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*Analysis of a novel X-
chromosomal recessive
glucokinase mutation within the
catalytic domain in a boy with
persistent hyperinsulinemic

hypoglycemia of infancy.
Mutations of the glucokinase gene (GK) have been shown to account for a number of forms of persistent hyperinsulinemic hypoglycemia of infancy (HHGI). Most reported mutations result in a defective GK protein through missense

mutations of the catalytic or regulatory domains of the protein. In this study, we identified a novel single-base mutation in exon 4 of the GK gene in a boy with glucokinase-deficient HHGI. This mutation resulted in a single base pair substitution, G to A, at codon

243 (c.724G>A). This A243 variant of GK cDNA introduced a premature stop codon at codon 242 (p.R242*) and resulted in a truncated GK protein of 150 amino acids with 32 amino acids missing from the C terminus. Because the single-base mutation in exon 4 resulted

in a protein truncation, we analyzed whether this truncated GK protein could be rescued by coexpression of wild-type (WT) GK cDNA. Both wild-type GK cDNA and the A243 GK cDNA were cloned into pCE

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