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Alcohol-Induced Cardiac Remodeling: An Age-Dependent Vulnerability

Background: Alcohol misuse is a persistent public health concern with increasing prevalence, particularly among older adults, placing this population at growing risk for alcohol-related morbidity. Alcohol-related cardiomyopathy is characterized by progressive myocardial functional deterioration, yet the drivers of this pathomechanism remain poorly understood.

Objectives: This study investigated the morphological and functional impact of chronic alcohol misuse by employing a chronic-*plus*-binge alcohol feeding paradigm in adult and aged mice to better understand the progression of alcohol-related heart disease and potential real-world implications for human cardiac health.

Methods: Male and female adult (12-week) and aged (72-week) mice were subjected to a 30-day Lieber-DeCarli chronic-*plus*-binge alcohol feeding regimen. Alcohol-fed groups received a liquid diet containing 5% ethanol, supplemented by oral ethanol binges (5 g/kg) on days 10 and 30. Control mice received an isocaloric maltose-dextrin control diet in combination with isocaloric liquid gavages. Cardiac function was evaluated via terminal invasive left ventricular pressure-volume catheterization. Cardiac tissue weights were measured and normalized to tibia lengths. Histological analysis quantified myocyte cross-sectional area and perimeter in transverse-oriented cardiac cross-sections.

Results: Hemodynamic assessments indicated that alcohol exposure significantly impaired left ventricular load- and heart rate-independent indices of systolic function, regardless of age. Alcohol-fed aged mice, however, showed significantly increased heart weight normalized to tibial length. Furthermore, histological analysis documented significantly increased myocyte cross-sectional area and perimeter in alcohol-exposed aged mice compared to age-matched controls or young mice, suggesting potential cardiac remodeling and a hypertrophic response following chronic-*plus*-binge alcohol feeding.

Conclusions: These findings demonstrate that chronic-*plus*-binge alcohol exposure may act as a driver of myocardial hypertrophy, particularly in aged mice, which may contribute to impaired cardiac performance. Our data indicates that as the heart ages, it experiences an increased vulnerability to alcohol-induced structural changes, which may affect hemodynamics. Overall, by identifying these vulnerabilities, this study sheds light on the importance of alcohol-consumption-related risks to cardiovascular health throughout aging.