# Impaired Fasting Glucose and Hepatic Insulin Resistance – a physiopathological relationship

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Hepatic Insulin Resistance (HIR) and Diabetes Mellitus (DM) present a physiopathological relationship, since increase of gluconeogenesis, reduce of glucogenesis, and diminished insulin secretion by pancreatic beta cells, carry together to higher levels of glucose. Brazil is the 8th country with largest number of cases of DM in the world. However, before the individuals becomes DM, these present a stage of dysregulation in their glucose metabolism called Impaired Fasting Glucose (IFG). It is believed that IFG can progress to diabetes within the next 5 to 10 years. Thus it is necessary to improve the knowledge about its physiopathological mechanisms, since reports in the literature are still scarce and discordant about the theme. This study aimed to investigate the prevalence of IFG and its relationship with HIR in 1,012 subjects in Pernambuco state Brazil. All subjects were 10h-fasting, and signed an informed term of consent. Glycemic and insulinemic plasma levels were determined by glucose oxidase and microparticles immunoassay, respectively. Hepatic insulin resistance (HIR) was determined when a score in the top quartile of the homeostasis model assessment for insulin resistance (HOMA-IR) was found. ANOVA, logistic regression and Pearson´s correlation were analysed ($p<0.05$). Hyperglycemia was found in 1/5 of subjects, and half of these presented IFG. In individuals of IFG, insulin and HOMA-IR values were significantly higher, when compared to normoglycemic individuals. IFG presented a significant odds ratio (5.5) to HIR. It was observed a stronger correlation between HOMA-IR and insulin than between HOMA IR and glucose values. This suggests that HIR might be a primary abnormality of IFG in this population.

Key-Words: Impaired Fasting Glucose, Hepatic Insulin Resistance, Physiopathological Mechanisms

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