language and whether they were on medical assistance at any time during the study period. The presence of a medication adjustment was tracked for metformin, thiazolidinediones, sulfonylureas (initiation or titration) and insulins (initiation only). Medication adjustment data was from the prescribing data in the medical record and thus represents physician actions. Results: There was a significant difference between African Americans and Caucasians on their initial A1c values (8.2% vs 7.3%; P=0.0001). Adjusted for the initial A1c, mean number of visits was similar between the two groups (16.9 vs. 15.8, P=.50). When change in A1c was calculated while controlling for initial A1c African Americans had less than half the decline found in Caucasians (P=.81) and medical assistance (P=.81) status were not related to the disparity in change value. Examination of medication adjustment for African Americans relative to Caucasians found that they were less likely to have adjustments of metformin (OR .69; P=.0021) and TZDs (OR .65; P=.0122) but no differences were found for Sulfonylureas (P=.49) or insulins (P=.30).

Conclusions: This study suggests that part of the racial disparity in glucose control is related to physician orders for medication intensification. This indicates a need for better understanding of the reasons for medication adjustment disparities and effective interventions to reduce them.

PS2-22:
Disease Management Strategies to Optimize Cardiovascular Risk in Type 2 Diabetes Mellitus

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Abstract: A number of disease management strategies have been developed in the last decade to reduce cardiovascular (CV) risk in adults with type 2 diabetes. On average such programs improve glycated hemoglobin (A1c) only 0.5%, and often have little or no effect on blood pressure (BP) control or low-density lipoprotein (LDL) control, which are critically important CV risk factors. In a large randomized Medicare demonstration program, diabetes disease management programs were unable to recover fees through cost savings to the Centers for Medicare and Medicaid Service (CMS). Available data suggest several strategies to improve the effectiveness and reduce the cost of diabetes disease management: (a) integrate disease management operationally and fiscally with primary care, (b) focus on treatment intensification, (c) focus on BP, LDL, aspirin use, and smoking cessation, as well as glucose control, and (d) individualize patient goals based on potential CV risk reduction potential and patient preference to maximize clinical benefit and return on investment for each patient.

PS2-30:
Study Recruitment Challenges for a Clinical Trial of Diabetes Education Interventions

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Background: While a scientifically sound study design is the basis for all clinical trials, activities such as patient recruitment present ongoing challenges. The availability of electronic data greatly enhances recruitment efforts, but also may bring new problems. The Journey for Control of Diabetes: the IDEA Study, a randomized clinical study conducted in two distant healthcare settings, utilizes an advanced electronic data system to identify and recruit eligible participants. We believe that communication of difficulties encountered could help researchers who face similar recruitment challenges.

Methods: The inclusion criteria included: Type 2 diabetes (DM), an A1C > 7 within 6 months of enrollment, and diabetes education naïve within the last year. Preliminary estimates of patient availability were calculated with the following assumptions: 1) two ICD-9 codes for Type 2 DM (10,000 patients); 2) 52% with A1C > 7 (5,200); 3) 60% with no DM education in the last 12 years (3,120); 4) 15% excluded with Type 1 or gestational DM (2,652); and 5) 15% willing to participate (397). Recruitment of 311 subjects was estimated to take five months. Results: Recruitment has been much slower than predicted (50% of subjects recruited at seven months). Recruitment results to date are: 1) 3,706 individuals identified as eligible using electronic data; 2) 156 enrolled; 3) 1,921 (50.4%) individuals not interested; 4) recent DM education (8.3%); and 5) Type 1 DM (2.1%). The electronic data on the pool of potential subjects is often out of date by the time the subject is screened and randomized. Refreshing the electronic data creates a moving target of potential candidates. A1C data and date of last diabetes education change at a rate faster than the recruitment mailing, screening and randomization process. Therefore, more time, programming, and staffing resources have been needed than originally anticipated to recruit study participants. Conclusions: This analysis of recruitment data demonstrates that initial recruitment capability estimates did not account for the inefficiencies created by outdated data and refreshed electronic encounter data. Daily tracking of all criteria for database accuracy has been a difficult and time-intensive task.

Genetics

C-B3-01:
Variation in Seven Obesity-Related Genes and Risk of Postmenopausal Breast Cancer

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Background/Aims: Obesity has been consistently associated with postmenopausal breast cancer risk. Proteins secreted by adipose tissue or involved in regulating obesity may play a role in breast tumor development. We conducted a nested case-control study among white, postmenopausal women from the American Cancer Society Cancer Prevention Study II (CPS-II) Nutrition Cohort to determine whether genes associated with obesity increase risk of breast cancer. Methods: Tagging single nucleotide polymorphisms (tagSNPs) were selected to capture common variation across seven candidate genes that encode adipose-related proteins: ADRB2, ADRB3, GHR, HSD11B1, IRS1, IRS2, SHC1. Thirty-nine SNPs were genotyped in 648 cases and 659 controls. Logistic regression models and haplotype analysis were used to examine the association between each tagSNP and risk of breast cancer while adjusting for matching factors and potential confounders. We also examined whether these SNPs were associated with measures of adult adiposity. Results: Two of five tagSNPs in HSD11B1 were associated with breast cancer (rs11807619, P=0.006; rs932335, P=0.0002). The rs932335 C allele was associated with a nearly two-fold increased risk of breast cancer (OR=1.83; 95% CI: 1.01–3.33 for C/C versus G/G). The rs11807619 and rs932335 were highly correlated (r2=0.74), and when modeled as a haplotype, only haplotypes containing the rs932335 C allele were associated with breast cancer. Three of the eleven SNPs for IRS2 were associated with breast cancer (rs4773082, P=0.007; rs2289046, P=0.016; rs754204, P=0.03). When these 3 SNPs were examined as a haplotype, only the haplotype that included the G allele of rs2289046 was associated with breast cancer risk adjusting for categorical and BMI (OR=0.74, 95% CI: 0.62–0.90 for TGC versus TGG). Background/Aims: Obesity has been consistently associated with postmenopausal breast cancer risk. Proteins secreted by adipose tissue or involved in regulating obesity may play a role in breast tumor development. We conducted a nested case-control study among white, postmenopausal women from the American Cancer Society Cancer Prevention Study II (CPS-II) Nutrition Cohort to determine whether genes associated with obesity increase risk of breast cancer. Methods: Tagging single nucleotide polymorphisms (tagSNPs) were selected to capture common variation across seven candidate genes that encode adipose-related proteins: ADRB2, ADRB3, GHR, HSD11B1, IRS1, IRS2, SHC1. Thirty-nine SNPs were genotyped in 648 cases and 659 controls. Logistic regression models and haplotype analysis were used to examine the association between each tagSNP and risk of breast cancer while adjusting for matching factors and potential confounders. We also examined whether these SNPs were associated with measures of adult adiposity. Results: Two of five tagSNPs in HSD11B1 were associated with breast cancer (rs11807619, P=0.006; rs932335, P=0.0002). The rs932335 C allele was associated with a nearly two-fold increased risk of breast cancer (OR=1.83; 95% CI: 1.01–3.33 for C/C versus G/G). The rs11807619 and rs932335 were highly correlated (r2=0.74), and when modeled as a haplotype, only haplotypes containing the rs932335 C allele were associated with breast cancer. Three of the eleven SNPs for IRS2 were associated with breast cancer (rs4773082, P=0.007; rs2289046, P=0.016; rs754204, P=0.03). When these 3 SNPs were examined as a haplotype, only the haplotype that included the G allele of rs2289046 was associated with breast cancer risk adjusting for categorical and BMI (OR=0.74, 95% CI: 0.62–0.90 for TGC versus TGG).