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Moderate and Heavy Alcohol Consumption Are Associated With Decreased Systolic Function After 8 Years of Follow-up

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Introduction: Excessive alcohol consumption is an important risk factors for cardiovascular disease, however, the underlying mechanisms are not well understood. Hypothesis: We assessed the hypothesis whether alcohol consumption is prospectively associated with unfavorable measures of cardiac structure and function. Methods: We used data from the Hoorn Study, a population-based, prospective cohort study. Data on self-reported alcohol consumption were collected with a validated food frequency questionnaire in 2000/2001(baseline for the current analyses). Echocardiography was performed in 2000/2001 in 582 participants and in 2007/2009 in 339 participants. Participants were classified into 5 categories based on self-reported alcohol consumption (glasses per week): 0 (non-drinkers), 0- 3 (light-drinkers), ≥3-7 (light to moderate drinkers), ≥7-14 (moderate drinkers) and ≥14 (heavy drinkers). Light drinking was considered the reference group. We studied the association of alcohol consumption with echocardiographic measures after 8 years of follow-up using linear regression analyses, adjusting for potential confounders. Results: The mean age was 69.8±6.5 years and 50% was female. After 7.4±0.5 years follow-up, moderate and heavy alcohol consumption were associated with a decreased left ventricular ejection fraction of -5.1% (-8.7, -1.4) for moderate and -4.8% (-8.8, -0.8) for heavy drinkers (Table). Heavy drinking was also associated with a decrease in left atrial volume index: -3.9mL/m² (-7.6, -0.2). No longitudinal

associations were found between alcohol consumption and left ventricular mass index. Conclusion: Both moderate and heavy drinking were associated with decreased systolic function after 8 years follow-up. The toxic effect of alcohol could lead to underfilling of the left atrium which could lead to lower systolic function. These findings may explain the increased cardiovascular risk among people with excessive alcohol use.

Table: Prospective associations of alcohol consumption on cardiac structure and function after 8 years follow-up in 220 Hoorn Study participants

| Outcome (95% CI) | Alcohol consumption in glasses per week | | | |
|---|---|------------------|-------------------------------|-------------------|
| | Non-drinker 0 (n=44) | Light 0-3 (n=92) | Light to moderate 3-7 (n=102) | Heavy ≥14 (n=92) |
| LV Ejection Fraction (%) | | | | |
| 0.1 (-4.2, 4.2) | 0.0 (ref) | -2.1 (-4.3, 0.1) | -4.4 (-6.5, -2.3) | -5.1 (-7.2, -3.0) |
| 0.1 (-4.4, 4.3) | 0.0 (ref) | -0.8 (-2.9, 0.3) | -4.0 (-6.1, -1.9) | -4.2 (-6.3, -2.1) |
| 0.1 (-4.6, 4.0) | 0.0 (ref) | -0.9 (-3.0, 1.1) | -4.2 (-6.3, -2.1) | -4.2 (-6.3, -2.1) |
| LV Volume Index (mL/m²) | | | | |
| 1.0 (-2.6, 0.6) | 0.0 (ref) | 0.7 (-0.8, 4.4) | -5.3 (-7.6, -3.0) | -4.8 (-7.2, -2.5) |
| 1.0 (-2.4, 0.3) | 0.0 (ref) | 1.6 (-1.2, 0.4) | -5.1 (-7.4, -2.8) | -4.7 (-7.0, -2.4) |
| 0.9 (-2.0, 0.7) | 0.0 (ref) | 1.7 (-0.8, 4.3) | -5.1 (-7.4, -2.8) | -4.7 (-7.0, -2.4) |
| LV Mass Index (g/m^{2.7}) | | | | |
| 0.2 (-2.4, 0.0) | 0.0 (ref) | 4.4 (-2.2, 0.4) | -1.0 (-3.1, 0.1) | 0.3 (-1.7, 0.1) |
| 0.2 (-2.8, 0.2) | 0.0 (ref) | 4.3 (-2.2, 0.4) | -1.0 (-3.1, 0.1) | 0.3 (-1.7, 0.1) |
| 0.1 (-4.0, 4.3) | 0.0 (ref) | -5.0 (-3.2, 0.3) | -1.0 (-3.1, 0.1) | 0.3 (-1.7, 0.1) |

LV, left ventricle; ref, reference; CI, confidence interval; mL, milliliter; g, gram; m², square meter; m^{2.7}, square meter to the power of 2.7; mL/m^{2.7}, milliliter per square meter to the power of 2.7; g/m^{2.7}, gram per square meter to the power of 2.7; m², square meter; m^{2.7}, square meter to the power of 2.7; mL/m^{2.7}, milliliter per square meter to the power of 2.7; g/m^{2.7}, gram per square meter to the power of 2.7.

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Remission/Cure of Autoimmune Diseases by a Lectin Limite Diet Supplemented With Probiotics, Prebiotics, and Polyphenols

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All autoimmune diseases are highly associated with increased rates of coronary artery and vascular disease secondary to immune cell attack on epithelial cells. The causes of autoimmune disease (AID) seem to be multifactorial. However, the idea that derangement of the microbiome, breaches of the intestinal barrier (leaky gut) and introduction into the human diet of plant defense molecules such as lectins, which are capable of molecular mimicry, prompted our group to investigate the application of a lectin limited diet, coupled with probiotics and prebiotics (The Pant Paradox Protocol) to impact biomarker proven autoimmune disease

activity in humans and their impact on endothelial biomarkers of inflammation. One hundred and two consecutive patients with immunoassay markers of autoimmune disease activity, i.e., RF, anti-CCP, ANA, Histone, etc, and signs and symptoms of RA, Lupus, Sjogrens, Crohns, Colitis, Scleroderma, Mixed Connective Tissue Disease, and biomarkers of endothelial inflammation, were enrolled into a program of elimination of major dietary lectins, consisting of all grains and pseudo grains, beans and legumes, peanuts, cashews, nightshades, squashes, and Casein A1 milk products (The Plant Paradox Program), supplemented with probiotics and prebiotics including resistant starches and polyphenol supplements. All pts initially low Vit D levels and low Omega 3 index and adiponectin levels above 16mg/dl. Biomarkers of inflammation, hs-CRP, TNF-alpha, IL-6, fibrinogen, myeloperoxidase and autoimmune markers were measured every 3 months. 95/102 patients achieved complete resolution of autoimmune markers and inflammatory markers within 9 months. The other 7/102 patients all had reduced markers, but incomplete resolution. 80/102 patients were weaned from all immunosuppressive and/or biologic medications without rebound. We conclude that a lectin limited diet, supplemented with pro and prebiotics, and polyphenols are capable of curing or putting into remission most autoimmune diseases.

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Adipose Tissue Palmitoleic Acid is Inversely Associated With Nonfatal Acute Myocardial Infarction in the Costa Rica Heart Study

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Background: Animal models have shown that adipose-derived palmitoleic acid may act as a lipokine by conferring resistance to diet-induced obesity; however, human epidemiologic studies investigating this relationship thus far have not provided data in support of this hypothesis. Because metabolic syndrome and cardiovascular disease are intricately linked with the former being a major risk factor for the latter, we hypothesized that adipose-derived palmitoleic acid may be inversely associated with myocardial infarction. **Objective:** We examined whether adipose tissue palmitoleic acid was associated with nonfatal acute myocardial infarction in a representative population of Costa Rican adults. **Methods:** Odds ratios of nonfatal acute myocardial infarction by quintiles of adipose tissue palmitoleic acid were calculated using conditional logistic regression in a case-control study of 1,828 cases and 1,828 controls matched by age, sex, and area of residence. **Results:** We observed an inverse relationship between nonfatal acute myocardial infarction and adipose tissue palmitoleic acid (OR for highest quintile compared to lowest quintile of palmitoleic acid: 0.54; 95% CI: 0.37, 0.79; *P* for trend: 0.0007). We additionally observed a significant positive association between adipose tissue palmitoleic acid and high-density lipoprotein (HDL) cholesterol, an important cardiometabolic risk factor for myocardial infarction. **Conclusions:** These data support the conclusion that adipose-derived palmitoleic acid may behave as a lipokine in the context of human myocardial infarction. This protective association may be partially explained by the increase in HDL cholesterol across quartiles of palmitoleic acid in our population of Costa Rican adults.

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