many forest lepidoptera (Myers 1993), and identifying the mechanisms driving baculovirus spread is important for understanding the population dynamics of these insects (Anderson & May 1980; Bowers, Begon & Hodgkinson 1993; Dwyer, Dushoff & Yee 2004).

Materials and methods

As in many insects, gypsy moth larvae that are infected with their baculovirus release infectious particles known as ‘occlusion bodies’

We intentionally designed our experiments to test for variability in behaviours that affect infection risk, rather than to test for effects of variability in behaviour on infection risk itself, for several reasons. First, we were only able to measure total area consumed, whereas risk of infection is also affected by how close a larva gets to a cadaver while it feeds. Second, we could not control for variability in physiological susceptibility independently of behaviours that affect exposure, yet variability in physiological susceptibility in gypsy moth larvae is known to be quite high (Dwyer et al. 1997). Larvae that ate similar areas of contaminated foliage may therefore have had very different infection risks. We therefore did not expect that our experiments would provide much evidence for effects of behaviour on infection risk. However, in three trials (the two trials using full-sibling feral insects and the trial using a laboratory strain), we nevertheless reared larvae individually on artificial diet for several weeks after exposure to determine which larvae had become infected. The resulting data did indeed show that the amount of leaf area consumed can affect infection risk, but they also showed no effects on infection risk of interactions between family and area eaten, as we expected. It is thus in turn difficult to demonstrate that heritability in cadaver-detection ability alters infection rates. These data are tangential to the main thrust of our work, and so they are presented as Supporting Information.

STATISTICAL METHODS

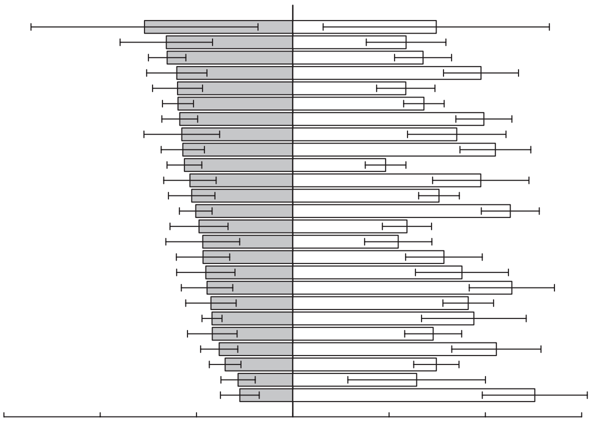
As we have described, Capinera

$$\mathrm{y_{ijkl}} = \mu + \mathrm{D_j} + \mathrm{F_{l(i)}D_j} + \mathrm{S_l D_j} + \mathrm{b_{k(il)}} + \epsilon_{\mathrm{ijkl}}. \qquad \text{eqn 5}$$

model has a ΔAIC value less than two, then the data cannot distinguish between that model and the best model. If one or more ΔAIC values are between two and three, support for the best model is only moderately strong; whereas if all values are greater than three, then support for the best model is very strong (Burnham & Anderson 2002). From this perspective,

Table 2. Akaike Information Criterion analysis of half-sibling experiments

Model	AIC	ΔAIC	AIC weights
Individual variation	−796·5	212·5	0
+ Presence of a virus disc	−956·0	53·0	0
+ Family's effect on consumption	−970·4	38·6	0
+ Family's effect on consumption + sire's effect on consumption	−968·6	40·4	0
+ Family's effect on detection + sire's effect on consumption	−1008·0	1·0	0·378
+ Family's effect on consumption + sire's effect on detection	−986·4	22·6	0
+ Family's effect on detection + sire's effect on detection	−1009·0	0·0	0·622



on branches in mesh bags in the field for 5 days, but in the first year of our study (conducted in July and August 2006), the control foliage was not held in the bags during this time. For our experiments in that year, this could have induced differences in foliage between the clean and virus-contaminated leaf discs due to mechanisms other than virus contamination. In our second year of experiments (conducted in 2007), however, including roughly half of the total individuals used in the study, we controlled for this effect by placing control foliage in bags alongside our infected bags. As we have already described, the results in the two different years were qualitatively consistent. More quantitatively, if we measure avoidance in terms of the difference in the amount consumed between clean and virus-contaminated discs, then the level of avoidance averaged across individuals was indistinguishable between the 2 years (year 1: mean 0·151 cm^2, SE 0·010; year 2: mean 0·130 cm^2, SE 0·010, two-sample t-test: t_{1221} = 1·51, P = 0·1324). It therefore appears that differences in the treatment of control foliage between years had no effect on our results.

Discussion

Our results confirm Capinera et al.'s result that gypsy moth larvae can detect and avoid leaves with infected cadavers. Larvae consumed significantly less contaminated foliage than control foliage in all of our trials. Our data also show that full- and half-sibling feral families differ in the amount of clean foliage consumed and in the extent to which they avoid contaminated leaves. These results suggest that there is a genetic component to the ability to detect virus-contaminated foliage, which is further supported by the observation that the genetically homogeneous laboratory strain did not vary in cadaver detection between families.

Experiments using full-sibling families do not rule out maternal effects, in which differences among egg masses stem from non-genetic attributes of the female parent, but previous work has suggested that such effects are weak in the gypsy moth (Myers, Boettner & Elkinton 1998; Erelli & Elkinton 2000). More directly, the occurrence of sire effects in the model that best described our half-sibling data suggest that cadaver avoidance is heritable, but the data also support the alternative model in which overall consumption is instead heritable. The family effects in our full-sibling trials may thus reflect genetic differences, but clearly more data are needed.

Larvae in our experiments also avoided contaminated foliage even when leaf discs were as much as 0·5 cm away from the cadaver. Spatial structure is known to have an effect on baculovirus transmission (Dwyer 1991; Hails et al. 2002; D'Amico et al. 2005), and we have demonstrated that the spatial scale at which larvae can detect cadavers is larger than a cadaver. It follows that larvae can avoid the virus even when it is at low concentrations, suggesting that small-scale spatial structure can have large effects on disease transmission. Indeed, Capinera et al. (1976) showed that larvae avoid even uninfected cadavers, and as the virus causes the breakdown of the larval integument, larvae that avoid infected cadavers may have been responding to cadaver components rather than to the virus.

Behaviour can thus play an important role in the transmission of insect baculoviruses. Anecdotal observations of larval behaviour in our experiments suggest that larvae consume foliage until they detect cadavers, and then they change position or stop feeding. Indeed, several individual feeding bouts were apparent on many discs, which is in accordance with reports of how gypsy moths feed in the wild (Heinrich 1979; Elkinton & Liebhold 1990). Thus, differences in leaf area consumed between the two discs are probably a reflection of smaller leaf bouts on the virus leaf discs, and differences between families probably result from different tolerances for the cadaver cue. Note that although vast numbers of different insect species are infected by nucleopolyhedroviruses (NPVs) (Miller 1997), to our knowledge behavioural mechanisms that affect NPV infection risk have been directly studied only in gypsy moths. Nevertheless, Dwyer (1991) provides indirect evidence that movement behaviour affects the risk that Douglas-fir tussock moth larvae (Orgyia pseudotsugata) become infected with tussock–moth NPV. Similarly, Hails et al. (2002) invoke small-scale spatial structure as a determinant of NPV transmission in the cabbage moth (Mamestra brassicae

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