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There are many risk factors for the development of adiposity including diet, environmental factors and genetic constitution. Genetic factors can influence the susceptibility of an individual to develop obesity. Genetic factors that contribute to the development of adiposity include a predisposition to a reduced energy expenditure (impairment of mitochondrial function), impaired energy utilization (insulin resistance), or increased food intake. Individuals who are prone to obesity in part are the result of a combination of reduced energy expenditure and increased food intake. Because reduced energy expenditure is common to all patients with obesity, there must be a co-contributing genetic factor(s). Obesity that is polygenic, in that it is the result of a number of genetic mutations, predisposes an individual to an increased incidence of type 2 diabetes mellitus and metabolic syndrome. This is in contrast to monogenic obesity, where there is a single genetic mutation that results in adiposity. Two regions of the human genome have been linked to an increase in the risk of being obese, namely the FTO locus (fat mass and obesity) on chromosome 16, and the MC4R locus (melanocortin 4 receptor) on chromosome 18 (Locke A, Scuteri A, Valle R. There are several animal models of obesity, including the agouti mouse. The agouti mouse was the first to be identified and its mutation was mapped to the agouti gene. The agouti mouse has a dominant mutation, named agouti, that results in an obese phenotype. The agouti protein is a member of the helix-loop-helix (HLH) family of transcription factors. The agouti protein acts as a transcriptional repressor. Thus, the mutation in the agouti gene results in the loss of function of the agouti protein. Loss of function of the agouti protein causes an increase in the rate of melanocyte proliferation in the skin. The agouti gene is the mouse homologue of the human FTO gene. The agouti gene is mutated in the agouti mouse, which has a recessive mutation that results in obesity. This gene is also a candidate gene for human obesity because a similar mutation is associated with human obesity in at least two groups of human subjects. The MC4R gene was first identified in a patient with congenital hypopituitarism. MC4R encodes a G-protein coupled receptor. Two loss

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