

As stanniocalcinas (STC 1 e 2) são glicoproteínas identificadas, primeiramente, no corpúsculo de Stannius de peixes com função hipercalcêmica. Em mamíferos esses hormônios são expressos em diferentes tecidos com ações parácrinas e autócrinas. As funções metabólicas desses hormônios ainda não foram esclarecidas. Estudos prévios, em nosso laboratório, demonstraram que as STCs modulam a via gliconeogênica renal. O objetivo do presente trabalho foi determinar a ação desses hormônios sobre o metabolismo da glicose em hepatócitos e células musculares esqueléticas de ratos. Foram usados ratos (*Rattus norvegicus*, n = 8) de 300 ± 50 g obtidos no CREAL ICBS-UFRGS. O protocolo experimental foi aprovado pelo Comitê de Ética Animal da UFRGS. Os ratos foram mortos por decapitação e o músculo gastrocnêmio e o fígado excisados, pesados e colocados em placa de Petri com tampão Krebs Riger Bicarbonato (KRB), pH 7.4. Os tecidos eram fatiados e incubados a 37°C (após gaseificação com carbogênio) por 1h em um incubador metabólico sob agitação constante e em presença de diferentes doses de STC1 ou STC2 (0.01ng/ml; 0.1ng/ml; 10ng/ml e 100ng/ml) mais 0,15 µCi de 1-[¹⁴C]-2-deoxi-D-glicose (39 mCi mmol⁻¹; Amersham International) para a determinação da captação da glicose. A análise estatística utilizada foi one-way ANOVA (p<0.05) e os resultados são apresentados como média± desvio padrão. As STC1 e 2 nas concentrações utilizadas no presente estudo não estimularam significativamente (p>0,05) a captação de glicose no fígado e no músculo de ratos. Estudos sobre o efeito das STCs sobre a oxidação de glicose, a síntese de glicogênio serão realizados para esclarecer o efeito deste hormônio sobre o metabolismo da glicose no fígado e no músculo de mamíferos.

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EFFECT OF PEPTIDES HORMONES STC1 AND STC2 ON GLUCOSE UPTAKE AND OXIDATION IN MAMMALS. ¹Gonçalves, A.S., ¹Rossetti, C.L., ¹Fontella, L., ¹Severo, L., Kucharski, L.C., ²Shein, V., ¹Da Silva, R.S.M., ¹Departamento de Fisiologia, ²Departamento de Bioquímica, Instituto de Ciências Básicas da Saúde, UFRGS, Porto Alegre, RS

Aim: The stanniocalcins (STC's) STC1 and STC2 are glycoproteins first identified in teleostean fishes as a hormone secreted by the corpuscles of Stannius with the function of avoiding hypercalcemia. These hormones are expressed in a variety of tissues in higher vertebrates too, but they seem to have autocrine and paracrine actions instead of the classical endocrine response found in fishes. The metabolic function of these hormones remains unknown. Previous studies in our laboratory found an inhibitory effect of these hormones on gluconeogenesis. The purpose of this study was to analyze the effects of the human isoform of STC1 e STC2 on the glycolytic pathway.

Methods and Results: We used male rats (*Rattus norvegicus*, n = 8) of 300 ± 50 g obtained from the ICBS-UFRGS CREAL. All animal procedures used in this study were in accordance with the principles of the Brazilian College of Animal Experimentation (COBEA), and the experimental protocol was approved by the Animal Care Committee of UFRGS. The rats were killed by decapitation and the gastrocnemius muscle and liver were excised, weighed and placed in Petri dishes with cold bicarbonate buffer, pH 7.4. The tissues were then sliced and incubated at 37°C with different doses of STC1 and STC2 (0.01ng/ml; 0.1ng/ml; 10ng/ml and 100ng/ml) with $0.15 \mu\text{Ci}$ of 1- ^{14}C -2-deoxy-D-glucose (39 mCi mmol^{-1} ; Amersham International) for determination of glucose uptake. For glucose oxidation the tissues were incubated in $500 \mu\text{L}$ incubation buffer in $0.15 \mu\text{Ci}$ [^{14}C]glucose ($230 \text{ mCi mmol}^{-1}$, Amersham International) plus 5 mM glucose. The incubations were performed in a Dubnoff incubator with constant shaking for 1 h for glucose uptake and 2 h for glucose oxidation. The statistical analysis was performed by one-way ANOVA ($p < 0.05$) and the data appear as mean \pm standard deviation. The glucose uptake was not significantly different in any of the doses of STC1 or STC2 ($p > 0.05$). In liver, the formation of $^{14}\text{CO}_2$ ($\mu\text{M}\cdot\text{G}^{-1}\cdot\text{h}^{-1}$) was reduced ($p < 0.05$) in the presence of STC2 in all tested doses ($p = 0.032$; $n = 4$; control: 0.0292 ± 0.00969 ; 0.01ng/ml: 0.0132 ± 0.00583 ; 0.1ng/ml: 0.0151 ± 0.00719 ; 10ng/ml: 0.0180 ± 0.00540 ; 100ng/ml: 0.0160 ± 0.00417). In muscle the inhibition of glucose oxidation by ST2 was dose-dependent. The inhibitory effect was observed only in the presence of 100ng/ml of this hormone ($p = 0.017$; $n = 4$; control: 0.0291 ± 0.0223 ; 100ng/ml: 0.00969 ± 0.000486). No significant effect of STC1 was observed on glucose oxidation or uptake in any tissue.

Conclusion: The hormone STC2 seems to be involved in glucose metabolism, inhibiting the oxidative pathway. On the other hand, STC1 did not affect the glucose uptake or oxidation in rat muscle and liver. Further studies to evaluate the enzymatic activity involved in the glycolytic pathway and lipid synthesis from glucose are needed to identify the actions of the STC2 hormone on glucose metabolism.

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