Can proline-rich polypeptides (PRPs) protect your brain and even boost brain function? Studies *in vitro* on animals and humans support that idea. The neuro-protective cytokines in PRPs have a remarkably stabilizing effect on cognitive function in Alzheimer’s disease patients. *In vitro* studies show that PRPs inhibit fibrils and amyloid plaques.[1] PRPs also modulate intracellular levels of reactive oxygen species (ROS), by regulating glutathione metabolism and antioxidant enzymes.[2] Gene expression analysis found that PRPs down-regulate genes involved in inflammatory pathways and increase levels of an Amyloid-beta (Aβ) hydrolyzing enzyme.[3] When given orally to mice, PRPs improve motor and sensory activities.[4] When mice are given either PRPs or plain colostrum, the PRP supplemented mice swim faster to a hidden platform.[5] PRPs also improve spatial learning and memory in older rats.[6]

Scientist Maria Janusz, of the Polish Academy of Sciences, has published numerous studies on PRPs and cognition, including work on their ability to slow down the progression of Alzheimer’s disease (AD). The first study that Janusz and colleagues conducted in humans was back in 1999. Forty-six mild or moderate AD patients in a placebo-controlled, double blind study were divided into 3 groups and randomly assigned to receive either Colostrinin (PRPs), selenium, or placebo. One cycle of the treatment lasted 3 weeks and was separated from the next cycle by a 2 week hiatus. Each patient received 10 cycles of treatment during the year of the clinical trial. According to psychiatrists, eight of 15 AD patients treated with PRPs improved, and the 7 other stabilised. In contrast, none of the 31 patients from the selenium or placebo groups with similar mild or moderate AD improved. Thirteen of the 15 on selenium remained stable, and 8 of 16 on placebo remained stable. The scientists concluded that oral administration of PRPs improves the outcome of AD.[7]
Cytokines: Messengers with a Mission

Cytokines play a pivotal role in our health. They are a diverse family of non-antibody soluble immunomodulatory proteins and peptides. You may have heard of some of them: interleukins, interferons, and growth factors. Cytokines allow cells to communicate with one another. Their synthesis is initiated by gene transcription and they are produced as and when needed by the immune system. They are commonly referred to as the ‘master regulators’ of the immune system although other cells can also induce cytokine expression. Cytokines can incite or prevent inflammation, promote cell growth, or influence cells to differentiate and become a specific cell type. Cytokines work by binding to receptors on the membranes of cells, which then triggers a cascade of signals that causes genes to start transcribing molecules. Cytokines can often “share” receptors: a cell membrane receptor for one cytokine can often respond to another cytokine in the same family.

Another 2002 placebo-controlled trial in 106 people with Alzheimer’s over a thirty week period showed that approximately 33% of patients stabilised or improved after 30 weeks of treatment.[8] According to a 2013 review article by Janusz and colleagues, PRPs modulate neurite outgrowth, suppress uncontrolled activation of cells, and reduce cellular damage. She concludes: “Biological response modifying activity of PRP/Colostrinin can play an important role in its use in the treatment of Alzheimer’s disease and suggests its application beyond neurodegenerative disorders.”[9]

PRP’s in clinical practice

Cognitive performance is a crucial component in maintaining independent living, and is influential to overall health as well. For those already presenting with signs indicative of cognitive decline, clinical evidence demonstrates that this PRP complex derived from Colostrum may be useful in stabilising or modestly improving measures of cognitive function — including memory, attention, and problem-solving. In addition to addressing symptoms associated with neurodegeneration, PRP shows promise in targeting the potential underlying causes of neurodegenerative processes, as well as those that support neuroplasticity.

References


