

THE RED MEAT DILEMMA:

Will It Give You Cancer?

Processed meat causes colorectal cancer and is associated with an increased risk of developing stomach cancer. Red meat probably increases risk of cancer and is most closely associated with an increased risk of colorectal, pancreatic and prostate cancer.

This news, from a report by the International Agency for Research on Cancer (IARC), a branch of the World Health Organization (WHO), created a media circus with vegetarians and meat lovers throwing nasty virtual punches while gritting their teeth and madly typing away on their keyboards. Steer clear of the emotional tug-of-wars attempting to steer you to one side of the meat-eating spectrum or the other, and you'll find out how this study fits into your risk of developing cancer.

Processed meat has been salted, cured, fermented, smoked or been through other processes used to preserve the meat and enhance flavor. Hot dogs, sausages, corn beef, beef jerky and canned meat are all examples of processed meat. Processed meats may also include other meats or meat byproducts such as blood.

Red meat is unprocessed mammalian muscle meat—beef, pork, veal, lamb, mutton, horse or goat meat, including minced or frozen meat. Red meat is usually consumed cooked.

The Study

The IARC (part of the World Health Organization, aka WHO) Working Group looked at more than 800 epidemiological studies on processed meat, red meat and cancer from many countries with diverse ethnicities and diets. "They relied on the strongest population-based studies with the best designs for their evaluation," states Alice G. Bender, MS, RDN, associate director of nutrition programs, American Institute for Cancer Research (AICR).

No study can completely rule out the possibility that other dietary factors associated with diets high in processed and red meat contribute to colorectal cancer. However, the large amount of data and consistent associations of colorectal cancer with consumption of processed meat across studies in different populations means it is unlikely that chance, bias and confounding variables influenced the results of this report. "Confounding factors known for links to cancer risk include BMI, smoking, physical activity, age, gender, alcohol use, NSAID use; [NSAIDs are nonsteroidal anti-inflammatory drugs such as ibuprofen] while dietary fiber and dairy products are linked with lower risk," states Karen Collins, MS, RDN, CDN, FAND, nutrition advisor to the American Institute for Cancer Research.

Though the IARC report may seem like earth shattering news, scientists from the World Cancer Research Fund (WCRF), an international nonprofit charity that includes several organizations including the AICR, write a number of reports as part of an ongoing initiative called the Continuous Update Project (CUP). "CUP reports are continuously updated and their conclusions on processed meat and colorectal cancer are based on cell studies, animal research, short-term human clinical studies of biomarkers and adenomas (benign polyps that are the starting point of the vast majority of colorectal cancers) AND longer-term observational population studies," says Collins. For several

years, WCRF has issued recommendations suggesting consumers avoid processed meat and limit intake of red meat to 11 ounces or less per week.

IARC placed processed meat into Group 1— it is cancer causing. Red meat was placed in Group 2A— it probably causes cancer. "For red meat, IARC said the evidence was strong, but not as consistent," states Bender. Several well-designed studies showed no association between red meat and colorectal cancer, which suggests that other diet and lifestyle factors could be responsible for the association between red meat and colorectal cancer. However, the Group 2A classification is based on strong mechanistic evidence for the association between red meat and cancer. Mechanistic evidence refers to mechanisms that support the development of cancer and in this case refers to a statistically significant association between red or processed meat consumption and lesions that may be cancerous as well as changes in markers of oxidative stress— suggesting red and processed meat cause damage to the body beyond which the body's antioxidant system can repair. Also, several known or suspected carcinogens (cancer-causing compounds) are formed in meat when it is cooked and also formed in the colon when we eat processed meat or red meat. The IARC report mentions three of these compounds: N-nitroso compounds (NOC), heterocyclic aromatic amines (HAA) and polycyclic aromatic hydrocarbons (PAH).

Heme iron in processed and red meat leads to the formation of NOC in the colon. In addition, nitrites, amines and amides are precursors to the formation of NOCs in the body. In fact, an estimated 45-75 percent of our total exposure to NOCs comes from our body's production of these compounds. NOCs, as well as PAHs, are also formed when meat is processed, including curing and smoking. HAAs and PAHs are formed when meat is cooked. Cooking meat on high temperatures as done during grilling, barbecuing and frying leads the greatest production of these chemicals. When fat and juices from meat drip onto fire, resulting in flames, PAHs develop and adhere to the meat like shellac. HAAs are formed when amino acids, sugars and creatine react at high temperatures. HAAs and PAHs must be metabolized by specific enzymes (a process called bioactivation) before they can damage DNA. Yet the activity of these enzymes varies between people and therefore, one's risk of developing cancer due to HAA and PAH exposure depends on how they metabolize these compounds.

Though considered mechanistic evidence for the relationship between processed and red meat and the development of cancer, these compounds are not unique to processed and red meat. NOCs are found in smoked fish, the malt in beer and whiskey production, pickled vegetables and foods stored under humidity leading to fungi that generate nitrosamines. PAHs are also found in air pollution. In addition, PAHs and HAAs are formed in fish and poultry (chicken, turkey, ducks, geese) cooked over high temperatures.

Is There a Difference Between Grass-Fed Beef Jerky and a Hot Dog?

Natural, uncured bacon, sausage and other processed meats that do not contain added nitrites or nitrates are still

possible that those who consume high BCAA content diets may have a relative deficiency of tryptophan in the diet.

Both tryptophan and 5-HTP have been reported to improve sleep onset. However, have they been shown to have weight-loss effects acting as serotonin precursors? Well, enough so that many "obesity specialists" recommend a 5-HTP/carbidopa combination to complement phentermine (a stimulant drug) for their patients.⁸ Carbidopa prevents the conversion of 5-HTP to 5-HT before it gets to the brain.⁹ In the report, one case was described where a patient lost 24 percent of initial bodyweight in six months using the 5-HTP/carbidopa combination without phentermine.

Of course, tryptophan and 5-HTP are typically taken as dietary supplements. So, it is gratifying to see published trials using randomized subjects. 5-HTP, taken at a modest dose of 900 milligrams per day, resulted in significant weight loss compared to the placebo group in a setting of a recommended caloric guideline, or eating ad lib (without restrictions).¹⁰ Further, there was a reduction in carbohydrate intake and reported earlier satiety (feeling full when eating). In type 2 diabetics, who are reported to have decreased brain serotonin activity, 5-HTP supplementation (750 mg/day) with no caloric restrictions resulted in weight loss, and reduced carbohydrate and fat intake.¹¹ Clearly, there is value to supplementing the diet to support serotonin and melatonin production, and availability for people seeking to lose weight.

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HEALTHY BODY AND A GOOD NIGHT'S SLEEP

The body is designed to function autonomously (without direction) when it is healthy. Part of good health is supporting the mass and function of healthy tissue (e.g., skeletal muscle, brown fat). When the serotonin pathways are active, they can aid not only in reducing excess calorie intake, but also stimulate brown fat activity. Melatonin is involved in a myriad of functions, but even before birth, it plays a role in the creation of brown and beige fat. These thermogenic fats, through the actions of uncoupling protein that separate energy production from calorie burning, can aid greatly in fat loss and maintaining a lean physique. Brown fat is designed to do what people try to accomplish with ephedrine/caffeine or DNP. Though both serotonin and melatonin can be supplemented through increasing tryptophan or 5-HTP, as well as taking melatonin orally, the actions at specific sites in the brain are key to gaining the complete benefits. Part of accomplishing this involves developing good sleep hygiene—regular and sufficient sleep times, avoiding bright lights prior to sleep and maintaining a quiet and cool room.

REFERENCES



Wakame Seaweed (Fucoxanthin) Burns Fat!

There are foods that most of us are never exposed to due to cultural or geographic limitations. When a food is consumed only by people in a certain region or culture, it allows researchers to see if different health aspects are present. It does not mean that the food, or cultural practice, causes the difference, just that there might be something present to look at more closely.

Wakame is called "brown seaweed" in the United States, and is a staple food ingredient in many Southeastern Asian regions. Though no single factor accounts for the difference, it is clear that the Western diet— you know the one ... cheeseburger, fries and a large diet soda— has played a role in the prevalence of obesity in the United States. Asian cultures avoided the obesity "contagion" until the United States was kind enough to export fast food.

Analysis of wakame revealed a carotenoid (the same class of chemical as vitamin A, astaxanthin, beta-carotene, etc.) called fucoxanthin that has been shown to affect a number of responses in animal studies, that suggest it may be an effective fat-loss agent for humans. Two recent reviews on fucoxanthin describe many of these effects.¹² Again, much of this relies on animal or "test tube" studies.

Fucoxanthin has repeatedly been shown to prevent or reduce the fat gain of rodents placed on high-fat diets; reduce "fatty liver," which is associated with insulin resistance and metabolic syndrome; improve insulin sensitivity; reduce inflammation in fat tissue; lower inflammatory cytokine (messenger signals) release from fat; increase enzymes associated with fat burning; and increase UCP-1 in white fat, which causes the fat cell to "waste" fat calories as heat. This is sometimes referred to as "beige fat," as white fat (which normally stores fat) acts like brown fat, which burns fatty acids and glucose to generate heat. All of these mechanisms are separate avenues of research for fat-loss drugs.

A pharmacokinetic study reported fucoxanthin is converted into the presumably active metabolite fucoxanthinol by intestinal and liver cells, reaching a maximal concentration in blood at four hours post-ingestion, with a seven-hour half-life.³ This is different from the rodent results, so it does emphasize the need for further human trials before any safety, efficacy and dosing recommendations can be given. Only a single human trial published in 2010 has been reported, though the findings were very promising.⁵ Among obese non-diabetic women, some with fatty liver disease (NASH), supplementation with a pomegranate oil and brown seaweed extract containing 2.4 milligrams of fucoxanthin was associated with an average loss of 11 pounds in 16 weeks, with significant losses in body and liver fat; liver enzymes and inflammatory markers were also lower. Further, resting energy expenditure was higher in those receiving the fucoxanthin supplement.

Given its role as a traditional food ingredient, fucoxanthin appears to be a product that can be reasonably included in a healthy person's diet if they wish to try to receive the benefits suggested by the existing literature. Based upon the rodent pharmacokinetic data, it appears that fucoxanthin's metabolites may accumulate with regular use, so it may not be necessary to use high doses, though the results may take weeks to months to be realized.⁴ ■

References

1. Muradian K, Vaserman A, et al. Fucoxanthin and lipid metabolism: A mini-review. *Nutr Metab Cardiovasc Dis* 2015 Jun 3. Epub. ahead of print
2. Gammon MA, D'Orazio N. Anti-obesity activity of the marine carotenoid fucoxanthin. *Mar Drugs* 2015 13 2199-2214
3. Hashimoto T, Ozaki Y, et al. Pharmacokinetics of fucoxanthinol in human plasma after the oral administration of kombu extract. *Br J Nutr* 2012;107:1566-9
4. Hashimoto T, Ozaki Y, et al. The distribution and accumulation of fucoxanthin and its metabolites after oral administration in mice. *Br J Nutr* 2009 Jul;102:242-8
5. Abidov M, Ramazanov Z, et al. The effects of Xanthigen in the weight management of obese premenopausal women with non-alcoholic fatty liver disease and normal liver fat. *Diabetes Obes Metab* 2010 12 72-81

