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Combating overweight and obesity among school children and adolescents through student counselling in Saudi Arabia

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Background: Overweight and obesity are health problems that can affect many children and adolescents around the world. The literature has identified that the prevalence of overweight and obesity is high in the Kingdom of Saudi Arabia (Saudi Arabia) in children and adolescents attending schools in Saudi Arabia. Student counselling practices can be a cost effective way to help students to deal with overweight and obesity in Saudi schools.

Purpose of Study: The aim of the study was to conduct an evidence-based review of the literature in order to suggest new approaches to applying student counselling services and programmes in order to directly combat overweight and obesity in schools in Saudi Arabia.

Sources of Evidence: The study used a non-empirical review of the literature on overweight and obesity and on student counselling in the United Kingdom, the United States, and Saudi Arabia. The study argues that in theory student counsellors in schools in Saudi Arabia are ideally placed to help to directly address and reduce the existing high prevalence of overweight and obesity in youths and adolescents attending schools in Saudi Arabia. This could be done using a combination of dietary interventions and counselling methods.

Conclusion: The research study concludes that student counselling in schools in Saudi Arabia could potentially be used to directly help to combat and reduce levels of overweight and obesity among school children and adolescents in the long term.

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CaMKII-dependent troponin-I phosphorylation contributes to the frequency-dependent acceleration of relaxation in ventricular myocytes

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Previous studies suggest that CaMKII activity is required for FDAR but the molecular targets remain elusive. We propose that CaMKII regulates FDAR by a mechanism that involves CaMKII-dependent alteration of myofilament sensitivity to Ca²⁺. [Ca²⁺]_i and sarcomere length were measured by IonOptix Ca²⁺ image system. Myofilament sensitivity to Ca²⁺ was assessed by measuring the gradient of cell length-fura2 trajectory during contraction and late relaxation. Increasing pacing rate from 0.5 Hz to 4 Hz in left ventricular (LV) myocytes accelerated Ca²⁺ decline and sarcomere relaxation time constants (from 152±13 ms to 60±5 ms and from 36±2 ms to 18±1 ms, respectively, p<0.05, n=27) and increased the length-fura2 trajectory gradient (ECa50 increased from 1.62±0.06 to 1.84±0.06, p<0.05) and shifted the trajectory loop to the right, indicating a consistency of FDAR with the reduction of myofilament sensitivity to Ca²⁺. Inhibition of PKA (H89, 1μM) or PKC (CHE, 1μM) had no effect on myofilament Ca²⁺ desensitization and FDAR, whereas CaMKII inhibitor KN93 (1μM) abolished frequency-dependent myofilament desensitization to Ca²⁺ and FDAR. We determined the Ser23/24 phosphorylation in LV myocytes and found that Ser23/24 phosphorylation was largely reduced by PKA and PKC inhibitors but not by CaMKII inhibition. However, a phospho-Ser-antibody showed that CaMKII inhibitor KN93 significantly reduced Tn-I phosphorylation in the Tn-I immunoprecipitates, indicating that CaMKII phosphorylates Tn-I at sites different from the PKA and PKC sites. Indeed, a co-immunoprecipitation of CaMKII and Tn-I has been detected. Our results suggest that FDAR is regulated by a frequency-dependent desensitization of myofilament sensitivity to Ca²⁺, in which CaMKII-dependent Tn-I phosphorylation plays a major role.

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