

Junk-food diets spur inflammation more than saturated fats alone

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(Medical Xpress) -- A diet based on American junk food could lead to more obesity-induced inflammation than a diet high in animal fat, according to a new study by researchers at the University of North Carolina at Chapel Hill.

The study was published this month in the <u>Public Library of Science</u> One (<u>PLoS ONE</u>).

The study analyzed <u>inflammatory responses</u> in rats fed different diets: control diets, a lard-based high-fat <u>diet</u> and a "cafeteria <u>junk-food</u>" diet consisting of nutrient-poor snacks such as salami, chocolate, cookies and chips.

"The diet that consisted of human junk food caused the most <u>inflammation</u> and dramatic metabolic changes," said Liza Makowski, Ph.D., assistant professor of nutrition at UNC's Gillings School of Global Public Health and the study's senior author.

A junk-food diet contains many ingredients associated with increased risk for coronary artery disease, stroke and Type 2 diabetes, including saturated fat, trans-fats, sodium and cholesterol. The diet also is low in protective nutrients such as fiber.

While it has been known for some time that <u>obesity</u> can cause inflammation in fatty tissue, Makowski said, this study is one of the first to show that a junk-food diet may cause dramatic alterations in certain



metabolites – molecular chemicals created when food is converted to energy.

These alterations may be responsible for obesity-induced inflammation and increased insulin resistance and could be a major contributing factor to metabolic syndrome, the cluster of factors that increase a person's risk for coronary artery disease, stroke and Type 2 diabetes.

The junk-food diet used in the study may be superior to high-fat diets for modeling modern human obesity trends, including exposure to energy-dense and nutrient-poor diets, early and rapid obesity development, and elevated markers of metabolic syndrome and inflammation.

"Biomarkers revealed in our study could be useful in future studies," said Makowski. "This needs to be replicated in human studies; it could be highly useful in future diabetes research."

Study co-authors from UNC's Gillings School of Global Public Health are Alex J. Freemerman, Ph.D., nutrition research associate; Pei-Fen Kuan, Ph.D., research assistant professor of biostatistics; Heather A. Brauer, Ph.D., postdoctoral research associate in epidemiology; and Melissa A. Troester, Ph.D., research assistant professor of epidemiology. Makowski, Kuan and Troester also are affiliated with the UNC Lineberger Comprehensive Cancer Center.

More information: www.plosone.org/article/info %3Adoi%2F10.1371%2Fjournal.pone.0038812

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