Press Release, March 15, 2021

Dr. Christiane Menzfeld
HEAD OF PUBLIC RELATIONS

Tel.: +49 (89) 8578-2824
menzfeld@biochem.mpg.de
www.biochem.mpg.de

@MPI_Biochem

Salt consumption controls autoimmune disease

Researchers at the Max Planck Institute (MPI) of Biochemistry show that increased salt consumption has no negative effect on disease progression but is rather beneficial in a mouse model of multiple sclerosis.

Multiple sclerosis (MS) is a chronic inflammatory disease of the nervous system. In this autoimmune disease, the myelin sheath of the nerve cells is attacked by the patient's own immune system. Several animal models are available to study the disease. Researchers at the Max Planck Institute of Biochemistry have now been able to show, contrary to the results of other studies, that moderately increased salt consumption in mice has no negative effect on the course of the disease. In transgenic mice that develop spontaneous MS-like disease, increased salt consumption led to a suppression of the disease. This study was published in the journal *PNAS*.

Sodium chloride, table salt, is an essential mineral that we must consume for a healthy life. However, excessive salt consumption is one of the known health risks, as it has been linked to cardiovascular and kidney diseases. Researchers are also interested in understanding the effect of excessive salt consumption in autoimmune and inflammatory diseases such as MS. Therefore, an animal model of multiple sclerosis called Experimental Autoimmune Encephalomyelitis (EAE) has been used in the past to study the effect of excessive salt consumption. It has been reported that it leads to exacerbation of the disease.

Different disease model, different result

Gurumoorthy Krishnamoorthy, head of the research group "Neuroinflammation and Mucosal Immunology" at the Max Planck Institute of Biochemistry, and his team now show an opposite finding. The research group leader explains "For our studies, we used a different mouse model that spontaneously develops MS-like symptoms. We have no evidence that increased salt consumption in the animals promoted or exacerbated the disease." Surprisingly, the scientists were even able to show that increased salt consumption suppressed the development of the autoimmune disease.

The blood-brain barrier

"For the analysis, we focused on the blood-brain barrier," reports Shin-Young Na, first author of the study. The blood-brain barrier is an important barrier between the bloodstream and central nervous system. It prevents the uncontrolled flow of substances as well as immune cells from the blood into the central nervous system. So-called tight junctions help with this diffusion barrier. These are membrane molecules that, as the

name suggests, create tight junctions between cells. "We could see that in the animals consuming an increased amount of salt, serum levels of the glucocorticoid hormone corticosterone were elevated. This increased level of corticosterone led to increased expression of tight junction molecules in the endothelial cells. As a result, the blood-brain barrier is strengthened and the entry of inflammatory T cells into the nervous system was blocked," Na further reports.

Gurumoorthy Krishnamoorthy says, "Our results show that moderately increased salt consumption has multiple and potentially beneficial effects on central nervous system autoimmunity in mice. I assume that the opposite effect compared to the previous studies is related to the use of different animal models where the blood-brain barrier is artificially opened through injection of pertussis toxin. This is not the case in our disease model and this model is closer to the early stage of MS disease in humans."

Original Publication:
S.-Y. Na, M. Janakiraman, A. Leliavski & G. Krishnamoorthy: High salt diet suppresses autoimmune demyelination by regulating the blood-brain barrier permeability. *PNAS*, March 2021.
DOI: https://doi.org/10.1073/pnas.2025944118

Caption:
In a surprising finding, increased salt consumption is found to regulate the blood-brain barrier permeability to suppress autoimmune disease development in mice.

Copyright:
Janvier, Adobe Stock

About Gurumoorthy Krishnamoorthy

Dr. Gurumoorthy Krishnamoorthy received his doctorate in neuroimmunology from the Max Planck Institute of Neurobiology and LMU. Subsequently, he worked as a postdoctoral fellow and as a project leader at the Max Planck Institute of Neurobiology. He was awarded the Sobek prize from the German MS society in 2009. Since 2015 he is leading a research group "Neuroinflammation and Mucosal Immunology" at the Max Planck Institute of Biochemistry in Martinsried supported by the European research council (ERC) starting grant and the Max Planck Society.

About the Max Planck Institute of Biochemistry

The Max Planck Institute of Biochemistry (MPIB) belongs to the Max Planck Society, an independent, non-profit research organization dedicated to top-level basic research. As one of the largest institutes of the Max Planck Society, 850 employees from 45 nations work here in the field of life sciences. In currently eight departments and about 25 research groups, the scientists contribute to the newest findings in the areas of biochemistry, cell biology, structural biology, biophysics, and molecular science. The MPIB in Munich-Martinsried is part of the local life-science-campus where two Max Planck Institutes, a Helmholtz Center, the Gene-Center, several bio-medical faculties of two Munich universities, and several biotech-companies are located in close proximity. http://www.biochem.mpg.de

Contact:

Dr. Gurumoorthy Krishnamoorthy
Neuroinflammation and Mucosal Immunology
Max-Planck-Institut für Biochemie
Am Klopferspitz 18
82152 Martinsried
Email: guru@biochem.mpg.de
https://www.biochem.mpg.de/krishnamoorthy

Dr. Christiane Menzfeld
Öffentlichkeitsarbeit
Max-Planck-Institut für Biochemie
Am Klopferspitz 18
82152 Martinsried
Tel. +49 89 8578-2824
Email: pr@biochem.mpg.de