

**DEPARTAMENTO DE BIOLOGIA E FARMÁCIA  
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**AVALIAÇÃO DAS CARACTERÍSTICAS BIOQUÍMICAS,  
SOCIODEMOGRÁFICAS, ANTROPOMÉTRICAS E DE  
POLIMORFISMOS GENÉTICOS EM CRIANÇAS E ADOLESCENTES  
COM PERFIL GLICÊMICO E INSULÍNICO ALTERADO**

Santa Cruz do Sul  
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## RESUMO

**Introdução:** A obesidade é um problema mundial que desencadeia diversas comorbidades, e somada aos fatores genéticos, pode estar associada ao desenvolvimento de resistência e sensibilidade à insulina. **Objetivo:** Avaliar as características bioquímicas, sociodemográficas, antropométricas e polimorfismos genéticos em crianças e adolescentes com perfil glicêmico e insulínico alterado. **Métodos:** Estudo transversal que abordou uma amostra de 176 crianças e adolescentes, com idade entre sete e 17 anos. Foram realizadas análises do perfil antropométrico, perfil lipídico e glicêmico, perfil insulínico, resistência e sensibilidade à insulina pelos modelos HOMA-IR (*Índice Homeostatic Model Assessment for Insulin Resistance*) e QUICKI (*Quantitative insulin sensitivity check index*), e análise molecular dos genes *RBP4* e *FTO*. Os dados foram analisados no programa estatístico SPSS v. 20.0. O estudo foi aprovado pelo CEP. **Resultados:** Foi possível observar que 39,8% da amostra apresentaram excesso de peso, e 15,9% e 47,7% da amostra apresentou HOMA-IR e QUICKI alterados, respectivamente. A faixa etária, circunferência da cintura (CC), percentual de gordura (%G), índice de massa corporal (IMC), glicose, triacilgliceróis (TAG) e insulina se associaram a HOMA-IR e QUICKI e colesterol HDL somente à HOMA-IR. A análise realizada com os Polimorfismos de Nucleotídeo Único (SNPs) mostrou que os portadores do alelo A de *FTO* rs9939609 e do alelo T de *RBP4* rs3758539 apresentaram maior mediana de HOMA-IR e insulina plasmática, e menor média de QUICKI. No entanto, ambos não mostraram relação significativa. Os níveis de glicose tiveram aumento significativo em portadores do alelo A do SNP rs9939609 do *FTO*. **Conclusões:** Esse estudo sugere a relação entre obesidade e resistência à insulina. Observamos a necessidade de mais estudos, principalmente aqueles que abordem a patogenia genética da RI, incluindo o estudo de adipocinas.

**Palavras chaves:** Resistência à insulina; Obesidade; Polimorfismo genético; Crianças; Adolescentes.

## ABSTRACT

**Introduction:** Obesity is a worldwide problem that triggers various comorbidities, and in addition to genetic factors, may be associated with the development of insulin resistance and sensitivity. **Objective:** Thus, the present study evaluated the biochemical, sociodemographic, anthropometric and genetic polymorphisms in children and adolescents with altered glycemic and insulin profile. **Methods:** This was a cross-sectional study that analyzed a sample of 176 children and adolescents, aged between seven and 17 years. The analysis of the anthropometric profile, lipid and glycemic profile, insulin profile, resistance and insulin sensitivity by the HOMA-IR (*Homeostatic Model Assessment for Insulin Resistance*) and QUICKI (*Quantitative insulin sensitivity check index*) and molecular analysis of the RBP4 and FTO genes were performed. The data were analyzed in the statistical program SPSS v. 20.0. The study was approved by CEP. **Results:** It was observed that 39.8% of the sample were overweight, and 15.9% and 47.7% of the sample presented HOMA-IR and QUICKI altered, respectively. Age, abdominal circumference, percentage of fat, BMI, glucose, triacylglycerols and insulin were associated to HOMA-IR and QUICKI and HDL-cholesterol only to HOMA-IR. Analyzes performed with the SNPs showed that the T allele of FTO rs9939609 and allele A of RBP4 rs3758539 had higher median HOMA-IR and plasma insulin, and lower mean QUICKI. However, both showed no significant relationship. Glucose levels had a significant increase in FTO allele A rs9939609. **Conclusions:** This study suggests the relationship between obesity and insulin resistance. We observed the need for further studies, especially those that address the genetic pathogenesis of IR, including the study of adipokines.

**Key words:** Insulin resistance; Obesity; Genetic polymorphism; Children; Adolescents.

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## INTRODUÇÃO

A falha das células  $\beta$ -pancreáticas resulta em complicações metabólicas como a resistência à insulina (RI) que vem sendo bastante abordada em estudos que investigam fatores desencadeadores de enfermidades maiores como o diabetes, aterosclerose e doenças cardiovasculares (VILA NOVA et al., 2016; VÁSQUEZ et al., 2015; KAUL; APOSTOLOPOULOU; RODEN, 2015). A obesidade é responsável por aumentar as chances de se desenvolver RI e a maioria dos indicadores antropométricos como Índice de Massa Corporal (IMC), percentual de gordura (%G) e circunferência da cintura (CC). Além disso, os indicadores bioquímicos como colesterol total (CT), Colesterol HDL (c-HDL), Colesterol LDL (c-LDL) e Triacilgliceróis (TAG) apresentam correlação positiva com essa desordem (GOBATO et al., 2014a).

Estudos com camundongos mostram a influência das dietas ricas em gordura no desenvolvimento de resistência à insulina (SMALL et al., 2017; BERGQVIST et al., 2017; NEMATİ et al., 2017). A dislipidemia está associada ao aumento do estado inflamatório, pois o metabolismo lipídico e a peroxidação são relevantes no seu desenvolvimento, e esse processo induz diretamente a resistência à insulina. Também se discute sobre a correlação de algumas adipocinas, produtos secretados principalmente pelo tecido adiposo visceral, contribuintes para a gênese de distúrbios como a desregulação glicêmica e insulínica e os genes codificantes dessas proteínas (SHAKER; SADIK, 2013; BASTOS et al., 2013). Fatores genéticos contribuem para uma alta variância tanto para a resistência à insulina quanto para sensibilidade avaliadas por diferentes métodos (HUANG et al., 2017).

Existem diversas maneiras de avaliar a resistência à insulina. O índice HOMA-IR (Homeostatic model assessment) e QUICKI (Quantitative Insulin Sensitivity Check Index) são frequentemente utilizados quando se trata de estudos populacionais amplos, devido ao custo e por serem métodos simples que estão entre os índices de substituição mais precisos para determinar a resistência e sensibilidade à insulina, sendo que em ambos é necessária a dosagem de glicose e insulina plasmática (PEÑA-ESPINOZA et al., 2017). Vindo de encontro a estes argumentos, o objetivo do presente estudo foi avaliar as características bioquímicas, sociodemográficas, antropométricas e polimorfismos genéticos em crianças e adolescentes com perfil glicêmico e insulínico alterado.

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