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Predicting early childhood obesity at infancy: a model for the New Zealand population

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Background: One in three children in New Zealand are overweight or obese by the time they start school. Internationally, several prediction models of early childhood obesity have been developed, but none exist for New Zealand's diverse population. Thus, the aim of this study was to develop, and validate, an early childhood obesity prediction model using data obtained in infancy, for use in the New Zealand population

Methods: A prediction model was developed using data from the Growing up in New Zealand (GUiNZ) prospective cohort (N = 6,853). The GUiNZ cohort was randomly split into derivation (70%), and validation (30%) populations. The model was also externally validated in two different longitudinal New Zealand-based cohorts: Prevention of Overweight in Infancy (POI) and the Pacific Islands Families Study (PIF).

Results: The derivation population consisted of 1,731 children. The following parameters were included in the final model: gestational age, maternal smoking during pregnancy, birth weight, maternal and partner BMI, and accelerated infancy weight gain. The model's discrimination was adequate, with an area under the receiving operating characteristic curve (AUROC) of 0.75 (0.72–0.78). The model's sensitivity was 70.1%, specificity was 65.6%, positive predictive value (PPV) was 27.7%, and negative predictive value (NPV) was 92.1%. 713 children were used for internal validation, and the AUROC produced was also adequate at 0.73 (0.68–0.78). Discrimination remained adequate for PIF (AUROC = 0.74 [0.66–0.82]), but improved for POI (AUROC = 0.83 [0.71–0.90]).

Conclusions: We have developed and validated a model, using birth and infancy data, for the prediction of early childhood obesity in the New Zealand population. The model's parameters are easily obtainable, thus we propose that use of this model could support targeted interventions to prevent early childhood obesity in New Zealand.

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The Color of Fat: brown, white, beige, and more

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Regulator of Calcineurin 1 (RCAN1) helps coordinate whole body metabolism and limit energy expenditure

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Obesity and Type 2 diabetes (T2D) are complex and inter-related metabolic diseases associated with insulin resistance and pancreatic β-cell dysfunction. We recently identified a single gene, Regulator of calcineurin 1 (Rcan1), as a candidate gene linking hyperglycemia and functional changes in T2D β-cells [1]. Rcan1 expression is also strongly correlated in both liver and adipose tissue with worsening metabolism. To determine whether such increasing levels of peripheral Rcan1 expression are causative in driving worsening metabolism, we placed Rcan1$^{-/-}$ mice under metabolic stress using a variety of different high fat diets. Rcan1$^{-/-}$ mice had an elevated resting metabolic rate and were resistant to diet-induced obesity. Furthermore, they maintained insulin sensitivity and showed no evidence of inflammation in visceral fat on a high fat diet. Cold-induced expression of the peroxisome proliferator-activated receptor gamma coactivator 1-alpha (Pgc1α) and uncoupling protein 1 (Ucp1) was significantly higher in the subcutaneous fat of Rcan1 KO mice compared to in wild type, indicative of an increased capacity for thermogenic "beiging" of white adipose. The effect was cell-autonomous, as cultured adipocytes depleted of Rcan1 also showed enhanced expression of Pgc1α and Ucp1 in response to an adrenergic stimulus. On an evolutionary scale, in the context of limited food resources, Rcan1-mediated suppression of adaptive thermogenesis would be beneficial, however, in the face of current caloric abundance, Rcan1-mediated suppression of these adaptive avenues of energy expenditure may contribute to the growing epidemic in obesity.

Reference

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