

Research links diseases to 'corroding' effect caused by high-salt diets

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Graphical abstract. Credit: Arteriosclerosis, Thrombosis, and Vascular Biology (2023). DOI: 10.1161/ATVBAHA.122.318439

When hungry thoughts arise, think twice before reaching for a salty snack.

New Brock University-led research shows how too much <u>salt</u> in a diet damages organs, leading to <u>chronic diseases</u> down the road.

This occurs because high salt levels peel away the protective layer on the



surface of <u>blood vessels</u> as well as the cells that line blood vessels, says the study, led by Brock Professor of Health Sciences Newman Sze.

"The process is similar to salt corroding the protective paint surface on a car, which can cause the metal body of the vehicle to rust," says Sze, who is Canada Research Chair in Mechanisms of Health and Disease.

The study, "Endothelial damage arising from high salt hypertension is elucidated by vascular bed systematic profiling," published in *Arteriosclerosis, Thrombosis, and Vascular Biology*, looked at how high salt levels impact glycocalyx and <u>endothelial cells</u>, which are the protective layer of proteins and cells that come into direct contact with blood circulating throughout the body.

Scientists have long established links between high salt intake and <u>blood</u> <u>pressure</u>, contributing to a host of health issues, including hypertension, left ventricular hypertrophy, and increased risk of stroke and <u>coronary</u> <u>heart disease</u>.

But it's unclear how high salt levels affect the body's endothelium and circulatory systems. The endothelium lines the innermost layer of blood vessel walls, ensuring that the body's tissues are supplied with nutrients and oxygen.

Sze and his team developed new methods to study the structure and function of blood vessels that experienced diet-induced hypertension with those of healthy diets to observe critical changes in the blood vessel structures.

The team discovered that consuming a diet high in salt leads to significant damage to the protective layer of glycocalyx proteins on the surface of the endothelium.



This damage can lead to a higher risk of stroke and <u>heart attack</u>. The receptors that regulate the inflammation of artery walls and blood coagulation are particularly affected, which can exacerbate these risks.

Sze and his team also discovered the presence of new biological molecules, called "biomarkers," that flag normal or abnormal cell processes.

The glycocalyx protein that is peeled away from the blood vessel continues to circulate in the patient's bloodstream and can be identified as a biomarker.

"We've identified new biomarkers for diagnosing blood vessel damage, identifying patients at risk of heart attack and stroke, and developing new drug targets for therapy for a range of blood vessel diseases, including heart, kidney and lung diseases as well as dementia," says Sze.

Diets high in salt are a huge problem in Canada and around the world. According to a World Health Organization (WHO) report released March 9, Canadians consume 9.1 grams of salt per day.

Meanwhile, the WHO recommends that the <u>average person</u> consume no more than five grams, or one teaspoon, of salt per day. The WHO has set a global target of reducing sodium intake by 30% by 2025.

"Many Canadians add extra salt to their food during cooking or at the table," says Sze. "Moreover, many Canadians, particularly children and <u>young adults</u>, eat processed and fast foods that have high level of salts."

Sze's recent study builds on his work as a Canada Research Chair.

He studies diseases that occur as people age, specifically diseases related to the lining of blood vessels becoming damaged. Sze's lab has developed



new research methods that investigate how the blood vessel lining deteriorates over time.

More information: Arada Vinaiphat et al, Endothelial Damage Arising From High Salt Hypertension Is Elucidated by Vascular Bed Systematic Profiling, *Arteriosclerosis, Thrombosis, and Vascular Biology* (2023). DOI: 10.1161/ATVBAHA.122.318439

Provided by Brock University

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