J Forensic Toxicol Pharmacol 2017, 6:2
DOI: 10.4172/2325-9841-C1-006

Thermodynamics of Cu^{2+}, Pb^{2+}, and Cd^{2+} sorption onto low molecular weight chitosan using Isothermal Titration Calorimetry (ITC)

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Chitosan and its derivatives possess valuable properties for its use as a sorbent for the removal of heavy metals from aqueous solution. In the present study, the thermodynamics of Cu^{2+}, Pb^{2+} and Cd^{2+} sorption onto low molecular weight chitosan (CS_8) using isothermal titration calorimetry (ITC) were investigated. Based on the ITC data, the stoichiometry data were 0.36±0.023, 0.813±0.015 and 0.029±0.006 for Cu^{2+}, Pb^{2+} and Cd^{2+}, respectively. The binding association constant (Ka) varied from $(1.74\pm0.333)\times10^{4}M^{-1}$ to $(17.3\pm18.9)\times10^{4}M^{-1}$. Also, all binding reactions to low molecular weight chitosan (CS_8) were enthalpically favored and the interaction between the sorbent and the metal ions were enthalpically not driven at 25°C. Furthermore, free energy of reaction values were all determined to be negative indicating spontaneous reactions. In conclusion, the ITC instrument was successfully used to measure directly the stoichiometry (N), binding association constant (Ka), the enthalpy change (ΔH) and the entropy change (ΔS).

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Double faced glucocorticoid and associated glucolipotoxicity on cardiovascular health- A commentary

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Statement of the Problem: The widely used potent anti-inflammatory agent, glucocorticoids, shows a behavioral paradox. Even though used extensively for treating severe pain and inflammation, in excess could results in metabolic and endocrine complications resulting in Cushing's syndrome like symptoms. Even though they produce major side effects following chronic treatment, the primary focus has been on direct genomic effects and very less emphasize has been put on their nongenomic effects. Non genomic mechanisms of glucocorticoids are mostly mediated by various stress kinases. When there is an over activation of this non genomic arm of glucocorticoids could result in metabolic complications that potentiate glucolipotoxic events in cardiovascular tissue.

Theoretical Orientation: In this commentary, I would like to shed light on this non genomic effect of glucocorticoids as shown by my own and other's work.

Findings: Using *in vivo* and *in vitro* model systems, we have shown that glucocorticoids in excess under normal physiological conditions could activate this non genomic pathway mediated by stress kinase pathways leading to glucolipotoxicity in the cardiovascular tissue.

Conclusion & Significance: Making use of this knowledge, we tried to interfere or inhibit the mediators in this non genomic signaling axis and was able to normalize the glucolipotoxicity and associated complications in the cardiovascular tissue. This information will really help to provide more awareness to the population who are on long term glucocorticoid treatment and also in long term stress situations.

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