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Title

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avoidance and excess hospitalization in the no-show group was $2,819,000 annually due to reduced hospital use alone. The results indicate that providing Diabetes EDRP visits improves A1c in a subgroup who continues ambulatory care, and those who show for the EDRP visit are approximately 50% less likely to be hospitalized within the next year.

24-OR
Impact of a Diabetes-specific Health Plan on ED and Inpatient Hospital Use

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Timely and appropriate ambulatory care can help prevent diabetes-related complications that lead to costly ED visits or hospital admissions. This study examined changes in annual adjusted rates of ED and inpatient hospital use associated with employers' purchase of a diabetes specific health plan, known as the Diabetes Health Plan (DHP), using employers who did not purchase DHP as concurrent controls.

We conducted a retrospective, employer-level, intent-to-treat analysis, using aggregate insurance claim and laboratory data from all employees and dependents with diabetes and pre-diabetes between 19-63 years of age, whether or not they were enrolled in DHP. Inverse propensity score weighting was used to adjust for employer level differences. Estimates were used to calculate the average treatment effect on the treated (ATET), or the difference between predicted rates of ED and inpatient hospital use among DHP employers and their predicted rates if they had not offered the DHP.

Results included 3 years of data from 9 DHP and 185 control employer groups. Unadjusted rates of ED use were similar in both groups at baseline (DHP 7.64%, control 8.60%) but ED use decreased at 2 years in DHP groups (DHP 4.50%, control 9.43%). ATET analysis showed no evidence of significant reduction in ED use at 1 year post DHP but did show a 3.8 percentage point predicted reduction (p=0.015) in the mean rates of ED use at 2 years post-DHP, representing a 46% decrease relative to the predicted baseline rate of 8.3% ED use. No evidence of significant association with predicted inpatient hospital use was found at 1 and 2 years post-DHP.

We found that DHP employers had a 46% reduction in the adjusted mean rates of ED use at 2 years post-DHP compared to the predicted mean rates of ED use had they not offered DHP. These findings suggest that health insurance benefit designs that decrease out of pocket costs for medications and preventive care can play an important role in decreasing costs for more resource intensive services such as ED use in diabetes and pre-diabetes.

TYPE 1 DIABETES IN YOUTH— MARKERS AND MANIFESTATIONS

25-OR
Hydrolyzed Infant Formula and Early Beta-Cell Autoimmunity

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Background: Early exposure to complex dietary proteins may increase the risk of beta-cell autoimmunity in children at genetic risk for type 1 diabetes. Based on pilot data, we tested the hypothesis that weaning to an extensively hydrolyzed formula decreases the cumulative incidence of diabetes-associated autoantibodies in young children. Methods: In this multicenter double-blind, randomized trial 2159 infants with HLA-conferred disease susceptibility and a first-degree relative with type 1 diabetes received either a casein hydrolysate or a conventional cow´s milk formula supplemented with 20% of the casein hydrolysate (control formula). Autoantibodies to insulin, glutamic acid decarboxylase, and the insulinoma-associated-2 molecule were analyzed during a median observation period of 7.0 years.

Results: Cow's milk antibody data confirmed adherence to the protocol. The unadjusted hazard ratio for positivity for two or more islet autoantibodies was 1.21 (95% CI, 0.94 to 1.54) among those randomized to the casein hydrolysate while the hazard ratio adjusted for HLA risk, duration of breast-feeding, study formula duration and consumption and region was 1.23 (95% CI, 0.96 to 1.58). There were no conspicuous differences in the rate of reported adverse events between the two groups.

Conclusions: The dietary intervention tested in this trial was safe but had no apparent effect on early markers of beta-cell autoimmunity. Its effect on progression to incident type 1 diabetes by the age of 10 years will be assessed in 2017.

26-OR
The Influence of the DQB1*0602 Allele on Predictors of Type 1 Diabetes (T1D) in the TrialNet Natural History Study (TNNHS)

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The DQB1*0602 (0602) allele is associated with a decreased risk of T1D among autoantibody positive individuals; however, the mechanism of its effect has not been well characterized. We thus studied the influence of the 0602 allele on autoantibody number and glycemia in 1,210 TNNHS participants (mean±SD age: 18.3±13.2 years). All were autoantibody positive relatives of T1D patients. There was an appreciable percentage of +0602s with ≥2 autoantibodies at baseline [19/81 (23%)]; however, the percentage was higher in -0602s [528/1,129 (47%); p<0.001). The sum of OGTT glucose values at 30, 60, 90 and 120 minutes (sumglu) was used as the glycemia measure. Baseline sumglu values of +0602s were significantly lower than values of -0602s (503±102 mg/dl vs. 533±106 mg/dl, p=0.016). The percentage of those with dysglycemia (fasting glucose: 110-125 mg/dl; 30-, 60-, and/or 90-minute glucose: ≥200 mg/dl; 120-minute glucose: 140-199 mg/dl) at baseline was also lower for the +0602s [(12/81 (15%) vs. 291/1,129 (26%); p=0.028]. There were 28/81 (35%) +0602s who had dysglycemia and/ or ≥2 autoantibodies. A six-month progression scale (PS6M) was used to assess glucose change. The PS6M indicates the extent of change in sumglu from baseline to 6-month OGTTs relative to what is expected for non-progressors to T1D. The PS6M is a robust predictor of T1D. PS6M values of +0602s were also lower (-8±71 mg/dl vs. 15±98 mg/dl, p=0.007). The negative value for the +0602s indicates that the average change was less than that for non-progressors. Only 1/81 (1%) +0602s developed T1D (follow-up: 2.3±1.8 years). In summary, although +0602s had lower percentages of multiple autoantibodies and dysglycemia, at least one of those risk markers was present in over one-third. Since T1D progression was rare, the 0602 allele was protective when risk seemed substantial. Thus, it appears that even after considerable disease progression, the 0602 allele acts to inhibit further progression to T1D.

27-OR
Serum B7-H4 mRNA Splice Variants as Biomarker for Diabetes

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Our study shows a moderate protein expression of B7-H4 in the islet β cells of normal human pancreas and a concomitant reduction in B7-H4 and insulin expression in the islets from patients with Type 1 Diabetes (T1D). The present study seeks to define the role B7-H4 plays in the pathophysiology within the islet β cells and whether B7-H4 transcript expression can be used as a biomarker for diabetes. B7-H4 mRNA spliced variants were detected by semi-quantitative RT-PCR using specific primers for B7-H4. Total RNA extracted from groups of serum samples of normal adults, pediatric age-matched controls (AMC), pediatric new-onset diabetics (New T1D), long-term diabetics [diabetes for < 2 years (LD<2yr)] and late diabetics [diabetes for 2-5 years (LD 2-5 yr)] were used for the study. For comparison, human islets were treated with oxidative stress agent tert-butyl hydroperoxide (tBH, at 200 µM for 5 h) to induce cell damage then checked for B7-H4 mRNA spliced variants. Agarose gel (1.8%) electrophorogram of RT-PCR products from serum samples showed B7-H4 mRNA spliced variants in two major bands: 200 and 300 bp, with the 200-bp band being more prominent. Band intensity estimation (mean±SEM) of the 200-bp form showed a significant reduction in new T1D (6519 ± 983, N=11, p<0.01), LD<2 yr (3470 ± 524, N=6, p<0.001) and LD 2-5 yr (1759 ± 62, N=6, p<0.001) compared to normal adults (11123 ± 383, N=13), while only LD<2 yr and LD 2-5 yr groups showed significant differences, both at p<0.001, compared to AMC (9320 ± 1519, N=12). Human islets displayed multiple B7-H4 mRNA spliced forms with the 200-bp form as the most prominent, tBH treated islets showed a significant reduction in 200-bp band intensity (3847 ± 320, N=6, p<0.001) when compare to untreated controls (5743 ± 238, N=7). Our data suggest that quantitative estimation of band intensity of the B7-H4 mRNA 200-bp form in serum from T1D groups reflects β-cell damage, as shown in the *in vitro* islet data, and that variation in the B7-H4 mRNA 200-bp form could be used as a biomarker for diabetes detection.