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Intrinsic Motivation and Glycemic Control in Adolescents with Type 1 Diabetes

Treatment for type 1 diabetes is often difficult for adolescents because of the multiple daily tasks required for successful management. Hence, adolescents who are more intrinsically motivated to manage their health might be more persistent with their diabetes care and consequently be in better glycemic control. We therefore examined the proportion of variance that intrinsic motivation contributed to HbA_{1c} in 43 adolescents diagnosed with type 1 diabetes relative to other disease-related and psychosocial factors that have been linked to glycemic control in cross-sectional research.

After receiving approval from the institutional review board, we recruited adolescents with a mean age of 14.14 ± 1.73 years from a university-affiliated diabetes clinic to participate in the study. All of the participants had been diagnosed with diabetes for a minimum of 1 year ($M = 5.85 \pm 4.53$ years), and none were on an insulin pump. The sample consisted mostly of girls (65%); 74% were Caucasian, and 26% were African American. The mean family income was in the \$30,000–45,000 range. The adolescents completed standardized measures of intrinsic/extrinsic motivation (Health Self-Determinism Index for Children), self-efficacy (Self-Efficacy for Diabetes Scale), family conflict (Family Environment Scale), diabetes-specific family behaviors (Diabetes Family Behavior Scale), and adherence to their diabetes regimen (Diabetes Regimen Adherence Questionnaire) while waiting for their medical appointment with the physician. Their parents completed a screening measure of behav-

ioral adjustment on the adolescent (Pediatric Symptom Checklist) and a general demographic questionnaire.

Bivariate correlations revealed that adolescents who were more intrinsically motivated to manage their health were more likely to report adhering to their treatment ($r = 0.38, P < 0.05$) but were also more likely to be in poorer metabolic control, as measured by HbA_{1c}, at the time of testing ($r = 0.43, P < 0.05$). The relation between intrinsic motivation and poor glycemic control was unexpected, but is consistent with research (1) suggesting that adolescents who are primarily responsible for their diabetes care tend to be in poor metabolic control. Family conflict was also found to be related to poor adherence ($r = -0.38, P < 0.05$) and to being in poor glycemic control at the time of testing ($r = 0.35, P < 0.05$). But intrinsic motivation was the only psychosocial variable that was related to HbA_{1c} 4 months later ($r = 0.41, P < 0.05$). The proportion of variance that intrinsic motivation contributed to future glycemic control, however, was not significant after controlling for baseline HbA_{1c} in hierarchical regression analyses. Given that baseline HbA_{1c} was highly correlated with follow-up HbA_{1c} ($r = 0.78, P < 0.0001$), further research on intrinsic motivation is worth pursuing with larger samples.

Although we observed significant relations between intrinsic motivation and both adherence and HbA_{1c}, we did not observe a significant relation between adherence and HbA_{1c} at baseline ($r = -0.10$) or at follow-up ($r = -0.06$), which is similar to reports in the literature (2). The present findings suggest that adolescents who are intrinsically motivated could be at risk for poor glycemic control because they are more likely to rely on their own internal cues and judgment for managing their health. Adolescents may lack the experience and objectivity to make medically sound judgments. Thus, frequent consultation with parents and medical staff may be recommended instead of encouraging adolescents to assume more personal responsibility for their diabetes care. This recommendation contradicts the popular practice of encouraging adolescents to manage their diabetes care autonomously, but may be warranted until they can successfully manage their diabetes independently.

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The Metabolic Syndrome in Inuit

Inuit have been considered to have a lower prevalence of diabetes and age-adjusted mortality from cardiovascular disease than the general population (1,2). This observation has prompted investigation of both traditional and newer cardiovascular risk factors. A new risk cluster called the metabolic syndrome, defined as three or more of 1) fasting glucose ≥ 6.1 mmol/l; 2) blood pressure $\geq 130/85$ mmHg; 3) triglycerides ≥ 1.69 mmol/l; 4) HDL cholesterol < 1.04 mmol/l in men or < 1.29 in women; and 5) waist circumference > 102 cm in men or > 88 cm in women (3), has not been evaluated in the Inuit. We thus determined the prevalence of the metabolic syndrome among 168 Inuit (48.2% women) and 53 Caucasian control subjects (38.5% women) who were residents in the arctic and had participated in a cardiovascular survey in 1989–1991 (2). Using the 2001 criteria, we found that Inuit had a lower prevalence of the metabolic syndrome (13.1%) compared with both regional Caucasian control subjects (20.8%) and Caucasian subjects from the contemporaneous 1988–1994 National Health and Nutrition Examination Survey (NHANES) III

(4) (23.8%, $P = 0.0013$). We next examined each of the metabolic syndrome components in Inuit and resident Caucasian control subjects. The frequencies of hypertriglyceridemia (7.1 vs. 24.5%, $P = 0.0005$) and of depressed HDL cholesterol (20.8 vs. 47.1%, $P = 0.0002$) were significantly lower in Inuit than in Caucasians. In contrast, the frequencies of high blood pressure (11.9 vs. 9.4%, $P = NS$), elevated fasting serum glucose (53.6 vs. 60.4%, $P = NS$), and increased waist circumference (28.6 vs. 18.9%, $P = NS$) were not different between Inuit and Caucasians. Furthermore, 32.1% of Inuit, compared with only 13.2% of Caucasians, were free of any positive metabolic syndrome variable ($P = 0.0072$).

Thus, Inuit studied in 1989–1991 had a lower prevalence of the metabolic syndrome compared with two independent Caucasian samples studied at the same time. Inuit had a favorable lipid profile, specifically lower triglycerides and higher HDL cholesterol, despite a trend to increased prevalence of higher waist circumference. Although genetic factors might have played a role, lower plasma triglycerides and higher plasma HDL cholesterol are both related to lifestyle factors, mainly activity level and diet. In this regard, it may be important that the consumption of marine-based fats by the Inuit study participants in 1989–1991 was high (2). Therefore, Inuit had a lower prevalence of the metabolic syndrome compared with Caucasians, which is consistent with the previous impression of lower cardiovascular disease and diabetes prevalence. Because lifestyle is changing dramatically in this “population in transition” (5), systematic reevaluation of the metabolic syndrome would be important in order to identify interval changes that could predict future increases in diabetes and cardiovascular disease (6).

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COMMENTS AND RESPONSES

The Association of NAD(P)H Oxidase p22phox With Diabetic Nephropathy Is Still Uncertain

Response to Hodgkinson, Millward, and Demaine

We believe that there are limitations in the study by Hodgkinson, Millward, and Demaine (1) about the association of the NAD(P)H ox-

idase p22phox polymorphisms with susceptibility to diabetic nephropathy. The case-control association studies on the polymorphisms in p22phox have been performed by a number of researchers, and their results are still in conflict, especially with regard to coronary artery disease. On the other hand, the allele frequencies of the C242T polymorphism have been found to be consistent in various Caucasian populations (2,3). The C242T allele frequencies in the control samples by Hodgkinson, Millward, and Demaine differ from those in previous reports, and the genotype frequencies also deviated significantly from Hardy-Weinberg equilibrium ($P = 0.025$), suggesting a nonrandom sampling or some technical error.

The estimation of haplotype frequencies in case-control association studies is often performed on the basis of a maximum-likelihood method with an expectation-maximization algorithm. It is usually impossible to determine whether an individual with the genotype Aa-Bb has haplotypes A-B and a-b or A-b and a-B. Moreover, the χ^2 test by contingency table is not appropriate for comparing the estimated haplotype frequencies. Hodgkinson, Millward, and Demaine did not describe how the haplotypes of their samples were determined. Tsai et al. (4) applied a proper method, referred to as permutation-based hypothesis testing, to evaluate the association between multilocus angiotensinogen gene polymorphisms and hypertension.

Although Hodgkinson, Millward, and Demaine reported a very low and striking P value, we believe the association of diabetic nephropathy with p22phox remains unsolved.

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