



Figure 4 The percentage of rare alleles in four related inbred lines. Allele frequencies were calculated in the 278 Chinese and US lines. Rare alleles are defined as those present in $\leq 5\%$ of the sequenced lines. B73 is known to be the ancestral line of 8112, 478 and Zheng58. All share the Iowa Stiff Stalk background.

Table 1 Mutation rates of base substitutions, short indels and copy number variations

	5003→478 ^a	8112→478 ^a	478→Z58 ^b
Number of years since breeding	26	26	24
Number of sites surveyed (all three generation with sequencing depth of ≥ 5)	170,469,103	317,986,593	488,466,696
Number of single-base mutations	179	164	441
Number of single-base mutations in genic regions	20	13	48
Number of single-base mutations in coding regions	6	9	13
Number of nonsynonymous single-base mutations	5	6	9
Single-base substitution rate (per site per year)	2.02×10^{-8}	9.92×10^{-9}	1.88×10^{-8}
Number of 1- to 10-bp indels	18	18	58
Mutation rate of 1- to 10-bp indels (per site per year)	2.03×10^{-9}	1.09×10^{-9}	2.47×10^{-9}
Number of genes surveyed	5,409	8,603	14,012
Number of CNV genes	103	113	305
CNV mutation rate (per gene per year)	3.66×10^{-4}	2.53×10^{-4}	4.19×10^{-4}

^aOnly the mutations transferred from 478 to Z58 were considered. ^bOnly the mutation sites showing no polymorphism in comparison of 8112 and 478 or 5003 and 478 were considered.

Corrigendum: A copy number variation morbidity map of developmental delay

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In the version of this article initially published, in Table 1 and its associated text, there was a calculation error in which the relative sizes of the case and control populations were set to be equal; because the size of the case population (15,767) was nearly double that of the control population (8,329), this resulted in erroneously inflated penetrance estimates. A simple definition of penetrance is used that is often applied in medical genetics—namely, the proportion of observed mutation carriers that are affected—to provide a metric that would be useful to clinical geneticists in a setting in which disease is heavily enriched, for example, in diagnosing children with developmental delay. That formulation is biased upwards with respect to population-level penetrance. Thus, in this corrigendum, an estimate more appropriate for population-level inference is provided assuming a general disease prevalence of 5.3% (*Am. J. Hum. Genet.* 42, 677–693, 1988) along with the more familiar odds ratio (OR) estimate. Importantly, all of these measures of penetrance are intrinsically limited by sampling error and imprecision in defining disease prevalence. We note that the mutation carrier counts, *P* values and other results in the original version of Table 1 are correct, and the key results and conclusions of the paper are unaffected. The error has been corrected in the HTML and PDF versions of the article.

Corrigendum: Genome-wide association analyses of esophageal squamous cell carcinoma in Chinese identify multiple susceptibility loci and gene-environment interactions

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