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Changing beliefs about role of genetics in body weight irrelevant to weight loss

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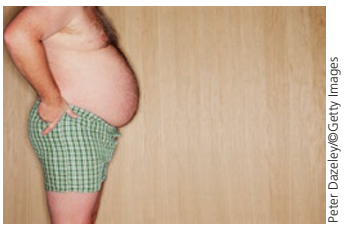

Peter Dazeley/©Getty Images

Holding a belief that body weight is controlled by genetic factors can influence behavior and motivation to lose weight, but beliefs may not affect weight loss. In this issue, McVay *et al.* report that nearly half of participants in a weight-maintenance study who believed that genes control their weight changed their minds after a year of participation, compared with a control group. The researchers state that this is the first report of change in perceptions about genetic causality for weight due to a weight-gain intervention program. However, change in attitude about the role of genetics in body weight did not affect weight loss after a year of study participation. In fact, participants who attributed their weight to genetics lost more weight than those who did not attribute their weight to genetics. By contrast, in the study control group, those who attributed their weight to genetics gained more weight after 18 months than those who did not attribute their weight to genetics. The data were obtained from the Shape Program, a trial that enrolled 185 black women between 25 and 44 years old. All study participants had a body mass index in the overweight range at enrollment. The study included behavior change goals and counseling, in addition to free access to local gyms. The research team hypothesized that changing a person's attitudes about the role of genetics may be an active ingredient in the intervention, but their results showed no association between change in attitudes and change in weight. *—Karyn Hede, News Editor*

Genetics of Charcot-Marie-Tooth disease gets even more complex

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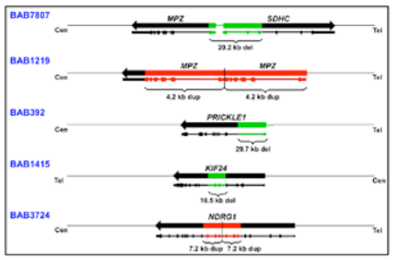

The neurological disorder Charcot-Marie-Tooth (CMT) disease is already among the most complex of the well-characterized genetic disorders, but it just got even more complicated. With a prevalence of 1 in 2,500, it is among the most common of inherited neurologic disorders. Although molecular studies have found all forms of Mendelian inheritance patterns in CMT, most cases can be traced to the peripheral myelin protein 22 (*PMP22*) gene. Pehlivan *et al.* evaluated 200 confirmed CMT patients in whom the genetic cause could not be identified using standard diagnostic techniques. The research team reports the first disease-associated autosomal recessive duplication with copy-number variants (CNVs) in a rare neuropathy gene, *NDRG1*. Using whole-exome sequencing (WES) combined with array comparative genomic hybridization, the researchers identified underlying genetic variants in five patients, detecting new CNVs in *MPZ* (two cases), *PRICKLE1*, *KIF24*, and *NDRG1*. The study revealed that in complex cases of unknown origin, WES combined with CNV analysis is necessary to elucidate the underlying genetics. The findings suggest that both single-nucleotide variants and CNVs can occur in multiple CMT genes and that these complex underlying genetics may contribute to complicated neurological CMT cases. *—Karyn Hede, News Editor*

NEWS BRIEFS

New rare genetic syndrome described

A new rare genetic syndrome likely combines the dual effect of copy-number variants in two chromosomal regions that had previously been described individually, according to an international research team. The study, published in the March issue of *Cold Spring Harbor Molecular Case Studies,* involved two brothers with cognitive and facial malformations as well as ambiguous genitalia. The researchers conducted chromosomal and whole-exome sequencing testing on the patients and their parents. Both patients have a terminal duplication of chromosome 16q involving 114 genes and a terminal deletion of chromosome 5p involving 50 genes. A literature search revealed previous reports of syndromes with some of the same clinical features as those of the patients in this study. The 5p terminal deletion is associated with speech delay. Craniofacial and genital-urinary abnormalities have been reported in patients with a terminal duplication of 16q. The authors suggest that changes to gene copy numbers in both genomic regions likely contribute to the complex array of physical abnormalities of the patients. "We anticipate that the unbiased dissection of large copy number variants similar in size to those identified in this study will become possible with the increased throughput of genome editing technologies in model organisms," the researchers write. *—Karyn Hede, News Editor*