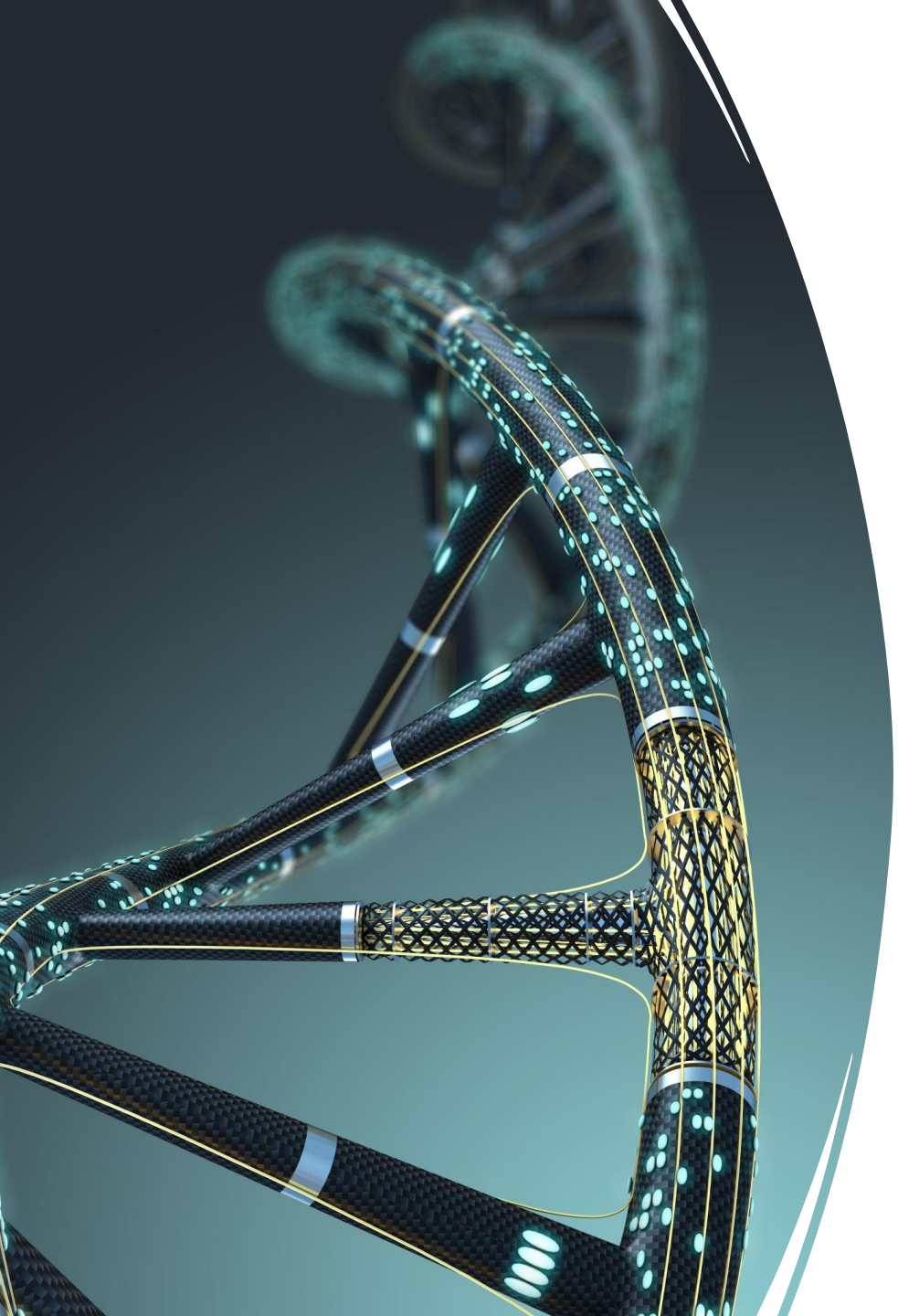

Exploring the Endocannabinoid System

Dr Rowan Thompson

With special thanks to Dr Stefan Broselid for his contribution to this presentation.

Disclosure of conflicts of interest

Doctor at Dispensed Clinic

Vice Chair Medical Cannabis Clinicians Society

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Medical Cannabis Clinicians Society

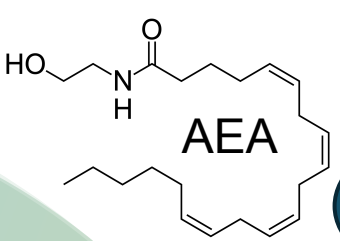
Learning Objectives

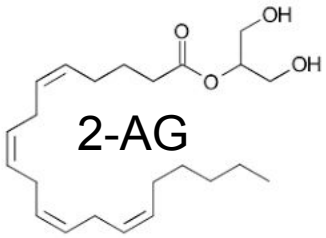
- By the end of this session, participants will be able to:
  - Describe the structure and key functions of the endocannabinoid system (ECS), its associated endocannabinoidome (eCBome) and its role in human physiology.
  - Recognise the relevance of ECS dysregulation as a contributing factor to a wide range of conditions
  - Explain how the ECS interacts with cannabinoids found in CBPMs and contributes to their therapeutic effects.
  - Apply a foundational understanding of the ECS to inform safe, evidence-based clinical decision-making.

Audience Poll

- Are you a clinician involved in the prescribing of medical cannabis?
- How would you rate your understanding of the Endocannabinoid System?
- Do you think diet is one of the most important contributors to our health?
- Do you discuss diet with 50% of your patients?
- 30%?
- 20%?
- 10%?
- 5%?

The components of the ECS

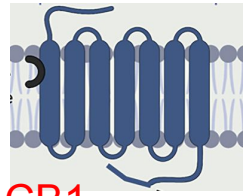


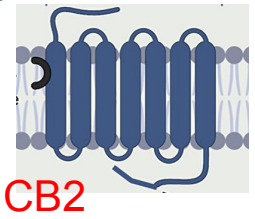


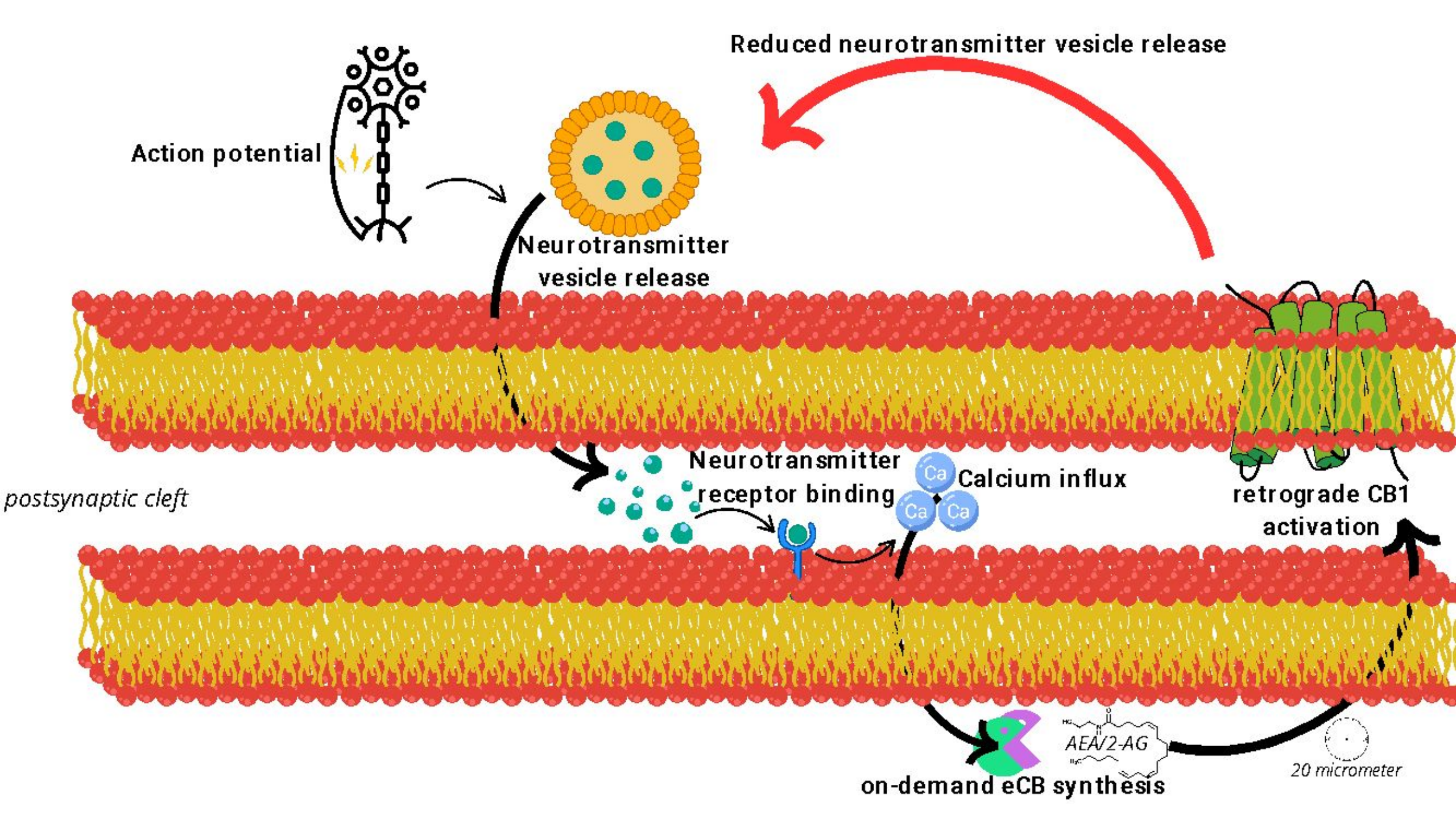
Endocannabinoids

Cannabinoid Receptors

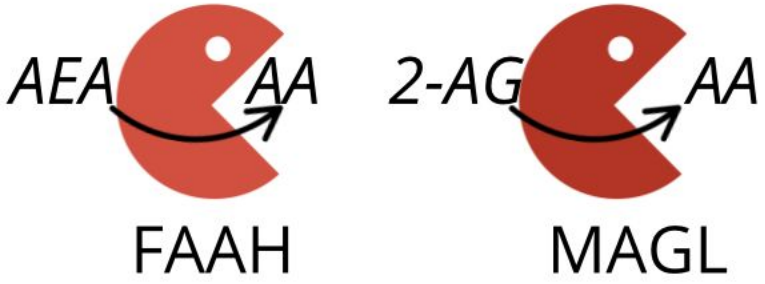
Enzymes






Reduced neurotransmitter vesicle release
Action potential
Neurotransmitter vesicle release
postsynaptic cleft
Neurotransmitter receptor binding
Ca
Ca
Ca
Calcium influx
retrograde CB1 activation
AEA/2-AG
on-demand eCB synthesis
20 micrometer

Metabolic Termination

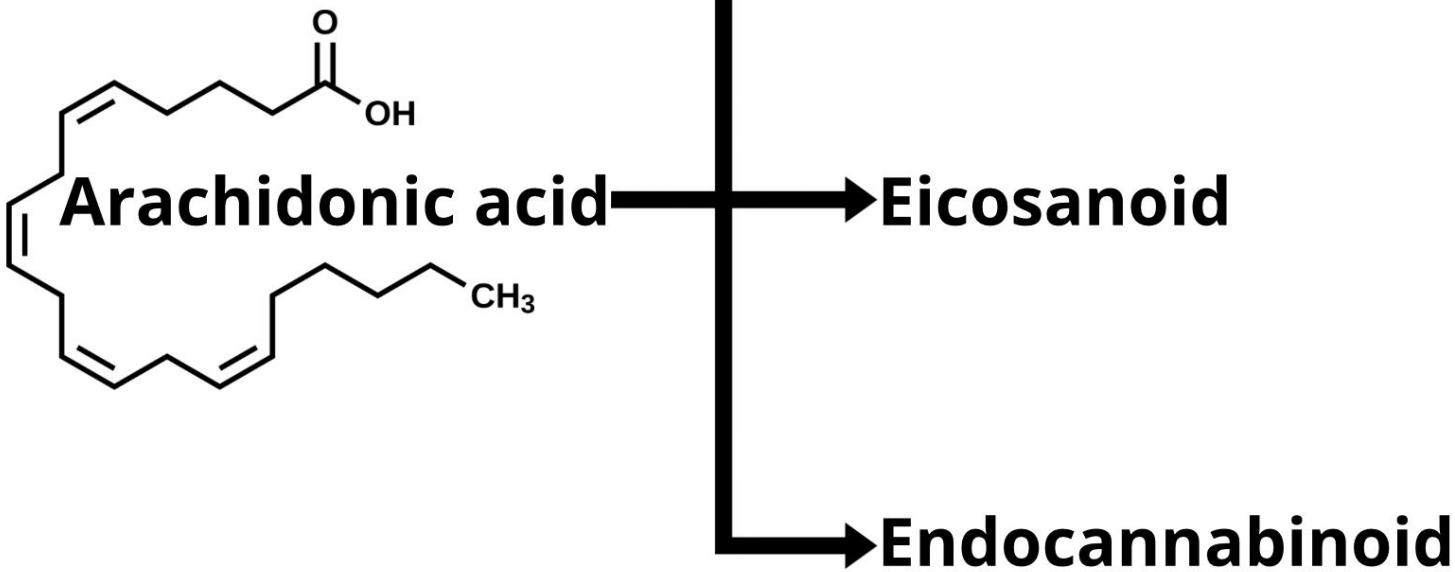


postsynaptic

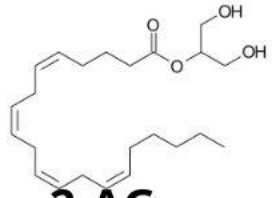
presynaptic

Half-life of AEA = 1.9–2.6 minutes (*Hillard et al., 1997*)

Half-life of 2-AG = 8-16 minutes (*Kozak et al., 2001*)

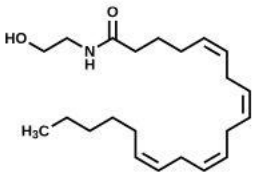


CB1 Receptor Overview



2-AG

full agonist



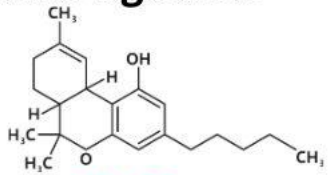
AEA

partial agonists

orthosteric ligands:

allosteric ligands:

CBD



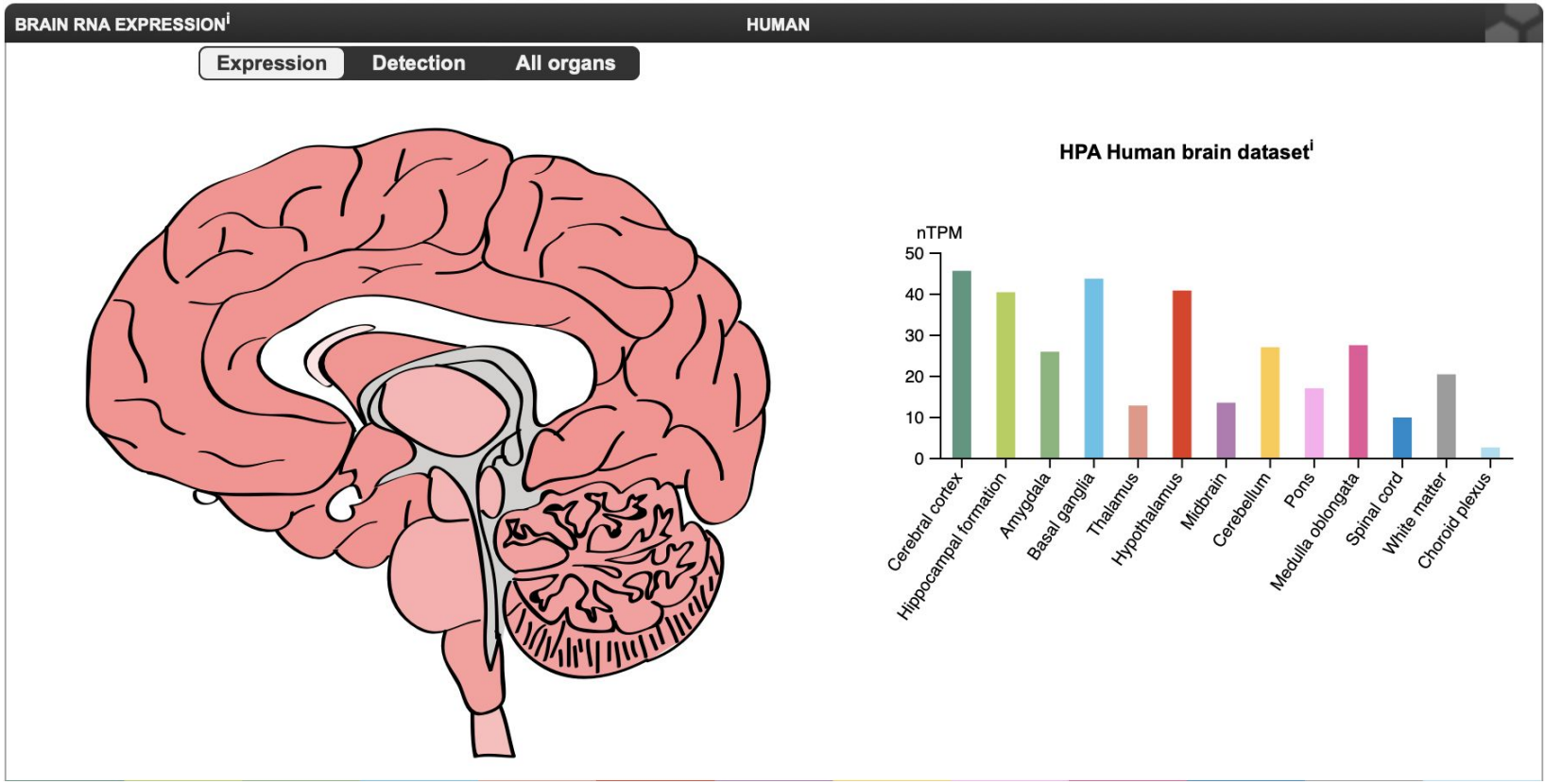
THC

Cannabinoid 1 Receptor

- **G protein signaling (Gai/o)**
- **Adenylate cyclase inhibition**
- **Ion channel modulation**
- **Gene expression changes**

CB1 Expression

THE HUMAN PROTEIN ATLAS



Brain Regions:

- High-density areas:
  - Basal ganglia (movement control)
  - Cerebellum (motor coordination)
  - Hippocampus (memory formation)
  - Cortex (executive function)
- Low-density areas noted:
  - Brainstem (vital functions)
  - Hypothalamus (homeostatic control)

Cellular Distribution:

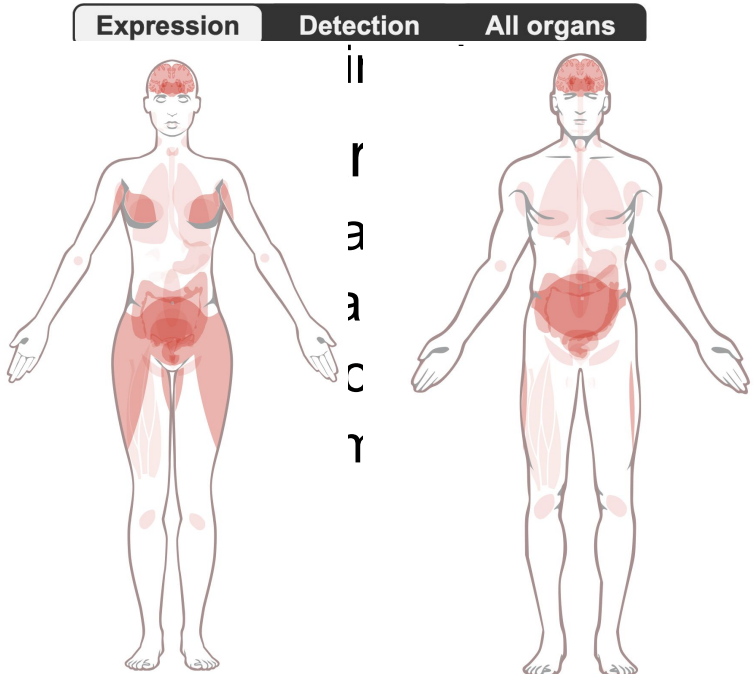
Peripheral CB1 Expression

Metabolic Tissues:

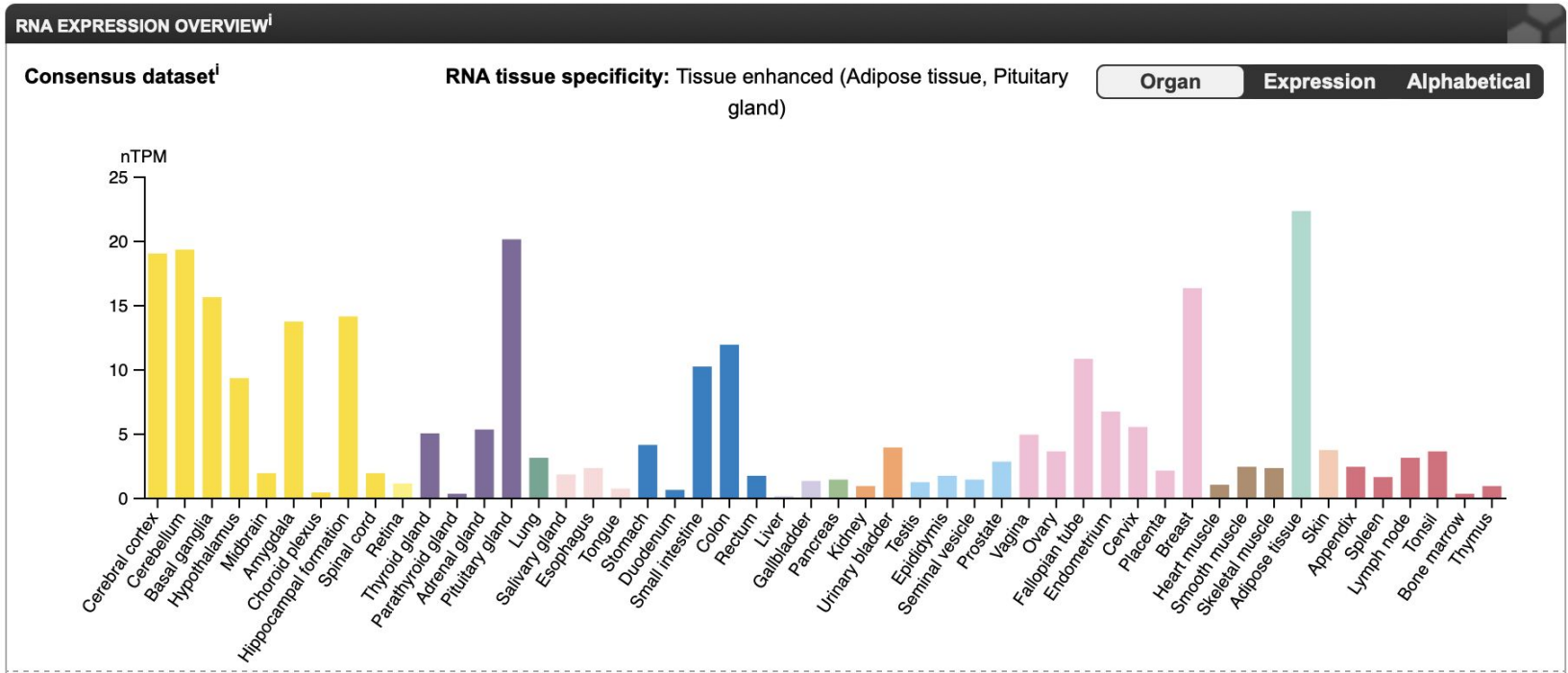
- Adipose tissue (white and brown)
- Liver
- Skeletal muscle
- Pancreatic β-cells

GI System:

- Enteric nervous system
- Vagal afferents

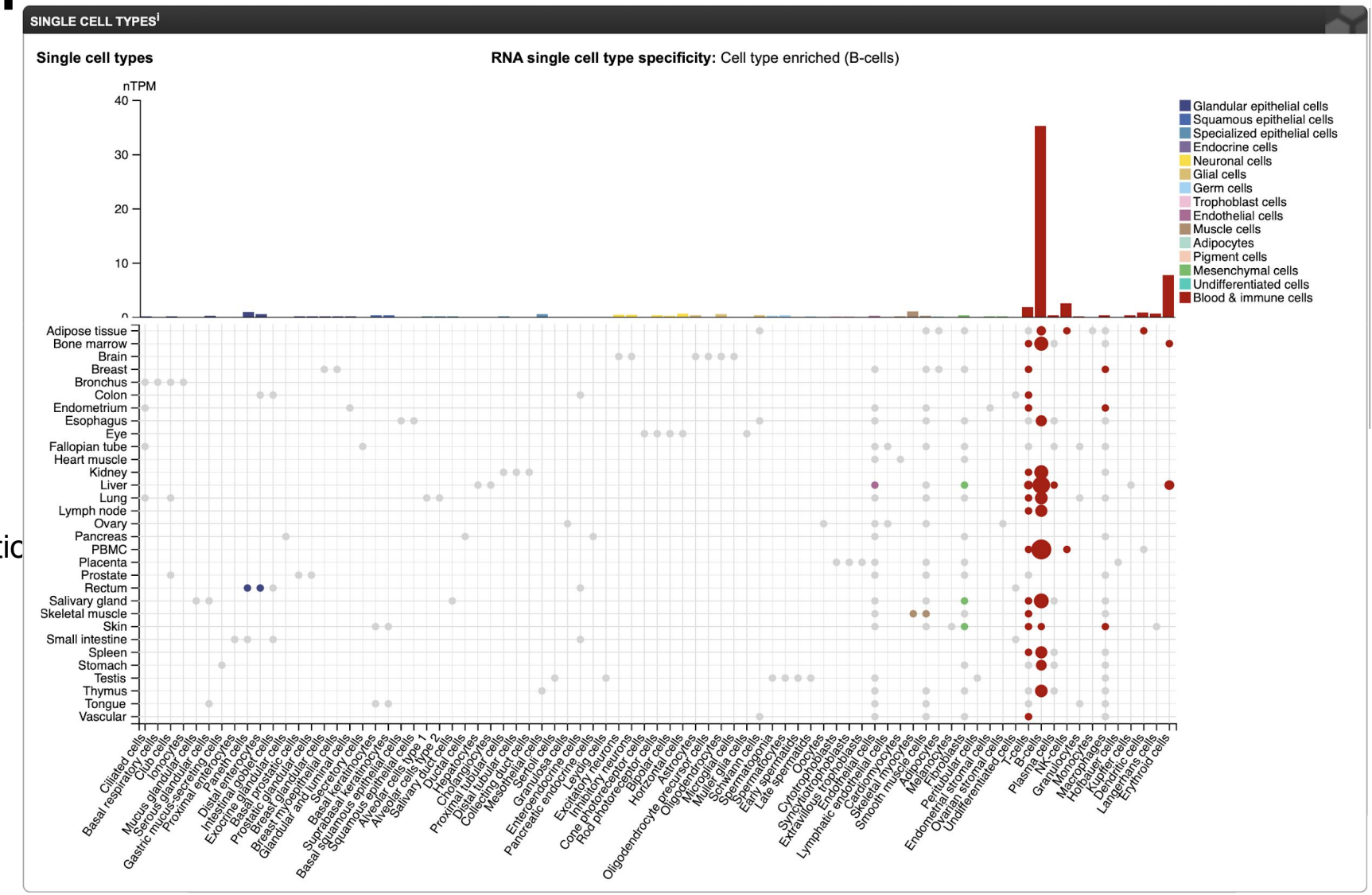


THE HUMAN PROTEIN ATLAS



CB2 Expression

1. Immune System Distribution:
   - Cell types expressing CB2:
     - B cells (highest expression)
     - Macrophages
     - T cells
     - Dendritic cells
     - Microglia
     - Natural killer cells
2. Dynamic Expression:
   - Baseline levels
   - Inflammation-induced upregulatic
   - Disease state changes
   - Tissue-specific patterns
3. Inflammatory Cascade:
   - Cytokine modulation
   - Microglial activation states
   - Resolution pathways
   - Immune cell migration

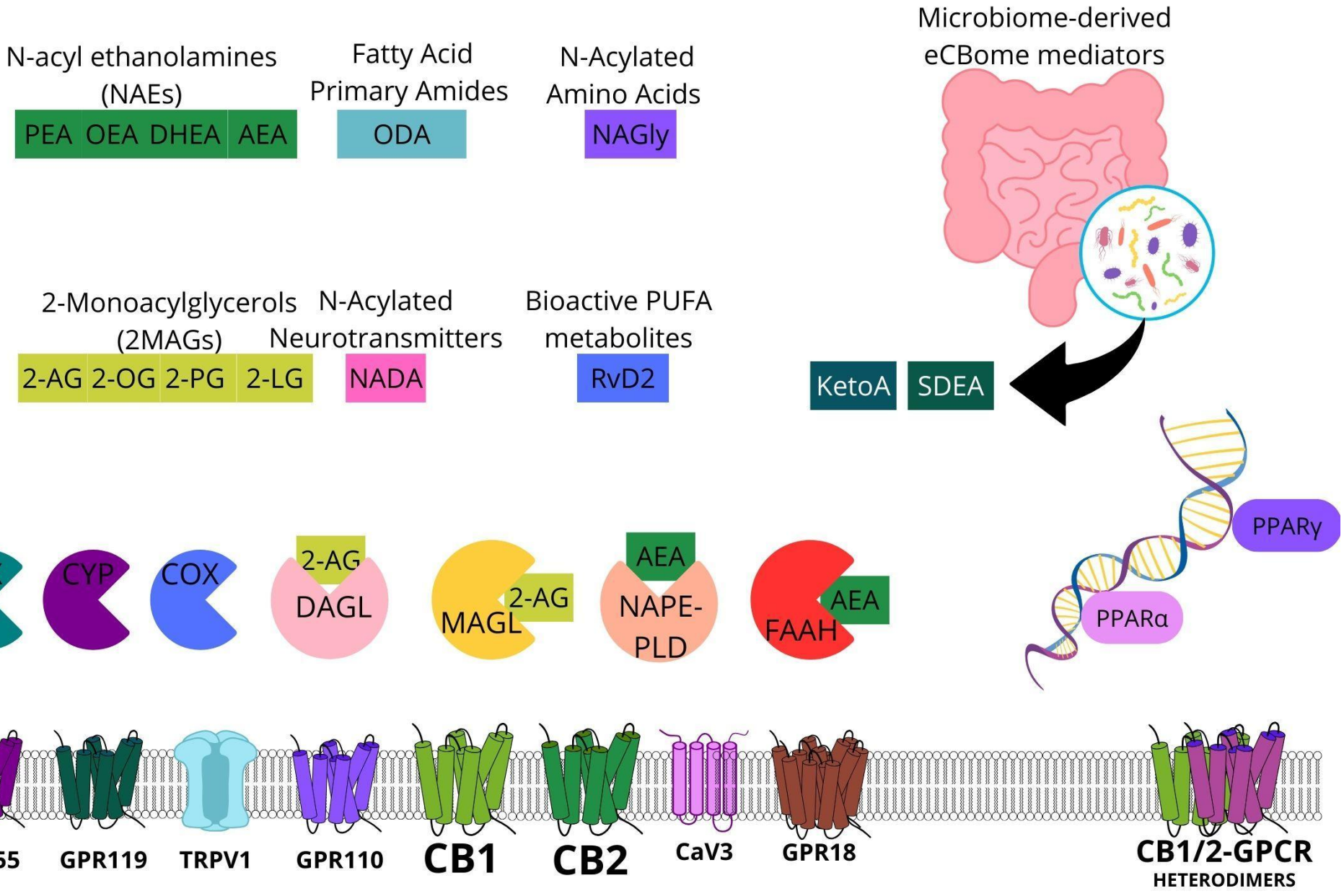


How does the Endocannabinoid System interact with other systems – the eCBome

- In and of itself, the basic endocannabinoid system as discussed has significant influences on mood, pain, inflammation and many other important physiological processes.
- However, its role stretches beyond this in its part of a wider interaction of other signalling molecules and receptors.
- This highlights how the ECS has a wider reaching involvement on our neurology, metabolism and even our microbiome.
- As we will see, cannabinoids like THC and CBD have a massive therapeutic potential for their interaction on the ECS. It is important to remember however that this plays only a part in how clinical interventions can target the ECS and the eCBome.

Slide courtesy of Dr Stefan Broselid

Overview of the eCBome

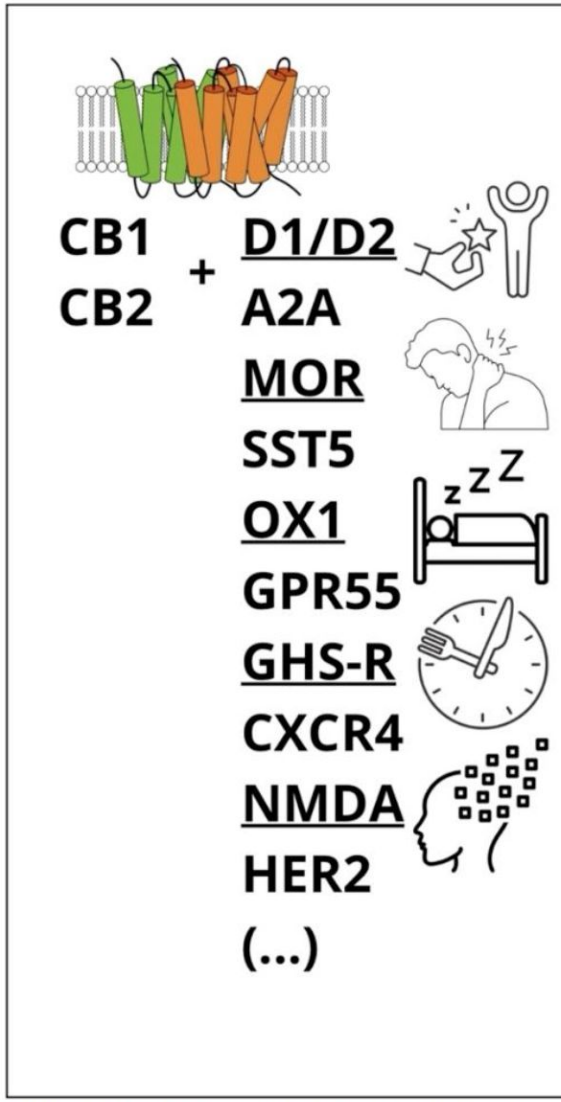


PEA	CB2, PPAR-α, TRPV1, GPR119, GPR55	Anti-inflammatory, analgesic, neuroprotective effects, wakefulness	Anti-inflammatory via PPAR-α activation, analgesic via TRPV1 desensitization, anti-nociception via CB2-mediated activation of endogenous noradrenergic system, neuroprotective antioxidant effects	[24], [67], [68], [69], [131]
LEA	PPAR-α, GPR119	Modulation of pain, inflammation, appetite	Activation of PPAR-α and GPR119, modulation of inflammatory and nociceptive pathways	[70]
DHEA	GPR110 and partial agonist of CB1 and CB2	Neurogenesis, synaptogenesis, anti-inflammatory effects	Activation of GPR110, partial agonism at CB1/CB2 receptors, anti-inflammatory and neuroprotective effects	[71], [72]
AEA	CB1, CB2, TRPV1, PPAR-α, CaV	Neuromodulation, pain modulation, appetite, mood, memory, thermoregulation, sleep	Retrograde signaling at synapses, modulation of neurotransmitter release, anti-inflammatory via PPAR-α and TRPV1 activation, immune cell modulation via CB2	[6], [7], [67]
2-AG	**CB1, CB2**, PPAR-γ, CaV	Neuromodulation, energy balance, immune function, pain modulation	Retrograde messenger, presynaptic inhibition of neurotransmitter release, anti-inflammatory via PPAR-γ activation, modulation of immune cells via CB2	[22], [73]
2-OG	GPR119	Regulation of glucose-dependent insulinotropic peptide	Activation of GPR119 on pancreatic islet cells, modulation of insulin secretion	[74]
2-PG	CB1	Functional CB1 receptor antagonist, modulation of endocannabinoid pharmacokinetics	Competitive antagonism at CB1 receptors, modulation of endocannabinoid metabolism and signaling	[75], [76]
2-LG	CB1	Partial agonist at CB1, suppression of endocannabinoid activity	Partial agonism at CB1 receptors, modulation of endocannabinoid signaling and effects	[77]
NAGly	GPR18, CaV	Suppresses tonic inflammatory pain	Activation of GPR18, modulation of inflammatory pain pathways	[78]
ODA	CB1, PPARα, CaV	Sleep induction, neuroprotection, neurogenesis	Oleamide (ODA) is a full gonist at CB1, activation of PPARα	[127], [128]
RsD2	GPR18, CaV	Resolution of inflammation, anti-inflammatory, neuroprotective	Activation of GPR18, modulation of inflammatory pathways and resolution of inflammation.	[129]
NADA	CB1, CB2, TRPV1, FAAH, CaV	Neuroprotection, pain modulation	Activation of CB1, CB2, TRPV1; inhibition of FAAH	[130]
SCFAs	GPR41, GPR43	Modulation of gut microbiome composition, influence eCBome mediator levels	Short Chain Fatty Acids (SCFAs) like butyrate, propionate, iso-propionate and acetate, modulate gut microbiome composition, in turn modulating eCBome mediator levels.	[31]
THC	CB1, CB2, GPR55, TRPV1, PPAR-γ, GPR18, opioid receptors, others	Pain relief, anti-nausea, appetite stimulation, psychoactive effects, modulation of immune responses, anti-inflammatory effects	Partial agonism at CB1 and CB2 receptors, activation of GPR55, TRPV1, PPAR-γ, GPR18 and others, modulation of various signaling pathways	[12], [13]
CBD	5-HT1A, GPR55, TRPV1, PPAR-α, PPAR-γ, CB1 (negative allosteric modulator), opioid receptors, others	Anti-inflammatory, anxiolytic, anticonvulsant, neuroprotective effects, modulation of immune responses, potential antiviral effects, analgesic effects	Negative allosteric modulation of CB1, activation of 5-HT1A, GPR55, TRPV1, PPAR-γ and others, modulation of various signaling pathways	[13], [14]
CBC	TRPV1, TRPA1	Anti-inflammatory, potential neuroprotectant	Activation of TRPV1 and TRPA1, modulation of inflammatory pathways	[79]
CBG	CB1, CB2, α2-adrenoceptors, PPAR-γ, 5HT1a	Analgesic, anti-inflammatory, neuroprotective	Partial agonism at CB1 and CB2 receptors, modulation of inflammatory and neuroprotective pathways, α2-adrenergic effects, antagonism at 5HT1a receptor	[80], [81]
CBN	CB1, CB2, TRPA1, PPAR-γ	Sedative, anti-inflammatory, analgesic, neuroprotective	Partial agonism at CB1 and CB2 receptors, activation of TRPV1 and PPAR-γ, modulation of inflammatory and neuroprotective pathways	[82], [83]
THCV	CB1 (antagonist), CB2 (agonist), TRPV1	Appetite suppression, glycemic control, neuroprotective	Antagonism at CB1 receptors, agonism at CB2 receptors, activation of TRPV1, modulation of metabolic processes	[84], [85]
BCP	CB2	Anti-inflammatory, analgesic, gastroprotective	Selective agonism at CB2 receptors, modulation of inflammatory pathways	[132]

Table 1 *eCBome-interacting molecules: Their targets, biological functions, mechanisms of action and supporting references.*

List of molecules shown to interact with the eCBome

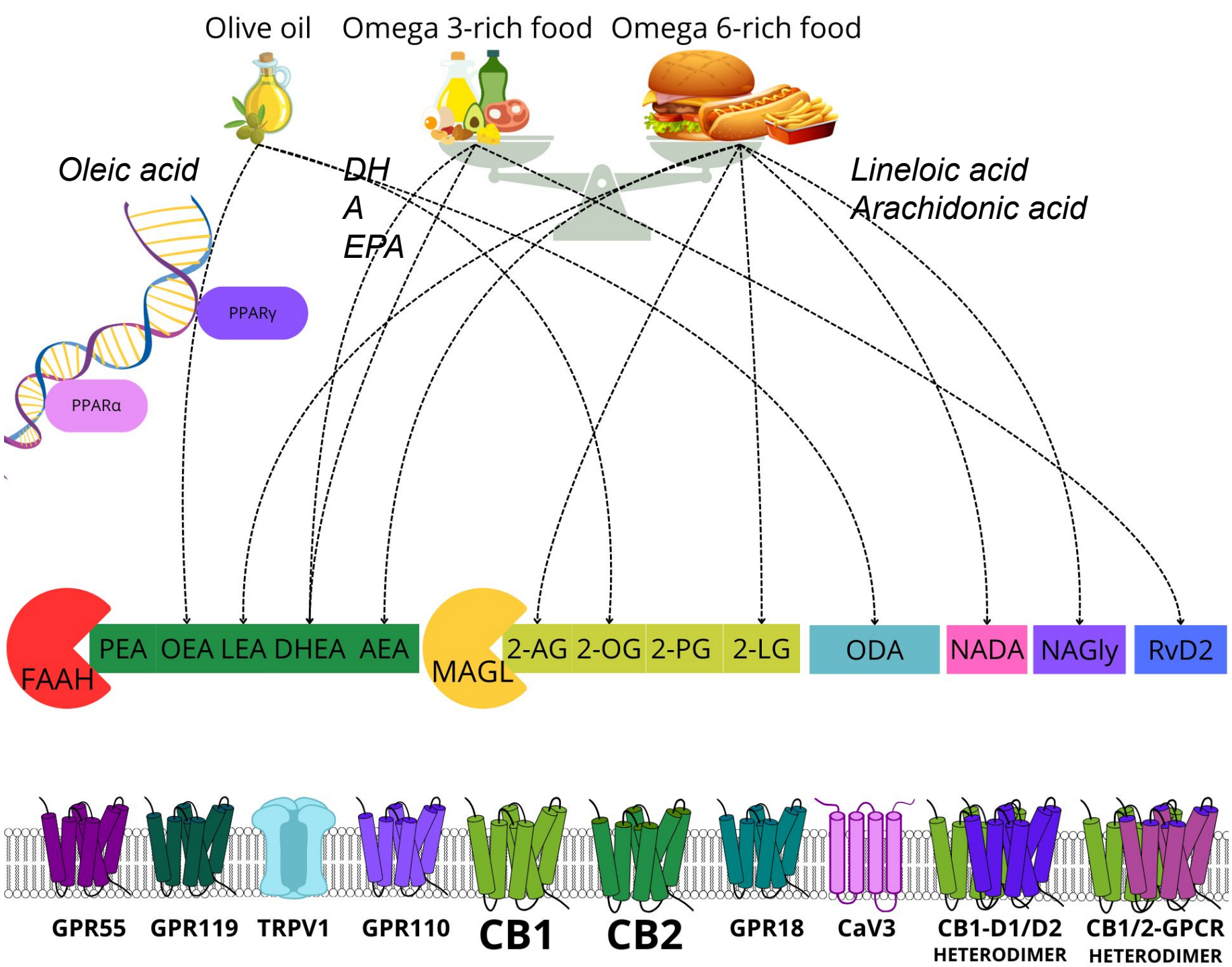
Endocannabinoid heterodimers



CB1 + GPCR heterooligomer	Additional Endogenous Heterodimer Ligands	Physiological Relevance	References
CB2	-	Modulation of immune function, inflammation, pain perception.	[10], [11]
D1/D2	Dopamine	Regulation of motor function, reward processing, cognition.	[86], [87]
μ-Opioid	Endorphins, Enkephalins	Pain perception, reward, respiratory depression.	[88], [89]
A2A	Adenosine	Modulation of locomotor activity, anxiety, neurodegeneration.	[90], [91]
NMDA	Glutamate	Modulation of glutamate signaling, potential neuroprotection in Alzheimer's disease.	[92], 93]
SST5	Somatostatin	Regulation of hormone secretion, neurotransmission, cell proliferation.	[94]
OX1	Orexins	Regulation of sleep/wakefulness, energy homeostasis.	[95], [96]
GPR55	NAGly, Lysophosphatidylinositol (LPI)	Modulation of bone physiology, neuropathic pain, cancer. Implicated in multiple sclerosis.	[97], [98], [19]
GHS-R	Ghrelin	Interactions between ghrelinergic and cannabinoidergic systems in CNS, impacting reward circuit plasticity.	[99]
CB2 + GPCR heterooligomer	**Endogenous Heterodimer Ligand**	**Physiological Relevance**	**References**
CB1	-	Modulation of immune function, inflammation, pain perception.	[10], [11]
GPR55	NAGly, Lysophosphatidylinositol (LPI)	Modulation of cancer cell migration, metastasis. Implicated in multiple sclerosis.	[16], [59]
CXCR4	SDF-1	Regulation of cancer cell migration, metastasis.	[17], [65]
NMDA	Glutamate	Modulation of glutamate signaling, potential neuroprotection in Alzheimer's disease.	[92]
HER2	N.A.	Modulation of tumor progression.	[18], [53]

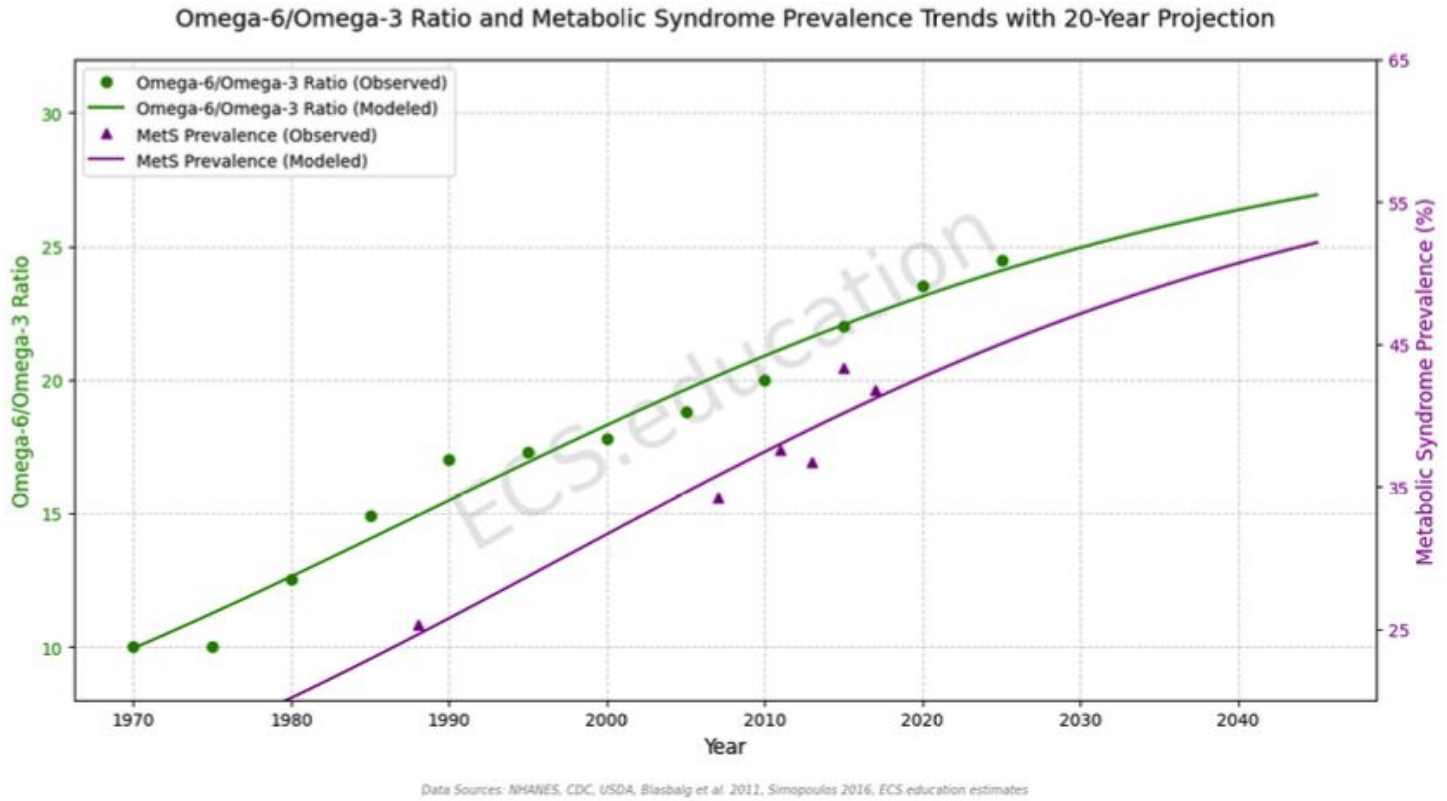
Table 3 *Endocannabinoid Receptor Heterodimers and Their Physiological Relevance*

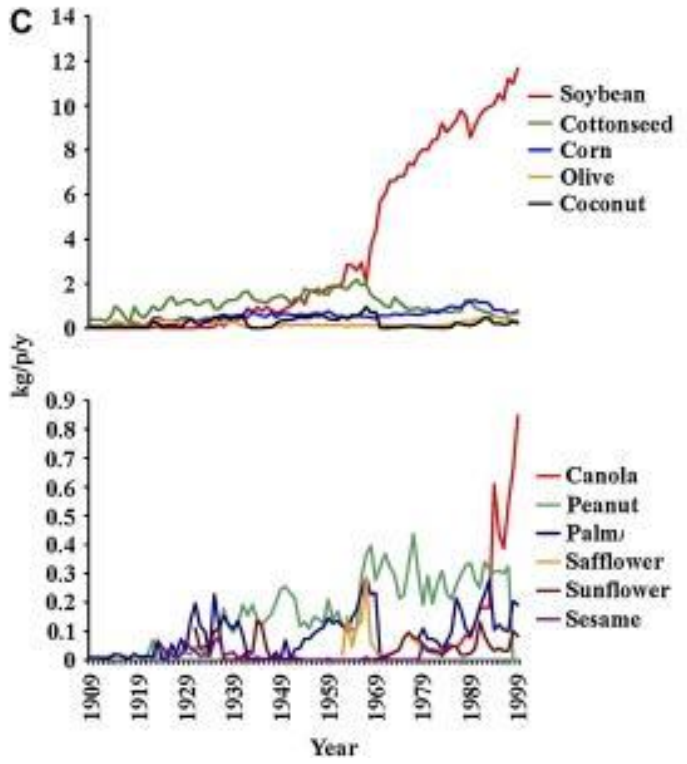
This figure illustrates various heterodimeric complexes formed between the endocannabinoid system (ECS) G protein-coupled receptors (GPCRs) and other receptor types, such as CB1-D1/D2, CB1-μ-Opioid, CB2-GPR55, CB2-CXCR4, and NMDA-CB1. The table provides details on the endogenous ligands that can modulate these heterodimers, their physiological relevance in processes like motor function, pain perception, cancer cell migration, and tumor progression, as well as relevant references supporting these interactions.

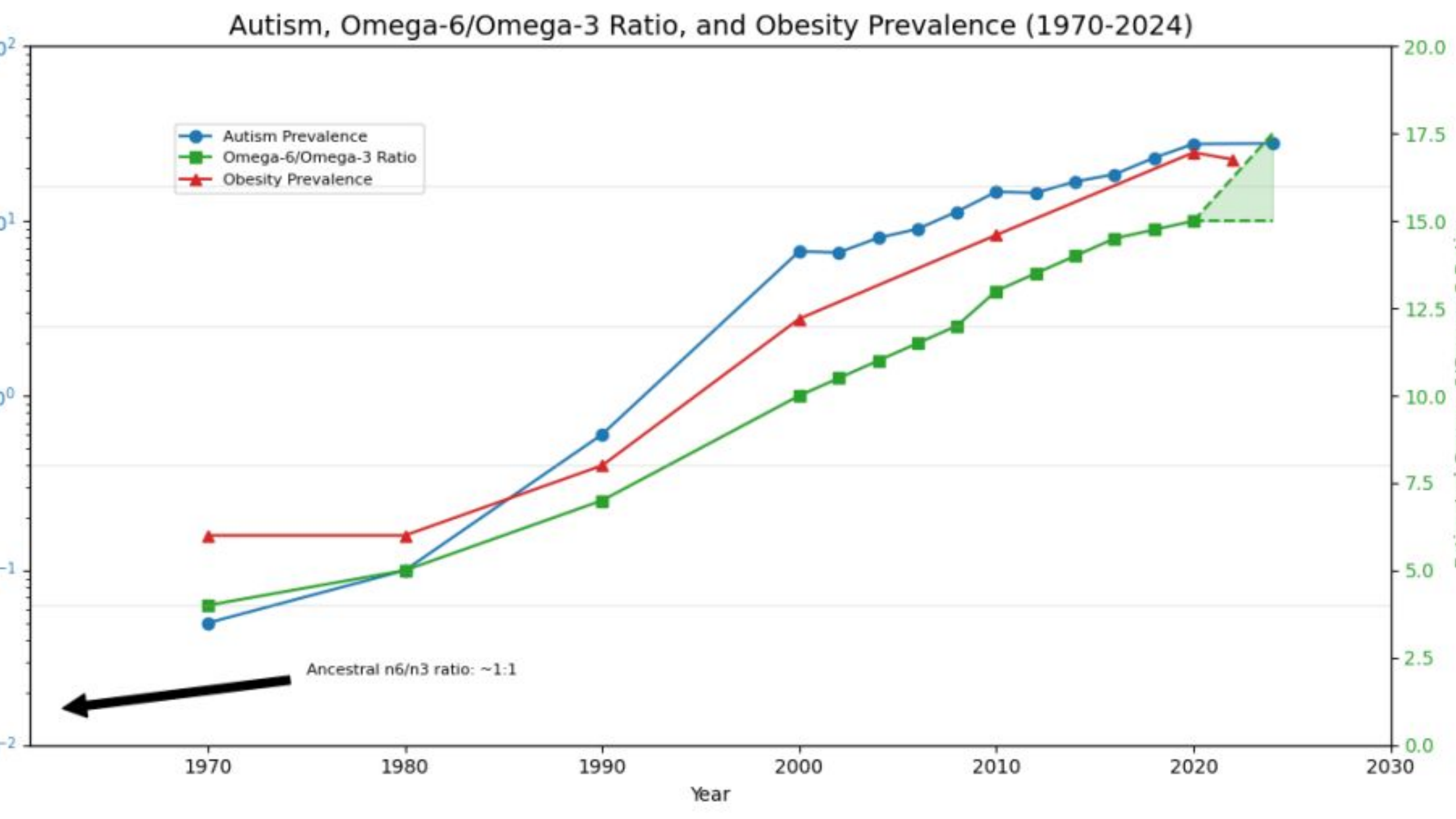

Olive oil
Omega 3-rich food
Omega 6-rich food
Oleic acid
DH
A
EPA
Lineloic acid
Arachidonic acid
PPARγ
PPARα
FAAH
PEA
OEA
LEA
DHEA
AEA
MAGL
2-AG
2-OG
2-PG
2-LG
ODA
NADA
NAGly
RvD2
GPR55
GPR119
TRPV1
GPR110
CB1
CB2
GPR18
CaV3
CB1-D1/D2
HETERODIMER
CB1/2-GPCR
HETERODIMER

Association between dietary fat intake and metabolic syndrome

Tissue-AA drives metabolic syndrome prevalence

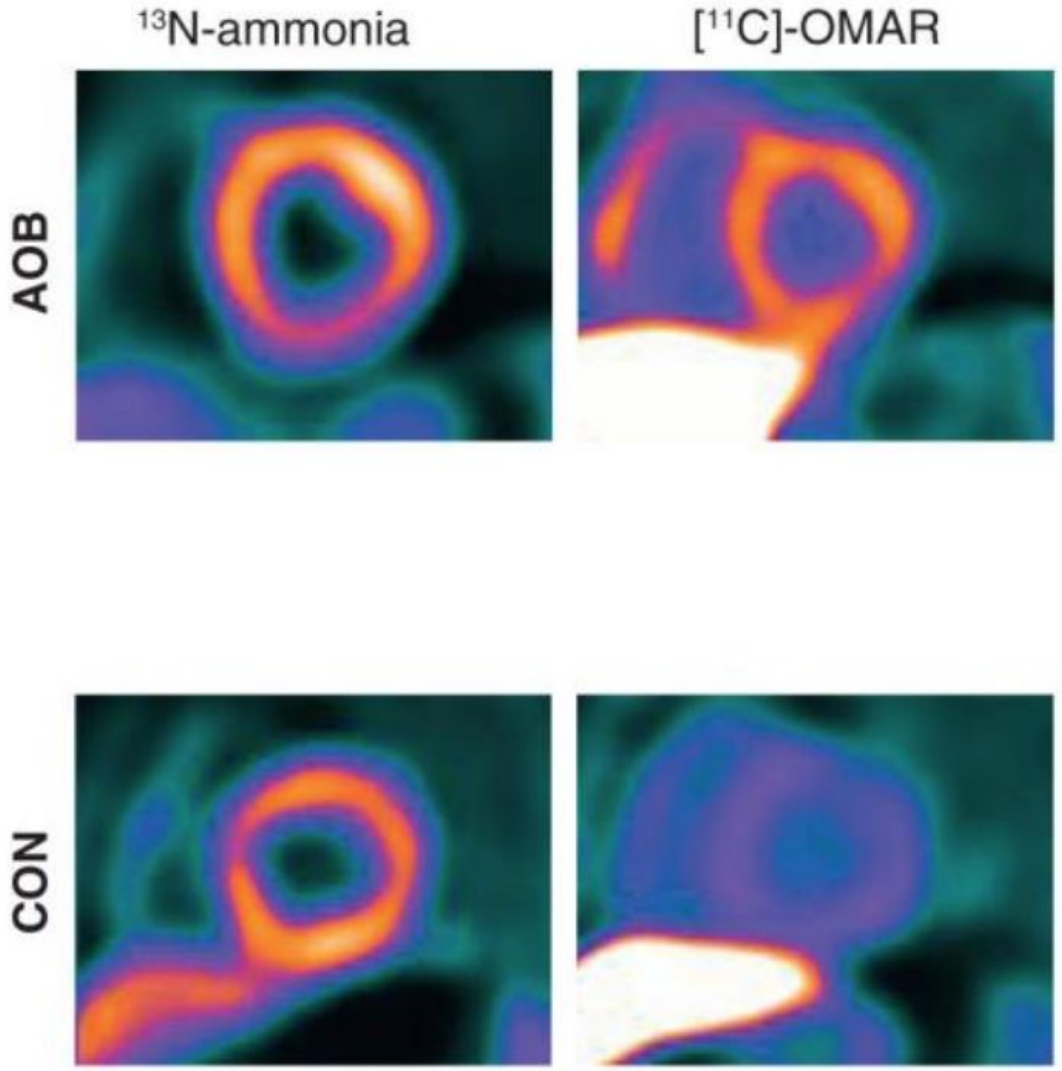





Autism, Omega-6/Omega-3 Ratio, and Obesity Prevalence (1970-2024)
Autism Prevalence
Omega-6/Omega-3 Ratio
Obesity Prevalence
Ancestral n6/n3 ratio: ~1:1
20.0
17.5
15.0
12.5
10.0
7.5
5.0
2.5
0.0
1970
1980
1990
2000
2010
2020
2030
Year

The ECS in cardiac tissues in obesity

- In healthy individuals with obesity (AOB) CB1 expression is notably higher on PET than in controls.



Research Articles

The Association Between the Dietary Fatty Acid Fraction and Healthy Life Expectancy: Global Spatiotemporal Epidemiology from 2010 to 2019

Yoshiro Shirai, Tomoko Imai, Chisato Abe, Ayako Sezaki, Keiko Miyamoto, Fumiya Kawase, ...show all

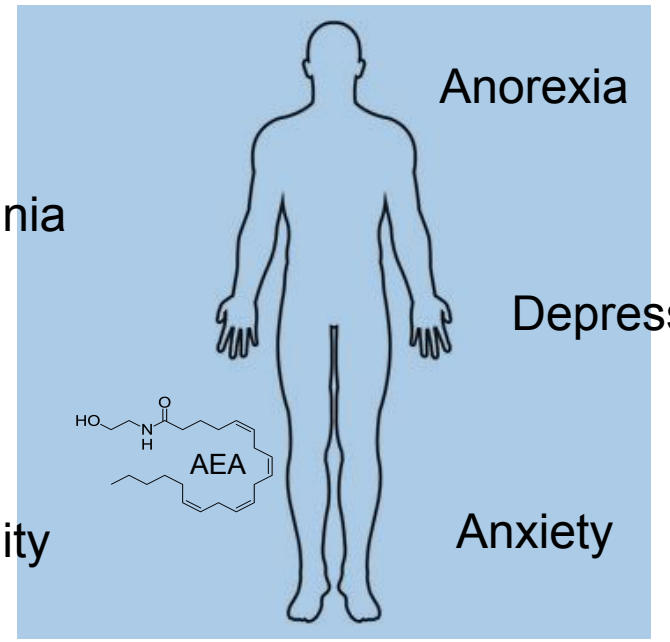
Pages 591-598 | Received 21 Nov 2024, Accepted 23 Feb 2025, Published online: 10 Mar 2025

Cite this article https://doi.org/10.1080/27697061.2025.2472656 Check for updates

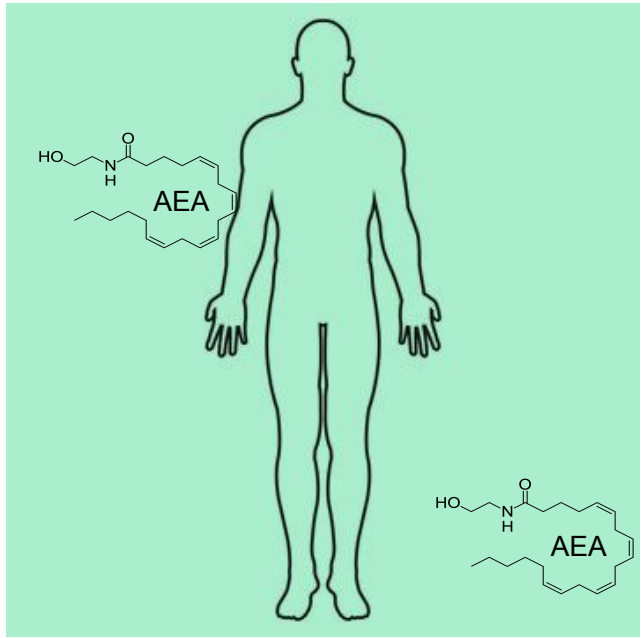
Summary on diet and the eCBome

- Healthy life expectancy in a population consuming high omega-3 diets is 2.6 years longer than in those with high omega-6 diets.
- Dietary supplementation with 3-4g of omega-3 per day has been shown to significantly reduce circulating levels of prostaglandins and cytokines.
- eCBome activity is, at least in part, substrate driven and determined by our dietary fat intake.

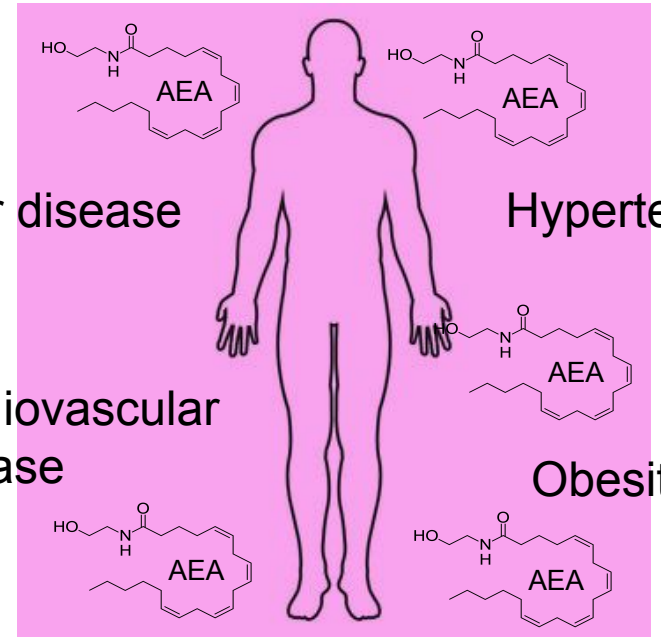
The ECS and pathology – the importance of ECS tone



Low ECS Tone



Normal ECS Tone



High ECS Tone

ECS Dysfunction Patterns - Reduced tonic signalling

Disease	Comment	Supporting References
Migraine	Strong evidence of reduced endocannabinoid tone in migraine patients.	Russo, 2016; Greco et al., 2010
Fibromyalgia	Clinical studies show low endocannabinoid levels in fibromyalgia.	Russo, 2016; Clauw, 2014
Irritable Bowel Syndrome (IBS)	IBS patients exhibit reduced endocannabinoid activity in the gut.	Russo, 2016; Storr et al., 2008
Anxiety Disorders	ECS dysregulation linked to anxiety-like behaviours in preclinical and clinical studies.	Hill et al., 2009; Morena et al., 2016
Autism Spectrum Disorder (ASD)	Reduced AEA and 2-AG levels are core features of ASD.	Karhson et al., 2018; Aran et al., 2019
Depression	Low endocannabinoid tone associated with mood dysregulation.	Hill et al., 2008; Gobbi et al., 2005
Post-Traumatic Stress Disorder (PTSD)	Hypofunction of ECS observed in PTSD patients.	Neumeister et al., 2013; Hill et al., 2018
Parkinson’s Disease	Defects in ECS metabolism linked to motor symptoms in Parkinson’s.	Pisani et al., 2005; van der Stelt et al., 2005
Multiple Sclerosis (MS)	ECS dysregulation contributes to spasticity and inflammation in MS.	Baker et al., 2001; Centonze et al., 2007

ECS Dysfunction Patterns – Excessive tonic signalling

Disease	Comment	Supporting References
Obesity	Elevated 2-AG levels correlate with increased appetite, fat storage, and CB1 overactivation.	Engeli et al., 2005; Di Marzo et al., 2001
Metabolic Syndrome	Increased endocannabinoid tone contributes to insulin resistance and dyslipidemia.	Di Marzo et al., 2004; Pagano et al., 2007
Non-Alcoholic Fatty Liver Disease (NAFLD)	CB1 overactivation promotes hepatic fat accumulation and inflammation.	Osei-Hyiaman et al., 2008; Mallat et al., 2011
Polycystic Ovary Syndrome (PCOS)	Elevated endocannabinoids linked to ovarian dysfunction and metabolic abnormalities.	Macut et al., 2018; El-Talatini et al., 2010
Endometriosis	Elevated levels of 2-AG and reduced expression of CB1 demonstrated in endometriosis	Maia et al 2019.
Addiction	CB1 overactivation in reward circuits contributes to substance use disorders.	Maldonado et al., 2006; Parsons & Hurd, 2015
Schizophrenia	Dysregulated ECS activity associated with psychotic symptoms and cognitive deficits.	Giuffrida et al., 2004; Leweke et al., 2012
Cancer (Certain Types)	Elevated endocannabinoids promote tumor growth and angiogenesis in some cancers.	Pisanti et al., 2013; Velasco et al., 2012
Cardiovascular Disease (CVD)	ECS overactivity linked to hypertension, atherosclerosis, and cardiac remodelling.	Pacher & Mechoulam, 2011; Montecucco & Di Marzo, 2012
Alzheimer’s Disease	Increased 2-AG and CB1-5HT expression in hippocampus of Alzheimer’s animal models.	Chen et al., 2011; Guida et al., 2024

Disorders with Mixed Phenotypes of ECS Dysfunction

Disorder	ECS Dysfunction	Comment	Supporting References
Anorexia Nervosa (AN)	Compensatory CB1 receptor upregulation; elevated AEA levels	ECS dysregulation reflects a maladaptive response to chronic starvation and altered reward processing.	Gérard et al., 2011; Monteleone et al., 2005
Schizophrenia	CB1 overactivation in some circuits; underactivation in others	Dysregulated ECS activity contributes to psychotic symptoms and cognitive deficits.	Giuffrida et al., 2004; Leweke et al., 2012
Bipolar Disorder	Fluctuating ECS tone	ECS dysfunction correlates with mood instability and altered reward processing.	Ashton et al., 2005; Chavarria-Siles et al., 2008
Bulimia Nervosa (BN)	Elevated AEA levels; altered CB1 receptor expression	ECS dysregulation impacts reward-related overeating and compensatory behaviors.	Monteleone et al., 2005; Frieling et al., 2009
Obesity with Food Addiction	CB1 overactivation in reward circuits	Excessive ECS signaling drives compulsive eating behaviors.	Di Marzo et al., 2009; Engeli et al., 2005
Endometriosis	Dysregulated CB1/CB2 signaling	ECS dysfunction contributes to pain, inflammation, and immune dysregulation in lesions.	3; McHugh et al., 2012

ECS Dysfunction Patterns

Pharmacological Approaches:

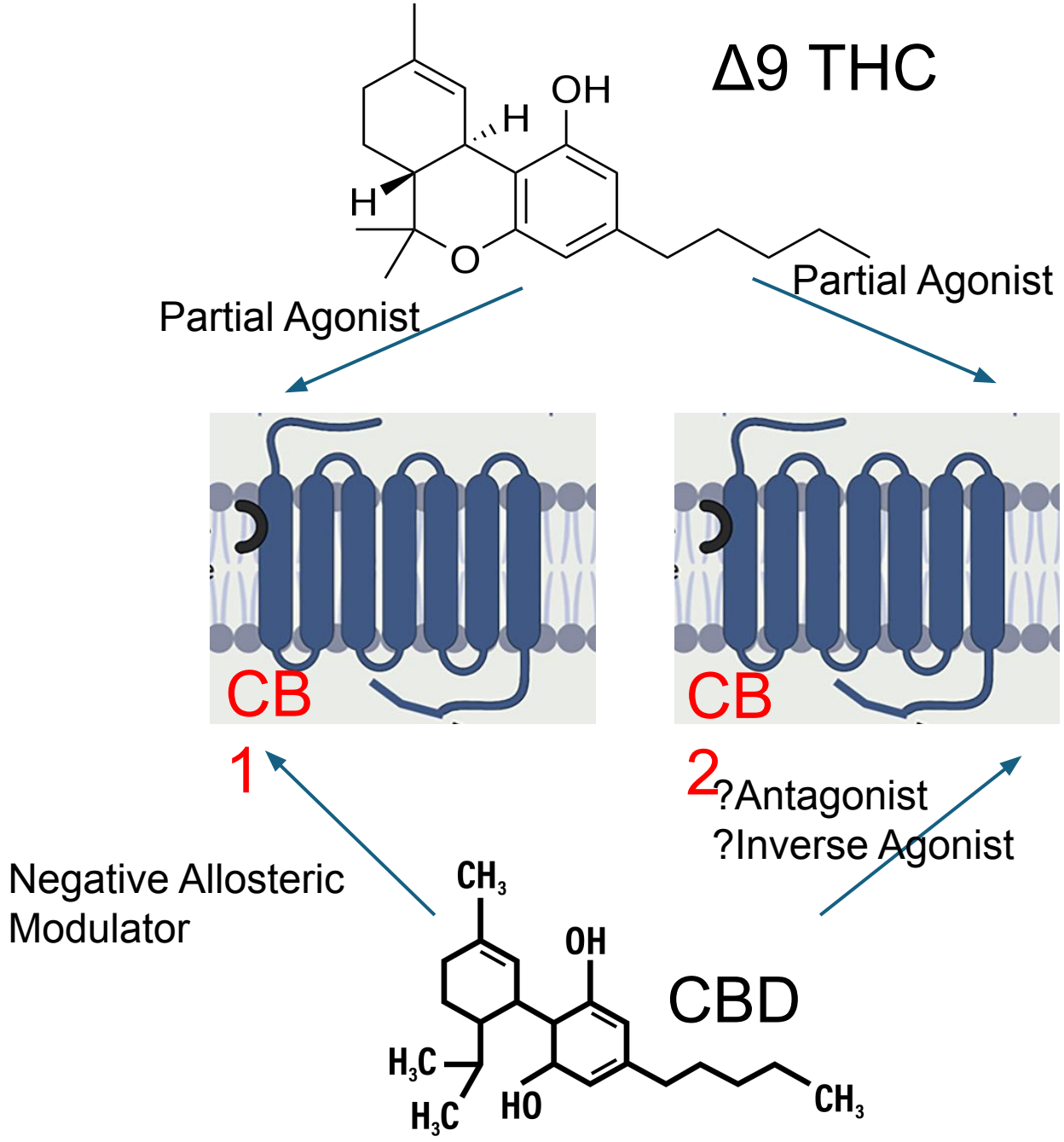
- CB1/CB2 Agonists: Directly activate cannabinoid receptors.
  - Example: Δ9-tetrahydrocannabinol (THC) for pain and spasticity.
- FAAH Inhibitors: Increase anandamide (AEA) levels by preventing its breakdown.
  - Example: PF-04457845 (investigational).
- MAGL Inhibitors: Elevate 2-AG levels by inhibiting its degradation.
  - Example: JZL184 (preclinical research).

Dietary Interventions:

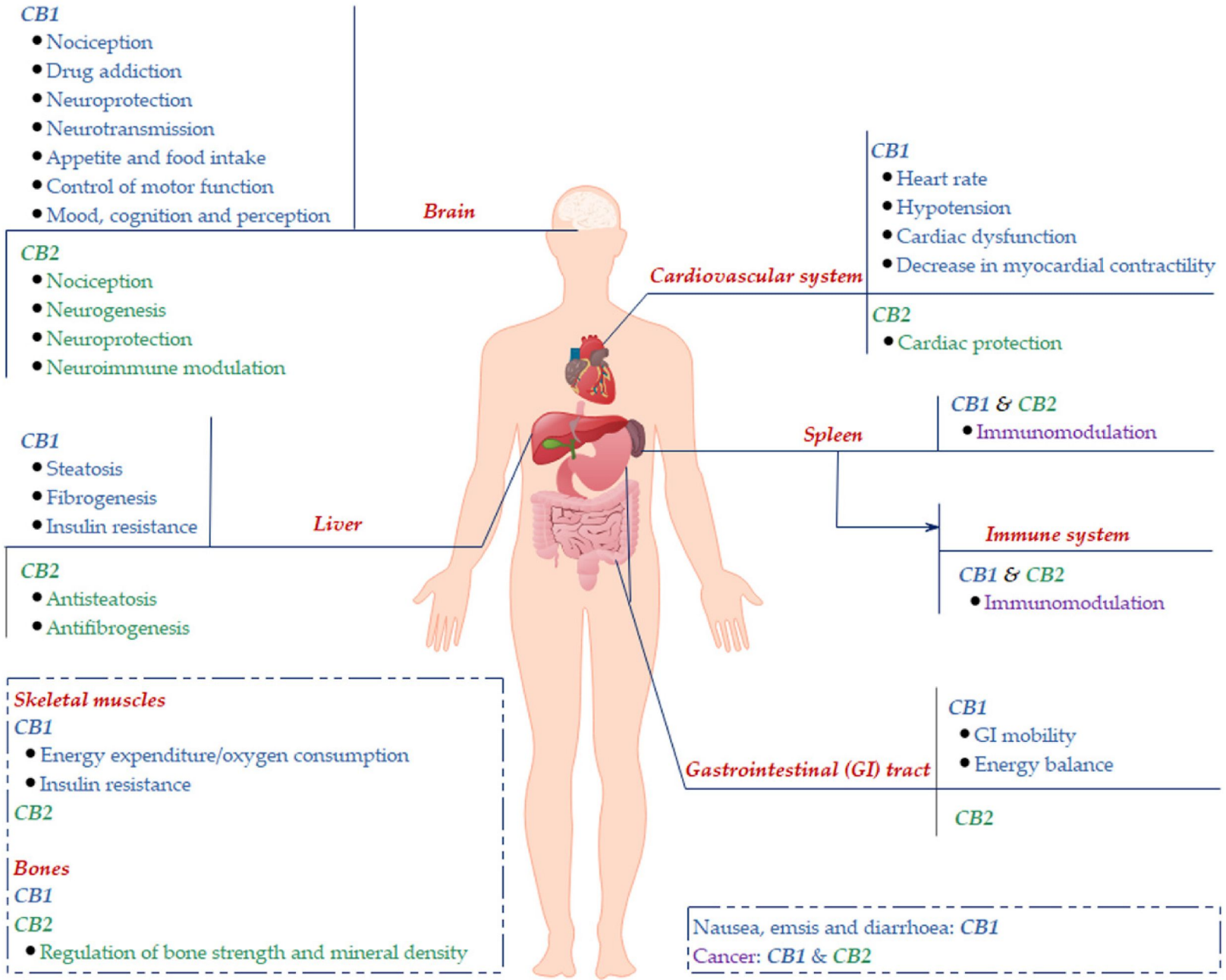
- Omega-3 Fatty Acids: Normalize endocannabinoid synthesis and receptor function.
  Sources: Fish oil, flaxseed, walnuts.
- Reduced Omega-6 Intake:
  - Mitigates excessive pro-inflammatory eicosanoid production.

Cannabinoid-Based Therapies:

Phytocannabinoids and their action on the ECS



Distribution of CB1 and CB2 receptors



The varying effects of THC

- Limited correlation between plasma THC levels and patient reported sedation.
- Vastly varying effects in dose-dependent response between naïve patients.
- Chronic THC usage dampens CB1 expression however managing the acute phase of THC use can be challenging in sensitive patient populations.

Potential clinical uses of CBD

Disease	Comment	Supporting References
Epilepsy inc. treatment resistant Epilepsy	CBD shown to be effective in treatment of treatment resistant epilepsy.	Reis et al., 2020; Gray et al., 20203
Anxiety and Depression	Anxiolytic effect of CBD established with role in reducing THC induced anxiety also significant.	Blessing et al., 2015
ADHD and Autism	Majority of significant benefit in management of paediatric ADHD and autism with cannabinoids associated with CBD	Black et al., 2018; Cooper et al., 2017; Amaro de Silva et al., 2022
Post Traumatic Stress Disorder	CBD in the management of PTSD with associated CUD remains an interesting therapeutic option	Rodas et al., 2024
Pain	As well as reducing THC induced side effects, the anti-inflammatory and analgesic potential for broad spectrum and isolate CBD has been demonstrated	Casedas et al., 2024
Inflammation	CBD demonstrated to reduce serum levels of pro-inflammatory cytokines in animal models	Henshaw et al., 2021
Schizophrenia	CBD shown to reduce the levels of positive psychotic symptoms and improve cognitive performance in patients with schizophrenia	McGuire et al., 2017
Substance Misuse	The potential for CBD to contribute to the management of alcohol and opioid abuse is significant.	Navarrete et al., 2021
Sleep	Anxiolytic effects of CBD can contribute to management of insomnia whilst minor cannabinoids like CBG and CBN may offer significant therapeutic potential.	AminLari et al., 2022; Lavendar et al., 2023

Potential clinical uses of THC

Disease	Comment	Supporting References
Sleep	Registry data supports cannabis for sleep however ongoing debate as to if THC is the optimal compound for this	Lynskey et al., 2023; AminLari et al., 2022; Kaul et al., 2021
Spasticity	Cannabis widely shown to be effective for spasticity and pain secondary to MS	Nielsen et al., 2018
Pain – Neuropathic and Nociplastic	Systematic reviews show possible significant benefit of THC in chronic pain whilst registry data is overwhelmingly positive	McDonagh et al., 2022; Athanasiou-Fragkouli et al., 2024
Opioid reduction	THC has been shown to reduce opiate use by more than 60% in some patient populations	Boehnke et al., 2016
Migraine	Systematic review on THC for migraine found it to be effective and well tolerated	Sherpa et al., 2022
Anxiety and depression	Literature divided on THC usage in anxiety and depression however these remain amongst the most prevalent indications for UK prescribing	Lev-Ran et al., 2013
ADHD and Autism	Minimal evidence for benefit of THC in paediatric ADHD however some adult studies show promise. Use of THC in autism should be secondary to CBD	Black et al., 2018; Cooper et al., 2017; Amaro de Silva et al., 2022
Epilepsy	Multiple case reports of drug and CBD resistant epilepsy being responsive to THC	Nowicki et al., 2022
Neurodegenerative Conditions	Potential for cannabis treatment to improve symptoms of PD	Urbi et al., 2021

The future of cannabinoid medicine – extending beyond symptom control

Journal of the American Heart Association
Volume 13, Issue 5, 5 March 2024
https://doi.org/10.1161/JAHA.123.030178

ORIGINAL RESEARCH

Association of Cannabis Use With Cardiovascular Outcomes Among US Adults

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BACKGROUND: We examined the association between cannabis use and cardiovascular outcomes among the general population, among never-tobacco smokers, and among younger individuals.

METHODS AND RESULTS: This is a population-based, cross-sectional study of 2016 to 2020 data from the Behavioral Risk Factor Surveillance Survey from 27 American states and 2 territories. We assessed the association of cannabis use (number of days of cannabis use in the past 30 days) with self-reported cardiovascular outcomes (coronary heart disease, myocardial infarction, stroke, and a composite measure of all 3) in multivariable regression models, adjusting for tobacco use and other characteristics in adults 18 to 74 years old. We repeated this analysis among nontobacco smokers, and among men <55 years old and women <65 years old who are at risk of premature cardiovascular disease. Among the 434 104 respondents, the prevalence of daily and nondaily cannabis use was 4% and 7.1%, respectively. The adjusted odds ratio (aOR) for the association of daily cannabis use and coronary heart disease, myocardial infarction, stroke, and the composite outcome (coronary heart disease, myocardial infarction, and stroke) was 1.16 (95% CI, 0.98–1.38), 1.25 (95% CI, 1.07–1.46), 1.42 (95% CI, 1.20–1.68), and 1.28 (95% CI, 1.13–1.44), respectively, with proportionally lower log odds for days of use between 0 and 30 days per month. Among never-tobacco smokers, daily cannabis use was also associated with myocardial infarction (aOR, 1.49 [95% CI, 1.03–2.15]), stroke (aOR, 2.16 [95% CI, 1.43–3.25]), and the composite of coronary heart disease, myocardial infarction, and stroke (aOR, 1.77 [95% CI, 1.31–2.40]). Relationships between cannabis use and cardiovascular outcomes were similar for men <55 years old and women <65 years old.

Key messages

Endocannabinoid Tone and the eCBome extends beyond cannabis, with both dietary fatty acids and exercise playing a significant role in ensuring healthy endocannabinoid tone.

The future of cannabis medicine extends beyond symptom control, with the eCBome offering a fantastic target for novel treatments aimed at a range of pathologies.

THC and CBD offer valuable therapeutic options however more focus is needed on how underlying endocannabinoid activity is affecting the action of these and other phytocannabinoids.

ECS teaching within Medical Schools

- The MCCS recognises the importance of delivering education on the ECS and its clinical implications at an undergraduate level.
- We have approached every mainland UK medical school offering to deliver teaching on this important area of physiology. We believe every medical school should offer its students the opportunity to learn about the ECS.
- If you are connected to a medical school or a university and feel you can help us in our goal, please get in touch.
- Additionally, if you are not yet a member of the MCCS, please visit us online at ukmccs.org to find out how you can join.
- contact@ukmccs.org

Medical Cannabis Clinicians Society