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Authors

Allen, Tara Shrout

Bhatia, Harpreet S

Wood, Alexis C

et al.

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State-of-the-Art Review: Evidence on Red Meat Consumption and Hypertension Outcomes

Tara Shrout Allen,[1] Harpreet S. Bhatia,[2] Alexis C. Wood,[3] Shabnam R. Momin,[3] and Matthew A. Allison[1,]

Hypertension (HTN) is a well-established risk factor for cardiovascular diseases (CVDs), including ischemic heart disease, stroke, heart failure, and atrial fibrillation. The prevalence of HTN, as well as mortality rates attributable to HTN, continue to increase, particularly in the United States and among Black populations. The risk of HTN involves a complex interaction of genetics and modifiable risk factors, including dietary patterns. In this regard, there is accumulating evidence that links dietary intake of red meat with a higher risk of poorly controlled blood pressure and HTN. However, research on this topic contains significant methodological limitations, which are described in the review. The report provided below also summarizes the available research reports, with an emphasis on processed red meat consumption and how different dietary patterns among certain populations may contribute to HTN-related health disparities. Finally, this review outlines potential mechanisms and provides recommendations for providers to counsel patients with evidence-based nutritional approaches regarding red meat and the risk of HTN, as well as CVD morbidity and mortality.

Keywords: blood pressure; hypertension; nutrition; public health; red meat; risk factor modification.

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INTRODUCTION: HYPERTENSION AND RED MEAT IN THE CURRENT CONTEXT

Hypertension (HTN) is a modifiable risk factor for cardiovascular disease (CVD), including ischemic heart disease, stroke, and heart failure.[1–3] The prevalence of HTN has increased in the United States, where an estimated 44% of adults meet the current criteria for the diagnosis of this condition.[4–7] Furthermore, between 2009 and 2019, US mortality rates attributable to HTN increased by an average of 34%, with significantly higher rates among Black individuals (56.7 per 10,000 non-Hispanic Black men) compared with White counterparts (25.7 per 10,000 non-Hispanic White men).[7,8]

The pathophysiology of HTN involves the complex interaction of genetics and lifestyle factors to include environment, physical activity, and dietary patterns. Even after accounting for potential confounders, accumulating evidence links the dietary consumption of red meat with a higher risk of HTN.[9–38] Conversely, when combined with heart-healthy diets, other studies report no significant association with HTN for specific types of red meat.[39–44] As such, it remains unclear how changes to contemporary red meat consumption patterns in the United States may serve as an applicable intervention to prevent and decrease HTN-related complications. Some variability in the findings may be attributable to inconsistent definitions of red meat. Based on the available literature, and for the purposes of this review, we defined and categorized "red meat" as unprocessed or processed and included pork-related products in the latter.

With approximately 74% of US adults reporting red meat intake per given day, red meat consumption in the United States is significantly higher compared to other countries.[45,46] Indeed, the United States is the second-highest consumer of red meat globally.[46] Among U.S populations, red meat consumption is particularly higher among adult men, those of lower educational and socioeconomic statuses (SES), and specific racial and ethnic groups.[12–14,45,47] Moreover, these populations are also more likely to consume processed red meat, which is associated with worse CVD and HTN outcomes than the unprocessed type.[45,47] Such differences in dietary patterns may perpetuate HTN-related health disparities and highlight the need for clear nutritional guidance related to red meat consumption.

THE CROSS-SECTION BETWEEN RED MEAT AND HTN

Extensive research demonstrates that blood pressure (BP) levels and HTN prevalence are distinctly lower among those who follow a diet devoid of meat.[48–51] Cross-sectional studies

Correspondence: Tara Shrout Allen (tshrout@health.ucsd.edu).
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[1]Division of Preventive Medicine, Department of Family Medicine, University of California, San Diego, San Diego, California, USA; [2]Division of Cardiovascular Medicine, University of California, San Diego, San Diego, California, USA; [3]USDA/ARS Children's Nutrition Research Center, Baylor College of Medicine, Houston, Texas, USA.

that specifically assess the relation of red meat and HTN have observed a positive correlation.[9–13] The association is reported for both systolic and diastolic BP parameters and a dose-dependent relation is suggested since higher tertiles of red meat consumption are associated with increasingly higher odds of HTN.[9–13] Of note, the data also demonstrate that white meat intake (such as poultry) is associated with more favorable HTN outcomes than red meat consumption.[9,27–29] Thus, studies that assess the exposure of general meat intake (which includes poultry and sometimes fish) must be carefully interpreted.

Data recently published from the U.S. National Health and Nutrition Examination Survey of 31,314 US adults found that, when compared with the lowest level of red meat intake, those in the fourth and fifth quintiles had a 29% and 39% higher odds of HTN, respectively (P-trend = 0.003).[9] Moreover, substituting just one serving of red meat per day with poultry, fish, eggs, dairy, or plant-based protein was associated with an 8%–15% lower odds of HTN.[9] Other cross-sectional studies found that red meat consumption is also associated with higher BP in general and more poorly controlled BP among those already with HTN.[12,13] For example, a cross-sectional study of participants on hemodialysis with HTN found that red meat, and particularly processed red meat, was significantly associated with higher BP levels.[13] Thus, red meat consumption, especially in certain populations, may confer a higher risk for uncontrolled HTN and associated CVD morbidity and mortality.

Since processed red meat has been consistently associated with a higher risk for HTN and CVD, it is critical to specify the type of red meat when evaluating studies that assess risk of HTN.[12,13] The higher risk for processed red meat may be related to sodium content, additives, and gut microbiome metabolism into deleterious metabolites (see below). In cross-sectional studies, and when compared to unprocessed red meat, systolic BP was significantly higher with processed red meat consumption.[12] Another important consideration throughout this review and among cross-sectional observations is the study sample, since, for example, Asian populations intake considerably less processed red meat compared to Western populations.[12,22] As such, some cross-sectional studies have found a null relation among Asian sub-populations when red meat type was not defined.[12,52] In contrast, the association is consistent among Black populations, which on average have significantly higher processed red meat intake than other cohorts.[34,47,53]

ASSOCIATION OF RED MEAT CONSUMPTION WITH CARDIOVASCULAR DISEASE, HTN, AND HTN OUTCOMES IN LONGITUDINAL COHORT STUDIES

Similar to cross-sectional studies, data from longitudinal studies indicate that increased red meat consumption is associated with a higher risk of HTN.[27–35] A meta-analysis of cohort studies demonstrated a positive association between red meat intake and HTN risk with a pooled multivariable-adjusted relative risk (RR) of 1.22 ($P < 0.001$).[30] Among the included studies, the Coronary Artery Risk Development in Young Adults (CARDIA) study followed participants for 15 years and found: (i) red meat consumption of ≥1–2 times/day was associated with a 20%–40% higher risk of HTN compared to <0.6 times/day and (ii) a positive dose-dependent relation across increasing quintiles of red meat consumption (p-trend = 0.004).[34] Another study followed 80,426 adults on the Dietary Approach to Stop Hypertension (DASH) diet (already low in red meat) and found that after multivariable-adjustment (for body mass index, physical activity, alcohol, total energy intake, smoking, family history, and educational status), the degree of red meat consumption still conferred a significantly higher risk of HTN (first vs. fourth quartile multivariable-adjusted hazard ratio 1.25, 95% CI, 1.11–1.42).[33] Importantly, red meat consumption among longitudinal studies has also been associated with increasing BP, more difficult to control HTN, and higher risk of stroke, heart failure, and mortality among those with HTN.[23,27,54,55]

Longitudinal studies that have assessed the risk of HTN have further demonstrated the importance of the type of red meat consumed (i.e., processed vs. unprocessed).[24,25,56] For example, one study observed a significantly higher risk of HTN in those who consumed ≥5 vs. <1 serving/week of processed red meat (5.0 g serving, 17% higher risk), but not with unprocessed red meat.[35] Similar to cross-sectional data, one cohort study did not observe a significant relation between red meat consumption and HTN; however, this study was among an Asian population and did not include data on processed vs. unprocessed red meat types.[12,41,52] Given that consumption and preparation patterns vary significantly between Asian and Western populations, caution is warranted in generalizing such observations to US adults.[12,24,25]

The link between red meat consumption and incident CVD morbidity and mortality is well described across longitudinal studies, and supported by a positive dose-dependent association, even amid populations with relatively low intake.[16–32,56–58] Recent systematic reviews of such cohort studies have also assessed evidence quality and concluded that red meat consumption is associated with adverse CVD outcomes, with one estimating a pooled RR of 1.37 for coronary heart disease per each additional daily serving.[26,57] The association between red meat consumption and CVD outcomes is at times attenuated after accounting for BP or HTN, suggesting that HTN may mediate the link.[21] Thus, cohort studies assessing red meat exposure specifically for BP and HTN outcomes provide valuable insight.

Historically, longitudinal observational studies have provided evidence that vegetarian, lacto-ovo, pescatarian, and poultry-inclusive diets are associated with significantly decreased risk of HTN and other adverse CVD outcomes when compared to diets with red meat.[30,34,48,59] More recent studies indicate that even in the context of a diet with some red meat consumption, the degree of plant-based intake may offset some of the adverse impact. Indeed, the CARDIA study with an omnivore diet found that plant-food was inversely associated with HTN risk in a dose-dependent manner (p-trend = 0.01).[34]

LIMITATIONS OF OBSERVATIONAL STUDIES: RED MEAT AND HTN

Observational studies can offer compelling evidence on the association between red meat consumption and HTN,

but results must be interpreted in the light of limitations inherent to the methodology. Although such data can provide epidemiologic and diet-disease relation insight, limitations are often underappreciated, especially by the general public and media outlets.[60]

One potential limitation is reverse causality, which can occur when individuals receive a diagnosis (i.e., HTN), or notice health metric changes (such as worsening BP), then change their diet in attempt to reverse these findings.[61] For example, initial epidemiological studies observed a J- or U-shaped association between alcohol use and BP, in which HTN risk was lower for light-to-moderate use but higher when alcohol was not consumed or consumed in high amounts.[62] However, this is not supported by other studies that better probe for causality and directionality, and the phenomenon is attributed to those who decreased or ceased alcohol use after an HTN diagnosis.[62] Given evidence that individuals tend to make beneficial lifestyle changes after an HTN diagnosis, it is possible that one dietary change would be a reduction in red meat.[63] In this case, the association between red meat and HTN risk and morbidity would be attenuated.

A second and more prominent limitation to consider is that of residual confounding. Although longitudinal studies with sufficient multivariable analysis, matching, and follow-up may reduce the likelihood of residual confounding, this likely remains an issue. Indeed, red meat consumption correlates with several factors shown to independently increase the HTN risk, such as SES, gender, age, central obesity, physical activity, smoking, and nutritional knowledge.[9,64,65] Moreover, associations may be influenced by wider dietary patterns, given that red meat consumption is also correlated with higher metabolic, saturated fat, and sodium intakes as well as with lower fruit, vegetable, whole grain, and fiber intakes.[47,65] Indeed, there is significant evidence on the effects of sodium intake and BP that are difficult to separate from processed meat consumption. Even with large, comprehensive cohort studies, it remains challenging to account for all confounding factors, and thus, to interpret observed diet-disease associations. Such factors may falsely attenuate or strengthen the observed association between red meat consumption and HTN outcomes and should be considered within the context of individual studies.

A third limitation is reliance on self-reported dietary surveys to measure dietary exposures. Such methods must contend with limitations, including possible recall and reporting biases. The gold standard for dietary assessment among self-report options remains aggregating responses across several 24-hour periods (24HRs), with at least one occurring over a weekend. In a 24HR, participants are asked to list, with prompts, all foods consumed within the past 24 hours. Another method is the use of food frequency questionnaires (FFQs), which are appealing for their lower burden to participants and ability to measure habitual intake. Though most FFQs only ask about a limited number of foods, red meat is quite common, and FFQs may be particularly helpful when assessing the effect of diet on HTN since these accrue long-term. Among smaller studies, differences between 24HRs and FFQs results can be notable, but among larger populations, mean red meat intake is often similar, showing less than a 20% difference in results.[66] As such, significant disparities can be noted by the dietary survey instrument utilized.

RANDOMIZED CONTROLLED TRIALS: RED MEAT AND HTN

Data from RCTs provide the strongest evidence for probing effects of specific exposures, such as red meat, on health outcomes. Although not all diet-disease relations can be practically evaluated by RCTs, those that have assessed the effect of red meat consumption on HTN outcomes provide insight to potential causality. It must be noted, though, that most of the presented RCTs, and nutritionally-based RCTs in general, do not have adequate follow-up to assess clinical outcomes such as incident CVD morbidity and mortality. Rather, currently available RCTs focus on risk factors and serial markers correlated with disease outcomes. With red meat consumption and HTN risk, the focus on BP changes during RCTs may reflect HTN risk and BP control. However, this may be influenced by bias including the inability to blind participants to diet and unanticipated changes related to the Hawthorne effect. Given these considerations, RCTs with a cross-over component, and that focus on event reduction after the intervention, are particularly useful.

Some of the earliest data come from RCTs that assessed the effect of plant-based diets on BP measures. For example, the Complete Health Improvement Program trial instructed participants on a plant-based diet, low in overall meat, and found that systolic and diastolic BP significantly decreased by 5.2% after just 30 days.[67] Another study followed adults with HTN for 1 year after a vegan diet intervention and found 90% were adherent. Notably, not only did BP significantly decrease (by -9 and -5 mmHg for systolic and diastolic parameters, $p<0.01$ and <0.05, respectively), but 77% were able to discontinue anti-HTN medication during the trial with a sustained BP-lowering effect.[68]

Among RCTs that specifically consider red meat as the exposure and measure BP outcomes, the overall data appear inconclusive. Two meta-analyses of RCTs evaluated BP outcomes related to overall red meat consumption of ≥ 0.5 vs. <0.5 servings/day and concluded that there was no significant difference. However, limitations of these analyses included heterogeneity among the individual studies and lack of data regarding processed vs. unprocessed red meat type.[43,44] When red meat type was accounted for in a different systemic review with meta-analysis, each additional 100g/day of overall red meat conferred a 14% higher risk of HTN (P-heterogeneity < 0.001; $N = 7$) and each additional just 50 g/day of processed red meat conferred a 12% higher risk of HTN (P-heterogeneity < 0.001; $N = 4$).[69] Other studies further demonstrate the importance of specific red meat characteristics, such as the type of red meat (processed vs. unprocessed), degree of saturated fat, and preparation methods, in regards to BP and HTN outcomes.[41,70]

Some studies appear to suggest that red meat consumption may not be as associated with adverse BP outcomes when in conjunction with heart-healthy diets. For example, the OmniHeart study recruited adults with pre-HTN or

HTN and compared the impact of a high-protein diet vs a high-carbohydrate diet on BP.[39] However, although this study has been cited to support red meat intake to improve HTN risk, consumption amounts were similar between the groups (0.9 servings/day for the high-carbohydrate diet and 1.1 servings/day for the high-protein diet).[39] Another RCT evaluated BP outcomes on a DASH-plus diet (DASH with 6 servings/week of lean red meat) vs. a non-DASH high-carbohydrate diet.[40] DASH-plus was associated with greater systolic BP reductions compared with the non-DASH diet (−5.6 vs. −2.7 mmHg, $P = 0.08$ overall and −6.5 vs. −5.2 mmHg, $P = 0.08$ among those with HTN, respectively), but the mediation analysis suggested that the significant effect was due to weight loss.[40] Furthermore, and when compared the standard DASH diet (systolic BP lowering of −7.1 overall and −11.5 mmHg among those with HTN), the results suggest that the DASH-plus addition of red meat consumption may have attenuated the beneficial BP-lowering effect, especially among those already with HTN.[40,71]

Such findings suggest that the variability among RCT results is in part attributable to the inconsistency of dietary compositions and methodologies, including red meat type and overall sodium intake. These characteristics are also important to consider when discussing possible underlying mechanisms for the relation between red meat consumption and BP, HTN, and adverse HTN-related outcomes.

POTENTIAL MECHANISMS: RED MEAT AND HTN

Many hypotheses exist regarding the association of red meat consumption, particularly that of processed red meat, with a higher risk of HTN and related complications (Figure 1). One mechanism that likely contributes is that of sodium content in red meat. It is well established that high sodium intake is associated with higher BP, risk of HTN, and poorly controlled HTN.[71] As reviewed in this article, some cohort studies have found that processed, but not unprocessed red meat consumption, is associated with HTN incidence.[24,25,56] Underlying these observations is that when compared to unprocessed meat, processed red meat contains approximately 400% more sodium content.[12,25,37] In this regard, some studies found that after adjustment for urinary sodium excretion, the relation between red meat and HTN was attenuated or became non-significant, thus further suggesting that sodium content contributes.[12] Indeed, the predicted BP effect from high sodium content is considered to account for more than two-thirds of the association between processed red meat consumption and coronary heart disease risk, and is also likely a mediator for the relation with heart failure.[72,73]

Other studies suggest that sodium content may not entirely explain how processed red meat intake confers a higher risk for adverse CVD and HTN outcomes. Some literature indicates that red meat also contains high concentrations of nitrite additives for food preservation, which have been linked to endothelial dysfunction and thus may separately confer a higher risk of elevated and dysregulated BP.[74] In the United States, processed red meats contain more nitrite additives than unprocessed red meats, perhaps up to an average of 50% more.[37,75]

Lastly, metabolomics may provide further insight into potential mechanisms that drive the association between red meat consumption and HTN as well as CVD. Small molecule metabolites in the serum can quantitively reflect dietary intake of specific foods and wider dietary patterns.[76] There is strong evidence that red meat consumption results in discrete metabolomic signatures.[77,78] In fact, red meat and processed red meat are rich in acylcarnitines, which are metabolized via intestinal microbiota and hepatic enzymes into trimethylamine-N-oxide (TMAO) (Figure 1).[77,78] This results in higher serum and urine concentrations of the metabolite.[79–81] Notably, TMAO is associated with several adverse effects (on fatty acid metabolism, endothelial dysfunction, atherosclerosis, oxidative stress, and vascular aging), and it is positively associated with the clinical Framingham risk score.[82] In an RCT with crossover and washout components, red meat consumption was associated with a greater than 2-fold increase in TMAO plasma and urine levels, and discontinuation led to reduced levels within 4 weeks, suggesting a reversible effect.[81] Red meat is associated with increased atherosclerosis, more than other foods high in saturated fat, and some studies suggest that this finding may be mediated in part by TMAO.[10,83] Moreover, TMAO may lead to HTN via arterial stiffness, which is directly correlated with elevated BP and HTN.[82,84]

The idea that the relation between red meat consumption and adverse CVD outcomes—including stroke, heart failure, and mortality—may be in part driven by the underlying effect on BP has been postulated widely in the literature.[21,23,38,43,54,55,85] Thus, the role of red meat on HTN is of particular importance. Future studies may further assess

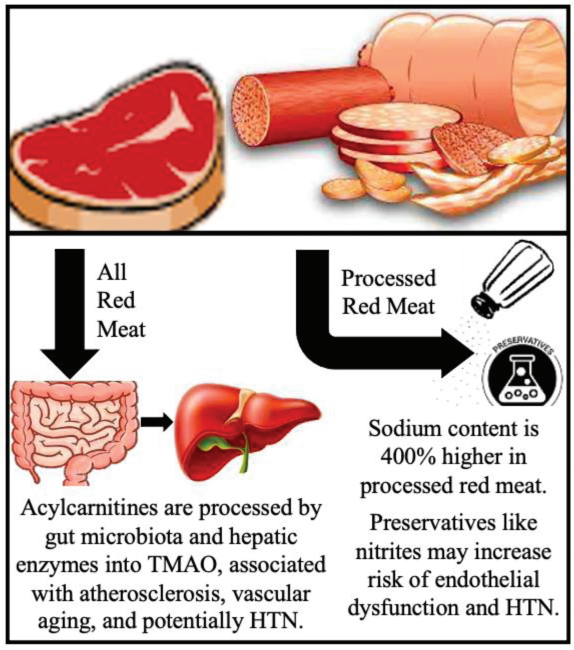

Figure 1. Red meat consumption, particularly that of processed red meat, may confer an increased risk of HTN via mechanisms including the effects of sodium content, nitrite preservatives, and the metabolite trimethylamine N-oxide (TMAO). Abbreviation: HTN, hypertension.

metabolomic signatures of red meat consumptions, including the processed and unprocessed types with specific measures of arterial stiffness, to better understand the association with BP and HTN outcomes.

SPECIAL POPULATIONS: RED MEAT AND HTN HEALTH DISPARITIES

As previously described, there are significant racial and ethnic differences in HTN prevalence.[7,8] Indeed, the prevalence of HTN in the United States is approximately 42% for those of African descent and 28% for White individuals.[7] Moreover, Black populations not only tend to develop HTN earlier in life, leading to a longer time to develop HTN-related morbidity and mortality, but they also have higher average BP and worse HTN prognoses compared to White counterparts.[86,87] Although poorly understood, pathophysiological differences in nocturnal BP, potassium levels, and salt loading have been implicated.[88–91] Effects, though, are multifactorial and potentially include broader factors such as SES, access to medical care, stress, food insecurity, and dietary habits.[9,64,87]

Studies demonstrate clear differences in red meat consumption and preparation patterns by race and ethnicity. On average, Black individuals consume more red meat than Whites (129 vs. 92 g/week, respectively), especially more of the processed type, with bacon and sausage accounting for much of the difference.[47,92,93] Black populations are also less likely to consume unprocessed beef and more likely to obtain fat intake from processed luncheon meats and fried preparations.[94] Even among Black individuals who follow a more plant-based diet, the consumption of overall and processed red meat is still above evidence-based recommended levels and higher than in comparable White populations.[95]

Studies have also shown that the response to BP interventions is different by race and ethnicity. For example, the DASH diet has been shown to be more effective in lowering BP and controlling HTN among Black populations.[96] Interestingly, serum levels of the metabolite 1-methylhystidine, a biomarker of red meat consumption in all populations, are only associated with BP in Black participants.[53] Taken together, these findings suggest that differences in dietary patterns related to red meat consumption may contribute to HTN-related health disparities. This highlights the need for more research into more personalized dietary approaches which are tailored to individual sociodemographic factors.

EVIDENCE-BASED COUNSELING ON RED MEAT INTAKE AND HTN: NUTRITION AS AN INTERVENTION

The efficacy of antihypertensive medications is currently estimated at 40%–70% and the gap in HTN control is not solely explained by medication non-adherence.[97,98] It is well established that diet can serve as an effective nonpharmacologic intervention.[50] Vegetarian diets are associated with lower HTN risk, and even when omnivore diets are high in plant-based criteria, the association is modified by amount of red meat consumption.[49–51] Although red meat can provide a source of protein, vitamins, micronutrients, and even satiety, the literature suggests it is not essential.[58,71] Given that red meat consumption is associated with adverse CVD and HTN outcomes, dietary counseling, especially among specific populations, has important public health implications to bridge evidence-based findings.

The European EAT-Lancet commission guidelines and the Harvard School of Public Health advise against any processed red meat consumption.[45,99] Regarding overall and unprocessed red meat consumption, the European, Canadian, and Mexican guidelines each recommend limiting intake to promote health, without more specific advice.[45] Currently, the U.S. Dietary Guidelines for Americans treat protein-based foods similarly and do not provide recommendations related to amounts of red or processed meat intake.[45] Based on observational studies presented in this review, it may be reasonable to recommend less than 50–100 g (one to two servings) per day of unprocessed red meat.[36,69] More importantly, data support a recommendation of zero to less than 50 g (one serving) per day of processed red meat to reduce the risk of HTN and CVD.[20,99] Still, RCT data on specific red meat consumption amounts and risk of HTN is currently inconclusive and many broader sociodemographic factors must be considered. Red meat is only one part of the diet, and as a whole, consumption should be generally decreased among US adults in regard to HTN.

Historically in the United States, red meat has been a more prominent source of protein intake compared to other countries, whereby nearly 75% of adults consume a serving of red meat daily. Contemporary research, mostly from observation cohort studies, suggests that consumption of red meat may be associated with adverse health outcomes to include HTN and the associated CVD morbidity and mortality. This higher risk may be due to added sodium content or the metabolism of red meat by the gut microbiome into deleterious metabolites. Additionally, consumption of processed, compared to unprocessed red meat, appears to incur a higher risk of HTN and CVD. Studies suggest that some of this risk can be mitigated by incorporating food choices that are not focused on red meat as the primary source of protein, such as vegetarian and pescatarian diets. Since there is limited clinical trial data to derive definitive conclusions, it is recommended that funding agencies prioritize this design so that clinicians, public health professionals, and the US population can be presented with robust information on the risks and benefits of red meat consumption. Finally, the effects of red meat consumption on the risk for HTN and subsequent CVD may be *different* by sex and race and ethnicity. Specifically, non-Hispanic Black men have the highest intakes of processed red meat as well as the highest rates of HTN and incident CVD. As such, this population may benefit the most from interventions to reduce red meat consumption.

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