

HIGH-FAT DIET DOES NOT INDUCE STRUCTURAL CHANGES OR OXIDATIVE STRESS IN HEART OF WISTAR RATS

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Introduction: Cardiovascular events have been linked with nutritional habits and the consumption of high fat diets is considered to increase the risk of events. **Objective:** To evaluate the influence of high-fat diet on heart morphological and functional parameters, molecular markers of heart failure, and oxidative stress. **Methods:** Adult Wistar rats were fed with standard rat chow (control) (n=18) or high-fat diet (HFD) (n=19) for 21 weeks. The animals were weighed, tested for insulin tolerance (ITT) and oral glucose tolerance (OGTT). The morphological and functional parameters were analyzed by echocardiography. After, the rats were killed and had the left ventricle (LV) dissected, weighted, and stored (-80°C) for further evaluation of biochemical, molecular, and inflammatory parameters. Data were analyzed by t-test. **Results:** The animals with HFD presented no difference in body mass when compared with those receiving standard diet. However, HFD caused an insulin resistance, evidenced by a 12% increase in area under the curve (AUC) of OGTT (p=0.002) and 16% increase AUC of ITT (p=0.001). HFD did not influence ventricular weight, diastolic diameters or ejection fraction, evidenced by echocardiography. Also, there were no disturbances in antioxidant enzymes, catalase and superoxide dismutase, or oxidative stress marker carbonyl. However, protein sulfhydryl oxidation could be found in heart from HFD fed group when compared with control group (7 nmol/mg prot vs. 4 nmol/mg prot, p=0.03). The expression of Nppa gene was not induced by HFD. The fetal gene Myh7 (30%, p=0,04) and the relationship between Myh7/Myh6 (63%, p=0,001) was decreased in HFD group. **Conclusion:** Despite clear influence on insulin resistance, high-fat diet did not caused any molecular imbalance that could indicate inflammation or damage. In the same way, cardiac architecture and function seemed to be preserved. Taken together, these data suggest that the heart is resistant to the early metabolic alteration caused by HFD.

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