EFFECTS OF 1?,25(OH)2-VITAMIN D3 AND CHOLECALCIFEROL IN VIVO ON GLYCEMIC AND LIPID PROFILES AND TOXICITY[1]

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Introduction: Insulin resistance is one of the most problematic cause of hyperglycemia. Vitamin D_3 and its metabolites are related to contribute for reducing type 2 diabetes (T2D) incidence and has been described to improve the blood glucose concentrations. However, these data are not well elucidated. **Aim:** To evaluate the effects of short-term treatment with 1α,25$(OH)_2$ Vitamin D_3 (1,25-D_3) and cholecalciferol (25-D_3) on glycemic profile, lipids, specific enzymes and calcium of insulin resistant and control rats. **Methodology:** Male *Wistar* rats (180 – 200 g, 60 days) (CEUA / UFSC / 2119280317) were randomly-divided into six groups (n = 6): I) Control (vehicle, 1 mg/ Kg); II) 1,25-D_3 (1 nM *i.p.*, 20 µg/ Kg); III) 25-D_3 (1.8 mM *o.g.*, 330 µg/ Kg); IV) Dexamethasone insulin resistance; V) 1,25-D_3 + dexamethasone, and VI) 25-D_3 + dexamethasone. 1,25-D_3 (*i.p.*) and 25-D_3 (*o.g.*) were administered together with dexamethasone (1 mg/ Kg/day, *i.p.*) for 5 consecutive days. For the ITT, rats were fasted for 2 h and blood glucose was measured before (time zero), and after 10, 30, and 40 min of human insulin administration (2 IU/ Kg, *i.p.*). Blood was collected from the tail vein to measure glycemia. KITT (% glucose/min) and AUC for glucose levels was calculated. At the end of ITT, serum was collected to measure AST, ALT, GGT, LDH, triglycerides, total cholesterol and total calcium concentrations. The liver was removed at the end of the ITT experiment and the glycogen content was quantified. **Results:** Dexamethasone induced insulin resistance. *In vivo*, 5 days treatment with 1,25-D_3 (*i.p.*) prevented insulin resistance in dexamethasone-treated rats. The body weights of rats of all groups remained unchanged during the treatment time. Treatment with 1,25-D_3 improved the activities of hepatic enzymes, lipid metabolism, and serum calcium concentrations in insulin-resistant rats. 1,25-D_3 did not change the hepatic glycogen concentrations. 25-D_3 (*o.g.*) did not affect insulin resistance, but significantly increased hepatic glycogen content in control and insulin resistant groups. **Conclusion:** 1,25-D_3 may prevent insulin resistance, improve lipid metabolism and is not related with liver toxicity. 25-D_3 has a role in increasing hepatic glycogen, however is not effective in preventing insulin resistance. **Financial support:** CNPq; PGFAR-

CAPES; PPG-BQA PROAP/UFSC.

Key-words: 1α,25(OH)$_2$-vitamin D$_3$; cholecalciferol; short-term treatment; insulin resistance.