

Table E1: Frequencies of genetic polymorphisms

Genetic polymorphisms	Frequency	Reference
<i>CYP2D6</i>	4% CYP2D6*4 homozygotes, 66% non-CYP2D6*4 homozygotes, 29% CYP2D6*4/wildtype heterozygotes, and 0.2% CYP2D6*4/CYP2D6*5 heterozygotes (where wildtype refers to non-CYP2D6*4 and non-CYP2D6*5 alleles.	Brown et al, 2000. Polymorphisms of the CYP2D6 gene increase susceptibility to ankylosing spondylitis. Hum Mol Genet 9:1563-6.
<i>PON1 L55M</i>	LL, LM and MM genotypes of PON 55 polymorphisms showed similar frequencies in Italy (39.9, 47.0, 13.1%) and Ireland (39.5, 48.6, 11.9%) 40% homozygous wildtype, 43% heterozygous, 17% homozygous mutant. The frequency of the L allele decreased when passing from northern to southern Europe	Rea et al, 2004. Exp Gerontol 39:629-35. Paraoxonase polymorphisms PON1 192 and 55 and longevity in Italian centenarians and Irish nonagenarians. A pooled analysis. Fortunato et al, 2004. Clin Chem. 50:2012-8. Paraoxonase and superoxide dismutase gene polymorphisms and noise-induced hearing loss. Deakin et al, 2002. J. Clin. Endocrinol. Metab., Mar 2002; 87: 1268 - 1273. Paraoxonase-1 L55M Polymorphism Is Associated with an Abnormal Oral Glucose Tolerance Test and Differentiates High Risk Coronary Disease Families
<i>PON1 Q192R</i>	QQ homozygotes 47%, QR heterozygotes 43%, RR homozygotes 10%	Fortunato et al, 2004. Clin Chem. 50:2012-8. Paraoxonase and superoxide dismutase gene polymorphisms and noise-induced hearing loss.
<i>GSTM1</i>	42-60% homozygous null in Caucasians	Garte et al, 2001. Cancer Epidemiol Biomarkers Prev. 10:1239-48. Metabolic gene polymorphism frequencies in control populations.

<i>GSTT1</i>	homozygous null 10-20% in Caucasians (varies greatly with ethnicity).	Rebbeck TR, 1997. Cancer Epidemiol Biomarkers Prev 6:733-743. Molecular epidemiology of the human glutathione S-transferase genotypes <i>GSTM1</i> and <i>GSTT1</i> in cancer susceptibility.
<i>GSTM3</i>	70.9% <i>GSTM3</i> *A homozygotes, 25.8 % <i>GSTM3</i> *A/ <i>GSTM3</i> *B heterozygotes and 3.4% <i>GSTM3</i> *B homozygotes. Allele frequencies were: <i>GSTM3</i> *A, 0.842; <i>GSTM3</i> *B, 0.158.	Inskip et al, 1995. Biochem J. 312: 713-6. Identification of polymorphism at the glutathione S-transferase, <i>GSTM3</i> locus: evidence for linkage with <i>GSTM1</i> *A.
<i>GSTP1</i> haplotype in exon 5 (Ile ₁₀₄ Val) and exon 6 (Ala ₁₁₄ Val)	For Ile ₁₀₄ Val : 42.9% homozygous Ile/Ile, 40.4% heterozygotes, 16.7% Val/Val For Ala ₁₁₃ Val 79.9% Ala/Ala, 17.9% Ala/Val heterozygotes, 2.1% Val/Val	Kelada et al, 2003. Glutathione S-transferase M1, T1 and P1 polymorphisms and Parkinson's Disease. Neuroscience Letters 337:5-8.
<i>NQO1C609T</i>	10% TT	Ref 11
<i>CYP1B1</i> Val ₄₃₂ Leu	Homozygous wildtype 36.3%, heterozygote 46.0% and homozygous mutant 17.7%	Ko et al, 2001. Cancer Res. 61(11):4398-404. Association of <i>CYP1B1</i> codon 432 mutant allele in head and neck squamous cell cancer is reflected by somatic mutations of p53 in tumor tissue.
<i>MAO-A</i> exon 14	C 81.2% ,T 18.8%	http://www.ncbi.nlm.nih.gov/SNP/ . Rs1137070 Accessed on 31st July 2006.
<i>MAO-B</i> intron 13	A 50%, G 50%	http://www.ncbi.nlm.nih.gov/SNP/ . Rs1799836 Accessed on 31st July 2006.
<i>SOD2</i> A16V	AA homozygotes 14%, AV heterozygotes 65%, VV homozygotes 21%	Fortunato et al, 2004. Clin Chem. 50:2012-8. Paraoxonase and superoxide dismutase gene polymorphisms and noise-induced hearing loss.
<i>EPHX3</i> Y113H	heterozygotes 39.8%, homozygous mutant 11.7%, allele frequency 0.316 in Caucasians	Garte et al, 2001. Cancer Epidemiol Biomarkers Prev. 10:1239-48. Metabolic gene polymorphism frequencies in control populations.
<i>EPHX4</i> H139R	heterozygous 35.3%, homozygous mutant 3.8%, allele frequency 0.215 in Caucasians	Garte et al, 2001. Cancer Epidemiol Biomarkers Prev. 10:1239-48. Metabolic gene polymorphism frequencies in control populations.

<i>DAT1</i> A ₁₂₁₅ G		
<i>DRD2A</i>	65% homozygous wildtype, 6.9% homozygous mutant 66.8% homozygous wildtype, 29.2% heterozygote, 4.0% homozygous mutant	Berlin et al, 2000. Int J Neuropsychopharmacol. 3:35-43. Dopaminergic drug response and the genotype (Taq IA polymorphism) of the dopamine D2 receptor. Costa-Mallen et al, 2000. J Neurol Neurosurg Psychiatry 69:535-537. Genetic polymorphism of dopamine D2 receptors in Parkinson's disease and interactions with cigarette smoking and MAO-B intron 13 polymorphism
<i>DRD2B</i>	74.5% homozygous wildtype, 23.8% heterozygote, 1.7% homozygous mutant	Costa-Mallen et al, 2000. J Neurol Neurosurg Psychiatry 69:535-537. Genetic polymorphism of dopamine D2 receptors in Parkinson's disease and interactions with cigarette smoking and MAO-B intron 13 polymorphism
<i>NAT2</i>	The frequencies of <i>NAT2</i> slow acetylator alleles vary greatly with ethnicity. The frequency of the slow acetylator phenotype is around 60% among Germans (1,2), 53% among American Caucasians (2), 63% among Poles (3), and 50% among Finns (4), 90% in Egypt (5) and only 5% among Japanese (6).	1. Lin H, Han C-Y, Lin BK, Hardy S. Slow acetylator mutations in the human polymorphic <i>N</i> -acetyltransferase gene in 786 Asians, Blacks, Hispanics, and Whites: application to metabolic epidemiology. Am J Hum Genet 52:827-834 (1993). 2. Cascorbi I, Drakoulis N, Brockmüller J, Maurer A, Sperling K, Roots I. Arylamine <i>N</i> -acetyltransferase (<i>NAT2</i>) mutations and their allelic linkage in unrelated Caucasian individuals: correlation with phenotypic. Am J Hum Genet 57:581-592 (1995). 3. Mrozikiewicz PM, Drakoulis N, Roots I. Polymorphic arylamine <i>N</i> -acetyltransferase (<i>NAT2</i>) genes in children with insulin-dependent diabetes mellitus. Clin Pharmacol Ther 56:626-634 (1994). 4. Hirvonen A, Pelin K, Tammilehto L, Karjalainen A, Mattson K, Linnainmaa K. Inherited <i>GSTM1</i> and <i>NAT2</i> defects as concurrent risk modifiers for asbestos-associated human malignant mesothelioma. Cancer Res 55:2981-2983 (1995). 5. Weber WW. The Acetylator Genes and Drug Response. New York:Oxford University Press,

		1987. 6. Grant DM, Hughes NC, Janezic SA, Goodfellow GH, Chen HJ, Gaedigk A, Yu VL, Grewal R. Human acetyltransferase polymorphisms. <i>Mutat Res</i> 376:61-70 (1997).
--	--	--