In this issue of Circulation: Heart Failure, Kaluza et al. report the results from their prospective follow-up trial on 37035 Swedish men, in which total and processed red meat consumption were associated with greater heart failure (HF) incidence and HF mortality during an =12-year follow-up. Compared with men consuming 0.5 servings/d of red meat (1 serving=75 g or 3 oz), those consuming 2.3 servings/d were 20% more likely to develop HF and 30% more likely to die from HF. This effect was largely attributed to processed red meat (eg, sausage and cold cuts); men consuming 1.2 servings/d of processed red meat were 28% more likely to develop HF and 43% more likely to die from HF compared with those consuming 0.2 servings/d. Although no association between unprocessed red meat consumption and HF risk was observed, this negative finding should be interpreted with caution because the difference between the groups with the highest and lowest consumption was <1 serving/d. The findings from Kaluza et al.1 support those from a 20-year follow-up on 21 120 male physicians in the United States,2 in which men consuming 1.4 servings/d of total red meat had a 20% higher HF incidence than those consuming 0.2 servings/d. In addition, Nettleton et al.3 observed a 27% increase in HF incidence for every 1 serving/d increase in red (mostly processed) meat consumption in 15 143 men and women in the United States; although this association became nonsignificant after accounting for hypertension, cardiovascular disease, diabetes mellitus, and other factors, this is not entirely surprising, because these factors could be part of the mechanistic connection between red meat intake and HF. From a broader health perspective, it is notable that meta-analyses, each of which represents numerous studies, demonstrated that red meat consumption (especially processed red meat) is positively associated with the risk of developing coronary artery disease,4 stroke,5 cancer,6 type 2 diabetes mellitus,4 and obesity and with all-cause mortality.7 Taken together, the studies of Kaluza et al.1 and others indicate that red and processed meat consumption are contributors to heart failure and other diseases and to mortality.

The mechanisms for the adverse effects of red and processed red meat consumption on heart failure risk are not clear, are likely multifactorial, and may depend on HF cause, with some factors increasing ischemic HF risk by promoting atherosclerosis, whereas others may promote nonischemic HF through more direct effects on the myocardium. Among the best recognized atherogenic compounds in red meat are saturated fat and cholesterol, both of which are present in greater quantities in processed red meat (eg, sausage and salami) than in unprocessed red meat. However, some researchers have questioned their role in the development of ischemic heart disease. It is also possible that red meat consumption, as a source of saturated and total dietary fat, causes transient but regular postprandial lipemia, which has adverse effects on the vasculature and also increases oxidative stress,8 which in turn promotes myocardial fibrosis and remodeling, and contractile dysfunction. Another candidate is heme iron, which gives red meat its color. Although dietary heme iron is more bioavailable than nonheme iron and may be useful for treating some types of iron deficiency, its absorption is poorly regulated, which increases the risk of clinical or subclinical iron toxicity. Iron toxicity may promote atherosclerosis through mechanisms involving oxidative stress and endothelial dysfunction9 and may promote fibrosis and remodeling of the heart, which cause contractile dysfunction. More recently, t-carnitine, which is abundant in red meat, has been found to undergo endogenous conversion to the atherogenic compound trimethylamine-N-oxide through a 2-step process involving the bacteria in the gut and hepatic metabolism10 and thus may contribute to ischemic HF. Finally, lipophilic organic pollutants such as dioxins are known to bioaccumulate up the food chain, making red and processed meat a major dietary source of these toxins.11 Evidence indicates that cardiac dysfunction is associated with elevated circulating levels of some of these organic pollutants.12

Red meat consumption might also be associated with an increased risk of HF through mechanisms that are not directly attributable to red meat itself. One possibility is that high intakes of red and other meat might displace micronutrient-rich plant foods from the diet and thus lead to micronutrient deficiencies that promote heart failure. Lower intakes of fruits and vegetables increase HF risk,14 and micronutrient deficiencies are common in patients with HF, including deficiencies in antioxidant nutrients, such as vitamin C and selenium.15 Furthermore, plant-rich dietary patterns that contain little or no red meat (ie, Dietary Approaches to Stop Hypertension [DASH] and Mediterranean diet patterns) are associated with lower risk of developing HF.16 Diets that are rich in red meat also tend to contain more energy (calories) and accordingly are associated with obesity,7 which increases HF risk. In contrast, calorie-restricted diets and low levels of adiposity, especially

The mechanisms for the adverse effects of red and processed red meat consumption on heart failure risk are not clear, are likely multifactorial, and may depend on HF cause, with some factors increasing ischemic HF risk by promoting atherosclerosis, whereas others may promote nonischemic HF through more direct effects on the myocardium. Among the best recognized atherogenic compounds in red meat are saturated fat and cholesterol, both of which are present in greater quantities in processed red meat (eg, sausage and salami) than in unprocessed red meat. However, some researchers have questioned their role in the development of ischemic heart disease. It is also possible that red meat consumption, as a source of saturated and total dietary fat, causes transient but regular postprandial lipemia, which has adverse effects on the vasculature and also increases oxidative stress, which in turn promotes myocardial fibrosis and remodeling, and contractile dysfunction. Another candidate is heme iron, which gives red meat its color. Although dietary heme iron is more bioavailable than nonheme iron and may be useful for treating some types of iron deficiency, its absorption is poorly regulated, which increases the risk of clinical or subclinical iron toxicity. Iron toxicity may promote atherosclerosis through mechanisms involving oxidative stress and endothelial dysfunction and may promote fibrosis and remodeling of the heart, which cause contractile dysfunction. More recently, t-carnitine, which is abundant in red meat, has been found to undergo endogenous conversion to the atherogenic compound trimethylamine-N-oxide through a 2-step process involving the bacteria in the gut and hepatic metabolism and thus may contribute to ischemic HF. Finally, lipophilic organic pollutants such as dioxins are known to bioaccumulate up the food chain, making red and processed meat a major dietary source of these toxins. Evidence indicates that cardiac dysfunction is associated with elevated circulating levels of some of these organic pollutants.

Red meat consumption might also be associated with an increased risk of HF through mechanisms that are not directly attributable to red meat itself. One possibility is that high intakes of red and other meat might displace micronutrient-rich plant foods from the diet and thus lead to micronutrient deficiencies that promote heart failure. Lower intakes of fruits and vegetables increase HF risk, and micronutrient deficiencies are common in patients with HF, including deficiencies in antioxidant nutrients, such as vitamin C and selenium. Furthermore, plant-rich dietary patterns that contain little or no red meat (ie, Dietary Approaches to Stop Hypertension [DASH] and Mediterranean diet patterns) are associated with lower risk of developing HF. Diets that are rich in red meat also tend to contain more energy (calories) and accordingly are associated with obesity, which increases HF risk. In contrast, calorie-restricted diets and low levels of adiposity, especially

The mechanisms for the adverse effects of red and processed red meat consumption on heart failure risk are not clear, are likely multifactorial, and may depend on HF cause, with some factors increasing ischemic HF risk by promoting atherosclerosis, whereas others may promote nonischemic HF through more direct effects on the myocardium. Among the best recognized atherogenic compounds in red meat are saturated fat and cholesterol, both of which are present in greater quantities in processed red meat (eg, sausage and salami) than in unprocessed red meat. However, some researchers have questioned their role in the development of ischemic heart disease. It is also possible that red meat consumption, as a source of saturated and total dietary fat, causes transient but regular postprandial lipemia, which has adverse effects on the vasculature and also increases oxidative stress, which in turn promotes myocardial fibrosis and remodeling, and contractile dysfunction. Another candidate is heme iron, which gives red meat its color. Although dietary heme iron is more bioavailable than nonheme iron and may be useful for treating some types of iron deficiency, its absorption is poorly regulated, which increases the risk of clinical or subclinical iron toxicity. Iron toxicity may promote atherosclerosis through mechanisms involving oxidative stress and endothelial dysfunction and may promote fibrosis and remodeling of the heart, which cause contractile dysfunction. More recently, t-carnitine, which is abundant in red meat, has been found to undergo endogenous conversion to the atherogenic compound trimethylamine-N-oxide through a 2-step process involving the bacteria in the gut and hepatic metabolism and thus may contribute to ischemic HF. Finally, lipophilic organic pollutants such as dioxins are known to bioaccumulate up the food chain, making red and processed meat a major dietary source of these toxins. Evidence indicates that cardiac dysfunction is associated with elevated circulating levels of some of these organic pollutants.

Red meat consumption might also be associated with an increased risk of HF through mechanisms that are not directly attributable to red meat itself. One possibility is that high intakes of red and other meat might displace micronutrient-rich plant foods from the diet and thus lead to micronutrient deficiencies that promote heart failure. Lower intakes of fruits and vegetables increase HF risk, and micronutrient deficiencies are common in patients with HF, including deficiencies in antioxidant nutrients, such as vitamin C and selenium. Furthermore, plant-rich dietary patterns that contain little or no red meat (ie, Dietary Approaches to Stop Hypertension [DASH] and Mediterranean diet patterns) are associated with lower risk of developing HF. Diets that are rich in red meat also tend to contain more energy (calories) and accordingly are associated with obesity, which increases HF risk. In contrast, calorie-restricted diets and low levels of adiposity, especially
in combination with a plant-based nutrient-rich diet, decrease oxidative stress and damage, reduce systemic inflammation, and improve cardiac diastolic function, suggesting a reduction in HF risk.17

Kaluza et al1 also report that processed red meat consumption is especially problematic for HF risk.1 This finding adds to existing literature that implicates processed red meat in the pathogenesis of numerous diseases. They describe the potential mechanisms by which additives in processed red meat might increase HF risk. These include sodium through effects on blood pressure, nitrates through effects on vascular function and insulin secretion (though controversial), phosphorous via effects on calcium–phosphate handling, and polycyclic aromatic hydrocarbons in smoked meat through their atherogenic effects. As described above, it is also conceivable that processed red meat is more strongly associated with HF risk than unprocessed red meat because it frequently contains more total and saturated fat and cholesterol. Undoubtedly, the task of identifying HF-promoting compounds in processed red meat is daunting, because processed meats are commonly mixtures of many tissues (ie, not solely muscle tissue) from multiple animal species and contain various additives for which health effects may not be known.

A complicating factor in studies on processed and unprocessed meat is the fact that classification of meat as processed or unprocessed is difficult and error prone, especially when performed with food frequency questionnaires or other instruments completed by study participants. Classification of products such as sausage as processed is straightforward in that the meat is ground, mixed with other meats, fats, flavorings, and salts, is repackaged into various forms and casings, and is cooked, smoked, and dried. At the other end of the spectrum, muscle tissue that has been removed from a freshly slaughtered animal without further handling or manipulation would be considered unprocessed (the meat industry often refers to this type of meat as minimally processed). However, a large portion of consumer-ready whole cuts of meat that would be commonly be considered unprocessed are enhanced by adding solutions of flavorings and preservatives, up to ≥10% of the total mass of the meat,18 through needle injection or other processes. The most common ingredients in the added solutions are water, sodium salts, and phosphate compounds,19 all of which are described in the article by Kaluza et al as being common in processed meats and having potential involvement in HF pathogenesis. Grocery store audits indicate that 21% of all packages of fresh meat in stores in the United States are enhanced, with 45% of pork and 16% of beef being enhanced.18 Enhanced meat is appealing from the perspective of industry and consumer in that it maintains better color, has less purge while on the store shelves, is more tender and moist, and has better taste than unenhanced products.19 However, because some of the ingredients in enhanced meat are the same as those from processed meat, it is conceivable that enhanced meat may have similar adverse health effects as processed meat. Certainly, there is a need for more research to evaluate and compare the health effects of unprocessed, processed, and enhanced red meat and to evaluate the health effects of individual additives in processed and enhanced meat. However, in the meantime, it might be prudent to consider enhanced meat another form of processed meat and to recognize the possibility that it may have the same adverse effects on health.

In summary, the research published by Kaluza et al in this issue of Circulation: Heart Failure adds to a growing body of evidence indicating that red meat and processed red meat (possibly including enhanced red meat) increase the risk of HF and have many other adverse effects on health. In light of this evidence, a limited intake of these foods is advisable and is consistent with some recommendations, such as the DASH eating Plan from the National Heart, Lung, and Blood Institute of the National Institutes of Health and the Harvard healthy eating pyramid. However, although the evidence that red meat consumption has adverse health effects accumulates, the per capita global meat consumption (including red meat) has been on the rise for many decades, which may have global health implications.

In the United States, where meat consumption is triple the global average, 58% of meat consumption is red meat and nearly a quarter of all red meat is processed.20 Furthermore, with the popularity of low-carbohydrate/high-protein diets such as the Atkins and paleolithic diets, many individuals perceive high levels of meat consumption, including red and processed red meat, to be healthful. In this context, there is an urgent need to educate patients and the general public about the adverse health effects of red and processed red meat consumption and about the importance of minimizing or eliminating these foods from the diet.

Disclosures

None.

References


Edward P. Weiss

Circ Heart Fail. 2014;7:549-551
doi: 10.1161/CIRCHEARTFAILURE.114.001459
Circulation: Heart Failure is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3289. Online ISSN: 1941-3297

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circheartfailure.ahajournals.org/content/7/4/549

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Heart Failure can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Heart Failure is online at:
http://circheartfailure.ahajournals.org//subscriptions/