Supplemental figures and references for

Compromised catalysis and potential folding defects in *in vitro* studies of missense mutants associated with hereditary phosphoglucomutase 1 deficiency

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Figure S1. **Selected PGM1 homologs from an alignment of widely diverse sequences within the enzyme family.**
Residues from (1) involved in PGM1 deficiency mutations are indicated by red triangles; functional polymorphisms (2, 3) characterized in Table 2 are shown by green triangles. Insertions in other sequences relative to the human sequence are not shown. Several key functional regions within the active site are highlighted by green boxes above the sequence. These are: i) the catalytic phosphoserine loop (residues 115-120); ii) the metal binding site (residues 288-293); iii) the sugar-binding loop (residues 375-379); and iv) the phosphate-binding site (residues 503-507). For a complete listing of active site residues in PGM1, see (4). Alignment performed using ClustalW (5); figure made with Jalview (6). Sequence conservation (% identity) indicated by blue shading as calculated by Jalview; conservation is calculated relative to human PGM1; shaded regions must have no gaps and identities of 45% (light blue) and 85% (dark blue). Overall amino acid identities between human PGM1 and other sequences in the alignment ranges from 24 – 54%. Sequences were selected from 28 seed members of phosphoglucomutase family PIRSF001493 from the Protein Information Resource (http://pir.georgetown.edu/pirwww/index.shtml). (Seed sequences omitted from this alignment had amino acid identities great than ~60% to human PGM1 or were redundant in the seed group.)

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