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Renal effects by animal toxins

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Animal toxin envenomation is an important health problem in the tropics. Animal toxins are complex compounds produced by animals for predation, digestion and protection, consisting of peptides, enzymes, chemicals and proteins. At the clinical level, acute kidney injury (AKI) with renal failure has been observed in animal toxin poisoning. The kidney, as a highly vascularized and excretory organ, is a prime target for such toxins. In this work, we review various toxins responsible for renal effects in isolated rat kidney system. Studies with venom from *Bothrops jararaca; B. jararacussu; B. moojeni; B. pirajai; B. insularis; B. marajoensis; B. eritromelas; B. leucurus; B. pauloensis; Crotalus durissus cascavella; Tityus serrulatus* scorpion and sea anemone *Bunodosoma caissarum*. The system provides perfusion pressure (PP), renal vascular resistance (RVR), urinary flow (FU), glomerular filtration rate (GFR), percentage of tubular sodium transport (%TNA^+), of chloride (%TCl^-) and potassium (%TK^+). For example *B. pauloensis* showed effects similar to *B. leucurus* and *Bothrops marajoensis.* The venom reduced PP, RVR, UF, GFR and %TNA^+. *B. moojeni* and *B. jararacussu* venom showed some toxic effects similar to the other above mentioned examples. These changes in activity can be explained also by the difference in concentration of certain components that act in synergy. In higher concentrations, some venom components can enhance or inhibit the action of others, promoting extremely contradictory or opposite effects. Nevertheless, animal toxins are an important area for research in physiology, pharmacology and drug discovery.

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Study of effects on the daily metabolism and the renal function of rats under high oral ingestion of sodium chloride

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Guanylin (GN), uroguanylin (UGN) and the bacterial heat-stable enterotoxin (ST) peptides comprise a new family of cyclic guanosine 3'-5' monophosphate (cGMP)-regulating agonist. Ingestion of a salt meal induces secretion of GN and UGN into the intestinal lumen, where they inhibit Na+ absorption and induces Cl-, HCO3- and water secretion. Simultaneously, these hormones stimulate renal electrolyte excretion by inducing natriuresis, kaliuresis and diuresis. The highly integrated mechanism allows the organism to maintain sodium balance by eliminating excess NaCl in the urine. However, their physiological regulation within the kidney has not been studied. The aim of this study was showing changes on daily metabolism and renal function of rat under high sodium chloride ingestion. Its effects were examined using Wistar rats maintained for ten days in metabolic cages. Control group received only water; two more groups received 1% and 2% solutions of sodium chloride. We daily analyzed urinary volume, weight, and food and water consumption. The renal function was evaluated using isolated perfused kidneys, in which the kidneys were perfused after ten days in metabolic cages, only with Krebs-Henseleit solution containing 6 g% of a previously dialyzed bovine albumin serum. All data were analyzed by ANOVA and Student t-test with level of significance set at $p<0.05$. Rat's weights of 2% group decreased after eighth day, compared with control group, while 1% group did not show significative weight lost. Urinary volume and water consume increased, in both treatments, from second day. Food consumption did not show significant among groups. In isolated kidney both treatments increase perfusion pressure (PP). The renal vascular resistance (RVR), urinary flow (UF), glomerular filtration rate (GFR) and the osmolar clearance (Cosm) increased in the 1% group compared with control group, however decreased in 2% NaCl group. Treatment with 2% NaCl decreased the sodium (%TNa^+, %$pTNa^+$), potassium (%TK^+, %pTK^+) and chloride (%TCl^-, %$pTCl^-$). These results suggest that a high salt ingestion on diet promote significant changes on daily metabolism and the renal function of rats.

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