

Table 2**Characteristics of the *ATM*, *CHEK2* and *ERBB2* tagSNPs and their association with breast cancer survival**

SNP ID	SNP name	Number of cases/ controls	Minor allele frequency ^a	Breast cancer deaths/ person-years ^b	HR (95% CI) ^{b,c}
<i>ATM</i>					
TAG1 ^d	rs4987886	1,220/1,440	0.06	70/10,660	0.86 (0.42–1.76)
TAG2 ^d	rs3092991	1,119/1,318	0.14	65/9,717	1.12 (0.71–1.77)
TAG3 ^d	rs1800057	1,144/1,346	0.03	66/9,958	0.62 (0.16–2.46)
TAG4	rs1801516	1538/1,500	0.15	185/12,421	0.99 (0.75–1.30)
TAG5	rs17107917	1,546/1,493	0.04	192/12,466	1.01 (0.63–1.64)
TAG6 ^d	rs227060	1,152/1,350	0.28	68/10,004	0.77 (0.52–1.14)
TAG7 ^d	rs664143	1,227/1,408	0.48	73/10,684	1.24 (0.89–1.73)
<i>CHEK2</i>					
TAG1	rs8135424	1,539/1,478	0.13	197/12,367	1.08 (0.83–1.41)
TAG2	rs5762749	1,516/1,472	0.35	191/12,172	0.78 (0.62–0.97)
TAG3	rs743185	1,547/1,491	0.12	192/12,471	1.21 (0.91–1.62)
TAG4	rs738722	1,501/1,444	0.25	187/12,083	0.72 (0.56–0.93)
TAG5	rs5762765	1,510/1,456	0.38	192/12,118	1.15 (0.94–1.41)
TAG6	rs2236142	1,541/1,471	0.31	192/12,398	1.25 (1.02–1.54)
<i>ERBB2</i>					
TAG1	rs2643195	1,494/1,458	0.32	186/12,052	1.09 (0.88–1.35)
TAG2	rs4252596	1,530/1,481	0.13	189/12,330	0.95 (0.70–1.30)
TAG3	rs2952155	1,459/1,407	0.25	184/11,666	1.02 (0.80–1.29)
TAG4	rs2952156	1,546/1,481	0.32	194/12,445	1.11 (0.89–1.37)
TAG5 ^e	rs1801200 ^e	1,548/1,485	0.26	193/12,463	1.00 (0.80–1.25)
TAG6	rs4252665	1,527/1,486	0.05	182/12,401	0.76 (0.45–1.27)
TAG7	rs3809717	1,532/1,478	0.31	194/12,310	0.96 (0.77–1.20)

^aIn controls.^bAmong women with breast cancer.^cHRs are assessed assuming co-dominance and show the increase/decrease in risk of death from breast cancer with each addition of the minor allele compared with homozygotes of the major allele.^dNot genotyped in the patients who participated through tissue sample donation.^eAlso named I655V.

ATM, ataxia-telangiectasia mutated; CHEK2, checkpoint kinase 2; CI, confidence interval; ERBB2, v-erb-b2 avian erythroblastic leukemia viral oncogene homolog 2; HR, hazard ratio; SNP, single nucleotide polymorphism; tagSNP, haplotype-tagging SNP.

Our study was population-based. All participants were born in Sweden between 1919 and 1944, a time at which foreign immigration to Sweden was still rare [57], which means population stratification is of limited concern in our study. To minimize exposure misclassification, we applied genotyping methods with low error rates (the Sequenom and Illumina methods have genotyping error rates of 0.5% and 0.3%, respectively), DNA samples were randomly assigned to the genotyping plates and the genotyping personnel were blinded to case-control status. Furthermore, we replicated genotype calls of 200 randomly selected SNPs for a subset of samples

using a separate genotyping method with >99.5% concordance.

The oestrogen and progesterone receptor status of tumours and S-phase fraction were assessed at seven different laboratories in Sweden, but it is doubtful that the genotype frequencies could be related to any interlaboratory differences. A large proportion of the information on receptor status, S-phase fraction and grade was missing. Assessment of receptor status and S-phase fraction was, to a large extent, dependent on the size of the tumour, but evaluation of the tumour grade was