Exhibit 13.1

Q1 2018

emerald™ HEALTH PHARMACEUTICALS

Developing medicines based on cannabinoid science

Jim DeMesa, MD, MBA
Chief Executive Officer

Important Notice

Emerald Health Pharmaceuticals Inc. (the Company) is currently “Testing the Waters” under Regulation A+ (Regulation A+) under the Securities Act of 1933, as amended. The offering will be made only by means of an offering circular. This presentation shall not constitute an offer to sell or the solicitation of an offer to buy, nor shall there be any sale of these securities in any state or jurisdiction in which such offer, solicitation or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction. No money or other consideration is being solicited at this time, and if sent in response to these materials, it will not be accepted. No securities may be sold, and no offer to buy securities can be accepted and no part of the purchase price can be received for an offering under Regulation A+ until an offering statement is qualified by the U. S. Securities and Exchange Commission, and any such offer may be withdrawn or revoked, without obligation or commitment of any kind, at any time before notice of its acceptance given after the qualification date. A person's indication of interest in a Regulation A+ offering is non-binding and involves no obligation or commitment of any kind.

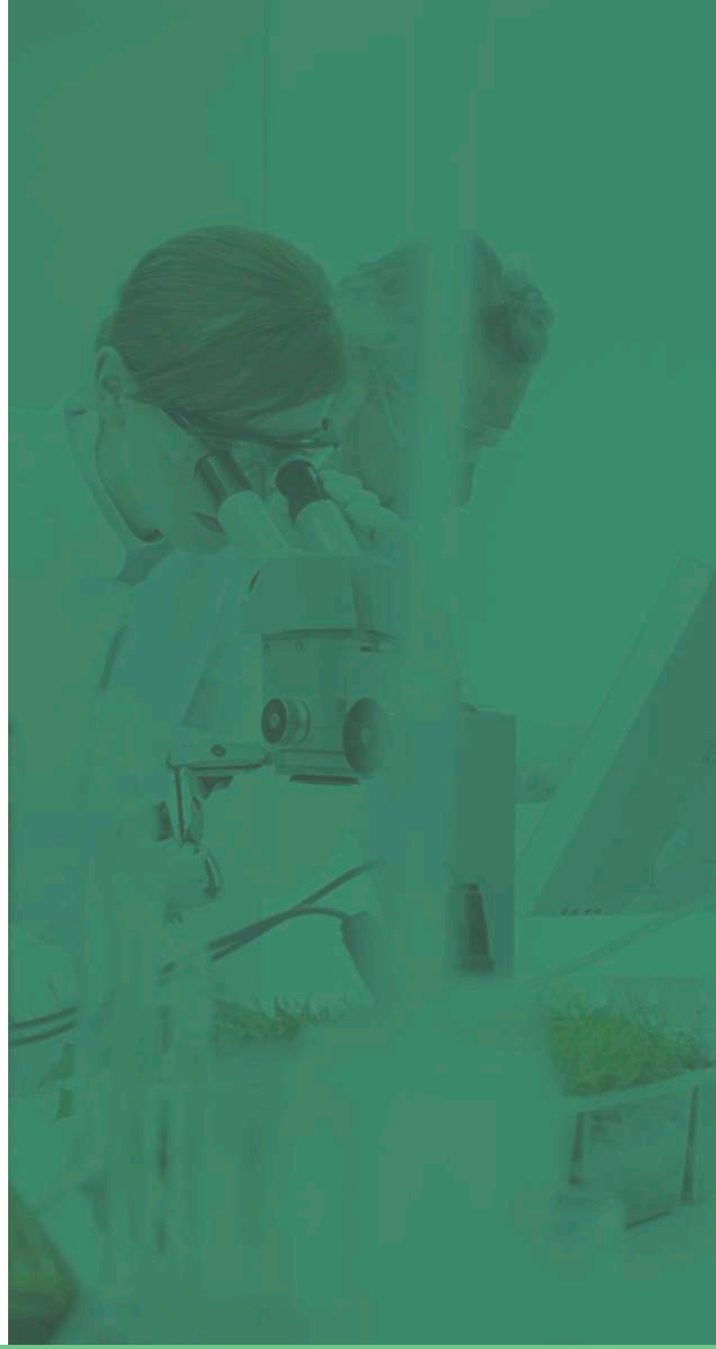
Cautionary Note Regarding Forward-Looking Statements

TO THE EXTENT STATEMENTS CONTAINED IN THE FOLLOWING PRESENTATION ARE NOT DESCRIPTIONS OF HISTORICAL FACTS REGARDING THE COMPANY, THEY SHOULD BE CONSIDERED “FORWARD-LOOKING STATEMENTS,” AS DESCRIBED IN THE PRIVATE SECURITIES LITIGATION REFORM ACT OF 1995, THAT REFLECT MANAGEMENT’S CURRENT BELIEFS AND EXPECTATIONS. YOU CAN IDENTIFY FORWARD-LOOKING STATEMENTS BY WORDS SUCH AS “ANTICIPATE,” “BELIEVE,” “COULD,” “ESTIMATE,” “EXPECT,” “FORECAST,” “GOAL,” “HOPE,” “HYPOTHESIS,” “INTEND,” “MAY,” “PLAN,” “POTENTIAL,” “PREDICT,” “PROJECT,” “SHOULD,” “STRATEGY,” “WILL,” “WOULD,” OR THE NEGATIVE OF THOSE TERMS, AND SIMILAR EXPRESSIONS THAT CONVEY UNCERTAINTY OF FUTURE EVENTS OR OUTCOMES. FORWARD-LOOKING STATEMENTS CONTAINED IN THESE PRESENTATIONS INCLUDE, BUT ARE NOT LIMITED TO, STATEMENTS REGARDING: (I) THE SUCCESS AND TIMING OF OUR PRODUCT DEVELOPMENT ACTIVITIES AND CLINICAL TRIALS; (II) OUR ABILITY TO DEVELOP OUR PRODUCT CANDIDATES; (III) OUR PLANS TO RESEARCH, DISCOVER, EVALUATE AND DEVELOP ADDITIONAL POTENTIAL PRODUCT, TECHNOLOGY AND BUSINESS CANDIDATES AND OPPORTUNITIES; (IV) OUR ABILITY TO DEVELOP AND MANUFACTURE OUR PRODUCT CANDIDATES AND TO IMPROVE THE MANUFACTURING PROCESS; (V) OUR ABILITY TO ATTRACT AND RETAIN KEY SCIENTIFIC OR MANAGEMENT PERSONNEL; (VI) THE ANTICIPATED TIMING OF CLINICAL DATA AVAILABILITY; (VII) OUR ABILITY TO MEET OUR MILESTONES; (VIII) OUR EXPECTATIONS REGARDING OUR ABILITY TO OBTAIN AND MAINTAIN INTELLECTUAL PROPERTY PROTECTION; AND (IX) THE IMPACT OF CAPITAL MARKET CONDITIONS ON US. FORWARD-LOOKING STATEMENTS ARE SUBJECT TO KNOWN AND UNKNOWN FACTORS, RISKS AND UNCERTAINTIES THAT MAY CAUSE ACTUAL RESULTS TO DIFFER MATERIALLY FROM THOSE EXPRESSED OR IMPLIED BY SUCH FORWARD LOOKING STATEMENTS. UNDUE RELIANCE SHOULD NOT BE PLACED ON FORWARD-LOOKING STATEMENTS. WE UNDERTAKE NO OBLIGATION TO PUBLICLY UPDATE ANY FORWARD-LOOKING STATEMENTS.
THE COMPANY’S INVESTIGATIONAL DRUG PRODUCTS HAVE NOT BEEN APPROVED OR CLEARED BY THE FDA.

Emerald Health Pharmaceuticals

Focused on developing patented synthetic cannabinoid-derived drug candidates to treat diseases with unmet medical need

Established: 2017
Headquarters: San Diego, CA, USA
Status: Private



Key Highlights

15 years of cannabinoid science, broad patent coverage, foundation is the endocannabinoid system (ECS)

Synthetic cannabinoid-derivative pharmaceutical drug developer

Multiple patented CBD & CBG derivatives for treating a range of disorders with unmet needs

Phase I human study planned for 2018

Orphan Drug status granted by FDA and EMA for scleroderma and by FDA for Huntington's disease

Experienced management in pharma/biotech

Why Cannabinoid-Derived Drugs?

NATURAL

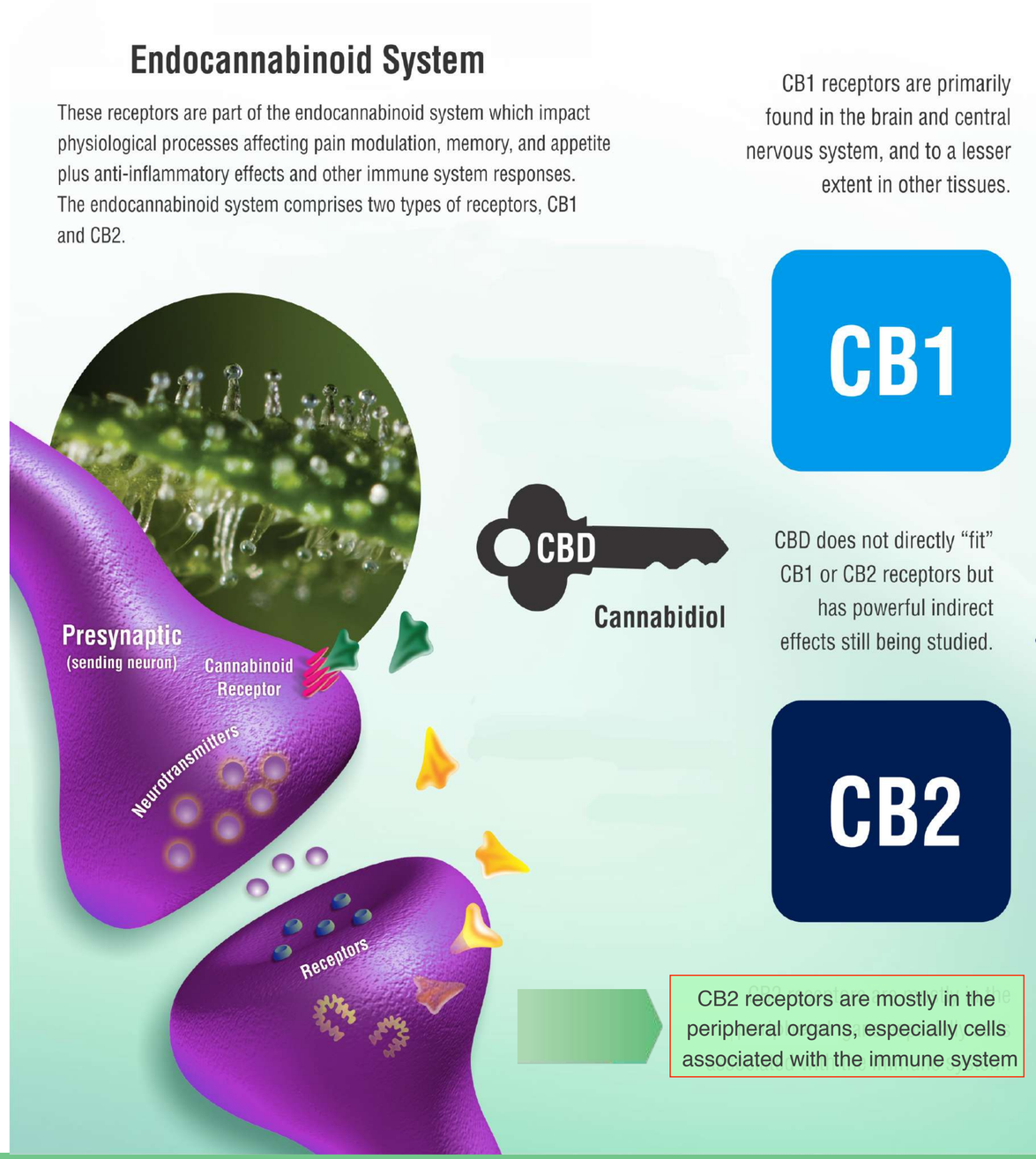
The endocannabinoid system (ECS) is an internal system in our bodies that has been shown to foster overall health with two primary receptors:
CB1 and **CB2**

THERAPEUTIC

Positive effects through influence on the ECS

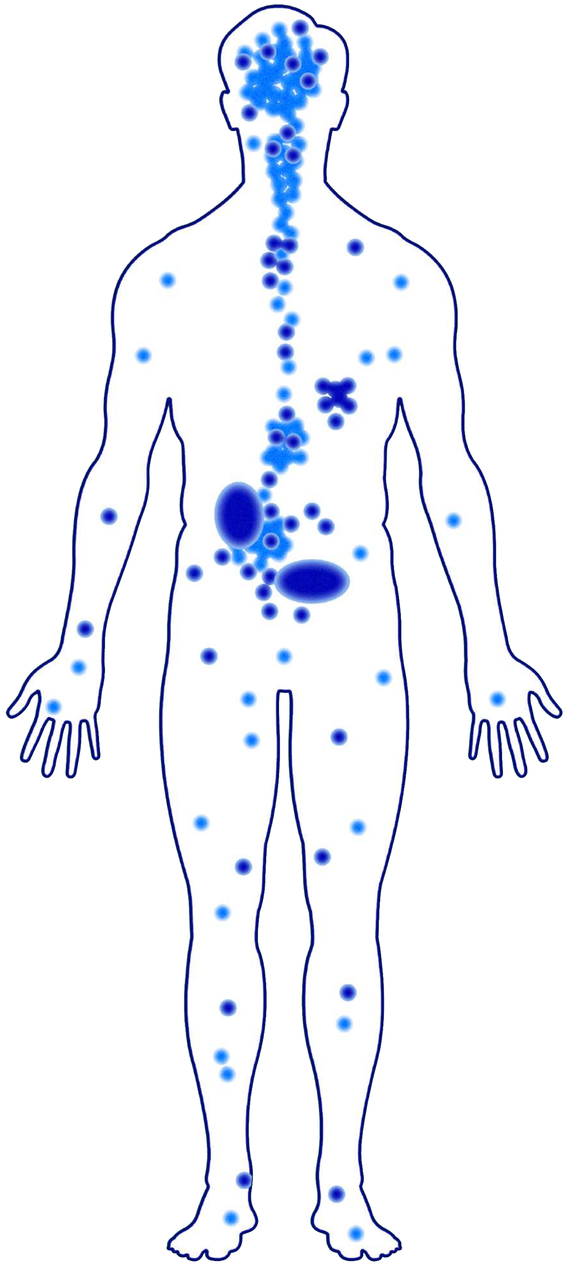
SAFE

Cannabidiol (CBD) & cannabigerol (CBG)
are non-psychotropic, with low toxicity



Why Patented Synthetic Drugs?

- **IMPROVE CDB & CGB** receptor targeting
- **FOCUS** on receptors that can treat diseases with unmet medical need
- **DEVELOP** composition of matter patented cannabinoid new chemical entities (NCEs)
- **CREATE** a strong IP portfolio

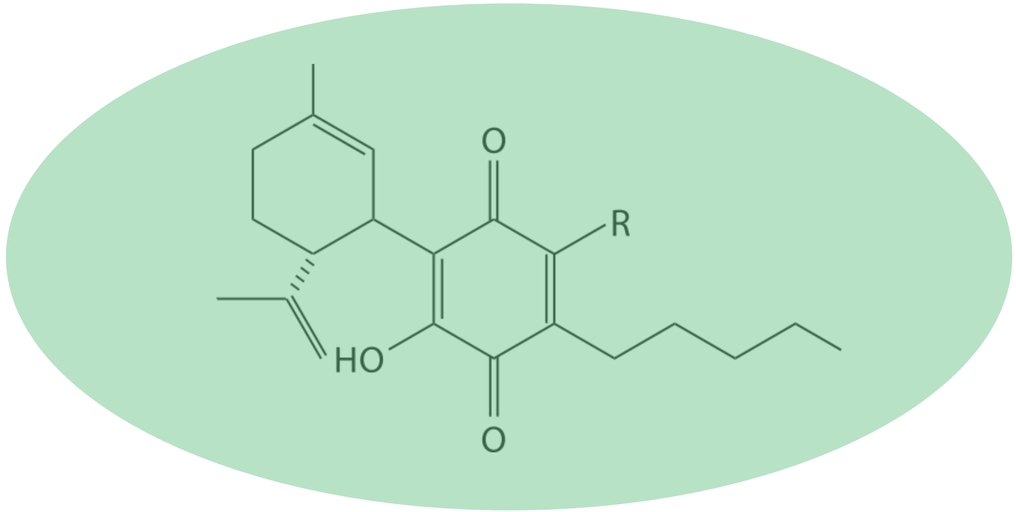


Patents: 6 Issued, 14 Pending

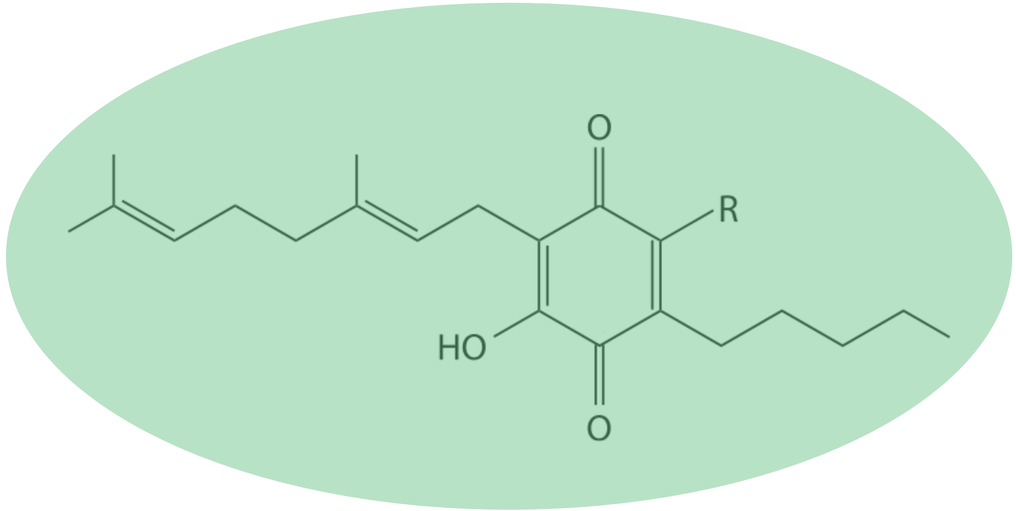
Molecules in our NCE Library

- Composition of matter patents
- Protection to 2035
- Potential for multiple products and indications

14 patented **CBD** derivatives



11 patented **CBG** derivatives



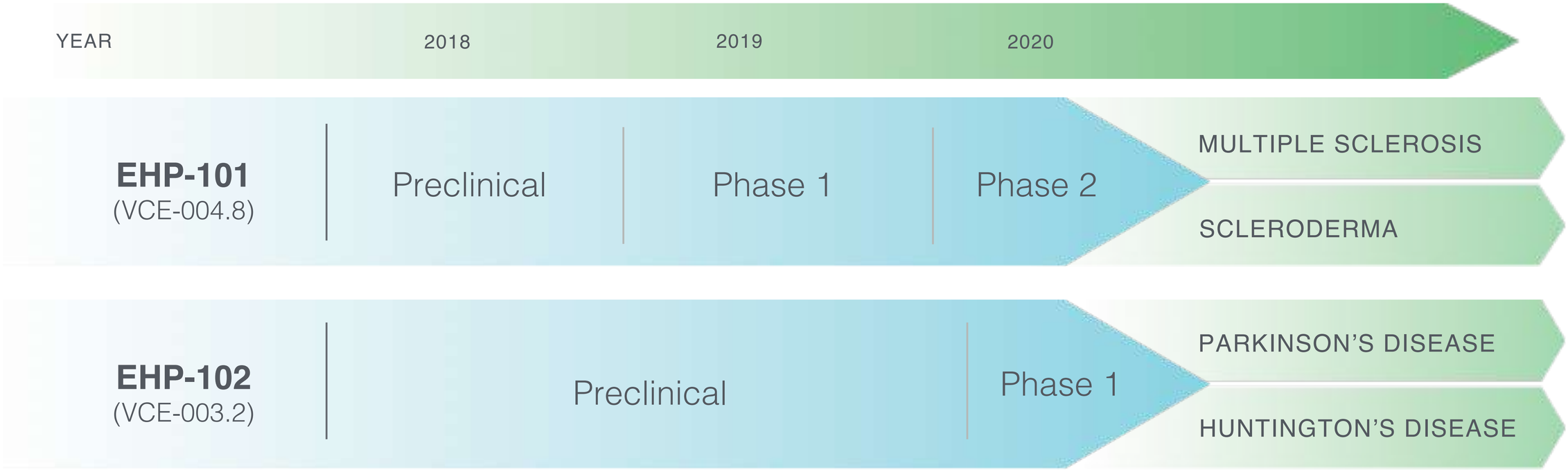
Significant Unmet Needs and Markets for 4 Initial Indications

Multiple Sclerosis	900,000 patients in 7 major markets*
Scleroderma	Orphan Designation granted in the U.S. and E.U.
Parkinson's Disease	10 million sufferers worldwide
Huntingdon's Disease	Orphan Drug Designation granted in the U.S.

**Data per National Multiple Sclerosis Society and Global Data*

Lead Product Candidates: Development Road Map

Proof-of-concept established for 4 initial indications



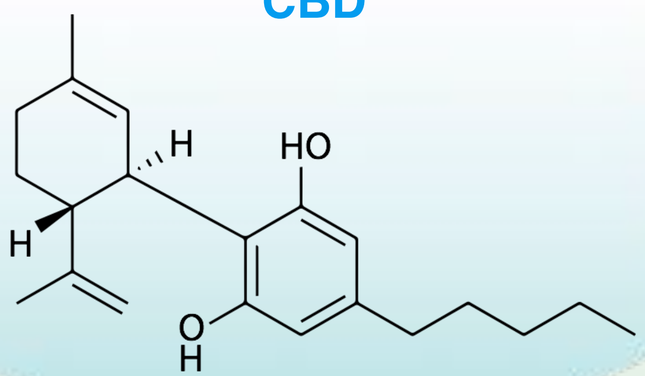
Lead Product Candidate: EHP-101

Cannabidiol (CBD) derivative

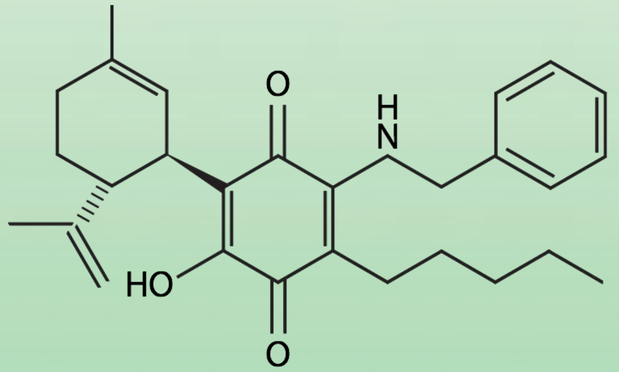
CDB:

- Does not bind to CB1
- Safe, anti-inflammatory, neuroprotective, analgesic, anti-proliferative
- Helps improve MS symptoms

CBD



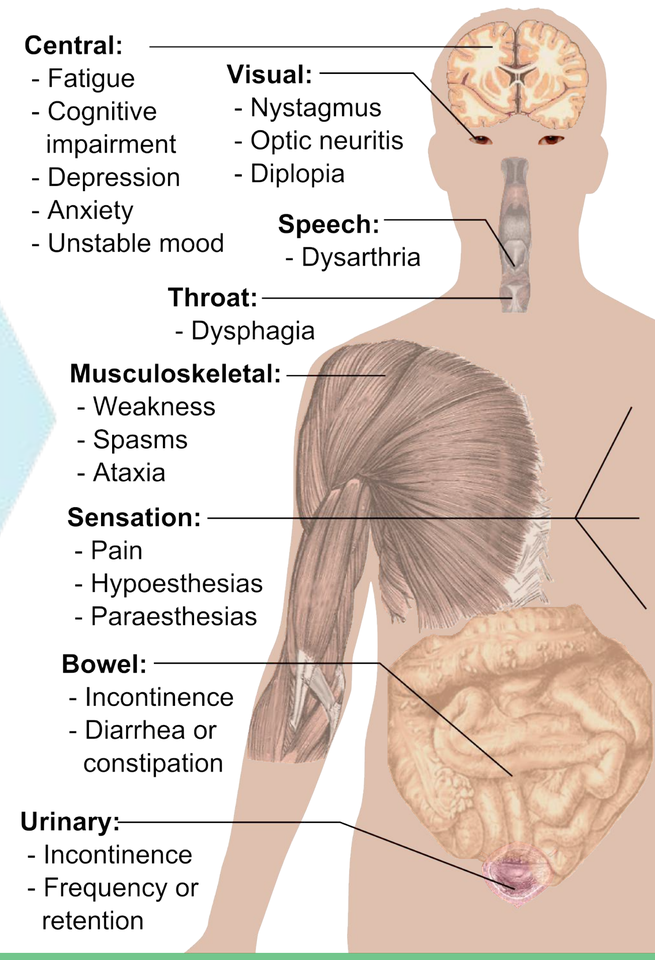
EHP-101



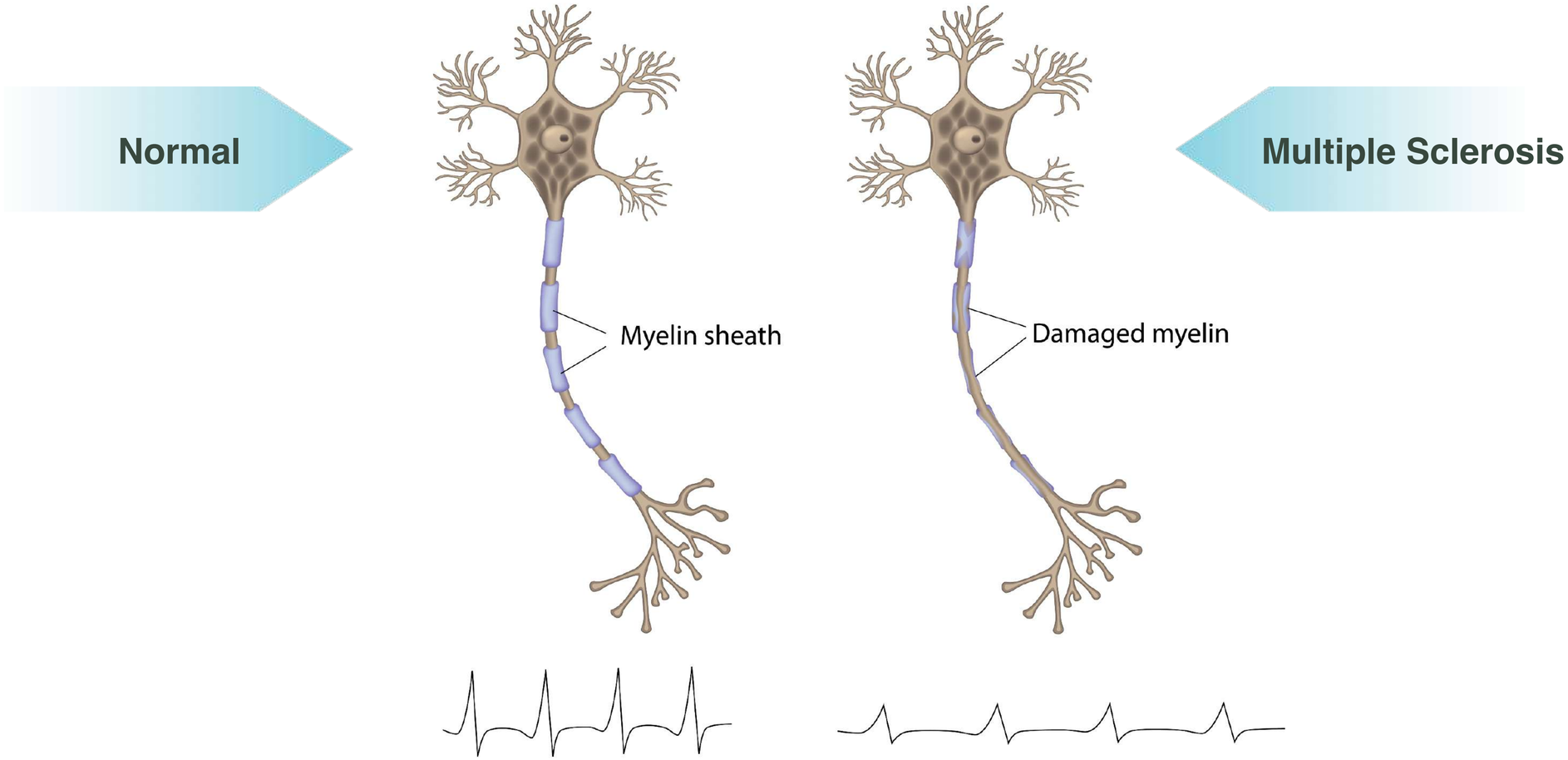
Why Multiple Sclerosis?

Chronic inflammatory, degenerative, demyelinating CNS disorder

- Our molecule targets the main receptors associated with MS
- Current medications are most effective only during the inflammatory phase; less potent as the disease transitions to a neurodegenerative process
- No effective disease-modifying drugs for progressive forms
- No therapies appear to re-myelinate damaged neurons



EHP-101 Can Potentially Re-Myelinate Nerves Damaged by MS

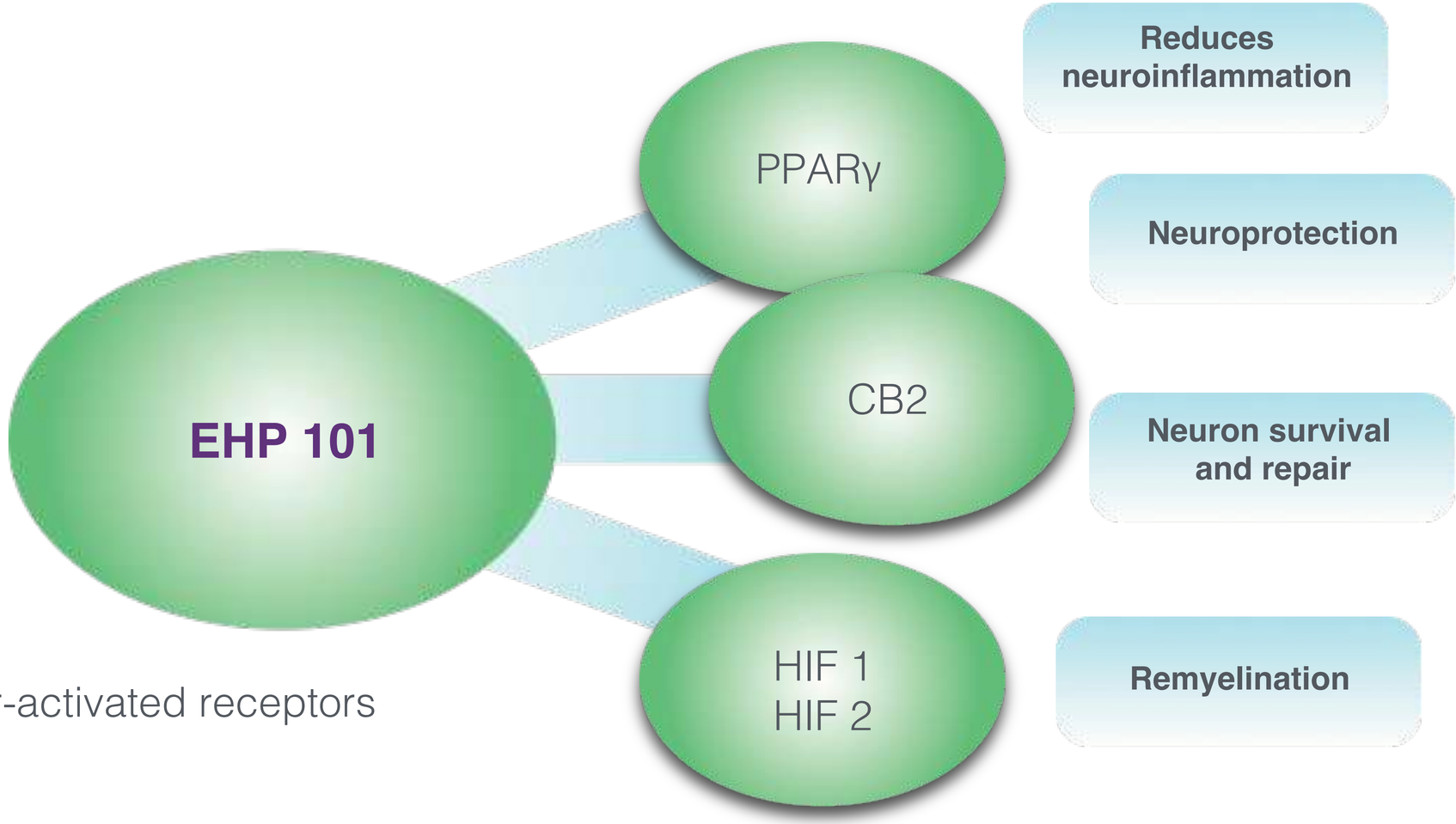


EHP-101: Designed for Mechanism of Action

Our strategy:

To improve on CDB's known positive effects by affecting validated targets in MS:

PPARγ, CB2 and HIF



PPAR: Peroxisome proliferator-activated receptors

HIF: Hypoxia inducible factor

EHP-101 Multiple Sclerosis: Suggestive of Safety & Efficacy

- MoA consistent with validated MS targets
- Efficacy shown in relevant animal models of MS
- Effective at very low doses
- Low toxicity seen at much higher than therapeutic doses

Human studies planned to start this year

Targets validated receptors related to MS

Oral treatment

Potentially disease-modifying

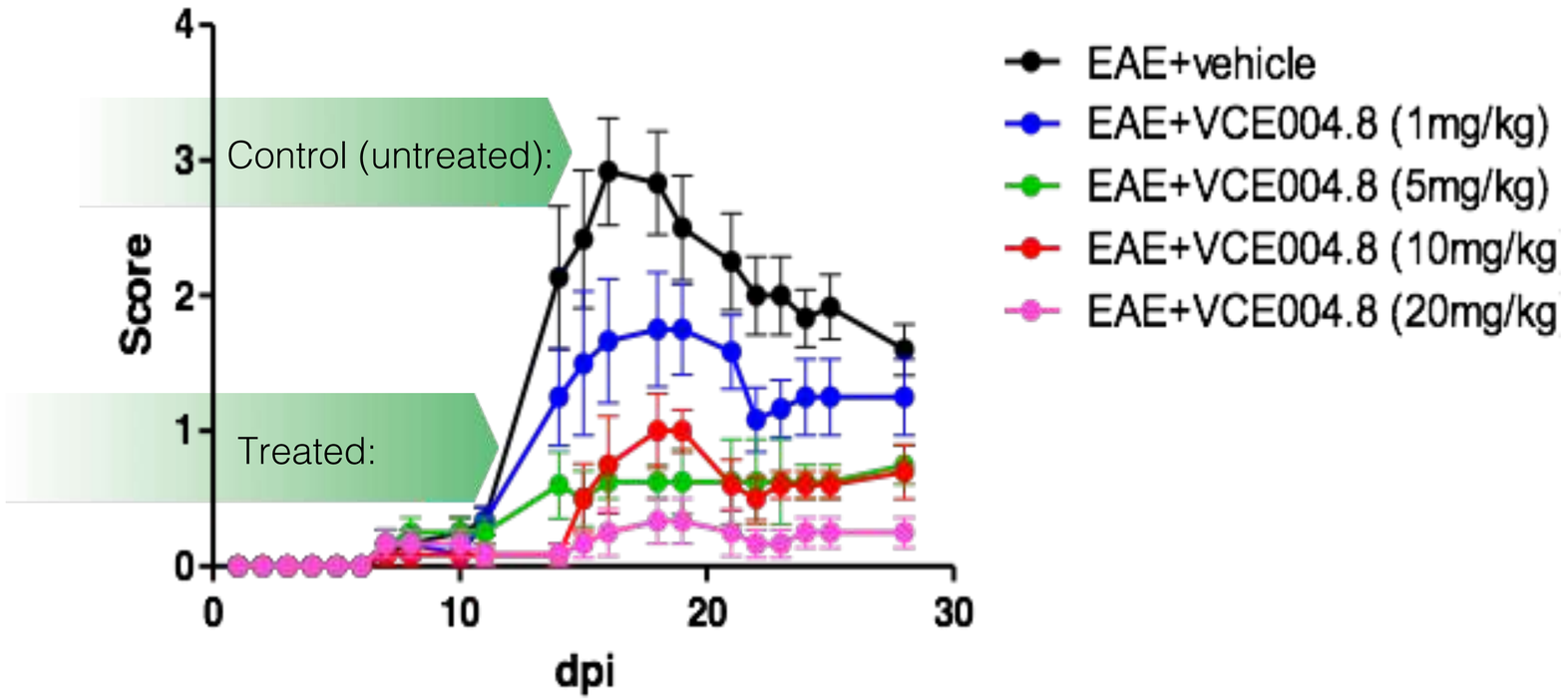
Broad IP protection

Phase 1 2018

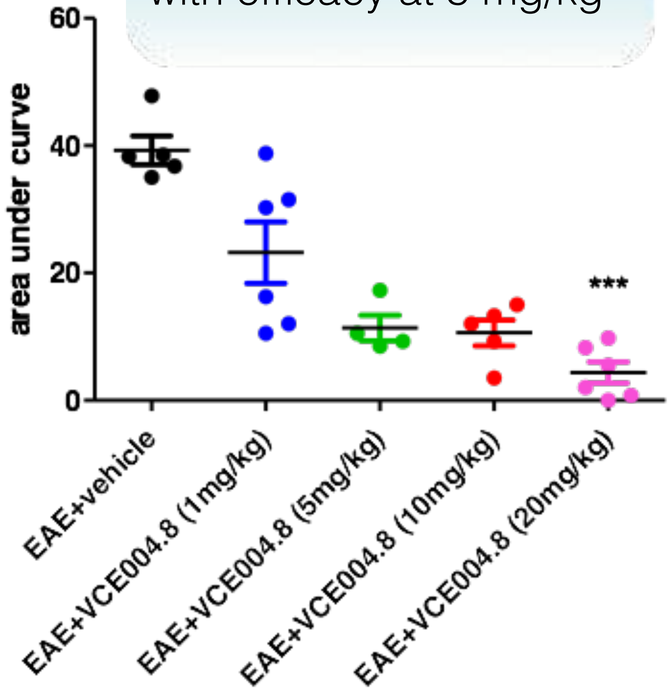
EHP-101 Multiple Sclerosis: Efficacy Demonstrated

Two widely accepted animal models of MS (EAE & TMEV)

Significant reduction in clinical signs and disease progression



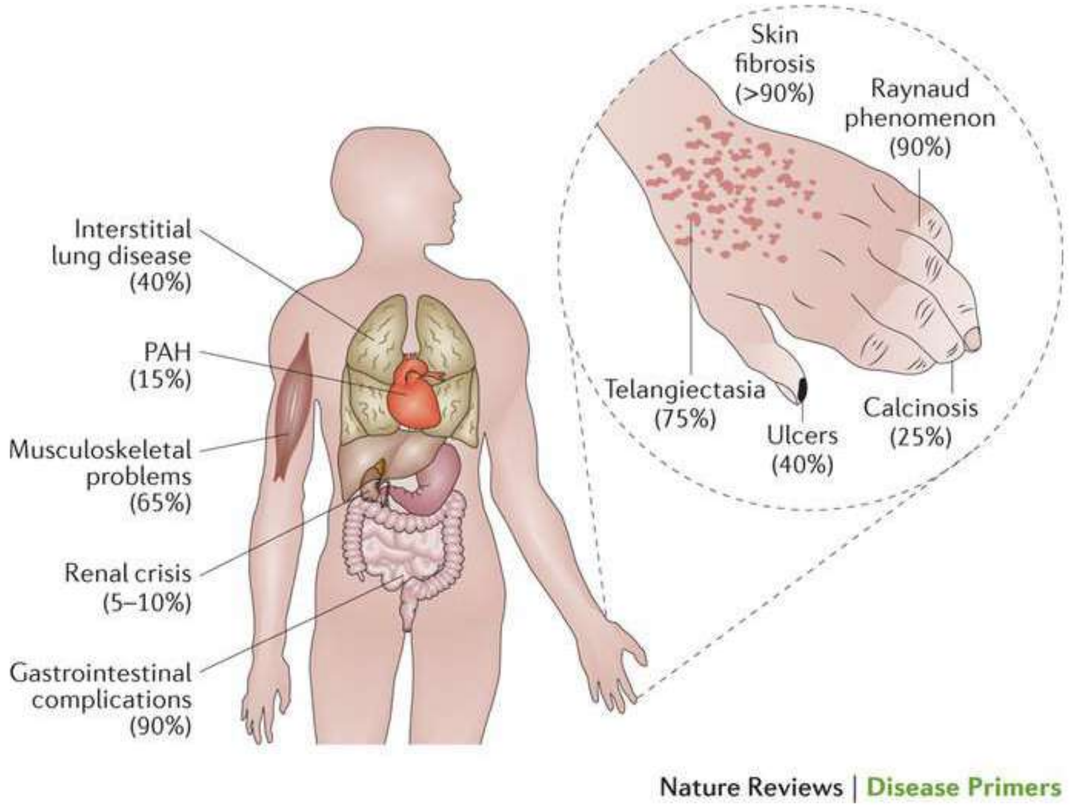
Dose response
with efficacy at 5 mg/kg



EHP-101 Second Indication: Scleroderma (Systemic Sclerosis or SSc)

Chronic, systemic autoimmune disease causing fibrosis of skin and internal organs

- Rare, life-threatening disease
- No SSc-specific approved drugs
- Current therapies not effective and have significant toxicities
- Lung fibrosis is a common cause of death (~60% mortality in 10 years)

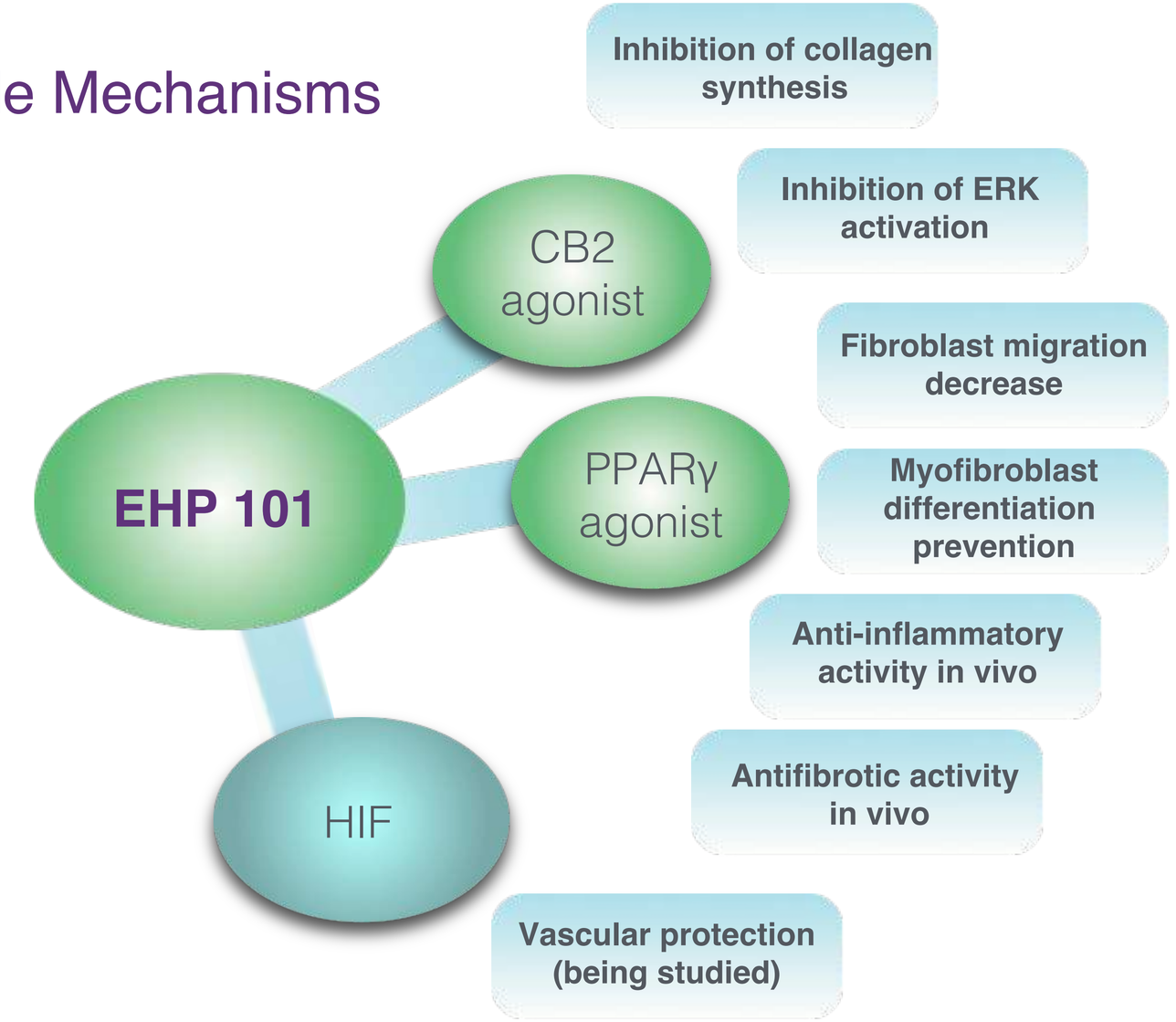


EHP-101 Targets PPARγ and CB2; Affects Scleroderma Through Multiple Mechanisms

- **PPARγ** and **CB2:** extensively studied molecular targets for the treatment of scleroderma*

- . Combined effect on PPARγ, CB2 and HIF not described for other types of marketed drugs

- Scleroderma is an orphan disease (no approved drugs; no cure)



Minghua et al, Tavarares et al, Akhmetshina et al, Del Rio et al

EHP-101: Regulatory Plan and Timeline

2017

H1 Initiated GLP tox studies and manufacturing for Phase I

H2 US FDA grant of Orphan Drug Designation (ODD) for systemic scleroderma (SSc)

EU EMA grant of ODD for SSc

2018

H1 Final clinical-enabling studies to be completed

Pre-IND meeting planned with US FDA (for Phase II preparation)

H2 Phase 1 human study planned in Australia
(single Phase I study expected to support Phase II in both MS & scleroderma)

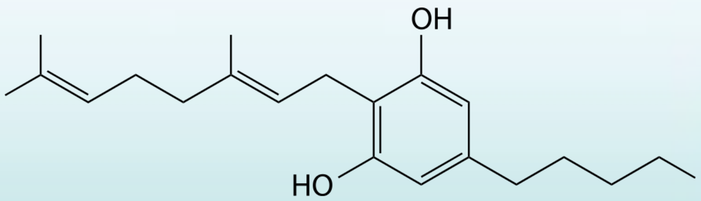
EHP-102: Second Product Candidate

Cannabigerol (CBG) derivative

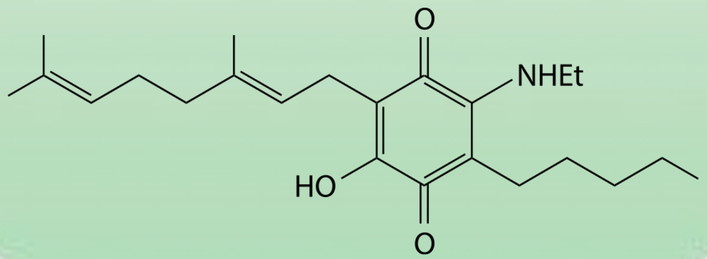
CBG:

- Does not bind to CB1 (non-psychotropic)
- Provides neuroprotection in models of Huntington's disease, partially through antioxidant and anti-inflammatory activity, and PPARγ modulation
- Suppresses norepinephrine, providing muscle relaxation and analgesic properties through effects on the CNS

CBG



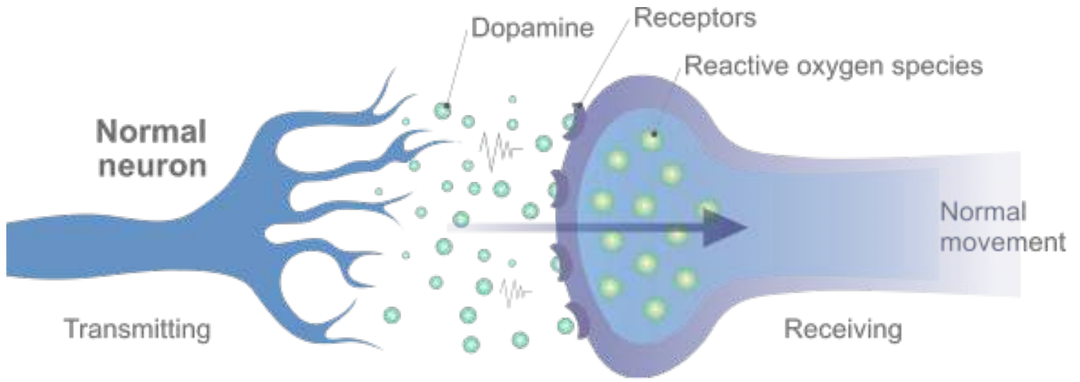
EHP-102



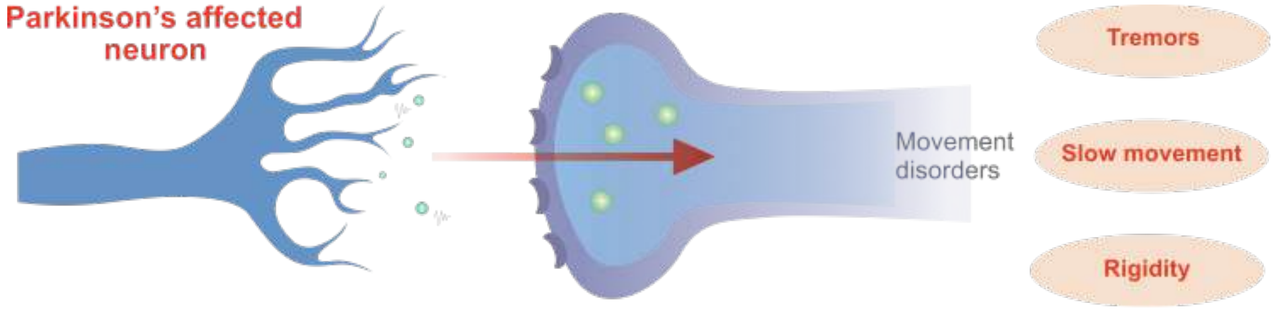
EHP-102: Parkinson’s Disease

Chronic, progressive neurodegenerative disorder with no current cure

- More than 10 million people worldwide have Parkinson's disease
- A disease where damaged neurons do not produce sufficient dopamine (dopamine helps transmit impulses from the brain to the muscles)



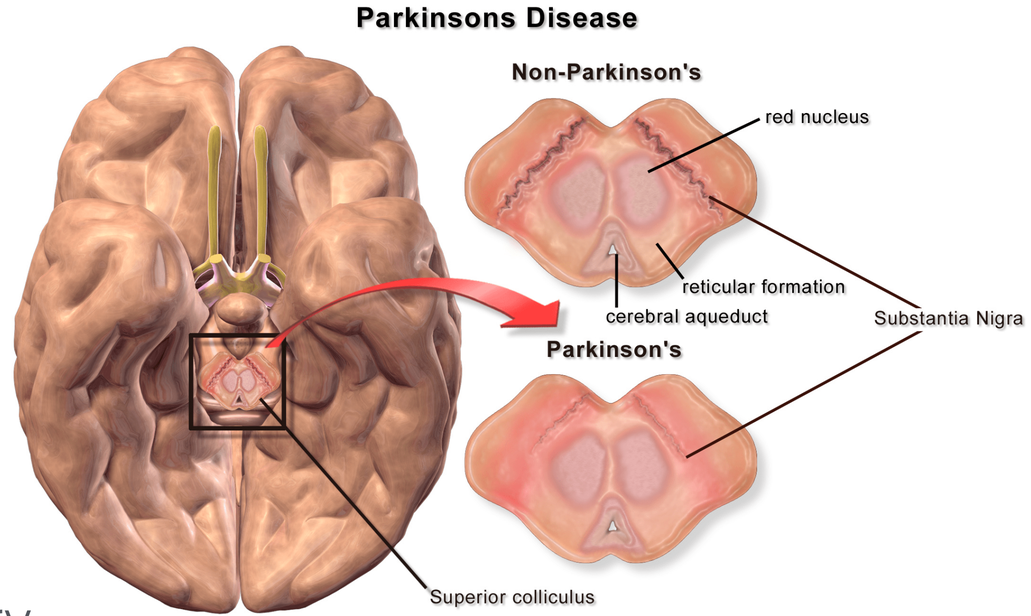
Parkinson’s Disease is a chronic, progressive neurodegenerative disorder with no current cure

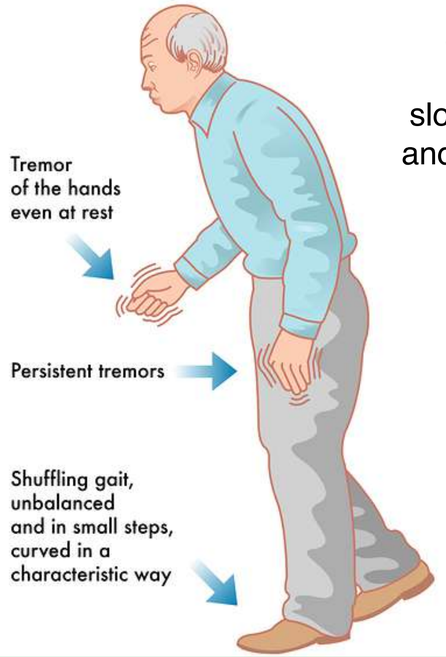


EHP-102: Parkinson’s Disease

Demonstrated efficacy in mouse models

- Provides neuroprotection, partially through PPARγ activity and reduction in proinflammatory mediators
- Improves clinical symptoms and recovers movement parameters (motor coordination and activity)
- Reduces inflammatory marker expression and prevents dopaminergic neuronal loss



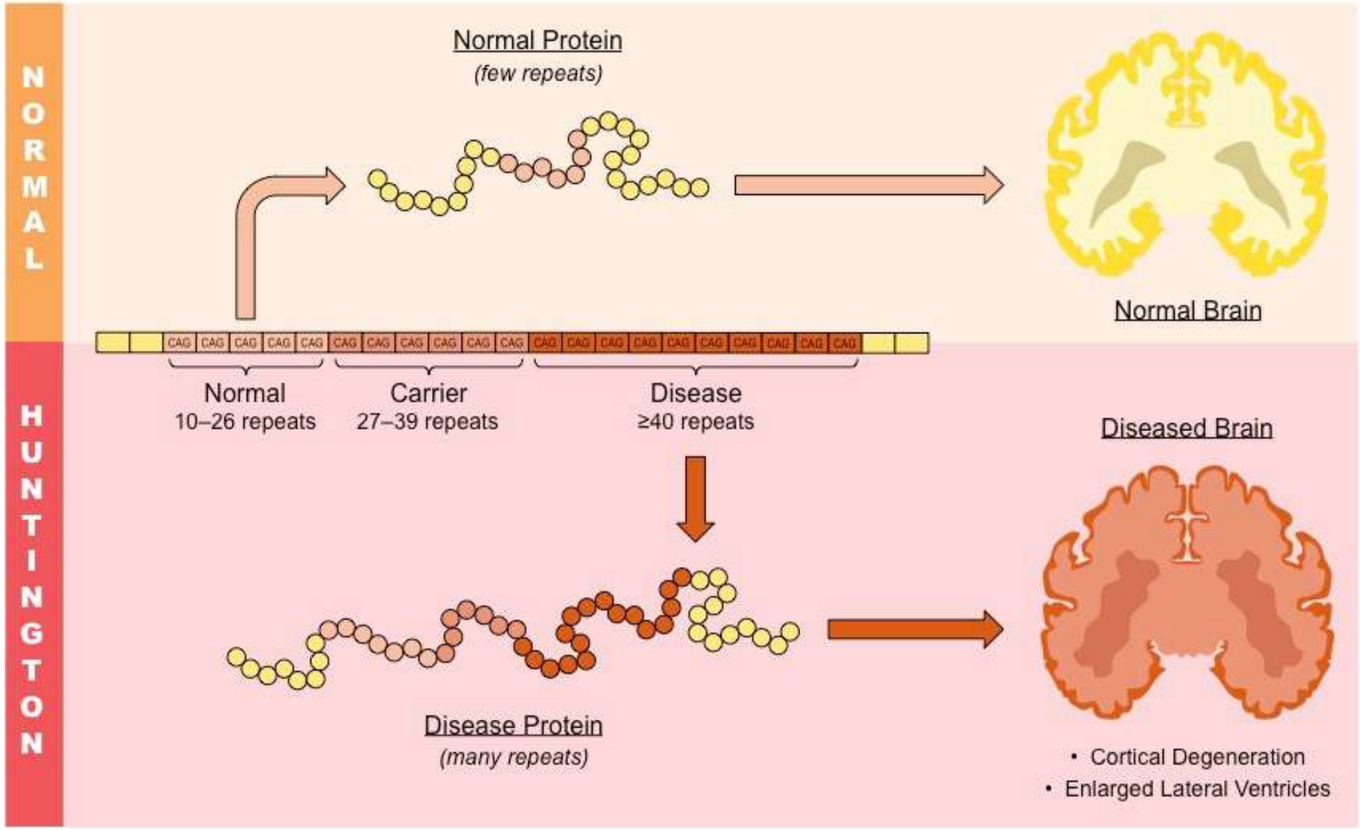


Symptoms include shaking, rigidity, slowness of movement, and difficulty with walking

EHP-102: Huntington's disease

Causes progressive breakdown of nerve cells

- An orphan disease
- EHP-102 Targets PPARγ with improved activity
- Also targets other pathways involved in neural survival (ERK 1+2)

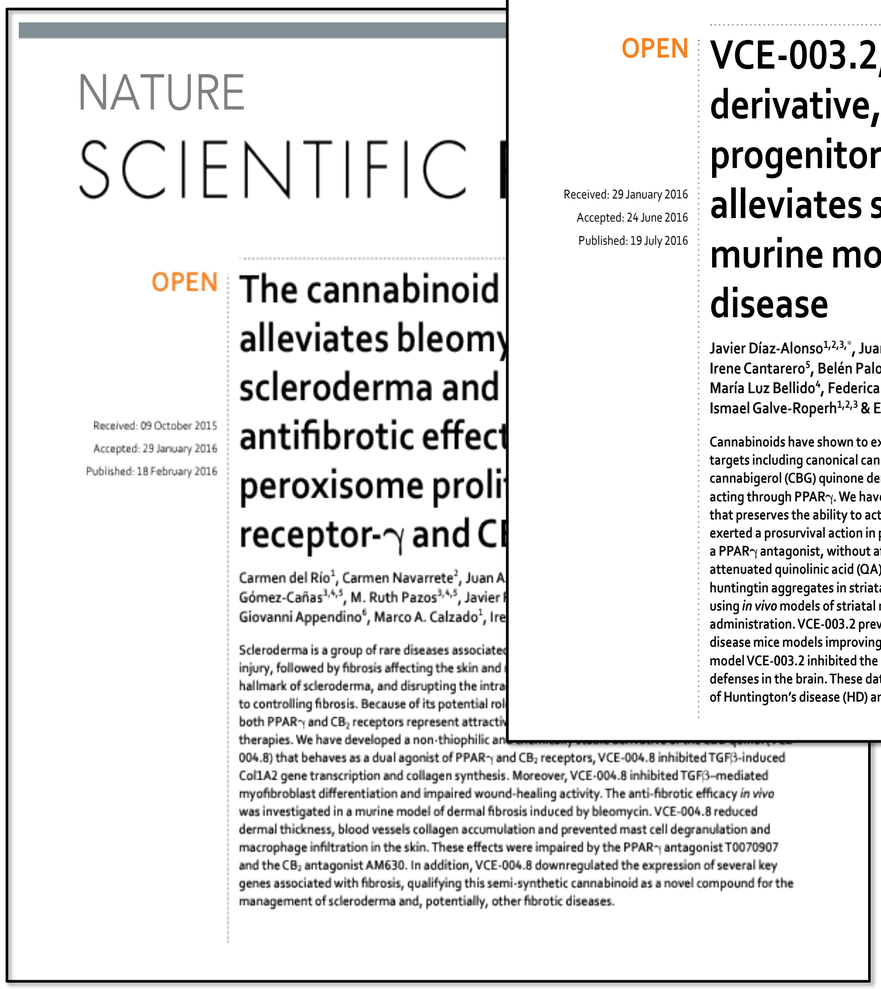


EHP-102: Current Status

Demonstrated preclinical efficacy

- Proof-of-concept established for PD & HD
- Orphan Drug Designation granted in US for HD
- Formulation & manufacturing development in progress
- Clinical-enabling studies to start in 2018
- Human development planned to begin in 2019

Publications



NATURE
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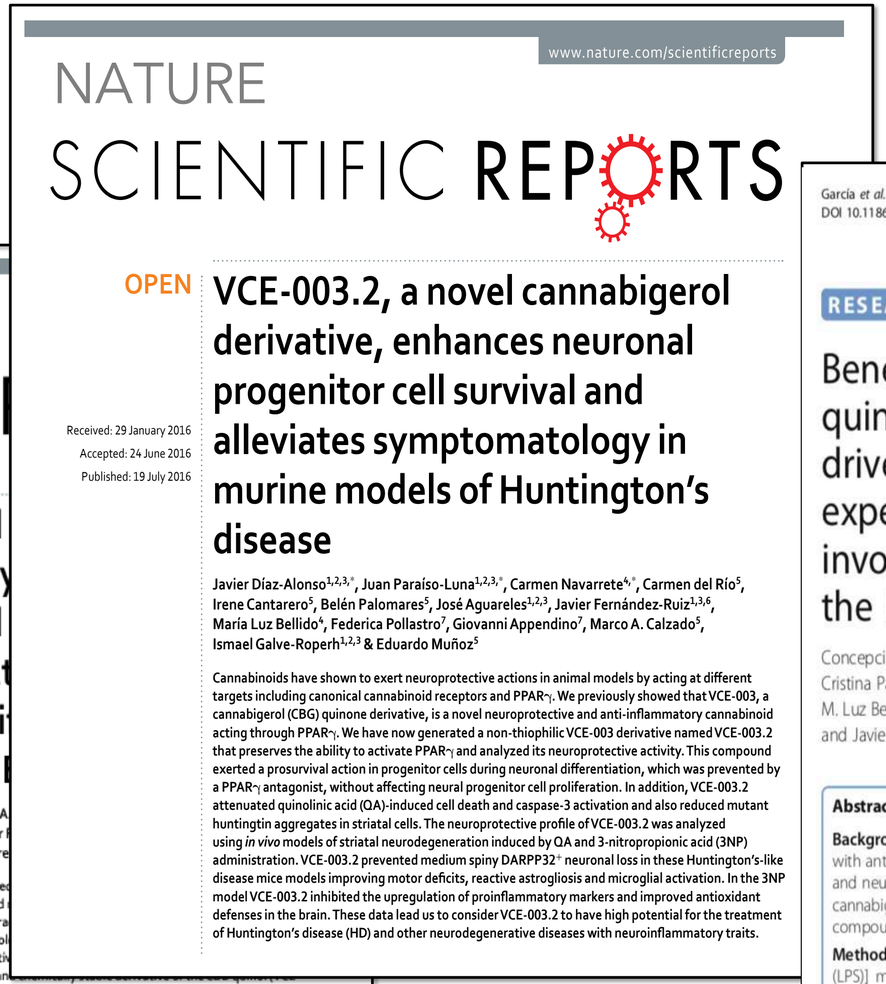
The cannabinoid
alleviates bleomy
scleroderma and
antifibrotic effect
peroxisome proli
receptor-γ and CB

Received: 09 October 2015
Accepted: 29 January 2016
Published: 18 February 2016

Carmen del Río[1], Carmen Navarrete[2], Juan A
Gómez-Cañas[3,4,5], M. Ruth Pazos[3,4,5], Javier
Giovanni Appendino[6], Marco A. Calzado[1], Ire

Scleroderma is a group of rare diseases associate
injury, followed by fibrosis affecting the skin and
hallmark of scleroderma, and disrupting the intra
to controlling fibrosis. Because of its potential rol
both PPARγ and CB_2 receptors represent attracti
therapies. We have developed a non-thiophilic an
004.8) that behaves as a dual agonist of PPARγ and CB_2 receptors, VCE-004.8 inhibited TGFβ-induced Col1A2 gene transcription and collagen synthesis. Moreover, VCE-004.8 inhibited TGFβ–mediated myofibroblast differentiation and impaired wound-healing activity. The anti-fibrotic efficacy *in vivo* was investigated in a murine model of dermal fibrosis induced by bleomycin. VCE-004.8 reduced dermal thickness, blood vessels collagen accumulation and prevented mast cell degranulation and macrophage infiltration in the skin. These effects were impaired by the PPARγ antagonist T0070907 and the CB_2 antagonist AM630. In addition, VCE-004.8 downregulated the expression of several key genes associated with fibrosis, qualifying this semi-synthetic cannabinoid as a novel compound for the management of scleroderma and, potentially, other fibrotic diseases.



www.nature.com/scientificreports

NATURE
SCIENTIFIC REPORTS

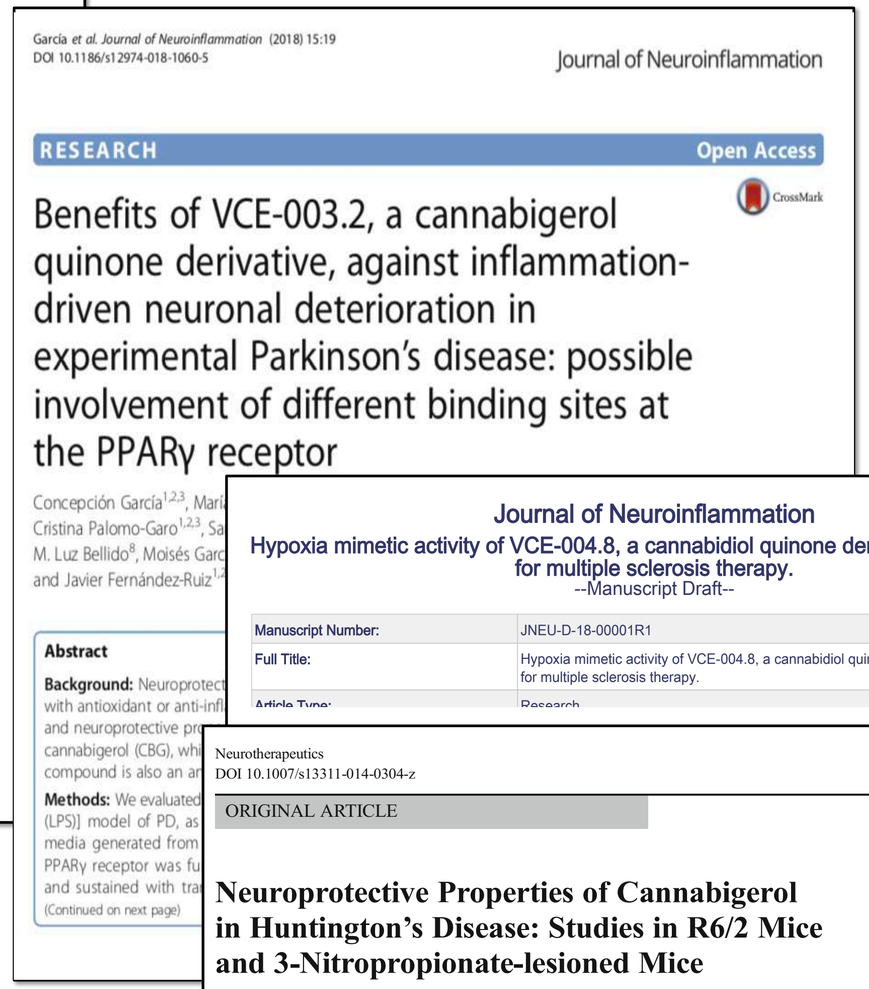
OPEN

VCE-003.2, a novel cannabigerol derivative, enhances neuronal progenitor cell survival and alleviates symptomatology in murine models of Huntington's disease

Received: 29 January 2016
Accepted: 24 June 2016
Published: 19 July 2016

Javier Díaz-Alonso[1,2,3,*], Juan Paraíso-Luna[1,2,3,*], Carmen Navarrete[4,*], Carmen del Río[5], Irene Cantarero[5], Belén Palomares[5], José Aguareles[1,2,3], Javier Fernández-Ruiz[1,3,6], María Luz Bellido[4], Federica Pollastro[7], Giovanni Appendino[7], Marco A. Calzado[5], Ismael Galve-Roperh[1,2,3] & Eduardo Muñoz[5]

Cannabinoids have shown to exert neuroprotective actions in animal models by acting at different targets including canonical cannabinoid receptors and PPARγ. We previously showed that VCE-003, a cannabigerol (CBG) quinone derivative, is a novel neuroprotective and anti-inflammatory cannabinoid acting through PPARγ. We have now generated a non-thiophilic VCE-003 derivative named VCE-003.2 that preserves the ability to activate PPARγ and analyzed its neuroprotective activity. This compound exerted a prosurvival action in progenitor cells during neuronal differentiation, which was prevented by a PPARγ antagonist, without affecting neural progenitor cell proliferation. In addition, VCE-003.2 attenuated quinolinic acid (QA)-induced cell death and caspase-3 activation and also reduced mutant huntingtin aggregates in striatal cells. The neuroprotective profile of VCE-003.2 was analyzed using *in vivo* models of striatal neurodegeneration induced by QA and 3-nitropropionic acid (3NP) administration. VCE-003.2 prevented medium spiny $DARPP32^+$ neuronal loss in these Huntington's-like disease mice models improving motor deficits, reactive astrogliosis and microglial activation. In the 3NP model VCE-003.2 inhibited the upregulation of proinflammatory markers and improved antioxidant defenses in the brain. These data lead us to consider VCE-003.2 to have high potential for the treatment of Huntington's disease (HD) and other neurodegenerative diseases with neuroinflammatory traits.



García *et al. Journal of Neuroinflammation* (2018) 15:19
DOI 10.1186/s12974-018-1060-5

Journal of Neuroinflammation

RESEARCH Open Access

CrossMark

Benefits of VCE-003.2, a cannabigerol quinone derivative, against inflammation-driven neuronal deterioration in experimental Parkinson's disease: possible involvement of different binding sites at the PPARγ receptor

Concepción García[1,2,3], Marí
Cristina Palomo-Garo[1,2,3], Sa
M. Luz Bellido[8], Moisés Garc
and Javier Fernández-Ruiz[1,2]

Abstract

Background: Neuroprotect
with antioxidant or anti-infl
and neuroprotective pro
cannabigerol (CBG), whi
compound is also an ar

Methods: We evaluated
(LPS)] model of PD, as
media generated from
PPARγ receptor was fu
and sustained with tra

(Continued on next page)

Journal of Neuroinflammation

Hypoxia mimetic activity of VCE-004.8, a cannabidiol quinone derivative: implications for multiple sclerosis therapy.

--Manuscript Draft--

Manuscript Number:	JNEU-D-18-00001R1
Full Title:	Hypoxia mimetic activity of VCE-004.8, a cannabidiol quinone derivative: implications for multiple sclerosis therapy.
Article Type:	Research

Neurotherapeutics
DOI 10.1007/s13311-014-0304-z

ORIGINAL ARTICLE

Neuroprotective Properties of Cannabigerol in Huntington's Disease: Studies in R6/2 Mice and 3-Nitropropionate-lesioned Mice

Sara Valdeolivas · Carmen Navarrete · Irene Cantarero · María L. Bellido · Eduardo Muñoz · Onintza Sagredo

Emerald Health Pharmaceuticals Summary

Foundation: Therapeutic effect of CDB and CBG on ECS

Library of patented NCEs derived from CBD and CBG with improved biologic activities targeting specific diseases

2 lead molecules, EHP-101 and EHP-102, being developed for 4 initial indications

Multiple additional molecules for new future indications

Emerald Health Pharmaceuticals Summary

- Combined MoA for EHP molecules not described with other drugs
- EHP-101 human study planned for 2018
- Orphan status granted for scleroderma and Huntington's
- Management team experienced in developing drugs and building companies

Experienced Pharma / Biotech Management Team

Jim DeMesa, MD, MBA
Chief Executive Officer

29 years in pharma product development and management, including preclinical and clinical trial management, and partnering with pharma companies. 15 years as CEO of public biotech companies.

Alain Rolland, PharmD, PhD
VP, Product Development

30 years of international leadership experience in R&D, strategic product management, and business development.

Mari-Luz Bellido, PhD, MBA
VP, European Operations

Molecular biologist with **10 years** experience in preclinical development of cannabis-based compounds.

Avtar Dhillon, MD
Chairman

Chairman of 5 public life science companies, led turnaround of NASDAQ:INO from $10m to $550m

Jill Broadfoot
Chief Financial Officer

31 years in biotech financial management at GW Pharma (NASDAQ GWPH), CFO, Vical (NASDAQ: VICL), Ernst & Young. BS in Business Administration and CPA.

Nancy Coulson
VP, Regulatory Affairs

30 years in global pharma and biotech regulatory management with J&J, BMS, and others.

Eduardo Muñoz, MD, PhD
Chief Scientific Officer

30 years in biomedical research, Professor of Immunology, author of 200 articles, patents and book chapters with nearly 5,000 citations.

Giovanni Appendino, PhD
Scientific Advisor

One of the worlds thought leaders in cannabinoid research; Keynote speaker at the 2014 ICRS Symposium; Professor ofPharmaceutical Chemistry at the University of Eastern Piedmont; Author of 250 articles and 10 book chapters.

Clinical Advisors:
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Juan-Antonio Garcia Merino, MD
SSc: John Varga, MD
Janet Pope, MD
Patricia Carreira, MD

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