An investigation into the diversity of genes of the innate immune system in the European Badger (*Meles meles*) and possible associations with trypanosome infection.

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Abstract

Toll-like receptor (TLR) genes encode for conserved proteins of the innate immune system which trigger pathways in response to pathogen invasion by recognising molecules essential for endoparasite survival. An endoparasite lives within the host and relies upon the host for its nutrition, here we consider it a pathogen when its presence causes detriment to the host. A pathogen is broadly anything which causes disease; causing specific symptoms in a specific location – in this case the symptoms would be the innate immune response to the parasite at its location, which would inevitably divert host energies to the creation of innate immune molecules. TLR genetic variation is rare and small mutations, such as single nucleotide polymorphisms (SNPs), can be correlated with enhanced disease resistance in some hosts. This research investigates TLR sequence diversity in European Badger (Meles meles) populations across the UK and possible associations with trypanosome infection. DNA from a collection of badgers from Woodchester Park, and other UK sites were available for investigation. As no badger TLR sequences were available or published, five PCR primer sets were successfully designed, using closely related species, to amplify the full badger TLR2 exon and exon 3 of TLR4. Sixty-one and fifty-nine badgers were sequenced across TLR2 and TLR4 (exon 3) respectively. Three TLR2 amino acid haplotype variants were found with two linked and one unlinked. No polymorphisms were found in the same badgers for TLR4. There was no significant relationship of polymorphism with trypanosome infection. Badgers ranging from St. Ives to Birmingham were found to be highly homogeneous with respect to TLR DNA sequence variation, and such low level variation was highly unusual in comparison to other TLR population studies.