1   **Body:** Hyperglycemia has been described as determined by total and central obesity, pancreatic β-cell dysfunction

2   and insulin resistance. **Objective:** We examined how these factors are related with different degrees of glucose

3   tolerance in consecutive patients of the Prediabetes (PDM) and Metabolic Syndrome clinic of Hospital de Clínicas de

4   Porto Alegre. **Methods:** A cross-sectional study with consecutive patients (n=135; age 52.6±11.8 years, mean ± SD,

5   women 76.6%) submitted to a protocol with assessment of metabolic parameters. Body size was estimated by body

6   mass index (BMI), body fat by electric bioimpedanciometry and central obesity by waist circumference. Based on a

7   2-h 75g glucose tolerance test, insulin sensitivity (Matsuda index; $10000/[(Glu_0 * Ins_0)*(Glu_{mean}*Ins_{mean})]$), β-cell

8   function (Insulinogenic index; $\Delta Ins_{30-0}/\Delta Glu_{30-0}$) were determined and glucose tolerance was defined as normal

9   glucose tolerance (NGT;26.7%), PDM (76.6%) and diabetes (DM;26.7%) according to the American Diabetes

10   Association classification. Comparisons among groups were made by chi-square and ANOVA. Multinomial

11   regression analysis was used to adjust for possible confounders using different levels of glucose tolerance as a

12   dependent variable. Statistical significance was determined by $P<0.05$. **Results:** Waist circumference (99.2±14.9 vs.

13   104.3±11.3 vs. 108.7±13.5, mean±DP, P=0.017) progressively increased with decreasing glucose tolerance. The

14   Matsuda index has also decreased with progressive glucose intolerance (4.7 [3.0-8.0] vs. 2.7 [1.8-4.1] vs. 2.0 [1.5-

15   2.8], $P<0.001$). Body fat was greater in those with PDM than in NGT and DM subjects (42.5±7.3 vs. 36.6±7.4 vs.

16   36.6±9.2, P=0,006). Sex and BMI did not differ among groups. Using different multinomial regression models,

17   increasing waist circumference and body fat were respectively determinants of DM and PDM whereas decreasing

18   insulinogenic index and Matsuda index were both determinants of PDM and DM. BMI was not associated with

19   abnormal glucose metabolism. **Conclusion:** Total and central obesity, insulin resistance and β-cell dysfunction were

20   the main determinants of abnormal glucose metabolism, namely PDM and DM. (Support: FAPERGS/FIPE-HCPA)

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