Hypertrophic cardiomyopathy: the genetic determinants of clinical disease expression

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CORRIGENDUM

In the March 2008 issue, Keren *et al.* stated that “Investigators in the Netherlands and South Africa have identified several *MYBPC3* [the myosin-binding protein C gene] mutations that exhibit founder effects that account for at least 30% of cases of HCM [hypertrophic cardiomyopathy] in their respective populations [Moolman-Smook JC *et al.* (1999) *Am J Hum Genet* **65**: 1308–1320; Tan HL *et al.* (2005) *Circulation* **112**: 207–213]”.

Although Moolman-Smook *et al.* have both reported numerous mutations in *MYBPC3* and founder effects in the South African HCM population, to date they have not found, nor reported, any HCM founder mutations in *MYBPC3*. Rather, they have identified founder mutations in the β -myosin heavy chain gene (*MYH7*), which account for approximately 25–30% of South African HCM cases.

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