Yes, Palmitoylethanolamide (PEA) has been shown to reverse or significantly improve symptoms and underlying pathologies in various neurological disorders in both preclinical and, in some cases, clinical settings. Its effects are largely attributed to its anti-inflammatory, neuroprotective, and immunomodulatory properties.

Here's a breakdown of its reported effects on specific neurological conditions:

- **Alzheimer's Disease (AD)**:
    - PEA has been shown to **prevent memory deficits** and **reduce or prevent amyloid-induced behavioral impairments** in animal models.
    - It **counteracts reactive astrogliosis** and **reduces neuroinflammation and neuronal damage**.
    - PEA can **improve neuronal survival** and **restore dendritic spine density**, which is crucial for synaptic function.
    - It **mitigates oxidative stress** by decreasing protein nitrosylation and reducing reactive oxygen species production.
    - Chronic oral administration of ultramicronized PEA (um-PEA) **rescued cognitive deficits**, **restrained neuroinflammation**, and **reduced increased hippocampal glutamate levels** in an AD mouse model.
    - PEA-Luteolin (PEALut) has been found to **induce an improvement of cognitive performances** in a patient with mild cognitive impairment (MCI), a precursor to dementia.
- **Parkinson's Disease (PD)**:
    - PEA **reduced the loss of dopaminergic neurons** and **reversed motor deficits** in animal models.
    - It **promotes neurogenesis** and **modulates microglial/astrocyte activation**.
    - Clinical studies have shown that um-PEA and PEALut supplementation **significantly improved both motor and non-motor symptoms**, **reduced dyskinesia**, and **lowered the incidence of camptocormia** in PD patients.
- **Multiple Sclerosis (MS)**:
    - PEA **reduced inflammation, demyelination, neuronal degeneration, and behavioral impairment** in animal models of MS.
    - Um-PEA therapy **significantly reduced serum cytokine levels and improved overall quality of life** in MS patients.
    - PEA was reported to **alleviate spasticity** in the hind limbs of spastic mice.
    - PEALut **reduced the severity of clinical signs** in an experimental autoimmune encephalomyelitis (EAE) model of MS through anti-inflammatory mechanisms.
    - It has been reported to **reduce neuropathic pain** in an MS patient.
- **Spinal Cord Injury (SCI)**:

    - PEA **reduced inflammation and tissue injury** associated with SCI and **improved motor function**.
    - Co-ultramicronized PEA/luteolin (co-ultraPEALut) **improved motor function and histological alteration** in mice with SCI.
    - PEA treatment **limited SCI-induced cytotoxic edema and infiltration of inflammatory cells** and **restored PPAR-δ and PPAR-γ expression**.
    - It **inhibited excessive autophagy** and regulated protein degradation in SCI models.
- **Traumatic Brain Injury (TBI)**:
    - Co-ultraPEALut **significantly decreased edema and brain infarction area and volume**, and **promoted functional and behavioral recovery** in mouse models, often outperforming PEA alone.
    - It **prevented increases in pro-inflammatory cytokines** (TNFα and IL-1β).
    - Co-ultraPEALut also **modulated apoptosis** and **inhibited autophagy**.
- **Stroke (Ischemic Stroke/Cerebral Ischemia/Vascular Dementia)**:
    - PEA therapy **prevented brain histological and cellular damage**, **reduced inflammatory cytokine levels**, and **prevented early blood-brain barrier disruption** in stroke models.
    - PEA **reduced neuroinflammation** by promoting anti-inflammatory macrophage phenotypes.
    - PEALut **improved neurological status, cognitive abilities, spasticity, pain, and independence in daily living activities** in stroke patients, suggesting it can **reduce the severity of neuroinflammation**.
    - PEA administration **rescues injured hippocampal neurons** and **improves memory, social behavior, and locomotor activity** in vascular dementia (VaD) models.
- **Frontotemporal Dementia (FTD)**:
    - PEA-LUT **reduced behavioral disturbances** and **improved frontal lobe functions** by modulating cortical oscillatory activity and GABA(B)ergic transmission.
    - It can **enhance GABAergic transmission** and **inhibit glutamate release**, offering anti-excitotoxic effects.
- **Neonatal Anoxia-Ischemia (AI)**:
    - PEA treatment **prevented neuroinflammation, reduced astrogliosis, and preserved cognitive functions**. It was able to **prevent memory deficits** associated with this condition.
- **Epilepsy (Seizures)**:
    - PEA has known **antiepileptic properties**.

    - It has been shown to **inhibit electroshock-induced and chemically induced seizures** in animal models.
- **Autism Spectrum Disorder (ASD)**:
    - PEA supplementation **improves social and nonsocial behaviors**, **reduces pro-inflammatory markers**, **modulates apoptosis**, and **increases hippocampal neurogenesis and neuroplasticity** in rodent models.
    - It has been shown to **improve expressive language** and **reduce overall autism severity**, including motor stereotypic behaviors, in human case reports.
- **Diabetic Neuropathy (DPN)**:
    - PEA has been shown to be **effective in relieving pain** in DPN.
    - It **significantly reduced pain symptoms** characteristic of diabetic neuropathy.
    - PEA **decreased inflammation, relieved mechanical allodynia, counteracted nerve growth factor deficit, improved insulin levels, and preserved pancreatic cell morphology** in rodent models.
- **Glaucoma and Retinopathy**:
    - PEA is considered a putative anti-inflammatory and retinoprotectant compound.
    - PEA intake **reduced intraocular pressure (IOP)** and **improved visual field indices** in glaucoma patients.
    - It **protected retinal ganglion cells** from neurotoxic damage.
- **Pain (Neuropathic, Chronic, Central)**:
    - PEA is a well-researched **analgesic, anti-inflammatory, and neuroprotective agent**.
    - It has shown **broad effectiveness for relieving occasional muscle pain** and **reducing the need for rescue medications**.
    - PEA consistently demonstrates a **favorable effect in managing chronic pain**, improving functional status and quality of life.
    - It can **reverse allodynia** in chemotherapy-induced peripheral neuropathy (CIPN) models, and combination with gabapentin produced **synergistic effects**.
    - PEA **improves nerve function and reduces neuropathic pain** in patients with painful neuropathy.
    - It **reduces pain intensity and spasticity** in post-stroke patients.
- **Myasthenia Gravis**:
    - Oral PEA supplementation **improved patients' response to repetitive nerve stimulation**, leading to **improved disease severity scores** and a **decrease in muscular fatigue**. This suggests a direct action on acetylcholine receptors.

PEA's therapeutic benefits often stem from its ability to modulate inflammation and its pleiotropic effects on various cellular targets and signaling pathways, including PPAR-α, GPR55, TRPV1, and indirect modulation of cannabinoid receptors (CB1 and CB2). The micronized and

ultramicronized formulations of PEA are noted for enhancing its bioavailability and therapeutic efficacy, especially for CNS disorders, by improving tissue absorption and crossing the blood-brain barrier. Furthermore, PEA is generally well-tolerated with a benign side effect profile.