Researchers uncover the weak and dizzy gene

Many of the patients I see at the Hoffman Center complain of chronic fatigue accompanied by extreme lightheadedness. They often describe weakness, especially after standing up, typically associated with palpitations, “spaciness” and anxiety. This is sometimes referred to as mitral valve prolapse syndrome. Typically, patients with weak and dizzy symptom constellations are diagnosed as having an anxiety disorder and are given potent antidepressant or antianxiety medication. And just as typically, they react poorly because such patients tend to be very sensitive to medications.

Moreover, drugs that are typically administered do not get to the underlying causes of the disorder, which have remained poorly understood—until the recent publication of a fascinating article in the *New England Journal of Medicine*. The technical name of the article really threw me until I realized that it addressed the concerns of my weak and dizzy patients—“Orthostatic Intolerance and Tachycardia Associated with Norepinephrine-Transporter Deficiency.”

The article is a remarkable piece of work in that it associates a vague and often-dismissed orphan symptom complex with a specific, detectable biochemical abnormality. The article then astonishingly takes one huge leap forward to implicate—for the first time—a specific gene with susceptibility to the syndrome.

According to the authors, what makes weak and dizzy people unusual is their inappropriate response to the simple action of standing up (or other normal forms of stress). Ordinarily, standing requires that blood vessels contract to keep gravity from allowing all your blood from pooling in your legs. This is accomplished through the release of small amounts of adrenaline, the body’s “fight or flight” hormone.

In normal individuals, excess adrenaline is quickly sopped up by efficient adrenaline transporters that whisk away the mischievous stress hormone before it can wreak havoc in other parts of the body, such as the heart or the brain. But in weak and dizzy individuals, the adrenaline transporter is defective and doesn’t do its job. The results are runaway symptoms that appear as if out of nowhere.

Of interest in this study is the fact that when identical twin sisters were studied, both were found to have the same adrenaline transporter gene defect, but one sister was profoundly affected, while the other only experienced mild symptoms. Moreover, some of the twins’ relatives also carried the gene, and some were weak and dizzy, while others were not.

This suggests that while genes may make some people susceptible to symptoms variously described as chronic fatigue syndrome, multiple chemical sensitivity or mitral valve prolapse, certain circumstances affect whether the gene will be expressed. These triggers may include stressful life experiences or personality traits, chemical exposure, viruses or other insults; head or neck injuries or inner ear disorders; dietary factors such as magnesium deficiency, reactive hypoglycemia or chronic dehydration; hyperventilation; or lack of physical conditioning.

With an understanding of the mechanisms underlying the weak and dizzy syndrome will come more effective therapies. Perhaps, with progress in genetic engineering methods being developed for repairing the specific gene defect underlying the condition.