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Idiopathic restrictive cardiomyopathy is part of the clinical expression of cardiac troponin I mutations

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Original citation: J. Clin. Invest.111:209–216 (2003). doi:10.1172/JCI16336. Citation for this corrigendum: J. Clin. Invest.111:925 (2003). doi:10.1172/JCI16336C1. The authors wish to correct errors that appeared in the Methods section and throughout the paper. The correct sentences are below. The authors regret the errors. Mutation analysis of TNNI3 by direct sequencing identified a 87A→G nucleotide substitution of exon 8 resulting in an Asp190Gly amino acid substitution that segregated with the disease in the family (maximal two-point lode score: 4.8). Direct sequencing of TNNI3 identified a 93G→A nucleotide substitution of exon 8, which resulted in an Arg192His amino acid substitution.

Corrigenda
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Idiopathic restrictive cardiomyopathy is part of the clinical expression of cardiac troponin I mutations

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Mutation analysis of *TNNI3* by direct sequencing identified a 87A→G nucleotide substitution of exon 8 resulting in an Asp190Gly amino acid substitution that segregated with the disease in the family (maximal two-point lode score: 4.8).

Direct sequencing of *TNNI3* identified a 93G→A nucleotide substitution of exon 8, which resulted in an Arg192His amino acid substitution.

MPDU1 mutations underlie a novel human congenital disorder of glycosylation, designated type If

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During the preparation of this manuscript for publication, errors were introduced into the author list. The corrected author list and affiliations appear below. The authors regret these errors.

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