

## News &amp; Comments

## Curcuma has an Anti-diabetic Impact

*Banaras Khan*

Diabetes Mellitus (DM) is a significant chronic metabolic condition defined by hyperglycemia, which leads to a slew of consequences including neurological disease, pancreatic disease, liver disease, cardiovascular disease, and blindness. The link between diabetes and neurodegenerative illnesses, such as Alzheimer's disease, is well understood. Advanced Glycation End Products (AGEPs) do not build up in the brain, but they have been discovered in senile plaques, where they can lower the solubility of proteins like Tau and amyloid (A). Few studies supported the hypothesis of brain insulin resistance because insulin was shown to improve memory in a few small studies.

In a recent investigation, the glycemic fluctuation was revealed to be a significant underlying cause of diabetic central neuropathy.

Furthermore, there is strong evidence that the gut microbiota plays a role in the pathogenesis of type 2 diabetes and that gut SCFA-producing bacteria influence hippocampal neurogenesis, casting doubt on the original explanation. The microbiota-gut-brain axis has long been considered a possible therapeutic target for central nervous system illnesses. For the first time in this experimental work, researchers looked at the effects of Curcuma on central neuropathy and gut microbiota in type 2 diabetic rats.

The Von Pain Measurement Instrument was used to calculate the MWT. Paw withdrawal generated by physical activity was not identified as a good effect. The experimental methodology was repeated three times at a 15 min interval, with the mean value being recorded at the end. MNCV was measured in the sciatic nerve of the terminally anaesthetized rat at the end of the experimental regimen. The serum was maintained at -80°C after blood samples were taken from the abdominal aorta in pre-incubated test tubes. The data from the current experimental study is presented as mean  $\pm$  SD.

Increased blood glucose levels, as well as diminished insulin and body weight, are prominent symptoms of diabetes. The activity of mechanical withdrawing threshold and motor nerve conduction velocity during neurology dysfunction. A change in lipid was identified during diabetes, and a similar outcome was observed in our experimental work. Diabetic neuropathy boosted brain damage by increasing the level of an inflammatory cytokine. The development of diabetes and its consequences is influenced by oxidative stress. Elevated the level of free radicals in diabetic neuropathy, which increased the level of oxidative stress in tissue. The current purpose of this study is to examine if Curcuma therapy aids in the restoration of the gut microbiota to its original state. When compared to diabetic rats, we discovered that Curcuma treatment affects microbial diversity, which is consistent with previous research. The findings revealed that the *Bacteroidetes* and *Firmicutes* phyla play a significant role in the gut microbiota of rats. When compared to normal control rats, diabetic rats revealed a significant shift in the gut microbiota, with lower percentages of *Candidatus Saccharibacteria* and *Firmicutes* and higher percentages of *Spirochaetes* and *Bacteroidetes* phyla. Direct signalling from the stomach to the central nervous system has been demonstrated using SCFA-GPR interactions.



Microglia, the brain's resident macrophages, rely on the gut microbiota's formation and function, and in rodents, SCFAs and GPR were required to maintain microglia homeostasis and the blood-brain barrier's integrity. A high-fructose diet caused gut dysbiosis and decreased SCFA, resulting in impaired colonic epithelial barrier impairment, induction of neuro-inflammation in the hippocampal and loss of neuronal in rodents, and neurodegeneration changes that could be protected by Curcuma treatment, according to recent research.

#### **MATERIALS AND METHODS**

Liu, Y., B. Huang, Z. Zhu and T. Zheng, 2022. Curcuma ameliorates diabetic neuropathy in streptozotocin induced diabetic rats via alteration of gut microbiota. *Int. J. Pharmacol.*, 18: 374-387.