opportunistic infections[15-17]. This is probably because of epidemiological differences that limit the opportunities for human-to-human infection with *Toxoplasma*. In the absence of cannibalism, intraspecies transmission of *T. gondii* occurs only in cats[1] or, rarely, by transplacental infection[8].

Given that the probes used here were randomly selected, it is unlikely that they are directly involved in producing the virulence phenotype. Hence, the high degree of genetic similarity among virulent strains of *T. gondii* suggests that these strains form a distinct subgroup. To test this possibility, we used a binomial calculation[15] to determine the probability of observing a set of virulent strains with the same multilocus genotype using the null hypothesis of random mating. The calculation gave the probability of obtaining identical genotypes for 10 virulent strains of 28 total strains (Table 1) as $P = 8.8 \times 10^{-6}$. Using the smaller number of strains in Table 2, for which a larger number of loci have been analysed, $P = 1.2 \times 10^{-5}$ for the seven loci that are unlinked. These probability values are very low, indicating that the virulent strains examined here are not in random mating equilibrium with the population as a whole. Nonvirulent strains also show allele frequencies that differ from expected values, although this pattern is much less pronounced than for virulent strains. It is evident from Tables 1 and 2 that several possible recombinant genotypes are not observed (most notably there are no virulent strains with alleles predominant in nonvirulent strains). These findings are consistent with clonal expansion of virulent strains of *T. gondii* from a single lineage. The broad host range of *T. gondii* and the absence of geographical barriers may have contributed to the global distribution of the virulent genotype.

Although clonality has been observed among parasitic protozoa, many of these do not have a definitive sexual phase in their life cycle[15,18]. In *T. gondii*, clonality occurs despite a well established sexual cycle[1] and evidence that it has contributed to the overall genetic diversity of several of these strains[19]. *T. gondii* is also highly unusual in that all virulent strains belong to a single clonal lineage. The mechanisms by which this clonality has arisen and is maintained cannot be determined from our study, but may involve exclusive self-mating and/or asexual reproduction. The ability to conduct classical genetic crosses in *Toxoplasma*[9,10,20] could be exploited to distinguish between these alternatives and to identify the loci contributing to virulence in toxoplasmosis.

The fact that virulent strains of *T. gondii* comprise a single clonal lineage, regardless of their host or geographic origin, has clinical and biological implications. Using PCR it should be possible to examine fresh isolates from pregnant women and AIDS patients to establish whether there is any correlation between disease manifestations and infecting strains. Our results unambiguously extend the phenomenon of clonality of virulent strains to a ubiquitous protozoan species which has a sexual cycle. □

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ERRATUM

A protein kinase homologue controls phosphorylation of ganciclovir in human cytomegalovirus-infected cells

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IN two figures in the paper in the 9 July issue, areas of shading failed to show up on the printed page. Revised versions below show the correct shading. The published figure legends are unchanged.

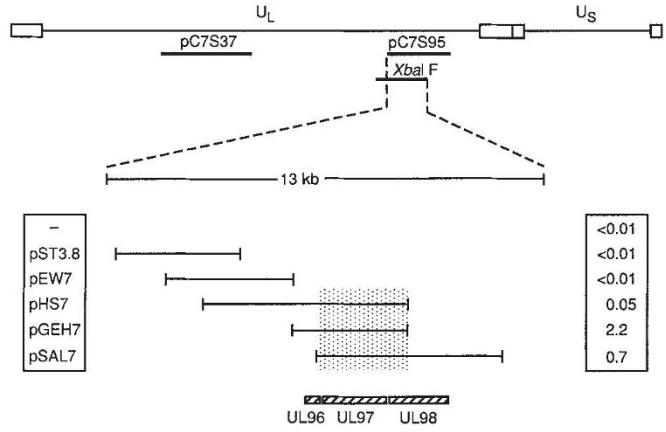

FIG. 1 Molecular mapping of ganciclovir-resistance markers of HCMV. The shaded box (lower centre) represents the 2.6-kb overlap (extending from *Sal*I to *Hin*dIII sites) of plasmids pHS7, pGEH7 and pSAL7 which were able to transfer ganciclovir resistance.

	amino acid				
HCMV UL97	622 -	AspGluValArgMetGly	- 9 -	GlyAlaAlaCysArgAlaLeu
HHV-6 15R	429 -	ArgGluAlaGlnLeuTyr	- 9 -	AspGluAlaCysArgLeuAsn
cAPK	220 -	AspTrpTrpAlaLeuGly	- 8 -	GlyTyrProProPhePheAla

FIG. 3 A 4-amino-acid deletion in the HCMV *UL97* gene product confers resistance to ganciclovir. The four amino acids deleted in ganciclovir-resistant mutant 759'D100 are shaded darkly and the five residues in subdomain IX of cAPK involved in substrate binding are lightly shaded.