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+\SR[LD DQG H[WUD FHOOXODU PDWUL[JHQH H[SUHVVLRQ LQ D sensitivity in black South African women

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Objective:Black South African (SA) women are more insulin resistant and have increased gluteal subcutaneous adipose tissue (SAT hypertrophy than white SA women. We tested the hypothesis that adipose tissue hypoxia and extracellular matrix (ECM) gene expression in gluteal and abdominal SAT is higher in black than white women, and associates with reduced insulin sensitivity (SI) in black women.

Methods:SI (frequently sampled intravenous glucose tolerance test), gluteal and abdominal SAT mRNA levels of hypoxia- and ECM-related genes were measured in normal-weight and obese premenopausal black (n=30) and white (n=26) SA women at baseline, a in black women, at 5-year follow-up (n=10).

Results:Compared to obese white women, obese black women had higher expression of hypoxia inducible factor 1 (HIF-1), collagen type V 1 (Col5a1) and collagen VI 1 Col6a1 and reduced vascular endothelial growth factor- (VEGF) expression in gluteal (p<0.05) but not abdominal SAT depots. Independent of body fatness, gluteal expression of HIF-1 (r=-0.55; p=0.01), Col5a1 (r=-0.41; p=0.05) and Col6a1 (r=-0.47; p=0.03) correlated with reduced SI in black women only. Over a 5-year follow-up, changes in gluteal HIF-1 (r=0.58; p=0.01), Col5a1 (r=0.82; p=0.02), and Col6a1 (r=0.88; p<0.00) expression correlated positively with the change in fasting insulin concentrations in black women.

Conclusion: Compared to their white counterparts, black women expressed higher levels of genes associated with hypoxia and collagen deposition, and that the associations between these genes and SI di ered by ethnicity. We thus propose that insulin resistan in black women may be related to higher ECM and hypoxia gene expression.

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Gastric leptin: An important factor that regulates food intake and body weight loss; studies towards clinical applications

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Cell biology studies on gastric leptin secretion have demonstrated that the hormone known to regulate appetite and food intake, is synthesized, packaged in granules and discharged through regulated secretion into the gastric lumen by the Chief cells of th gastric mucosa. Leptin present in the gastric juice is tightly associated to the soluble isoform of its receptor. e leptin-leptin receptor complex allows the leptin molecule to survive the harsh conditions of the gastric juice. Leptin vehiculated from the gastric cavity to the duodenal lumen interacts with membrane-bound leptin receptors located on the apical membrane of the duodenal epithelial cells. Leptin is then internalized and released at the basal pole of the intestinal cells towards the submucosa. It penetrates the blood strea and reaches its hypothalamic target cells where regulation of food intake takes place. Since leptin is normally present in the gastr juice, we evaluated the e ciency of an oral administration of exogenous leptin for the control of food intake. Experiments were performed on normal and obese rodents as well as on large mammals, pigs and dogs. Exogenous leptin given orally in the form of pill is able to reduce food intake and to trigger weight loss in all animals. Compared to leptin secreted by the adipose tissue, gastr leptin appears as an important and favorabletargetfor clinical application.

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