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Clinical Studies Implicating Obesity in Autoimmunity and Type-1 Diabetes

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About the Study

Several works have examined the impact of birth weight, weight increase, and obesity in the development of islet autoimmunity, the progression from single antibodies to numerous antibodies, and the development of type-1 diabetes. The first studies comprised community-based cohort and epidemiological case-control studies that linked a type 1 diabetes diagnosis to raised birth weight and early childhood weight gain in infancy when compared to a control group. The multiple prospective cohort studies from birth found a greater rate of early weight increase and absolute BMI z-score as predictors of islet autoimmunity development. Particularly, the Environmental Determinants of Diabetes in the Young (TEDDY) study, a multi-country cohort following children at risk for type-1 diabetes based on their HLA-DR-DQ genotype, investigated the associations between weight in the first few years of life and progression from single to multiple antibodies and subsequent type-1 diabetes development. Weight z-scores at 12 and 24 months were associated with an increased risk of progression to multiple antibodies, and a higher rate of early childhood weight gain was associated with progression from autoimmunity to type 1 diabetes in those whose initial presenting autoantibody was directed against glutamic acid decarboxylase. Furthermore, there may be a link between weight increase and an earlier age of onset of type 1 diabetes.

Extensive studies has looked at the relationship between BMI and the course of type 1 diabetes after autoimmune onset in older children and adults. Comparisons of autoantibody status by BMI at diagnosis were equivocal at first. A cross-sectional study of 263 children under the age of 19 years at the time of initiation discovered no significant relationship between the quantity of antibodies and obesity measurements such as BMI and waist circumference. A second study investigated the relationship between BMI, BMI percentile, and insulin resistance (measured by HOMA-IR) with the progression from autoimmunity to diagnosis in the TrialNet Pathway to Prevention (PTP) cohort, which follows individuals at risk for type-1 diabetes based on family history and islet autoantibody status.

CeBMI enhanced the risk of type-1 diabetes development in adults, but only in particular age and gender cohorts, especially males over 35 years of age and women younger than 35 years of age, implying some effect from sex hormones. Insulin resistance certainly impairs -cell function in kids with type-1 diabetes and may enhance immunological activation in at-risk people, lending credence to the accelerator theory.

Higher BMI at diagnosis was associated with greater decline in fasting C-peptide levels among teens 10-18 years at 1 year follow-up in a pooled European cohort of youth with type-1 diabetes, even after adjusting for glycemia, suggesting that BMI-related insulin resistance is contributing to cell dysfunction in this population.

A study evaluating T-cell autoreactivities to neuronal diabetes-associated autoantigens, which are often found early in type-1 diabetes pathophysiology before antibodies develop, revealed that this resulted in immunological activation. Youth with the highest quintile of BMI or an increased waist circumference at the initiation of insulin-dependent diabetes had greater islet-associated T-cell autoreactivities, indicating that visceral adiposity-associated insulin resistance may promote T-cell autoimmunity. Furthermore, with the exception of one kid who did not have autoantibodies, all exhibited signs of T-cell autoimmunity, indicating some immune activation among children who would normally be categorised as having type-2 diabetes. Obese adolescents with newly diagnosed type-1 diabetes exhibit altered pro-inflammatory profiles, which may contribute to the accelerated development.

Adipocytes release a number of pro-inflammatory and anti-inflammatory adipokines that are linked to insulin resistance. Obese children with newly diagnosed type 1 diabetes exhibit greater levels of pro-inflammatory markers (leptin, visfatin, chemerin, TNF, CRP) and lower levels of anti-inflammatory markers (adiponectin, omentin) than non-obese peers with type-1 diabetes. While having two or more autoantibodies at diagnosis indicates a greater degree of autoimmunity and is related with higher adiponectin and lower leptin levels, these associations were lost when BMI was adjusted for, indicating that obesity is the major cause of aberrant adipokines.