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The Hawaiian and Cardiovascular Performance Genetics Studies – Research News

Our research into the effects of genetic variation in elite athletes and in cardiovascular disorders has kicked off excellently with some really interesting results.

The first gene we have completed working on is called alpha actinin 3, and it makes a protein (of the same name) that helps hold muscle fibres together, especially muscle fibres involving fast twitching muscle actions. There is a variation in this gene that shortens the alpha actinin 3 protein in a way that stops it from working, so it can't hold the muscle fibres together. People have two versions of each gene, one inherited from their mother and one from their father, so some people may have two normal versions of alpha actinin 3, or two short versions or one of each.

You might expect that people who end up with two short versions of alpha actinin 3 would have some problem with their muscles if they can't anchor the fibres in them together, but there is a closely related gene (alpha actinin 2) that can pick up the slack and hold the muscles together for people who don't have a normal alpha actinin 3. Alpha actinin 2 isn't quite the same as alpha actinin 3 though, and other researchers have shown that alpha actinin 3 is best for fast movements, and that alpha actinin 2 works better for slower movement. We thus thought that endurance athletes might tend to have more short versions of alpha actinin 3, because slower and more consistent action is required for these sports. And so, we tested it.

Our results showed that about 26.5% of our participants had two normal versions of the gene, 50.0% of participants had one of each and 23.5% had two short versions of alpha actinin 3. These numbers are about in line with the normal rates of variation for people originating in North America, Australia and Europe. When we looked at what factors determined how fast an athlete finished their event, it turns out that the gene variation in alpha actinin 3 didn't have any effect at all. This is probably because each different variation group has a tradeoff in abilities. People with only normal alpha actinin 3 can run faster, but need to slow down more to rest, while those with only short alpha actinin 3 don't need to rest, but can't quite get the speed going, and those with one of each sitting nicely in the middle.
We also found that an athlete's age and sex had strong effects on finishing time, with each decade adding about 40 minutes to finishing time and females finishing about an hour after males. There was also a weak (about 2.8% of variation) effect caused by the continent of origin and we're not sure exactly why that should be. Given that we were already accounting for genetic factors, it may be a result of different training availability or styles.

Interestingly, even though which version(s) of alpha actinin 3 an athlete have didn't alter their finishing time, we did find that the elite endurance athletes in this research did have significantly different alpha actinin 3 variations when compared to power and sprint athletes of European and African origin, who tend to have more normal versions of alpha actinin 3.