Strike three for GLI3

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Nature Genet. **17**, 259–260

Due to an error on our part, the legend to Fig. 1 (below) was not printed in its entirety. We regret this error.

Fig. 1 *GLI3* mutation and consequent effect (an hypothesis). Full-length *GLI-3* consists of seven conserved domains, including the DNA-binding, zinc-finger and microtubular anchor domains. Beneath the amino-terminal end of the depicted protein are bars that represent proteins that are predicted to arise from truncating mutations in GCPS, PHS and PAP-A; the hatched portion of the bar indicates the known range in length of the predicted truncation products. These may act to repress or activate transcription. Phenotypic manifestations of the disorders include anterior or preaxial polydactyly (pre), posterior or postaxial polydactyly (post), central or insertional polydactyly (cen), syndactyly (syn), hypothalamic hamartoma (HH), hypertelorism (HT) and visceral anomalies (V). The colour of symbols and boxes in and indicate the proposed mechanism by which each phenotype is proposed to occur; Rep, repressor; Act, activator. Speckled boxes indicate variant features.

errata

Heterozygote advantage for HLA class-II type in hepatitis B virus infection

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Nature Genet. **17**, 10–11 (1997).

There was a typographical error in table 1 (printed below) under the category HLA-B. 113 heterozygotes were cleared of hepatitus B; not 13 as originally printed. The author regrets this error.

Table 1 • The numbers of homozygotes and heterozygotes for HLA class-I and class-I loci in Gambians who had cleared HBV or were persistently infected with this virus

	HLA-A		HLA-B		HLA-C		HLA-DR-DQ	
	Cleared	Persistent	Cleared	Persistent	Cleared	Persistent	Cleared	Persistent
Homozygotes	47	24	37	17	70	35	49	46
Heterozygotes	103	54	113	61	67	29	346	172
Odds ratio	1.03 [0.55–1.94]		1.17 [0.58–2.38]		0.87 [0.46–1.64]		0.53 [0.33–0.84]	
P value	NS		NS		NS		0.004	

NS: not significant. Subject phenotyping: HBV infection status was determined with standard serological criteria. Uninfected individuals were negative for antibodies to the HBV core antigen (anti-HBc). Individuals with self-limiting infection were anti-HBc positive and HBV surface antigen (HBsAg) negative. Individuals with persistent infection were anti-HBc (IgG) positive and HBsAg positive. Individuals who were anti-HBc (IgM) positive and HBsAg positive were excluded, as they are assumed to be in the acute phase of the disease, as were HIV antibody–positive individuals. MHC genotyping: HLA class-I antigens were typed serologically and HLA class-II haplotypes were determined using RFLP analysis as previously described[6]. RFLP MHC class-II haplotypes and the serological MHC class I serotypes encompass some allelic sequence diversity[12]. This is limited, however, and the functional relatedness of variants within a serological or RFLP defined type (as borne out by extensive experience in transplantation matching) allow homozygosity for these types to be used as a measure of functional homozygosity while preserving statistical power. Not all samples were typed serologically for HLA class-I antigens. Statistical analysis: The likelihood of persistent infection in heterozygotes is indicated by the odds ratio with 95% confidence intervals. *P* values were calculated with the χ^2 test.

Early diabetes and abnormal postnatal pancreatic islet development in mice lacking Glut-2

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Nature Genet. **17**, 327–330 (1997).

Dr. Jih-I Yeh's name was mistakenly listed as J.-Y. Wu when originally printed. The author regrets this error.