



Summary

Involvement of brain function and networks after exposure to extrinsic stimuli: a possible underlying mechanism in multiple chemical sensitivity Kenichi Azuma, PhD

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Dr. Kenichi Azuma's presentation offered a scientifically rigorous exploration of the neurological and physiological mechanisms underlying Multiple Chemical Sensitivity (MCS), focusing specifically on how external chemical stimuli impact brain function. As an environmental health expert at Kindai University in Japan, Dr. Azuma drew upon a series of neuroimaging and behavioral studies conducted over several years to build a case for the biological basis of MCS.

He began by defining MCS as a chronic disorder marked by heterogeneous, non-specific symptoms in response to everyday chemical exposures, such as those found in perfumes, cleaning products, and industrial compounds. His research focused on how these exposures alter brain network activity, particularly in the prefrontal cortex. Using neuroimaging techniques, his studies demonstrated that MCS patients show significant activation in the prefrontal cortex when exposed to odorants, compared to unaffected individuals. This hyperactivation supports the theory that MCS involves a sensitized central nervous system. Hence, the brain networks of a person with MCS may act more strongly in response to chemicals, relative to people without MCS.

Dr. Azuma further explained that this response could be triggered either by a single, high-dose exposure or by the accumulation of smaller exposures over time. He emphasized that these reactions are not governed only by one's sense of smell, suggesting that the brain's response occurs even when chemical levels are below conscious detection thresholds. Using brain activation maps, he provided visual evidence demonstrating differences in oxygenated hemoglobin flow in key regions, such as the orbitofrontal cortex, signalling greater activation in areas responsible for processing sensory and emotional stimuli, between MCS patients and healthy controls.

Another key point in his presentation was the concept of delayed recovery. Even after exposure had ended, MCS patients exhibited prolonged activation in certain brain regions, pointing to

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lasting neurological effects that could explain why symptoms persist well beyond the initial contact with chemicals.

Dr. Azuma also discussed the broader health and diagnostic implications of his findings. For instance, MCS symptoms are often mistaken for psychological or respiratory conditions such as anxiety or asthma. His research, however, indicates that the condition stems from neurological dysfunction rather than a psychiatric or pulmonary cause. He touched on the challenges of differentiating MCS from other illnesses and stressed the need for more precise diagnostic criteria based on objective biomarkers.

In conclusion, Dr. Azuma called for continued research into the neurological underpinnings of MCS. He emphasized that a more nuanced understanding of how chemical exposures affect brain networks could pave the way for improved diagnostics, interventions, and public health policies that recognize and accommodate individuals with MCS. His work represents a significant contribution to the global effort to legitimize MCS as a biologically grounded condition, challenging outdated notions that it is merely psychological or imaginary.

Citations

- Azuma K, et al. (2013) Changes in cerebral blood flow during olfactory stimulation in patients with multiple chemical sensitivity: A multi-channel near-infrared spectroscopic study. *PLoS ONE* 8(11): e80567. doi:10.1371/journal.pone.0080567.
- Azuma K, et al. (2015) Assessment of cerebral blood flow in patients with multiple chemical sensitivity using near-infrared spectroscopy—recovery after olfactory stimulation: a case-control study. *Environ Health Prev Med* 20(3):185–194.
- Azuma K, et al. (2016) Association of odor thresholds and responses in cerebral blood flow of the prefrontal area during olfactory stimulation in patients with multiple chemical sensitivity. . *PLoS ONE 11*(12): e0168006.