Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Oesophageal cancer					
Tsutsumi *et al*. (1993), Japan	Cancer of the oesophagus	***CYP2E1***	5	See comments	Prevalence of **5B* allele was significantly higher in cases than in controls
Lucas *et al*. (1996), Brest, France, study period not reported	Cancer of the oesophagus	***CYP2E1 Rsa*I and *CYP2E1 Dra*I**	62	NS	There was no significant difference in genotype distribution and allele frequencies of *CYP2E1* among the groups studied
Morita *et al*. (1997), Osaka and Hyogo, Japan, 1992–1995	Cancer of the oesophagus SCC	***CYP2E1 Pst*I/*Rsa*I** **1A*/**1A* (wild-type) **1A*/**5B* + **5B*/**5B*	 34 18 + 1	 1.0 1.0 (0.5–2.0)	No adjustment
Hori *et al*. (1997), Tokyo, Japan, study period not reported	Cancer of the oesophagus SCC	***CYP2E1 Pst*I/*Rsa*I** **1A*/**1A* **1A*/**5B* **5B*/**5B*	 49 24 6	NS	No adjustment. No significant association
Tanabe *et al*. (1999), Hokaido, Japan, 1994–1997	Cancer of the oesophagus SCC	***CYP2E1 Rsa*I** **1A*/**1A* **1A*/**5B* **5B*/**5B*	 8 10 1	NS	No adjustment. No significant association
Tan *et al*. (2000), Linxian, Henan Province, China, 1997–1998 Results for *CYP2E1 Dra*I are from Lin *et al*. (1998)	Cancer of the oesophagus	***CYP2E1 Rsa*I** **1A*/**5B* + **5B*/**5B* **1A*/**1A* ***CYP2E1 Dra*I** CC CD + DD	 66 77 + 7 25 16 + 4	 1.0 4.8 (1.8–12.4) 1.0 1.5 (0.7–3.6)	Update of Lin *et al*. (1998), which had reported increased risk of oesophageal cancer associated with **1A*/**5B* + **5B*/**5B* genotypes versus **1A*/**1A* (in models not adjusted for tobacco use). Adjusted for age, sex and tobacco smoking
Chao *et al*. (2000), Taipei, Taiwan, China, 1997–1999	Cancer of the oesophagus	***CYP2E1 Pst*I/*Rsa*I**	88	NS	There was no significant difference in genotype distribution and allele frequencies of P4502E1 among the groups studied

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Gao *et al*. (2002), Huaian, Jiangsu Province, China, 1999–2000	Cancer of the oesophagus	***CYP2E1 Rsa*I**			Adjusted for age, sex, smoking and drinking habits, and consumption of meat, pickled vegetables, raw vegetables and garlic
		**1A/*1A*	55	1.00	
		**1A/*5B*	31	1.13 (0.60–2.13)	
		**5B/*5B*	7	1.23 (0.40–3.77)	
		**1A/*5B* + **5B/*5B*	38	1.15 (0.64–2.07)	
Lu *et al*. (2005), Xinjiang, China, 1998–2000	Cancer of the oesophagus SCC	***CYP2E1 Rsa*I**			No adjustment
		**1A/*5B* + **5B/*5B*	23	1.00	
		**1A/*1A*	91	11.13 (5.84–21.22)	
Yang *et al*. (2005), Aichi, Japan, 2001–2004	Cancer of the oesophagus	***CYP2E1 Rsa*I**			Adjusted for age, sex, tobacco smoking and alcohol drinking
		**1A/*1A*	110	1.00	
		**1A/*5B*	47	0.69 (0.45–1.07)	
		**5B/*5B*	7	1.11 (0.40–3.10)	
		**1A/*5B* + **5B/*5B*	57	0.73 (0.48–1.10)	
Li *et al*. (2005), Cape Town, South Africa, study period not reported	Cancer of the oesophagus SCC	***CYP2E1 Rsa*I**			Adjusted for age, sex, tobacco smoking and alcohol consumption
		**1A/*1A*	184	1.00	
		**1A/*5B*	5	0.58 (0.18–1.91)	
		***CYP2E1 Pst*I**			
		**1A/*1A*	184	1.00	
		**1A/*5B*	5	0.49 (0.16–1.46)	
		***CYP2E1 Dra*I**			
		DD	122	1.00	
		CD	66	5.90 (3.25–10.7)	
		CC	1	0.27 (0.02–2.56)	
Wang *et al*. (2006b), Huaian, Jiangsu Province, China, 2002–2003	Cancer of the oesophagus SCC	***CYP2E1 Rsa*I**	107	NS	There was no significant difference in genotype distribution and allele frequencies of *P4502E1* among the groups studied

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Rossini *et al*. (2007), Rio de Janeiro, Porto Alegre and Sao Paulo, Brazil, 2000–2006	Cancer of the oesophagus SCC	***CYP2E1 (*5B)***			No adjustment
		1A*/1A*	107	1.00	
		**1A*/*5B	11	1.58 (0.70–3.55)	
		*5B/*5B	5	3.58 (0.84–15.27)	
		**1A*/*5B + *5B/*5B	16	1.91 (0.94–3.89)	
		CYP2E1 (*6)			
		1A*/1A*	99	1.00	
		**1A*/*6	19	1.31 (0.71–2.42)	
		*6/*6	2	4.40 (0.39–49.17)	
		**1A*/*6 + *6/*6	21	1.40 (0.77–2.55)	
Qin *et al*. (2008), Xinjiang, China, 2005–2007	Cancer of the oesophagus	***CYP2E1 PstI/RsaI***			No adjustment was reported. MTHFR and CYP2E1 had a significant interaction: individuals with the MTHFR677 (C/T + T/T) and CYP2E1**1A*/**1A* genotypes had a 7.41-fold (95% CI: 3.60-15.25) risk of developing oesophageal cancer compared with those who carried the MTHFR677CC and CYP2E1 **1A*/*5B* + *5B/*5B* (*P* <0.001)
		1A*/1A*	94	1.00	
		**1A*/*5B*	23	0.37 (0.22–0.62)	
		*5B/*5B*	3	0.19 (0.05–0.66)	
		1A*/*5B* + *5B/*5B* **1A*/1A*	26 94	1.00 3.00 (1.82–4.96)	
Guo *et al*. (2008), Lanzhou, Gansu Province, China, 2004–2007	Cancer of the oesophagus SCC	***CYP2E1 PstI/RsaI***			Variables for which the results were adjusted were not reported
		1A*/*5B* + *5B/*5B* **1A*/1A*	16 + 7 57	1.00 2.82 (1.23–6.55)	
Cancers of other parts of the upper aerodigestive tract (UADT)					
Jahnke *et al*. (1996), Germany, study period not reported	Laryngeal SCC	***CYP2E1 RsaI/PstI***		NS	There was no significant difference in genotype frequencies of *P4502E1* between cases and controls
Lucas *et al*. (1996), Brest, France, study period not reported	UADT cancer	***CYP2E1 RsaI* and *CYP2E1 DraI***	96	NS	UADT cancer included cases with cancer of the mouth, pharynx and larynx. There was no significant difference in genotype distribution and allele frequencies of *P4502E1* among the groups studied

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Hildesheim *et al*. (1997), Taipei, Taiwan, China, 1991–1994	Nasopharyngeal cancer	***CYP2E1 Rsa*I**			Update of Hildesheim *et al*. (1995). Adjusted for age and sex
		**1A/*1A*	229	1.0	
		**1A/*5B*	108	0.83 (0.60–1.2)	
		**5B/*5B*	27	2.6 (1.2–5.7)	
		***CYP2E1 Dra*I**			
		DD	209	1.0	
		CD	125	0.89 (0.65–1.2)	
		CC	30	1.9 (0.98–3.7)	
Hung *et al*. (1997), Taipei, Taiwan, China, 1996	Oral cancer	***CYP2E1***			Only male participants. Adjusted for age and ethnicity
		Overall			
		**1A/*1A*	20	1.0	
		**1A/*5B*	19	1.8 (0.9–3.8)	
		**5B/*5B*	2	1.8 (0.3–10.7)	
		**1A/*5B* + **5B/*5B*	21	1.8 (0.9–3.9)	
		Betel quid non-chewers			
		**1A/*1A*	4	1.0	
		**1A/*5B* + **5B/*5B*	7	4.7 (1.1–20.2)	
		Betel quid non-chewers			
		**1A/*1A*	16	1.0	
		**1A/*5B* + **5B/*5B*	14	0.8 (0.2–3.3)	
González *et al*. (1998), Oviedo, Spain, 1993–1994	Head and neck cancers	***CYP2E1 Pst*I**		NS	Head and neck cancer cases included cases with cancer of the, pharynx, larynx and floor of the mouth. There was no significant difference in genotype frequencies of *P4502E1* between cases and controls
		**1A/*1A*	61		
		**1A/*5B*	11		
		**5B/*5B*	3		

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Matthias *et al*. (1998), Berlin, Germany, 1994–1996	Oral/pharyngeal cancer	***CYP2E1 Rsa*I/*Pst*I**			UADT cancer included cases with cancer of the mouth, pharynx and larynx. Adjusted by age and sex
		**1A/*5B* + **5B/*5B*	7 + 0	1.0	
		**1A/*1A*	115	0.9 (0.3–2.7)	
		***CYP2E1 Dra*I**			
	Laryngeal cancer	CD + CC	18 + 0	1.0	
		DD	100	1.2 (0.6–2.5)	
		***CYP2E1 Rsa*I/*Pst*I**			
		**1A/*5B* + **5B/*5B*	16 + 1	1.0	
		**1A/*1A*	240	0.8 (0.3–1.9)	
		***CYP2E1 Dra*I**			
		CD + CC	33 + 1	1.0	
		DD	195	1.3 (0.7–2.6)	
Katoh *et al*. (1999), Kitakyushu, Japan, 1992–1998	Oral cancer	***CYP2E1 Rsa*I**			Adjusted for age, sex and smoking status
		**1A/*1A*	53	1.00	
		**1A/*5B*	36	1.52 (0.82–2.79)	
		**5B/*5B*	3	0.94 (0.17–5.10)	
Morita *et al*. (1999), Japan, 1996	Head and neck SCC	***CYP2E1***		NS	There was no significant difference between patients and controls in the distribution of the polymorphisms of *CYP2E1*
		**1A/*1A*	14		
		**1A/*5B*	9		
		**5B/*5B*	0		
Tanabe *et al*. (1999), Hokaido, Japan, 1994–1997	Head and neck cancer	***CYP2E1 Rsa*I**		NS	Head and neck cancer cases included cases with cancer of the oral cavity, pharynx and larynx. There was no significant difference in genotype distribution and allele frequencies of *P4502E1* among the groups studied
		**1A/*1A*	10		
		**1A/*5B*	7		
		**5B/*5B*	1		

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Bouchardy *et al*. (2000), France, 1988–1992	Oral/pharyngeal cancer	***CYP2E1 Rsa*I**			UADT cancer included cases with cancer of the oral cavity, pharynx and larynx. Adjusted for age, sex and tobacco smoking
		By alcohol drinking			
		≤80 g/day			
		**1A/*1A*	41	1.0	
		**1A/*5B + *5B/*5B*	6	2.1 (0.6–7.2)	
		> 80 g/day			
		**1A/*1A*	63	2.5 (1.4–4.2)	
		**1A/*5B + *5B/*5B*	6	7.2 (1.4–38.2)	
		***CYP2E1 Dra*I**			
		By alcohol drinking			
		≤80 g/day			
		DD	35	1.0	
	Laryngeal cancer	CD + CC	12	1.8 (0.8–4.2)	
		> 80 g/day			
		DD	57	2.4 (1.1–4.3)	
		CD + CC	12	5.8 (1.9–18.2)	
		***CYP2E1 Rsa*I**			
		By alcohol drinking			
		≤80 g/day			
		**1A/*1A*	43	1.0	
		**1A/*5B + *5B/*5B*	6	2.0 (0.6–6.8)	
		> 80 g/day			
		**1A/*1A*	73	2.3 (1.4–3.9)	
		**1A/*5B + *5B/*5B*		0.9 (0.1–10.3)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Bouchardy *et al*. (2000) (contd)		***CYP2E1 Dra*I**			
		By alcohol drinking			
		≤80 g/day			
		DD	38	1.0	
		CD + CC	11	1.7 (0.7–4.1)	
		> 80 g/day			
		DD	61	2.2 (1.3–3.7)	
		CD + CC	13	4.6 (1.5–14.1)	
Liu *et al*. (2001), Philadelphia and New York, USA, 1994–1997	Oral cancer	***CYP2E1 Rsa*I/*Pst*I**			Adjusted for age, sex, race, study center, tobacco use and alcohol consumption. A significant increase in the *CYP2E1 *1A/*1A* genotype was observed in oral cancer cases as compared to controls in subjects who smoked ≤24 pack-years (*P*= 0.033).
		**1A/*1A*	160	1.00	
		Other genotypes	14	0.51 (0.22–1.20)	
		Smoking ≤24 pack-years			
		**1A/*1A*	50	1.00	
		Other genotypes	0	Reduced risk (*P*= 0.033)	
		Smoking >24 pack-years			
		**1A/*1A*	106	1.00	
		Other genotypes	14	0.70 (0.26–1.84)	
Zavras *et al*. (2002), Athens, Greece, 1995–1998	Oral SCC	***CYP2E1 Rsa*I**		See comments	93 cases. Genetic variation at the *CYP2E1 Rsa*I was almost entirely absent, with only 1 case and 1 control heterozygous for the variant. No statistical analyses were performed

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Gattás *et al*. (2006), Sao Paulo, Brazil, 2000–2003	Head and neck cancers	***CYP2E1 Pst*I**			Head and neck cancer included cases with cancer of the oral cavity, pharynx and larynx. Adjusted for age and sex
		**1A/*1A*	90	1.0	
		**1A/*5B*	13	2.3 (0.84–6.34)	
	Oral cavity cancer	**1A/*1A*	31	1.0	
		**1A/*5B*	7	3.6 (1.29–11.56)	
	Pharyngeal cancer	**1A/*1A*	39	1.0	
		**1A/*5B*	4	1.6 (0.43–6.13)	
	Laryngeal cancer	**1A/*1A*	17	1.0	
		**1A/*5B*	5	1.6 (0.30–8.51)	
Sugimura *et al*. (2006), Nagoya, Japan, 1988–2004	Oral SCC	***CYP2E1 Rsa*I**			Adjusted for age, sex, tobacco smoking and alcohol consumption
		**1A/*1A*	72	1.00	
		**1A/*5B*	39	1.26 (0.76–2.07)	
		**5B/*5B*	11	3.38 (1.22–9.36)	
		**1A/*1A* + **1A/*5B* + **5B/*5B*	111	1.00	
			11	3.13 (1.15–8.52)	
		***CYP2E1 Dra*I**			
		DD	59	1.00	
		CD	45	0.97 (0.59–1.58)	
		CC	18	2.28 (1.06–4.91)	
		DD + CD	104	1.00	
		CC	18	2.36 (1.14–4.86)	
Boccia *et al*. (2008), Rome, Italy, 2002–2006	Head and neck SCC	***CYP2E1 Rsa*I**			Head and neck cancer cases included cases with cancer of the larynx, oropharynx, hypopharynx and paranasal sinuses. Adjusted for age and sex
		**1A* allele		1.00	
		**5B* allele	10	0.72 (0.33–1.63)	
		***CYP2E1 Dra*I**			
		D allele		1.00	
		C allele	15	0.87 (0.43–1.76)	
Buch *et al*. (2008), Pittsburgh, USA, 2000–2004	Oral and pharyngeal SCC	***CYP2E1 Rsa*I**		NS	There was no significant difference in genotype distribution and allele frequencies of *CYP2E1* among the groups studied
		**1A/*1A*	176		
		**1A/*5B* + **5B/*5B*	14		

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Soya *et al.* (2008), Pondicherry, India, 2003–2006	Oral SCC	***CYP2E1 (*1B)***			Adjusted for age, sex, tobacco smoking, tobacco chewing and alcohol consumption
		A2A2	124	1.00	
		A2A1	57	1.46 (0.84–2.54)	
		A1A1	6	0.44 (0.11–1.80)	
		CYP2E1 (*5)			
		**1A/*1A*	179	1.00	
		**1A/*5B + *5B/*5B*	8	1.64 (0.48–5.58)	
		CYP2E1 (*6)			
		DD	121	1.00	
		CD	60	1.22 (0.72–2.09)	
	Pharyngeal SCC	CC	6	0.97 (0.29–3.27)	
		CYP2E1 (*1B)			
		A2A2	85	1.00	
		A2A1	51	1.87 (0.92–2.43)	
		A1A1	5	0.45 (0.10–2.00)	
		CYP2E1 (*5)			
		**1A/*1A*	138	1.00	
		**1A/*5B + *5B/*5B*	3	0.62 (0.13–3.08)	
		CYP2E1 (*6)			
	Laryngeal SCC	DD	85	1.00	
		CD	51	1.35 (0.80–2.29)	
		CC	5	0.86 (0.23–3.17)	
		CYP2E1 (*1B)			
		A2A2	55	1.00	
		A2A1	23	1.51 (0.75–3.04)	
		A1A1	2	0.26 (0.04–1.88)	
		CYP2E1 (*5)			
		**1A/*1A*	77	1.00	
		**1A/*5B + *5B/*5B*	3	0.87 (0.17–4.44)	
		CYP2E1 (*6)			
		DD	42	1.00	
		CD	34	1.66 (0.87–3.17)	
		CC	4	1.12 (0.27–4.60)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Ruwali *et al*. (2009), Lucknow, India, study period not reported	Head and neck cancer	***CYP2E1 Rsa*I**			Head and neck cancer included cases with squamous cell carcinoma of the oral cavity, pharynx, or larynx.
		Non-alcoholics			
		**1A/*1A*	206	1.00	
		**1A/*5B* + **5B/*5B*	4	1.61 (0.35–7.31)	
		Alcoholics			
		**1A/*1A*	121	1.00	
		**1A/*5B* + **5B/*5B*	19	3.65 (1.20–11.09)	
		***CYP2E1 Dra*I**			
		Non-alcoholics			
		DD	133	1.00	
		DC + CC	77	1.37 (0.93–2.02)	
		Alcoholics			
		DD	76	1.00	
		DC + CC	64	2.87 (1.60–5.12)	
Olivieri *et al*. (2009), Sao Paulo, Brazil, study period not reported	Head and neck SCC	***CYP2E1 Rsa*I**			The distribution of **5B* allele showed a greater incidence in head and neck SCC than in control subjects. No adjustment
		**1A* allele		Increased risk (*P*< 0.001)	
		**5B* allele			
Lung Cancer					
Persson *et al*. (1993), Stockholm, Gothenburg, Malmo and Lund, Sweden, study period not reported	Cancer of the lung	***CYP2E1 Dra*I**		NS	195 lung cancer cases. A part of cases and controls were from a prospective study. Less frequent distribution of **5B* allele among lung cancer patients as compared to controls suggests that people carrying the **5B* allele might be at lower risk for developing lung cancer (**5B* allele frequency: 2% in cases; 5% in controls).
		DD	160		
		CD	33		
		CC	0		
		***CYP2E1 Taq*I**		NS	
		A2A2	148		
		A1A2	34		
		A1A1	0		
		***CYP2E1 Rsa*I**			
		**1A/*1A*	176		
		**1A/*5B*	8	Reduced risk for **5B* allele (*P*= 0.02)	
		**5B/*5B*	0		

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Hirvonen *et al*. (1993), Helsinki, Finland, in a 3-year period (dates not reported)	Cancer of the lung	***CYP2E1 Dra*I**		NS (*P*= 0.44)	No adjustment
		DD	85		
		CD	14		
		CC	2		
		***CYP2E1 Rsa*I**		NS	
		**1A/*1A*	118		
		**1A/*5B*	3		
		**5B/*5B*	0		
Kato *et al*. (1994), Baltimore, USA, 1985–1992	Cancer of the lung	***CYP2E1 Dra*I**			Update of Kato *et al*. (1992). Controls were patients with chronic obstructive pulmonary disease or a history of >40 pack-years of tobacco smoking or patients with cancer at anatomical sites other than lung or urinary bladder. No difference in distribution of *CYP2E1* genotypes between controls and cases with different histologic subtypes of lung cancer
		DD	46	1.00	
		CD	12	1.57 (0.59–4.18)	
		CC	0	–	
Watanabe *et al*. (1995), Saitama, Japan, study period not reported	Cancer of the lung	***CYP2E1***		NS	No adjustment. No difference in distribution of *CYP2E1* genotypes between controls and cases with different histologic subtypes of lung cancer
		**1A/*1A*	207		
		**1A/*5B*	96		
		**5B/*5B*	13		
Hamada *et al*. (1995), Rio de Janeiro, Brazil, 1991–1992	Cancer of the lung	***CYP2E1 Rsa*I**			Adjusted for age, sex and race
		**1A/*1A*	102	1.00	
		**1A/*5B*	11	0.85 (0.36–2.02)	
		**5B/*5B*	0	–	
London *et al*. (1996), Los Angeles, USA, 1991–1994	Cancer of the lung	***CYP2E1 Rsa*I**			Adjusted for age, sex, race and tobacco smoking
		**1A/*1A*	328	1.00	
		**1A/*5B*	13	0.72 (0.35–1.46)	
		**5B/*5B*	0	–	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Oyama *et al*. (1997), Kyushu, Japan, 1990–1994	Cancer of the lung	***CYP2E1 Rsa*I**			No adjustment was reported
		**1A/*1A* + **1A/*5B* **5B/*5B*	119 7	1.00 1.38 (0.59–3.26)	
	Cancer of the lung SCC	**1A/*1A* + **1A/*5B* **5B/*5B*	48 5	1.00 2.45 (0.92–6.48)	
	Cancer of the lung adenocarcinoma	**1A/*1A* + **1A/*5B* **5B/*5B*	71 2	1.00 0.66 (0.16–2.83)	
el-Zein *et al*. (1997), Texas, USA, study period not reported	Cancer of the lung	***CYP2E1***			Only smokers were recruited. No adjustment was reported
		All lung cancers			
		**1A/*1A*	47	1.00	
		**1A/*5B* + **5B/*5B*	7	2.40 (0.52–15.8)	
		Lung adenocarcinoma			
		**1A/*1A*	20	1.00	
		**1A/*5B* + **5B/*5B*	7	18.9 (1.02–351)	
Wu *et al*. (1997), Houston, Galveston and San Antonio, USA, study period not reported	Cancer of the lung	***CYP2E1***			Study participants were African American or Mexican American.
		**1A/*5B* + **5B/*5B*			
		Never-smoker	1	1.0	
		Ever-smoker	15	1.3 (0.1–11.0)	
		**1A/*1A*			
		Never-smoker	7	6.7 (0.8–56.4)	
		Ever-smoker	114	16.3 (2.1–126.8)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Le Marchand *et al*. (1998), Hawaii, USA, 1992–1997	Cancer of the lung	***CYP2E1 Dra*I**			Adjusted for age, sex, ethnicity, smoking status, years of smoking, (years of smoking)^2, number of cigarettes smoked/day, and saturated fat and total vegetable intakes
		DD	240	1.0	
		DC	93	1.0 (0.7–1.6)	
		CC	5	0.2 (0.1–0.7)	
		***CYP2E1 Rsa*I**			
		**1A/*1A*	269	1.0	
		**1A/*5B*	66	0.8 (0.5–1.3)	
		**5B/*5B*	2	0.1 (0.0–0.5)	
Persson *et al*. (1999), Beijing, China, study period not reported	Cancer of the lung	***CYP2E1 (*5B)***			No adjustment was reported
		**1A/*1A*	48	1.00	
		**1A/*5B*	26	0.78 (0.43–1.43)	
		**5B/*5B*	2	0.50 (0.12–2.05)	
		CYP2E1 (*6)			
		**1A/*1A*	47	1.00	
		**1A*/*6	24	0.65 (0.35–1.19)	
		*6/*6	5	1.06 (0.33–3.36)	
Quiñones *et al*. (2001), Santiago, Chile, study period not reported	Cancer of the lung	***CYP2E1 Dra*I**			No adjustment
		DD	34	1.0	
		D/C	22	1.3 (0.6–2.7)	
		CC	2	0.6 (0.09–3.9)	
		***CYP2E1 Rsa*I/ *Pst*I**			
		**1A/*1A*	45	1.0	
		**1A/*5B*	14	0.8 (0.3–1.7)	
		**5B/*5B*	0	–	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Minegishi *et al*. (2007), Chiba and Tokyo, Japan, 2002–2003	Cancer of the lung	***CYP2E1***			Adjusted for age, sex, and smoking (pack-years). The genotype distribution was not in Hardy-Weinberg equilibrium in the control population [so the finding of an association with lung cancer is most likely a false-positive result]
		Overall			
		**1A/*1A*	300	1.00	
		**1A/*5B*	175	0.83 (0.60–1.15)	
		**5B/*5B*	30	4.66 (1.36–16.0)	
		**1A/*5B* + **5B/*5B*	205	0.93 (0.68–1.29)	
		By alcohol intake			
		**1A/*1A*			
		Non-drinker	72	1.00	
		≤31.6 g/day	95	1.81 (0.97–3.38)	
		>31.6 g/day	133	1.67 (0.86–3.21)	
		**1A/*5B* + **5B/*5B*			
		Non-drinker	48	1.00	
		≤31.6 g/day	59	1.74 (0.91–3.35)	
		>31.6 g/day	98	2.56 (1.16–5.65)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Eom *et al*. (2009), Cheongju and Cheonan, Korea (Republic of), 2001–2006	Cancer of the lung	***CYP2E1***			Adjusted for age and sex; results by alcohol drinking were additionally adjusted for smoking cumulative use; results by smoking were also adjusted for alcohol drinking level
		By alcohol intake			
		Non-drinkers			
		**1A/*1A*	132	1.00	
		**1A/*5B* + **5B/*5B*	49	0.56 (0.34–0.91)	
		<108 g/week			
		**1A/*1A*	44	1.00	
		**1A/*5B* + **5B/*5B*	30	1.19 (0.64–2.21)	
		≥108 g/week			
		**1A/*1A*	78	1.00	
		**1A/*5B* + **5B/*5B*	54	1.11 (0.63–1.94)	
		By smoking			
		Non-smokers			
		**1A/*1A*	47	1.00	
		**1A/*5B* + **5B/*5B*	25	1.11 (0.60–2.08)	
		<28 pack-years			
		**1A/*1A*	59	1.00	
		**1A/*5B* + **5B/*5B*	30	0.84 (0.46–1.53)	
		≥28 pack-years			
		**1A/*1A*	148	1.00	
		**1A/*5B* + **5B/*5B*	78	0.72 (0.46–1.13)	
Cancer of the stomach					
Kato *et al*. (1995), Tokyo, Japan, 1993–1994	Cancer of the stomach	***CYP2E1 Rsa*I**			Results with regard to *helicobacter pylori* infection were shown in Kato *et al*. (1996). No adjustment was reported
		**1A/*1A*	90	1.00	
		**1A/*5B*	54	1.04 (0.67–1.63)	
		**5B/*5B*	6	0.57 (0.22–1.50)	
Kato *et al*. (1997), Japan, study period not reported	Cancer of the stomach	***CYP2E1***			284 gastric cancer cases. No adjustment was reported. Possible overlap with Kato *et al*. (1995)
		**1A/*1A*		1.00	
		**1A/*5B*		1.05 (0.74–1.49)	
		**5B/*5B*		1.02 (0.47–2.20)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Nishimoto *et al*. (2000), Sao Paolo, Brazil, 1991–1994	Cancer of the stomach	***CYP2E1 Rsa*I**			Study participants were Japanese Brazilians, second-generation Japanese Brazilians and non-Japanese Brazilians. The number of participants with AA genotype was 1 among Japanese and 0 among non-Japanese Brazilians. Adjusted for age, sex, ethnicity, trimester of hospital admission, smoking meat consumption
		Japanese			
		**1A/*1A*	31	1.00	
		**1A/*5B* + **5B/*5B*	28	0.70 (0.30–1.62)	
		Brazilians			
		**1A/*1A*	178	1.00	
		**1A/*5B* + **5B/*5B*	11	0.42 (0.17–1.02)	
Cai *et al*. (2001), Changle, Fujian Province, China, 1996–1998	Cancer of the stomach	***CYP2E1***			No adjustment was reported
		**1A/*1A*	58	1.00	
		**1A/*5B*	27	1.50 (0.74–3.07)	
		**5B/*5B*	6	7.34 (0.84–166.6)	
		Alcohol non-drinkers			
		**1A/*1A*	26	1.00	
		**1A/*5B* + **5B/*5B*	14	1.83 (0.70–4.77)	
		Alcohol drinkers			
		**1A/*1A*	32	3.14 (1.42–6.99)	
		**1A/*5B* + **5B/*5B*	19	18.58 (6.47–53.37)	
Wu *et al*. (2002), Taipei, Taiwan, China, 1996–1999	Cancer of the stomach	***CYP2E1 Pst*I/*Rsa*I**		Reference group: **1A/*1A* + **1A/*5B*	The risk estimate remained significant after adjustment for gender, histological subtypes (diffuse, intestinal, mixed), location (cardia, body, antrum/angle), and stage (early, advanced). The prevalence of CYP2E1 *Dra*I polymorphism was similar in controls and gastric cancer cases (P=0.18).
		**1A/*1A*	215		
		**1A/*5B*	108		
		**5B/*5B*	33	2.9 (1.4–6.4)	
Gao *et al*. (2002), Huaian, China, 1999–2000	Cancer of the stomach	***CYP2E1 Rsa*I**			Adjusted for age, sex, smoking and drinking habits, and consumption of meat, pickled vegetables, raw vegetables and garlic
		**1A/*1A*	58	1.00	
		**1A/*5B*	31	1.07 (0.59–1.96)	
		**5B/*5B*	9	1.50 (0.54–4.18)	
Nan *et al*. (2005), Cheongju and Eulji, Korea (Republic of), 1997–2002	Cancer of the stomach	***CYP2E1***		NS	110 cases and 220 controls. Polymorphisms of the CYP2E1 gene was not associated with the risk of gastric cancer either with or without hypermethylation in the promoter of the hMLH1 gene.

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Boccia *et al*. (2007), Rome, Italy, study period not reported	Cancer of the stomach	***CYP2E1***			Adjusted for age and sex
		All participants			
		*Rsa*I polymorphic allele	5	0.54 (0.20–1.50)	
		*Dra*I polymorphic allele	15	1.33 (0.67–2.65)	
		Never smokers			
		*Rsa*I polymorphic allele	4	0.86 (0.26–2.88)	
		*Dra*I polymorphic allele	10	1.59 (0.67–3.79)	
		Ever smokers			
		*Rsa*I polymorphic allele	1	0.20 (0.02–2.70)	
		*Dra*I polymorphic allele	5	0.99 (0.31–3.13)	
		Never drinkers			
		*Rsa*I polymorphic allele	0	–	
		*Dra*I polymorphic allele	1	0.18 (0.70–2.46)	
		Ever drinkers			
		*Rsa*I polymorphic allele	5	0.86 (0.28–2.68)	
		*Dra*I polymorphic allele	14	3.70 (1.45–9.37)	
Cancer of the colorectum					
Kiss *et al*. (2000), Baranya, Hungry, study period not reported	Cancer of the colorectum	***CYP2E1 Pst*I**			No adjustment was reported
		**1A/*1A*	124		
		**1A/*5B*	37		
		**5B/*5B*	2		
		**1A* allele		1.00	
		**5B* allele		1.91 (1.05–3.52)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Le Marchand *et al*. (2002), Hawaii, USA, 1994–1998	Cancer of the colorectum	***CYP2E1 Rsa*I**			*CYP2E1* insertion refers to 5' 96-bp insertion variant in the gene. Adjusted for age, sex, ethnicity, pack-years of cigarette smoking, lifetime recreational physical activity (hours), lifetime aspirin use (months), body mass index 5 years ago, years of schooling, and intakes of non-starch polysaccharides from vegetables and calcium from foods and supplements
		Colorectum			
		**1A/*1A*	384	1.0	
		**1A/*5B*	116	0.8 (0.6–1.1)	
		**5B/*5B*	21	0.9 (0.5–1.6)	
		**1A/*5B* + **5B/*5B*	137	0.8 (0.6–1.1)	
		Colon			
		**1A/*1A*	271	1.0	
		**1A/*5B*	78	0.8 (0.6–1.1)	
		**5B/*5B*	19	1.2 (0.6–2.2)	
		**1A/*5B* + **5B/*5B*	97	0.8 (0.6–1.1)	
		Rectum			
		**1A/*1A*	120	1.0	
		**1A/*5B*	42	0.9 (0.6–1.3)	
		**5B/*5B*	3	0.4 (0.1–1.3)	
		**1A/*5B* + **5B/*5B*	45	0.8 (0.6–1.3)	
		CYP2E1 insertion			
		Colorectum	357	1.0	
		0	133	1.3 (1.0–1.8)	
		1	21	0.9 (0.5–1.6)	
		2	154	1.3 (0.9–1.7)	
		1 + 2			
		Colon	261	1.0	
		0	88	1.2 (0.9–1.7)	
		1	12	0.7 (0.3–1.2)	
		2	100	1.1 (0.8–1.2)	
		1 + 2			
		Rectum	104	1.0	
		0	49	1.7 (1.1–2.6)	
		1	9	1.4 (0.6–3.1)	
		2	58	1.6 (1.1–2.5)	
		1 + 2			

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Landi *et al*. (2005), Barcelona, Spain, 1996–1998	Cancer of the colorectum	***CYP2E1 Taq*I-**			+ / + genotype refers to more frequent genotype. + / – refers to heterozygote genotype. Adjusted for age and sex. None of the phenotypes of *CYP2E1* were found to be associated with colorectal cancer after adjusting for multiple comparison
		+ / +	274	1.00	
		– / +	82	0.83 (0.58–1.18)	
		– / –	5	0.79 (0.22–2.79)	
		***CYP2E1 Pst*I+**			
		**1A/*1A*	323	1.00	
		**1A/*5B*	17	1.05 (0.51–2.14)	
		**5B/*5B*	1	–	
		***CYP2E1 Rsa*I**			
		**1A/*1A*	305	1.00	
		**1A/*5B*	15	1.56 (0.65–3.75)	
		**5B/*5B*	0	–	
		CYP2E1 -333T>A	240	1.00	
		+ / +	95	0.88 (0.62–1.24)	
		– / +	14	1.01 (0.45–2.25)	
		– / –			
		CYP2E1 -71G>T	327	1.00	
		+ / +	29	0.56 (0.34–0.92)	
		– / +	1	–	
		– / –			
van der Logt *et al*. (2006), Nijmegen, the Netherlands, study period not reported	Cancer of the colorectum	***CYP2E1 Pst*I/*Rsa*I**			Adjusted for age and sex
		**1A/*1A*	333	1.0	
		**1A/*5B* + **5B/*5B*	23 + 1	1.1 (0.5–2.4)	
		***CYP2E1 Dra*I**			
		DD	324	1.0	
		D/C + CC	38 + 3	1.8 (0.9–3.8)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Gao *et al*. (2007), Huian and Jintan, Jiangsu Province, China, 2000–2002	Cancer of the colorectum	***CYP2E1 Rsa*I**			Adjusted for age and sex
		Colorectum	Non-smokers		
		1A*/5B* + **5B*/**5B*	275	1.00	
		5B*/5B*	5	2.20 (1.31–3.70)	
			Smokers		
		1A*/5B* + **5B*/**5B*	145	1.34 (0.93–1.92)	
		5B*/5B*	8	1.41 (0.50–3.96)	
			Non-drinkers		
		1A*/5B* + **5B*/**5B*	313	1.00	
		5B*/5B*	9	1.41 (0.89–2.24)	
			Drinkers		
		1A*/5B* + **5B*/**5B*	107	1.86 (1.28–2.24)	
		5B*/5B*	4	5.42 (1.65–17.40)	
		Colon	Non-smokers		
		1A*/5B* + **5B*/**5B*	57	1.00	
		5B*/5B*	4	1.95 (0.99–1.88)	
			Smokers		
		1A*/5B* + **5B*/**5B*	41	1.11 (0.66–1.88)	
		5B*/5B*	2	0.91 (0.18–4.57)	
			Non-drinkers		
		1A*/5B* + **5B*/**5B*	62	1.00	
		5B*/5B*	2	1.09 (0.50–2.38)	
			Drinkers		
		1A*/5B* + **5B*/**5B*	36	1.50 (0.89–2.55)	
		5B*/5B*	4	4.74 (1.10–20.40)	
		Rectum	Non-smokers		
		1A*/5B* + **5B*/**5B*	105	1.00	
		5B*/5B*	10	2.30 (1.32–3.99)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Gao *et al*. (2007) (contd)			Smokers		
		1A*/5B* + **5B*/**5B* **5B*/**5B*	88 6	1.48 (0.98–2.25) 1.75 (0.57–5.42)	
			Non-drinkers		
		1A*/5B* + **5B*/**5B* **5B*/**5B*	112 8	1.00 1.55 (0.95–2.53)	
			Drinkers		
		1A*/5B*+ **5B*/**5B* **5B*/**5B*	81 6	2.07 (1.37–3.14) 5.75 (1.65–20.05)	
Fan *et al*. (2007), Jiashan, Zhejiang Province, China, 1989–2005	Cancer of the colorectum	***CYP2E1 C-1019T***			Adjusted for age, sex and alcohol consumption
		Non-smoker		–	
		CC	60	–	
		CT + TT	62		
		Smoker			
		CC	31	1.00	
		CT + TT	51	1.83 (0.76–4.40)	
		≤35 pack-years			
		CC	18	1.00	
		CT + TT	32	2.42 (0.91–6.46)	
		>35 pack-years			
		CC	12	1.00	
		CT + TT	18	1.49 (0.49–4.53)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Morita *et al*. (2009), Fukuoka, Japan, 2000–2003	Cancer of the colorectum	***CYP2E1 Rsa*I**			*CYP2E1* insertion refers to 5' 96-bp insertion variant in the gene. Adjusted for age, sex, area, cigarette smoking, alcohol consumption, body mass index, type of job, physical activity and parental colorectal cancer
		Colorectum			
		**1A/*1A*	412	1.00	
		**1A/*5B*	237	0.91 (0.73–1.14)	
		**5B/*5B*	36	0.86 (0.53–1.38)	
		**1A/*5B* + **5B/*5B*	273	0.91 (0.73–1.12)	
		Colon			
		**1A/*1A*	215	1.00	
		**1A/*5B*	146	1.07 (0.82–1.40)	
		**5B/*5B*	23	1.12 (0.64–1.95)	
		**1A/*5B* + **5B/*5B*	169	1.08 (0.83–1.39)	
		Rectum			
		**1A/*1A*	191	1.00	
		**1A/*5B*	88	0.73 (0.54–0.99)	
		**5B/*5B*	11	0.56 (0.56–1.14)	
		**1A/*5B* + **5B/*5B*	99	0.71 (0.71–0.95)	
		CYP2E1 insertion			
		Colorectum			
		0	388	1.00	
		1	257	1.14 (0.92–1.43)	
		2	39	1.78 (1.07–2.96)	
		1 + 2	296	1.20 (0.97–1.49)	
		Colon			
		0	225	1.00	
		1	132	1.01 (0.77–1.32)	
		2	27	2.28 (1.29–4.01)	
		1 + 2	159	1.12 (0.87–1.45)	
		Rectum			
		0	154	1.00	
		1	123	1.39 (1.04–1.86)	
		2	12	1.46 (0.71–2.98)	
		1 + 2	135	1.40 (1.06–1.85)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Hepatocellular carcinoma					
Yu *et al*. (1995), Taipei, Taiwan, China, 1998–1992	Hepatocellular carcinoma	***CYP2E1 Pst*I** **1A/*5B* + **5B/*5B* **1A/*1A* ***CYP2E1 Dra*I** CC + CD DD	 5 25 8 21	 1.0 2.9 (1.0–8.0) 1.0 1.6 (0.7–3.9)	A cohort of 4841 male, asymptomatic, long-term HBV carriers and 2501 male non-carriers 30-65 years of age
Ladero *et al*. (1996), Spain, study period not reported	Hepatocellular carcinoma	***CYP2E1*** *All participants* **1A/*1A* **1A/*5B* **5B/*5B* *Alcohol intake >50 g/day* **1A/*1A* **1A/*5B*	 90 11 0 29 8	NS 1.00 5.18 (1.65–16.22)	In multivariate analyses which included age, hepatitis B and C virus serum markers, previous liver state and tobacco use, the **1A/*5B* genotype was also significantly associated with risk of hepatocellular cancer among heavy drinkers (*P*= 0.022; risk estimate was not provided)
Lee *et al*. (1997a), Seoul (Republic of Korea) and Fukuoka (Japan), 1994–1995)	Hepatocellular carcinoma	***CYP2E1*** **1A/*1A* **1A/*5B* + **5B/*5B* *Non-habitual drinkers* **1A/*1A* **1A/*5B* + **5B/*5B* *Habitual drinkers* **1A/*1A* **1A/*5B* + **5B/*5B*	 104 67 59 28 45 39	 1.0 1.9 (0.8–4.4) 1.0 1.4 (0.5–3.8) 1.0 2.6 (0.5–13.6)	No adjustment was reported

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Koide *et al*. (2000), Nagoya, Japan, 1994	Hepatocellular carcinoma	***CYP2E1***			The crude OR (95% CI) for **1A*/**1A* versus **1A*/**5B* + **5B*/**5B* genotypes was 0.45 (0.21–0.99). Overall results were adjusted for age and sex. Results by smoking status were adjusted for hepatitis C infection
		1A*/5B* + **5B*/**5B*	29 54	1.00 0.67 (0.16–2.88)	
		1A*/1A*			
		Nonsmokers			
		1A*/1A*		1.00	
		1A*/5B* + **5B*/**5B*		0.36 (0.07–1.95)	
		Smokers			
		1A*/1A*		2.07 (0.6–7.18)	
		1A*/5B* + **5B*/**5B*		6.22 (0.67–57.74)	
Wong *et al*. (2000), Edinburgh, United Kingdom, study period not reported	Hepatocellular carcinoma	***CYP2E1***			46 cases of hepatocellular carcinoma and 375 healthy blood donors (controls). No adjustment was reported
		*Rsa*I **5B* (vs. **1A*) allele		0.53 (0.01–3.35)	
		*Dra*I C (vs. D) allele		1.40 (0.55–3.10)	
		*Taq*I A1 (vs. A2) allele		0.73 (0.31–1.52)	
Yu *et al*. (2002), Haimen, China, 1995–1997	Hepatocellular carcinoma	***CYP2E1***			Adjusted for age and sex
		1A*/5B* + **5B*/**5B*	48 83	1.00 1.36 (0.81–2.28)	
		1A*/1A*			
Munaka *et al*. (2003), Kitakyushu, Japan, 1997–1998	Hepatocellular carcinoma	***CYP2E1***			Adjusted for age, sex, drinking status and viremia
		1A*/1A*	45	1.00	
		1A*/5B* + **5B*/**5B*	32	5.77 (1.24–27.39)	
Kato *et al*. (2003), Tokyo, Japan, 1993–1999	Hepatocellular carcinoma	***CYP2E1***			No adjustment
		1A*/1A*	57	1.00	
		1A*/5B* + **5B*/**5B*	36	0.91 (0.48–1.60)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Cancer of the female breast					
Choi *et al*. (2003), Seoul, Republic of Korea, 1995–2001	Cancer of the female breast	***CYP2E1***			Adjusted for age and family history of breast cancer
		All women			
		**1A/*1A*	232	1.0	
		**1A/*5B*	83	0.9 (0.59–1.23)	
		**5B/*5B*	14	1.3 (0.55–2.96)	
		Premenopausal			
		**1A/*1A*	154	1.0	
		**1A/*5B*	55	0.9 (0.58–1.51)	
		**5B/*5B*	6	0.7 (0.22–2.30)	
		Postmenopausal			
		**1A/*1A*	78	1.0	
		**1A/*5B*	28	0.7 (0.40–1.32)	
		**5B/*5B*	8	3.1 (0.8–11.46)	
Wu *et al*. (2006), Kaohsiung, Taiwan, China, 1999–2001	Cancer of the female breast	***CYP2E1***			Adjusted for age, menopausal status, and polymorphism in *GSTM1*
		**1A/*1A*	162	1.00	
		**1A/*5B*	94	1.00 (0.69–1.49)	
		**5B/*5B*	6	0.39 (0.08–0.76)	
Other cancers					
Anwar *et al*. (1996), Egypt, study period not reported	Cancer of the urinary bladder	***CYP2E1***			The P value (Fisher's exact test) for **1A/*5B* genotype versus **1A/*1A* was 0.48
		**1A/*1A*	22	1.00	
		**1A/*5B*	0	–	
		**5B/*5B*	0	–	
Brockmöller *et al*. (1996), Berlin, Germany, 1991–1994	Cancer of the urinary bladder	***CYP2E1 mutations***			
		-1019T	372	NS	
		9930G	362	NS	
		7666T	341	NS	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Farker *et al*, (1998a), Jena and Halle, Germany, study period not reported	Renal cell/urothelial cancer	***CYP2E1 Rsa*I/*Pst*I**			No adjustment was reported. In another publication based on 224 cases, Farker *et al*. (1998b) reported a higher risk associated with **1A/*5B* genotype of *CYP2E1 Rsa*I/*Pst*I and CD genotype of *CYP2E1 Dra*I among women, whereas these genotypes had inverse association with the risk in men. These estimates were based on small numbers. In analyses for both sexes combined, there was no association between any of the above polymorphisms and risk of renal cell/urothelial cancer.
		**1A/*1A*	256	NS	
		**1A/*5B*	16		
		**5B/*5B*	1		
		***CYP2E1 Dra*I**			
		DD	233	NS	
		CD	38		
		CC	2		
Murata *et al*. (2001), Mie, Yokohama and Chiba, Japan, 1995–1996	Cancer of the prostate	***CYP2E1***			Adjusted for age
		**5B/*5B*	5	1.0	
		**1A/*5B*	39	1.0	
		**1A/*1A*	71	1.4 (0.85–2.17)	
Yang *et al*. (2006a), Nanjing, Jiangsu Province, China, 2003–2005	Cancer of the prostate	***CYP2E1 Dra*I**			Update of Yang *et al*. (2006a). No adjustment was reported
		T/T	145	1.00	
		T/A	73	0.79 (0.54–1.16)	
		A/A	7	0.43 (0.17–1.08)	
		***CYP2E1 Msp*I**			
		A/A	166	1.00	
		A/G	50	0.93 (0.61–1.44)	
		G/G	9	1.24 (0.47–3.29)	
		***CYP2E1 Pst*I (*Rsa*I)**			
		**1A/*1A*	156	1.00	
		**1A/*5B*	65	0.67 (0.46–0.99)	
		**5B/*5B*	4	0.31 (0.10–1.00)	

Table 2.77. Studies of *CYP2E1* genotype-associated risk for cancer

Reference, study location, period	Cancer and site	Genes involved	No of cases/deaths	Relative risk (95% CI)[1]	Comments
Yang *et al*. (2009), Nanjing, Jiangsu Province, China, 2003–2005	Cancer of the prostate	***CYP2E1***			No adjustment was reported. Probable overlap with Yang *et al*. (2006a)
		Overall			
		**1A/*5B* +	32	1.00	
		**5B/*5B*	77	1.71 (1.04–2.82)	
		**1A/*1A*			
		By smoking			
		No smoking			
		**1A/*5B* +	10	1.00	
		**5B/*5B*	19	1.30 (0.53–3.20)	
		**1A/*1A*			
		≤700 cigarettes/year			
		**1A/*5B* +	12	1.08 (0.41–2.90)	
		**5B/*5B*	20	1.44 (0.58–3.54)	
		**1A/*1A*			
		>700 cigarettes/year			
		**1A/*5B* +	10	1.12 (0.40–3.14)	
		**5B/*5B*	38	2.80 (1.20–6.56)	
		**1A/*1A*			
Lee *et al*. (1997b), Korea (Republic of), study period not reported	Cancer of the pancreas	***CYP2E1***		NS	No adjustment

NR, not-reported; NS, non-significant; SCC, squamous cell carcinoma; UADT, upper aerodigestive tract