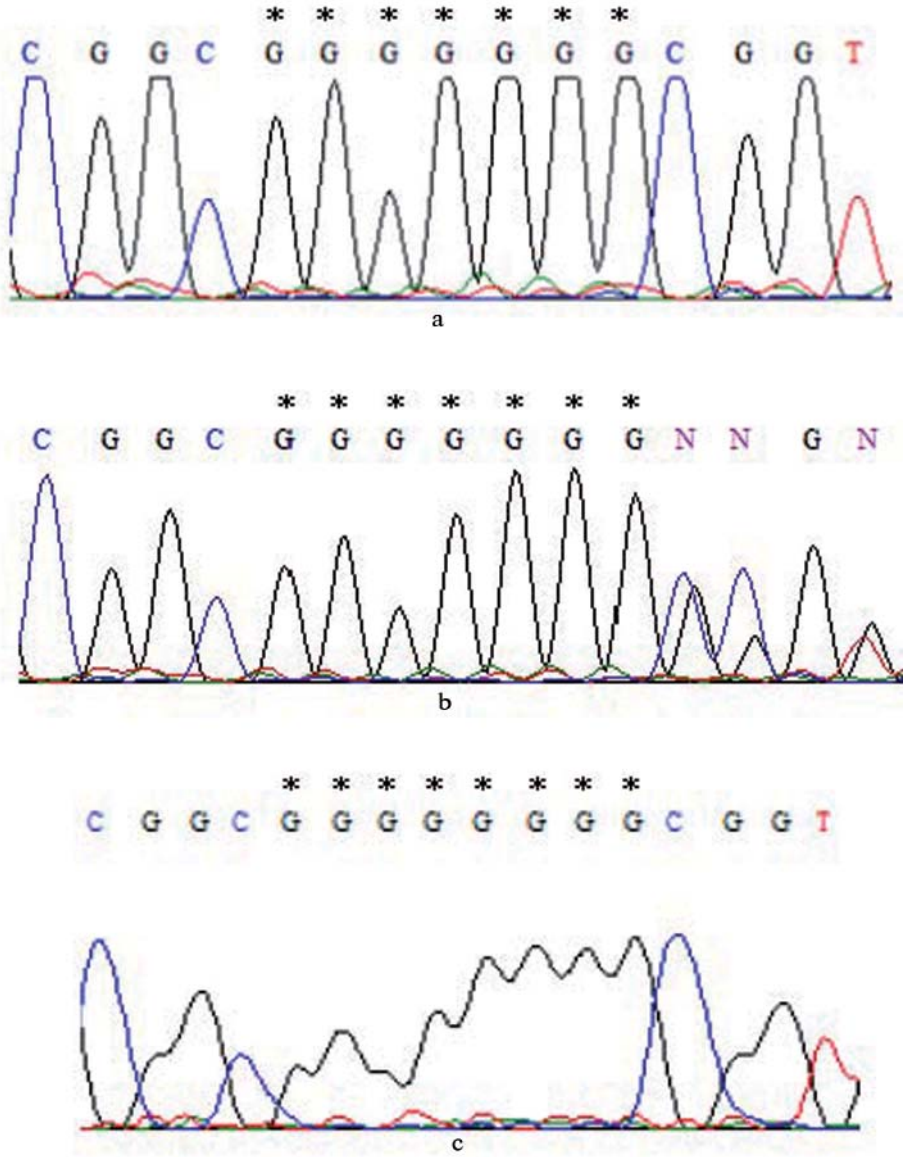

Figure A.1. Promoter polymorphisms of *p53* at positions −824 to −818. (a–c) Genomic DNA was amplified by PCR with a forward primer at positions −918 to −899 and a reverse primer at positions −669 to −690. The nucleotide sequences were determined by direct sequencing of the PCR products; the sequencing reactions were performed using a reverse primer at positions −669 to −690. *Nucleotides at positions −824 to −818 in the *p53* promoter show G-to-G insertions (b and c) in the opposite DNA strand to the *p53* promoter sequence. "N" indicates the heterozygous nucleotides CGGT and GCGG (b)

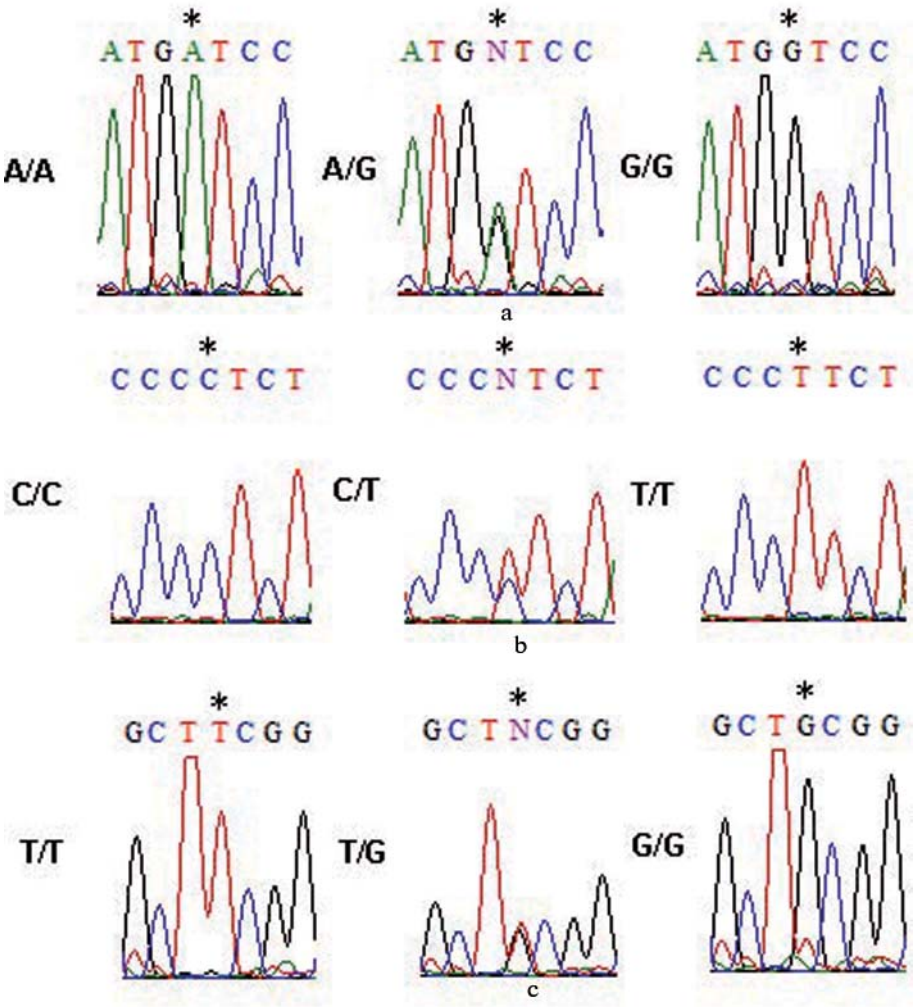

Figure A.2. Exon and intron polymorphisms of *MDM2* at positions −628, −466, and −215. (a–c) Genomic DNA was amplified by PCR with a forward primer at positions −725 to −704 and a reverse primer at positions +96 to +75. The nucleotide sequences were determined by direct sequencing of the PCR products; the sequencing reactions were performed using forward primers at positions −725 to −704 and −310 to −289. *Nucleotides at position −628 (a) in the *MDM2* exon 1 and positions −466 (b) and −215 (SNP309) (c) in the *MDM2* intron 1 show nucleotide substitutions of A-to-G (a), C-to-T (b), and T-to-G (c). "N" indicates the heterozygous nucleotides and includes the nucleotides adenine and guanine (a), cytosine and thymine (b), and thymine and guanine (c)

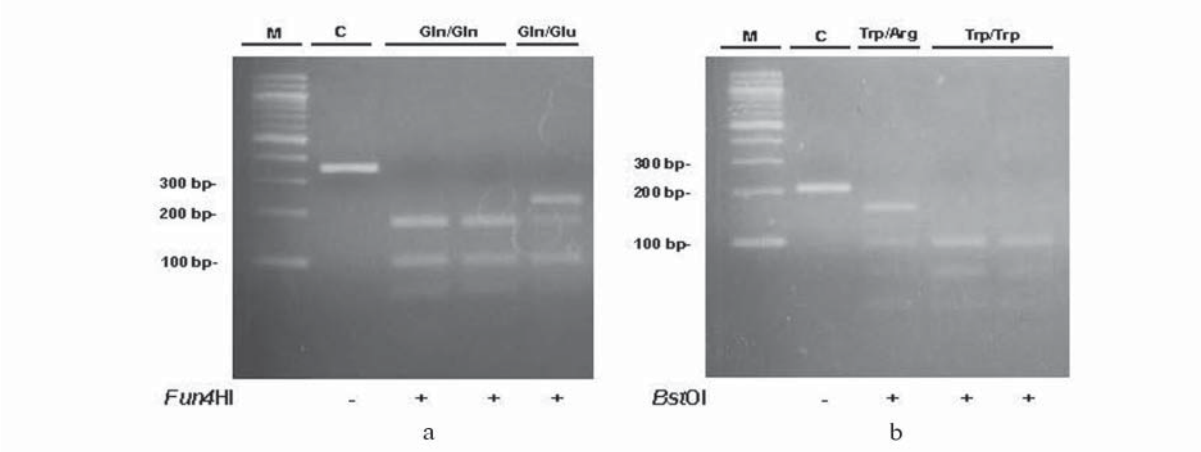

Figure A.3. RFLPs of β_2- and β_3-AR at codons 27 and 64. (a and b) Genomic DNA was amplified by PCR with the forward and reverse primers shown in "Materials and methods". The PCR products were treated with the indicated restriction enzyme and then separated by 3 % agarose gel electrophoresis. "M" indicates a 100-bp DNA ladder. Gln/Gln (which produces 174-, 97-, 55-, and 27-bp fragments) and Trp/Trp (which produces 99-, 62-, and 30-bp fragments) express homozygous glutamine residues at codon 27 (a; β_2-AR) and tryptophan residues at codon 64 (b; β_3-AR), respectively. Gln/Glu (which produces 229-, 174-, 97-, 55-, and 27-bp fragments) and Trp/Arg (which produces 161-, 99-, 62-, and 30-bp fragments) indicate heterozygous nucleotides coding for glutamine and glutamic acid at codon 27 (a; β_2-AR) and tryptophan and arginine at codon 64 (b; β_3-AR), respectively. Some small fragments generated by treatment with restriction enzymes were too small to be resolved by the gel electrophoresis

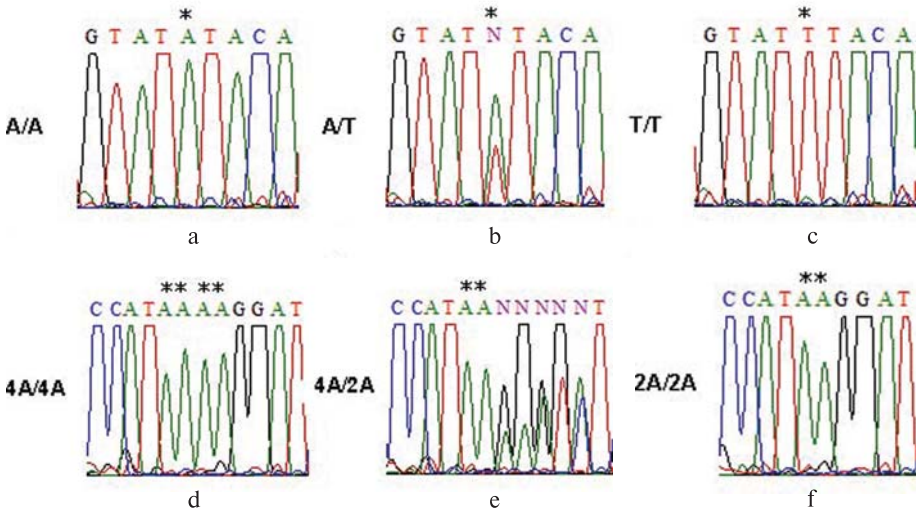

Figure A.4. Promoter polymorphisms of $p14^{ARF}$ at position −2610 and −2221 to −2218. (a–f) Genomic DNA was amplified by PCR with forward primers at positions −2986 to −2965 and −2702 to −2682 and reverse primers at positions −2532 to −2553 and −2157 to −2178. The nucleotide sequences were determined by direct sequencing of the PCR products; the sequencing reactions were performed using a forward primer at positions −2702 to −2682 and −2300 to −2279. *Nucleotides at positions −2610 and −2221 to −2218 in the $p14^{ARF}$ promoter show nucleotide substitutions of A-to-T (b and c) and nucleotide deletions of AA (e and f). "N" indicates the heterozygous nucleotides adenine and thymine (b) and nucleotides AAGGA and GGATC (e)

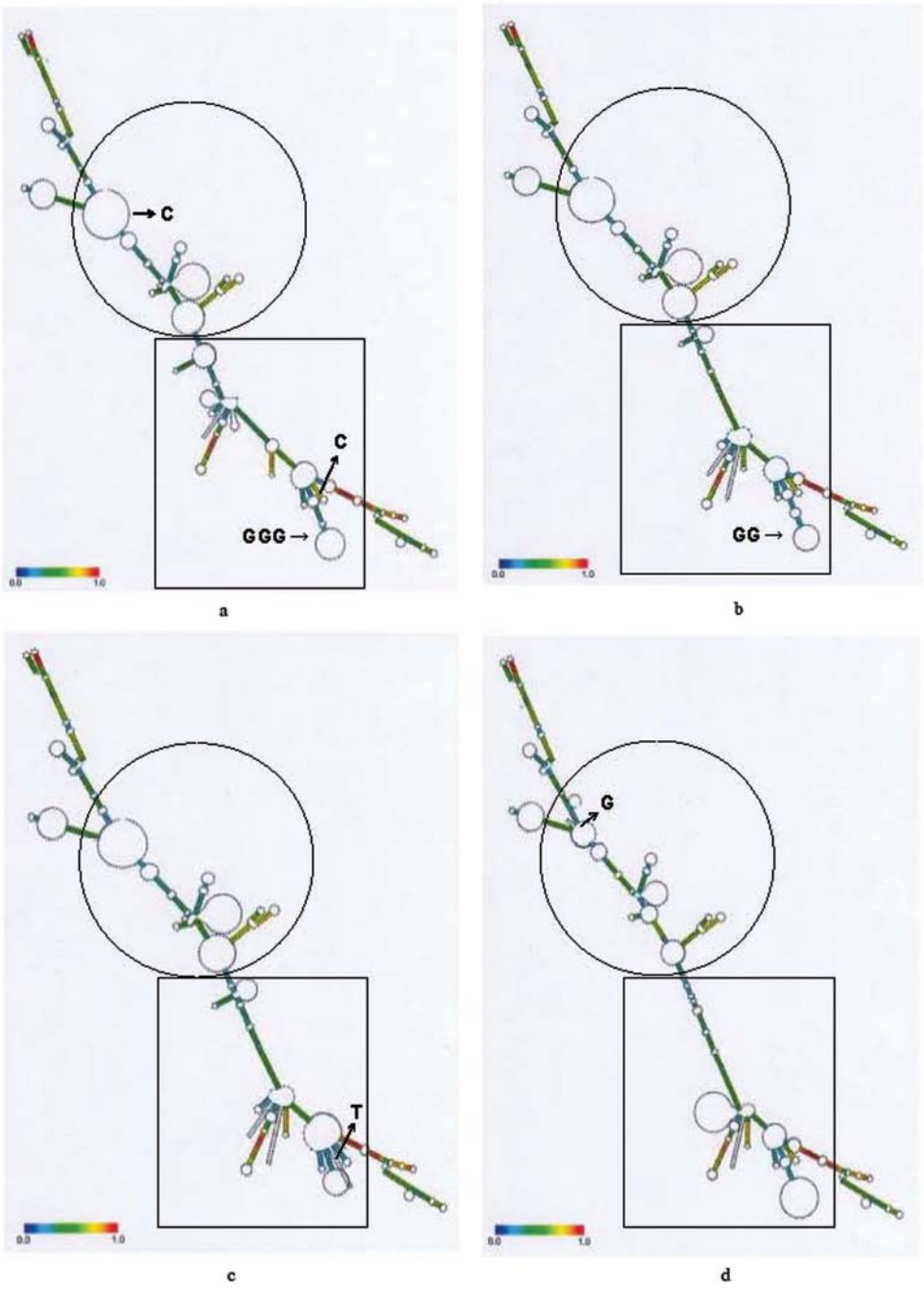

Figure A.5. Putative RNA secondary structures harboring a *PTEN* 5′-UTR polymorphism. The −465 to −463 positions with nucleotides GGG (a) or GG (b), the −404 position with nucleotide C (a) or T (c) (in a square), and the −9 position with nucleotide C (a) or G (d) (in a circle) in the *PTEN* 5′-UTR are indicated by arrows. Partial differences in the RNA secondary structure are expressed in the indicated frames with a circle (d) and square (b, c, and d)

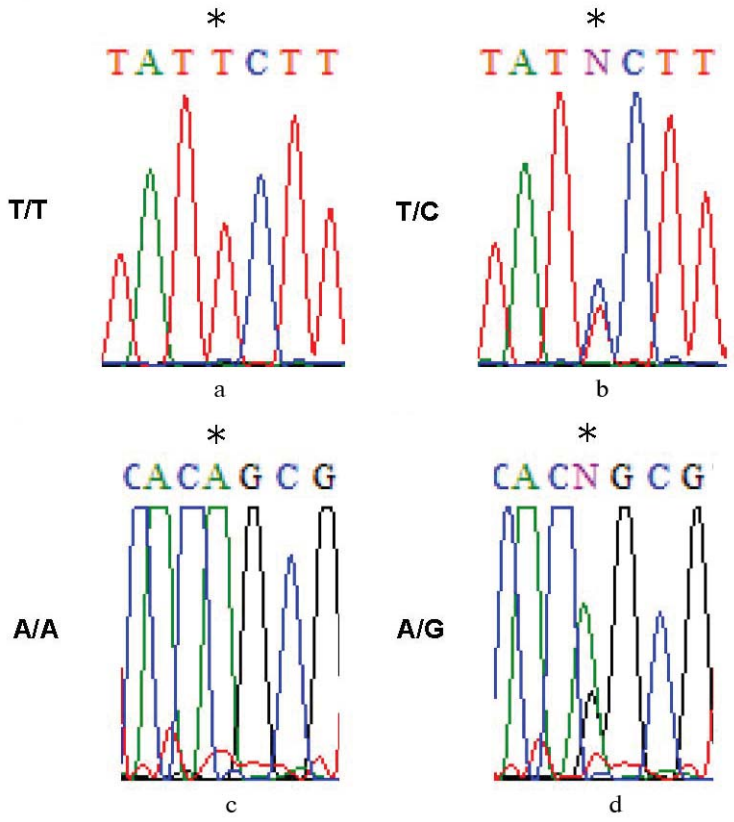

Figure A.6. Promoter polymorphisms of *p14*^{ARF} at position −3631 and of *p16*^{INK4a} at position −191. (a–d) Genomic DNA was amplified by PCR with a forward primer at positions −3756 to −3734 and a reverse primer at positions −3267 to −3289 of *p14*^{ARF} or with a forward primer at positions −485 to −465 and a reverse primer at positions +214 to +194 of *p16*^{INK4a}. The nucleotide sequences were determined by direct sequencing of the PCR products; the sequencing reactions were performed by using forward primers at positions −3756 to −3734 of *p14*^{ARF} and positions −248 to −228 of *p16*^{INK4a}. Nucleotide positions are numbered by considering the position of nucleotide G [56] in *p14*^{ARF} (accession no. AF082338.1) and nucleotide A at the initiation site for translation in *p16*^{INK4a} (accession no. X94154.1) as +1. *Nucleotides at position −3631 (a and b) in the *p14*^{ARF} promoter and position −191 (c and d) in the *p16*^{INK4a} promoter show nucleotide substitutions of T-to-C (b) in *p14*^{ARF} and A-to-G (d) in *p16*^{INK4a} "N" indicates the heterozygous nucleotides thymine and cytosine (b) and adenine and guanine (d)

Table A.1

***p14*^{ARF} and *p16*^{INK4a} promoter polymorphisms in normal Japanese individuals**

Individual number	*p14*^{ARF}				*p16*^{INK4a}
	−4924	−3631	−2610	−2218 to 2221	−191
I	C/T	T/C	A/T	4A/2A	A/G
IV	T/T	T/T	T/T	2A/2A	A/A