Grilled, seared foods may add to waistlines, disease risk

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Kee-Hong Kim found that the chemicals created by some cooking methods, including grilling, may allow immature fat cells to survive and accumulate lipids, blocking a cellular process that would normally kill those cells. Credit: Purdue Agricultural Communication photo/Tom Campbell

(Medical Xpress)—A steak slapped onto a hot barbecue will leave the meat with black grill lines that add flavor and aroma, but the chemicals contained in charred, seared and fried foods may over time kick-start the body's ability to add new fat cells and increase the risk of age-related

diseases, a Purdue University study shows.

Over time, the human body shuts down the ability of young fat cells to mature and accumulate lipids. But grilling, searing and frying create glycated proteins, which result from proteins chemically bonding with sugar.

"When you put proteins and sugars together at high temperatures, there is a chemical reaction, and that creates flavor and texture, which we think of as good things," said Kee-Hong Kim, an assistant professor of food science. "Research suggests that these glycated proteins are involved in age-related diseases like cardiovascular disease."

Kim wanted to see whether glycated proteins affect the speed at which precursor, or immature, fat cells turn into mature fat cells. Using a cell culture, Kim saw no change in how quickly those immature cells accumulated lipids, which is stored as fat in cells, but he did notice something else.

"Older animals don't generally accumulate new fat cells. Those precursor cells lose their ability to become mature as we age," Kim said. "But when exposed to glycated proteins, immature fat cells started to differentiate and accumulate lipids like they would in a younger animal.

"When we continuously consume glycated proteins we might turn on the ability of precursor cells to mature," said Kim, whose findings were published in the Journal of Biological Chemistry.

Kim found that the byproducts of glycated proteins - advanced glycation end products, or AGEs - interfere with cellular processes that should kill immature fat cells in older animals. That means those animals, or people, may accumulate more fat cells than they should, and those cells store compounds that can lead to inflammation and certain types of diseases.

AGEs interact with a protein called p53, which usually begins cell death and aging programs for immature fat cells. With p53 disrupted, the immature fat cells survive and can accumulate lipids.

"It's really interesting that a single food component could contribute to a number of diseases," said Chih-Yu Chen, a doctoral student in Kim's laboratory. "This could cause people to think about their food preparation and diet choices."

Kim is investigating the relationship between obesity and a number of chronic illnesses such as diabetes, cardiovascular disease and some types of cancer. He believes glycated proteins may be a factor in some of those diseases.

"It's not immediately toxic, but if you're exposed over a long period of time, some portions of the glycated materials accumulate in the cells or tissues, and over time, that contributes to inflammation and oxidative stress," Kim said.

Next, Kim would like to confirm his findings in an animal model. The Purdue Research Foundation, a Ralph W. and Grace M. Showalter Research Trust Award, and Purdue startup funds supported his work.

More information: An Advanced Glycation End Products (AGEs)-The Receptor for AGEs Axis Restores Adipogenic Potential of Senescent Preadipocytes through Modulation of p53 Protein Function, Chih-Yu Chen, Allison Martorano Abell, Yang Soo Moon, and Kee-Hong Kim

ABSTRACT
Impaired adipogenic potential of senescent preadipocytes is a hallmark of adipose aging and aging-related adipose dysfunction. While advanced glycation end products (AGEs) derived from both foods and endogenous

non-enzymatic glycation, and AGEs-associated signaling pathways are known to play a key role in aging and its-related diseases, the role of AGEs in adipose aging remains elusive. We show a novel pro-adipogenic function of AGEs in replicative senescent preadipocytes and mouse embryonic fibroblasts, as well as primary preadipocytes isolated from aged mice. Using glycated bovine serum albumin (BSA) as a model protein of AGEs, we examined that glycated BSA restores impaired adipogenic potential of senescent preadipocytes in vitro and ex vivo. However, glycated BSA showed no effect on adipogenesis in non-senescent preadipocytes. AGEs-induced the receptor for AGEs (RAGE) expression is required for the pro-adipogenic function of AGEs in senescent preadipocytes. This is through RAGE-dependent impairment of p53 expression and p53 function in regulating p21 expression in senescent preadipocytes. We also observed a direct binding between RAGE and p53 in senescent preadipocytes. Taken together, our findings reveal a novel pro-adipogenic function of AGEs-RAGE axis in p53-regulated adipogenesis of senescent preadipocytes, providing new insights into aging-dependent adiposity by diet-driven and/or endogenous glycated proteins.

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