervention. Incorporating BCP and OEA supplementation into a daily routine, in addition to adopting healthy lifestyle practices, can harness the therapeutic benefits of these powerful supports to metabolic well-being. BCP and OEA are more than promising—they're here now, with clinical backing, practical results, and a future full of therapeutic potential.

Looking Ahead: The Future of Metabolic Supplements

Blair Medical Group's physician formulated **BCPlus Metabolic Blend Capsules** combine a **proprietary nano-powder BCP** with olive derived **OEA**, into an Enteric-coated, time-released delivery for optimal absorption. This development reflects the latest in nutraceutical innovation.

As interest in natural, research-backed therapies grows, **BCP and OEA stand out as leaders** in the next generation of metabolic health tools. Their use aligns with integrative and preventive medicine strategies—supporting energy balance, hormone regulation, and long-term wellness.

Visit our website to learn more or place an order.
Go to: www.blairmedicalgroup.com
Or click on the QR code to left.

About the author, Dr. Philip Blair

Retired Colonel (Dr.) Philip Blair is a board-certified Family Physician licensed in Washington State. He graduated from West Point in 1972 and attended the University of Miami School of Medicine and trained as a family physician. He had assignments in Georgia, Louisiana, Washington, Oklahoma, Texas, Hawaii, Kansas, Italy, Korea, Germany, and the Gulf War.

In 2014, he was introduced to cannabidiol. Since then, he has treated several thousand patients with CBD in his clinical practice. Currently, he is researching the terpene β-Caryophyllene as an alternative to medicinal cannabis. Blair Medical Group SPC offers physician-formulated BCP products that everyone can use to support, restore, and activate the endocannabinoid system, as well as address chronic pain and inflammation-related conditions. BCPlus products are available at blairmedicalgroup.com.

Dr. Blair is also available for private consultations and speaking engagements. Please contact him at DrBlairMD@icloud.com and (360) 991-4791.

About Blair Medical Group

Blair Medical Group is dedicated to the development and dissemination of evidence-based, integrative endocannabinoid therapies. We focus on supporting healthcare providers and patients with pioneering technology in product formulation, research, and clinical tools that promote sustainable wellness.

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We welcome your comments and reserve the right to revise this publication and/or make improvements or changes to the products or programs described in this publication at any time, without notice.

Appendix

References and Detailed Diagrams

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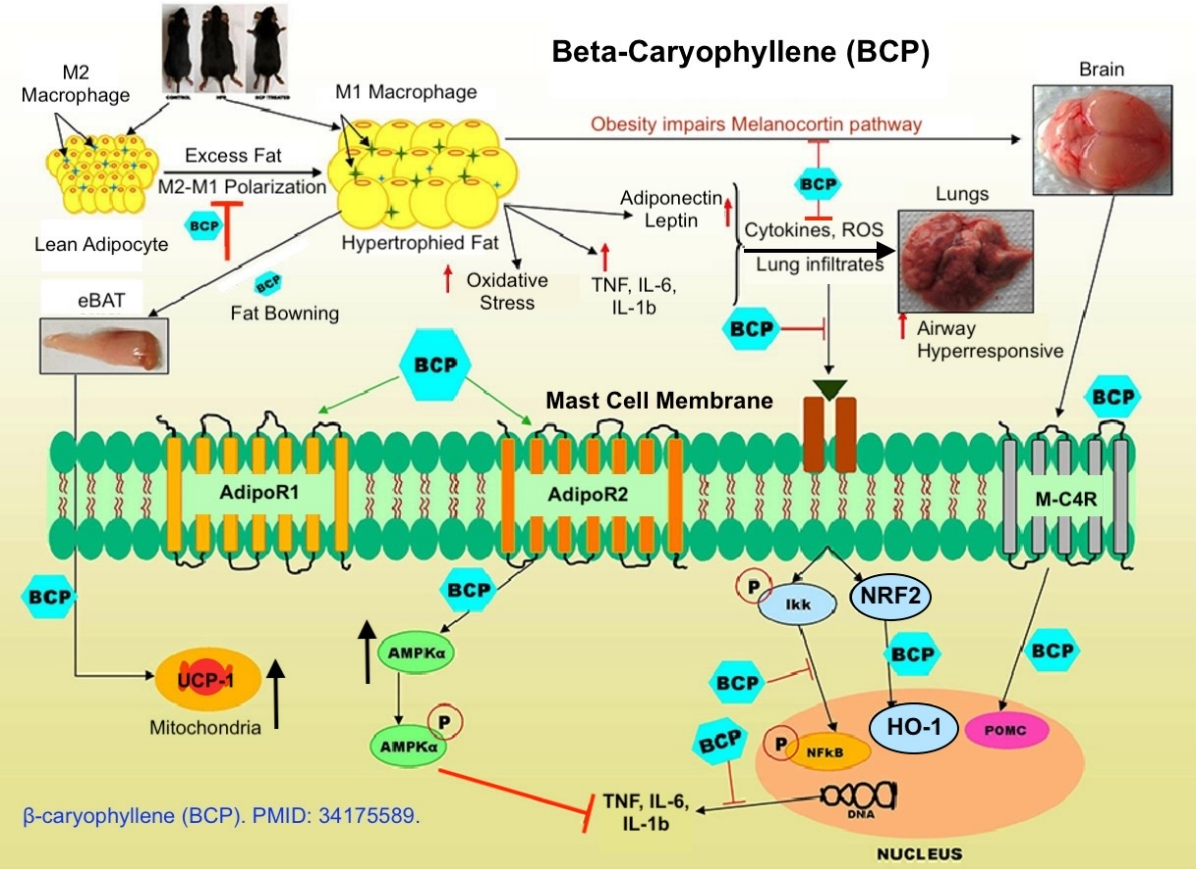
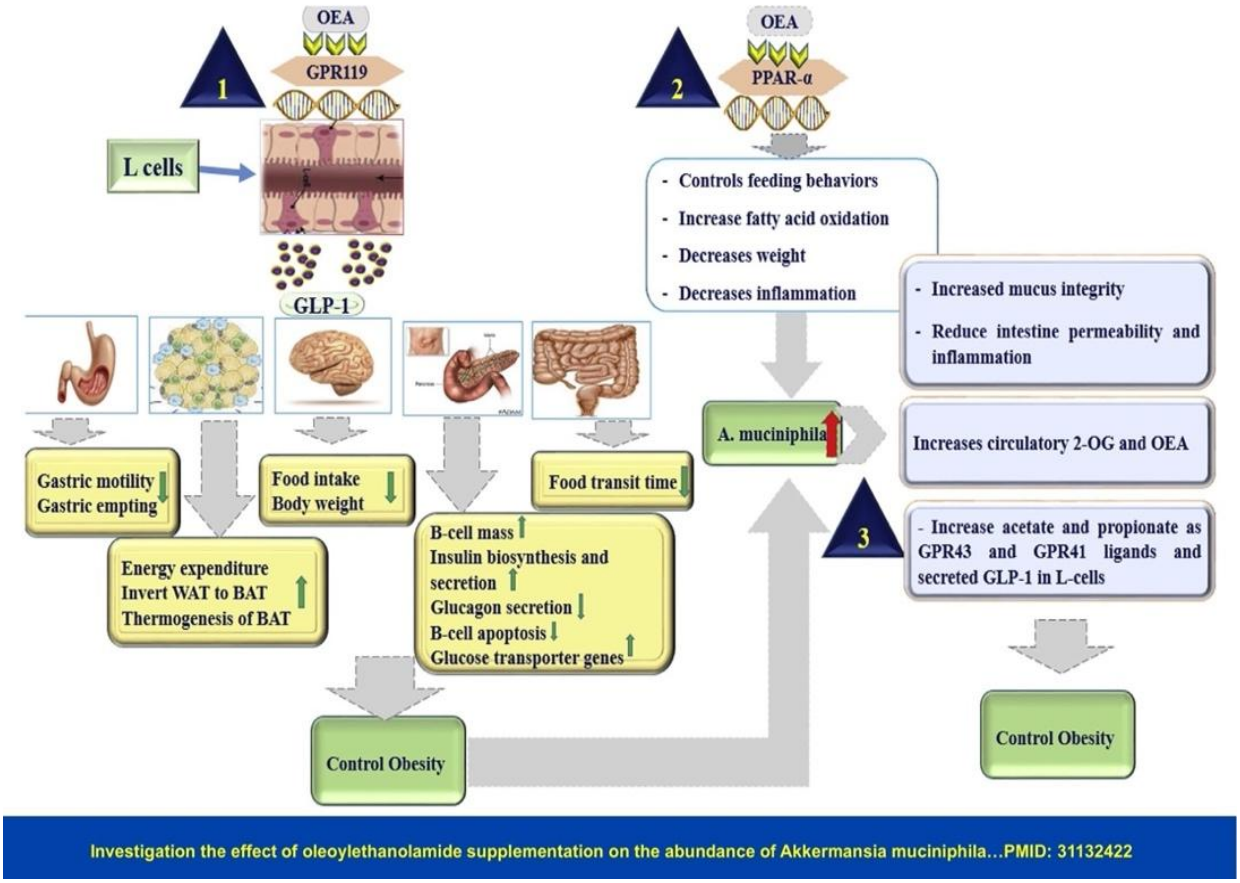
Diagram of BCP Effects on Body Systems



This diagram illustrates how **beta-caryophyllene (BCP)** interacts with cellular pathways to influence obesity-related processes. BCP binds to receptors such as AdipoR2 and CB_2R, activating downstream signals that enhance mitochondrial function, stimulate AMPK activity, and promote antioxidant defenses (Nrf2, HO-1).

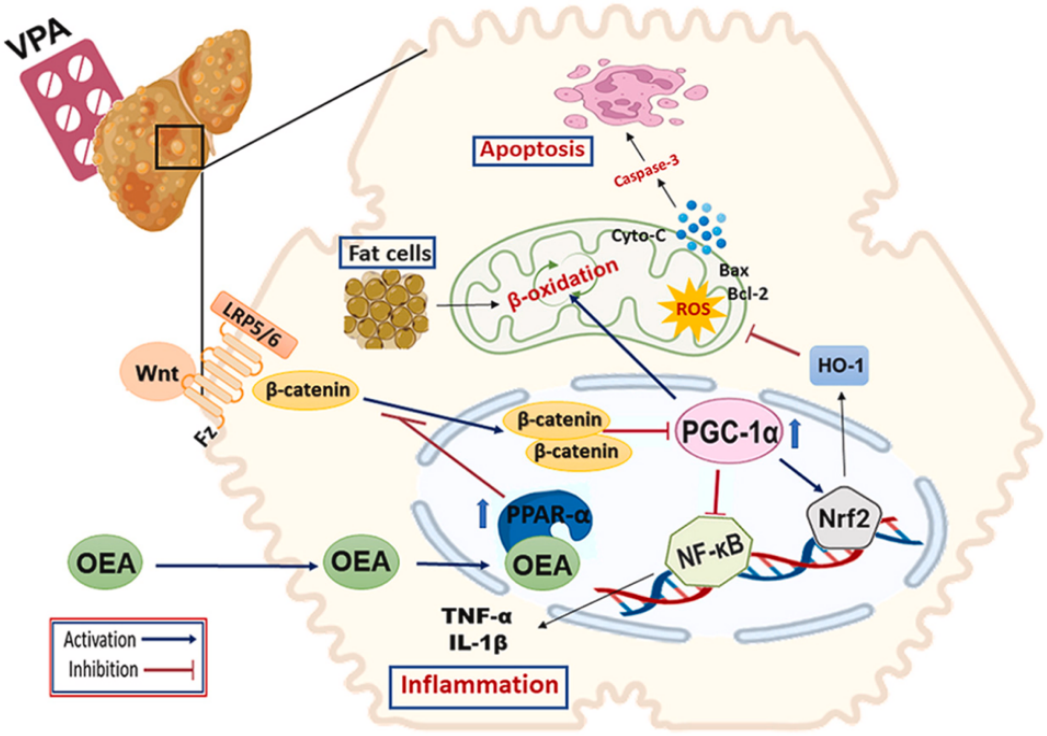
These mechanisms counteract oxidative stress, reduce inflammatory cytokines (TNF-α, IL-1β, IL-6), and support healthier adipose tissue function. In obesity, dysregulated adipocyte signaling and excessive inflammation impair these pathways, contributing to altered brain, lung, and metabolic responses.

OEA: Diagrams of Fatty Living Pathways



This diagram illustrates the multi-pathway effects of oleoylethanolamide (OEA) on obesity control, highlighting its activation of GPR119 and PPAR-α receptors, regulation of gut microbiota (A. muciniphila), and downstream impacts on satiety, fat oxidation, inflammation reduction, and energy metabolism.

HOW OEA ACTIVATION MECHANISM WORKS



This diagram depicts how **oleoylethanolamide (OEA)** activates PPAR-α and related pathways to promote fat oxidation, reduce oxidative stress (ROS), suppress inflammation, and prevent apoptosis in fat cells—ultimately supporting metabolic health and cellular resilience.

-End of Whitepaper-