Neural tube defect (NTD) is a general term for a congenital malformation of the central nervous system in which the neural tube fails to close properly. Spina bifida meningomyelocele (SBMM) results from a lack of closure below the head, with spinal cord and meninges involvement. Individuals with SBMM have varying physical handicaps which can generally be associated with the level of the lesion along the spine. Derangements in maternal glucose metabolism have been implemented as causative in NTD formation. This study was designed to explore genetic variants in 12 genes known to be involved in glucose homeostasis and their relationship with the location of the rostral edge of the spinal lesions in patients with SBMM. We hypothesized that SNPs in these 12 genes, either singularly or in combination, modify the timing of the closure of the neural tube in such a way as to alter the location of the rostral edge of the lesion. 473 individuals with SBMM were genotyped for 40 SNPs within these 12 genes. Individuals were placed into categories based on how confident we were of their rostral lesion location, and based on two major ethnicities (Caucasian and Hispanics of Mexican descent). Analysis of variance (ANOVA) was used to compare the location of the rostral edge of the lesions of our sample population with SNP and haplotype genotypes. One SNP, LEPR_P1019 was significant in all of the population data sets. INSR_S366 was significant for the all subjects with lesion levels of confidence 1, and the Caucasian subset. LEPR_S343 was found to be significant for all subjects with rostral lesion levels of confidence 1, but not for either of the major ethnic groups. No haplotypes were found to be significant in the entire sample set of confidence 1, or the Caucasian subset. However, two haplotypes were found to be significant in the Mexican subset (LEPR_N656K/ LEPR_P1019, LEPR_S343/ LEPR_R223Q). Our results suggest that differential expression of these SNPs may affect the timing of the closure of the neural tube in such a way as to modify the location of the rostral edge and ultimately the degree of severity of SBMM in these patients. Further studies are needed in order to elicit how these SNPs work together to affect neurulation.