



Figure 7. The English grain aphid, *Sitobion avenae*, a major pest of cereals in Europe and elsewhere and seemingly a highly migratory species. Fourth instar individual showing developing wing buds, and (top left) a winged adult.

# Big issues in population biology: lessons from a greenfly

## Presidential Address (Part 3)

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**Geographic populations:** What is an aphid population exactly? Can it be defined?

It is a well-established fact that many national aphid populations, especially those in the New World and Australasia, have been derived from introduced insects, probably very small founder populations, perhaps only a single individual in some instances. In most such cases, immigration probably results from human agency, i.e. aphids on imported plant material, root stocks, tubers, etc. However, instances appear to have occurred where winged aphids have travelled vast distances across landmasses and oceans to found new colonies (Bowden & Johnson, 1976; see Loxdale *et al.*, 1993 and references therein).

With insects such as *S. avenae*, a lack of any very clear IBD relationship (genetic divergence *vs.* geographical distance; Loxdale *et al.*, 1985; Llewellyn, 2000; Llewellyn *et al.*, 2003; Miller, 2000; Miller *et al.*, 2003) hinders attempts to derive the migratory range or source of migrants

from one region to another, at least over the time scales studied (usually several years). Such a lack of correlation does not readily fit with expectations of 'island' models of dispersal and population genetic structuring (Wright, 1931, 1951; Slatkin, 1985), nor with isolation by distance (Wright, 1943) and 'stepping stone' models (Slatkin, 1985, 1993; see Llewellyn, 2000 and Miller, 2000 for discussions). The generally poor IBD correlations may be compared with the strong IBD relationship shown for European badgers (*Meles meles* L.) which suggests that animals in one particular area are probably resident and that there is little gene flow between geographically-close populations which may have originated from a common refugium (Pope *et al.*, 2006; see their Fig. 4). With the tansy aphid, plotting measures of genetic divergence *vs.* geographical distance (on a log scale) failed to show any sign of IBD until populations were separated by around 470 km (Massonnet *et al.*, 2002; Massonnet & Weisser, 2004). Metapopulations show significant local genetic heterogeneity, but

clearly there is enough gene flow to offset the seasonal effects of drift and selection, with allopatric differentiation only apparent at large geographic scales. Hence, interpretation of such data must be treated with caution and probably gene/genotype frequencies are rarely, if ever, in equilibrium in terms of the dual influences of selection and drift (Roderick, 1996). Other data collected for *S. avenae* clearly show that some genes and multilocus genotypes are common across southern England and large parts of northern France (e.g. clone 53; Fig. 6), thereby revealing that the Channel is in all probability not a geographical barrier to migration and hence inter-population gene flow (Llewellyn, 2000).

At the very large geographical scale, the introduction of the Spotted Alfalfa aphid, *T. trifolii*, forma *maculata* into the USA in the 1950s (Blackman & Eastop, 2000), *M. dirhodum* into New Zealand in the early 1980s (Nicol *et al.*, 1997) and the Russian Wheat aphid, *Diuraphis noxia* (Mordvilko) into South Africa and the USA in the late 1970s and mid-1980s, respectively

(Blackman & Eastop, 2000), are all examples of the accidental importation of economic pests in recent historical times. With the aid of chromosomal and molecular markers, clues to the origins of such introductions have been forthcoming in recent years. Thus for example, *M. persicae* in Australia probably derived from European stock (Wilson *et al.*, 2002), *Sitobion* aphids in Australia and New Zealand came from Taiwan and Australia, respectively (Wilson *et al.*, 1999), whilst *S. avenae* in Chile were probably also of European origin (Figueroa *et al.*, 2005). In applied entomology, sometimes markers cannot prove exactly the source of an immigrant insect, but they can definitively say where it has probably *not* come from, e.g. the Californian 1989 and 1991 outbreaks, where samples of the medfly, *Ceratitis capitata* (Wiedemann) (Diptera: Tephritidae) differentiated by restriction fragment analysis of mtDNA did not apparently derive from Hawaiian populations of the insect (Sheppard *et al.*, 1992; see also Mun *et al.*, 1999 in the case of rice planthoppers).

### Box 6 *Aphids as crop pests*

That some aphid species were pests of growing crops causing direct feeding damage was certainly known by the end of the 18th century (e.g. Marsham, 1798; see Fig 4 Loxdale, *Antenna* 31(4)), whilst the ability of these insects to transmit plant viruses was established during the first few decades of the 20th century (e.g. Harris, 1940). As global pests they cause serious economic losses to a wide variety of crops and house and garden plants (Tatchell, 1989; Blackman & Eastop, 1994, 2000), whilst the cost of combating these insects, mainly by pesticides, is also huge (e.g. Riley *et al.*, 1997).

### *Clonal selection: survival of the fittest... or the luckiest?*

Whatever the mode of transport, aphid lineages are tested on arrival on their host plant/s by selection, be this abiotic or biotic. In fact, it may be disadvantageous for aphids acclimatised to certain ecological parameters, including photoperiods, to move long distances where they may be maladapted (Loxdale *et al.*, 1993 and references therein; but see also Lushai *et al.*, 1996 which contradicts this viewpoint). In the case of insecticide resistant genotypes, a susceptible individual moving to a sprayed area is clearly at a selective disadvantage compared with a resistant or perhaps better still, cross-resistant genotype (Loxdale *et al.*, 1993). A single aphid can found an

entire population (including sexual morphs in the case of holocyclic aphids), indeed a national population as aforementioned. The problem with obligate asexuals or species with rare sex is that the genome is linked, leading quickly to linkage disequilibrium of loci, and whilst one or a few loci may be selected in one direction, the remainder of the genome is dragged with it in a kind of mass 'hitchhiking' effect (see also Wilson & Sunnucks, 2006). Scenarios can be imagined where this quickly leads to wild swings in gene/genotype frequency as asexual lineages are selected in one direction and then later in another (see below). Sexual recombination, by reshuffling the genome, may be beneficial in the longer term in reducing the worst

effects of maladapted linkages of alleles. But even so, some loci will still react epistatically and other genes undoubtedly have negative pleiotropic influences, as apparent in the highly-resistant forms of *M. persicae* (Foster *et al.*, 2000).

In *M. persicae*, four main resistant genotypes occur in the field in the U.K. – S (susceptible), R<sub>1</sub>, R<sub>2</sub>, R<sub>3</sub>, directly related to the level of amplification and expression of the carboxylesterase E4/FE4 genes (80 x more genes in R<sub>3</sub> compared with S), which confers resistance primarily to carbamates and organophosphates and to a lesser extent, pyrethroids (Field *et al.*, 1999; Foster *et al.*, 2000). These are assayed either using electrophoretic, immunoassay techniques, or PCR (polymerase

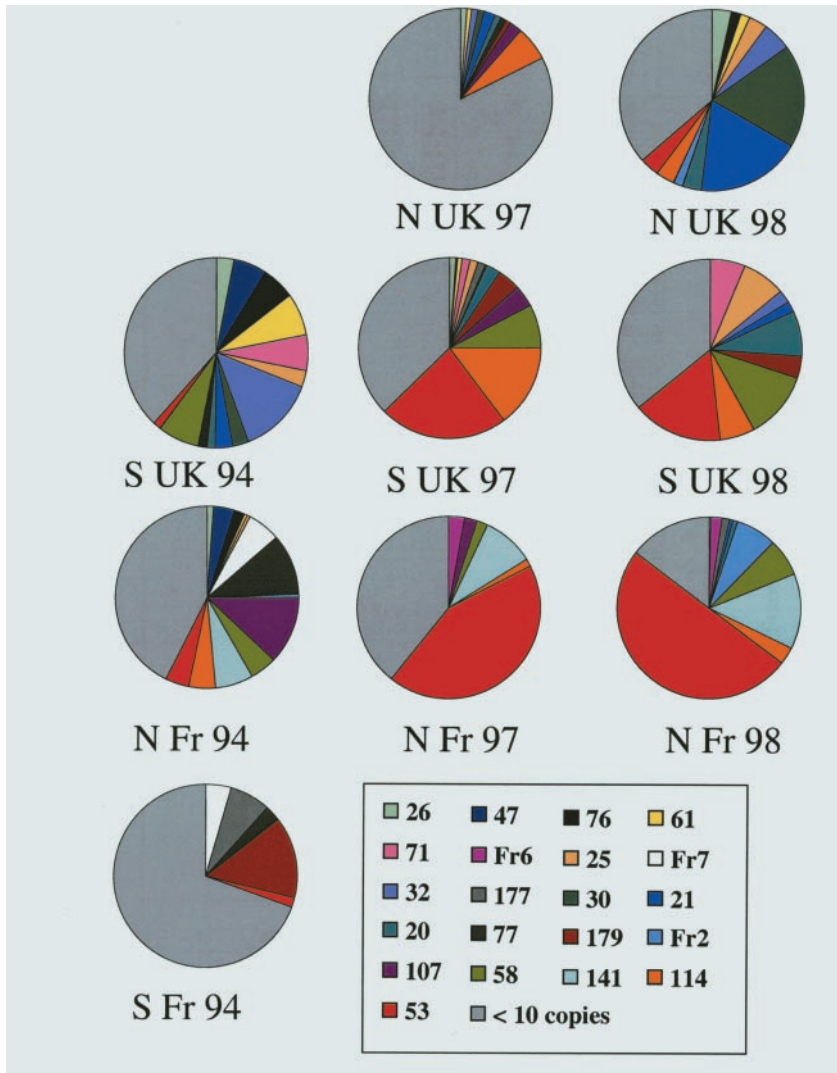


Fig. 8. Pie diagrams showing that some *S. avenae* genes and multilocus (microsatellite) genotypes are common across southern England and large parts of northern France. This suggests that the Channel is not a barrier to aerial movement, and hence gene flow in this migratory pest species. Frequencies in each collection of the 21 genotypes represented by 10 + aphids overall; unique genotypes or genotypes found in < 10 times overall are represented by pale grey segment. Abbreviations; N = north, S = south, numbers = year, i.e. 1994, '97 or '98. (From: Llewellyn, 2000).

chain reaction)-based methods, the last involving specific primers (Anstead *et al.*, 2005; Devonshire, 1989; Field *et al.*, 1999; Foster *et al.*, 2000). The highly E4 resistant genotypes (R<sub>2</sub>+) are also associated with a translocation of autosomes 1 and 3 (A1,3; Blackman *et al.*, 1995; Foster *et al.*, 2000; Field & Blackman, 2003). In addition, the various resistant genotypes may have other resistant mechanisms in association with the E4 genotype. These include MACE (modified acetylcholinesterase), which confers resistance to chemicals blocking the normal hydrolytic function of the enzyme acetylcholinesterase in the nerve synaptic junction and hence cause paralysis of the insect, and *kdr*

(‘knockdown resistance’) as well as *super-kdr*, mutated forms of the gene which controls the expression of the allosteric binding site of the sodium gating channel in the insect nerve and which confer resistance to pyrethroids (Denholm *et al.*, 1999; Anstead *et al.*, 2005). It is possible that the genotypes bearing the *kdr* and *super-kdr* mutations have a common origin, although due to the short time scale of resistance development, multiple independent origins of these are more likely (Anstead *et al.*, 2005).

The possession of the *kdr* gene has several pleiotropic effects on fitness in resistant *M. persicae*, including response to alarm pheromone (Foster

*et al.*, 2000) and vulnerability to parasitoid attack (Foster *et al.*, 2007 in press). As a consequence, whilst R<sub>2</sub> and R<sub>3</sub> genotypes may be highly selected in the growing season as a consequence of chemical selective pressure, in the autumn and winter they tend to be selected out of the population and decline in frequency, as seen from their reduced occurrence in 12.2 m high suction trap samples (Foster *et al.*, 2002). Their frequency is thus generally very much reduced the following spring of any given year (see Foster & Devonshire, 2007 for a review). The frequency of the various S and R genotypes is found to fluctuate at various sites in the U.K. during the growing season (Foster *et al.*, 2000, 2002), a situation probably reflecting selection in relation to changing patterns of insecticide usage and fitness costs associated with insecticide resistance. It may also reflect the fact that highly resistant asexual lineages of *M. persicae* have linked genomes bringing about non-random associations between the resistance mechanisms and selection operating on asexual lineages (Foster *et al.*, 2002). Hence when pesticide selection pressure is reduced, the resistant geno-phenotypes may be otherwise maladapted (Foster *et al.*, 1998). Furthermore, in Scottish population samples, resistant genotypes (R, MACE and *kdr*), arbitrarily linked with microsatellites at a number of loci, are seen to fluctuate within natural populations, although why some lineages associated with MACE/*kdr* should have declined extensively in sites sampled since 2003 is a mystery, but may be related to selection affecting other aspects of the geno-phenotype as earlier discussed (Fenton *et al.*, 2005; Kaspruwicz, 2006).

As also mentioned earlier, evidence has been found for latitudinally-dependent clinal changes in aphid nuclear gene frequencies in turn related to climate and life cycle (e.g. Simon *et al.*, 1999; Llewellyn *et al.*, 2003). Similar clines have also been observed in French populations of *R. padi* in relation to non-recombinant mtDNA haplotypes (i.e. haplotype 1), monitored in populations sampled from south-west to northern France (Martinez-Torres *et al.*, 1997). At a smaller geographic scale, Llewellyn *et al.* (2004) found evidence for



Figure 9. An apterous adult peach-potato aphid, *Myzus persicae*, with her progeny. This species has evolved resistance to insecticides in many places around the world, including the U.K. Here it comprises several genotypes which range from susceptible to highly resistant. The nature of the resistance in this species is providing important insights not only into the biochemical and genetic mechanism/s of resistance, but also how these mechanisms evolve and are maintained – or not – in the field when subject to selection (insecticide selective pressure as well as natural selection).

clonal/lineage selection of *S. avenae* multilocus microsatellite genotypes in wheat fields in southern England, possibly related to host plant cultivar, which is not surprising considering the other clear evidence for host plant preference in *Sitobion* aphids (e.g. De Barro *et al.*, 1995a,b; Haack *et al.*, 2000; Lushai *et al.*, 2002). Similarly, Vorburger (2006) has found evidence for some degree of host selection in predominantly asexual populations (i.e. particular microsatellite multilocus genotypes) of *M. persicae* in Australia on some hosts (e.g. *Solanum physalifolium*), as has Kasprowicz (2006) for the same species in Scotland on *Brassicas*. Sunnucks *et al.* (1998) also provide evidence that chromosomal changes (rearrangements) may be associated with host plant preference/adaptation in aphids of the genus *Sitobion*. Whether GPGs truly exist in aphids is debatable, but my view is that since the broad thrust of evolution is mainly towards specialisation of one form or another, probably they don't (but see Van Doninck *et al.*, 2002 in the case of ancient asexual ostracods). Empirical host plant performance studies by Vorburger *et al.* (2003), involving different life cycle forms, seemingly deny the existence of GPG's. For sure, linkage of the loci of the asexual aphid genome would, on theoretical grounds at least, make the idea of GPGs seem unlikely.

#### Was Lamarck partially right all along?

It is well established that cells in the animal body communicate using chemical signals (and more recently, it has been discovered that they also do so via tiny cell protrusions; Rustom *et al.*, 2004) and that all sorts of scenarios are thereby effected during development, wound healing, etc., including by apoptosis, the normal series of events that lead to cell death and sometimes replacement. Cancer cells avoid apoptosis (Krauss, 2003). It is therefore perhaps not surprising that it has now been documented that chemicals in the environment directly or indirectly affect the switching on and off of genes and therefore regulate development and/or behaviour. After all, animals and plants have lived within ecological communities and indeed have had 'ecologies', perhaps for many millions of years, and just as 'no man is an island, entire of itself' (Donne, 1624), nor seemingly is an aphid. Jean-Baptiste Lamarck's (1744-1829) ideas of genetic changes being passed from one generation to another ('inheritance of acquired characteristics'; see Browne, 1996) seemed ludicrous to earlier generations of biologists and probably still do to many today. But the genome is now acknowledged to be a much more dynamic system than hitherto envisaged, even 10-15 years

ago, possessing as it does a plethora of mutational mechanisms known to influence gene sequence and even order (e.g. inversion polymorphisms related to transposon-rich 'hotspots'; see Lushai *et al.*, 2003), the latter perhaps being a principal mechanism in sympatric speciation of insects like *Drosophila* (Ortiz-Barrientos *et al.*, 2002).

Whether or not rearrangement of the actual sequence order (by mutational processes of one form or another and still open to selection) are widespread among insect species (see for example, Al-Barrak *et al.*, 2004 and references therein), certainly chemicals or other stimuli (i.e. mechanical) passed between individuals may, and in some instances certainly do, trigger phenotypic changes, including gross changes – for example, phase changes in desert locusts, *Schistocerca gregaria* Forskål (Orthoptera: Acrididae) (Ferenz & Seidelmann, 2003), presumably by affecting gene expression (see Kang *et al.*, 2004). These changes of course have generational consequences, probably not evolutionary ones as such, although they may do in some insect species and in certain ecological-environmental-physiological scenarios, perhaps by a process of genetic accommodation (Braendle & Flatt, 2006).

Aphid crowding elicits contact/tactile stimuli via



Fig. 10a,b. Darwin and Lamarck. Darwin's views of *natural selection* as being the main mechanism of evolution are generally widely accepted and Lamarck's views on *inheritance of acquired characters* largely discredited, certainly in the 19th and 20th centuries. However, both lived many years before even the discovery of the gene (in 1910 by Thomas Hunt Morgan), let alone our modern appreciation of the 'dynamic genome' recently discovered using molecular biological techniques. It now appears that the physical environment of a living organism does indeed impact on the genome, certainly on the expression of genes (e.g. by epigenetic means) – and can lead to so-called 'transgenerational effects', as seen in aphids and other organisms.

mechanoreceptors on the antennae and legs which triggers development of winged forms, but only if one or more insects jostle others (Lees, 1967). Crowding causes the next generation to become winged in some species, e.g. vetch aphid, *Megoura viciae* (Buckton) and the pea aphid, *A. pisum*, and the generation after as winged adults (3<sup>rd</sup> generation) are most reluctant to produce winged offspring (to some researchers, a transgenerational effect, to others, a maternal effect). In other aphid species, crowding of mothers and offspring is especially important and crowding can act on early stage nymphs to effect wing development in an individual (Jim Hardie, pers. comm.). Similarly, the release of alarm pheromone [(E)-]-farnesene, EBF], which has structural similarities with JH and is synthesized *de novo* in a feedback loop with this compound (Mondor & Roitberg, 2003), can cause species-dependent transgenerational changes promoting wing development in aphid colonies subject to predation (parasitoids or predators; Kunert *et al.*, 2005;

Podjasek *et al.*, 2005). Perhaps EBF does this by acting as a competitive JH binding site mimic (blocker), thereby reducing the titre of JH in the haemolymph and promoting alatform production rather than apterisation of aphid larvae (Lees, 1980; Hardie, 1980), although this is disputed as being unlikely (J. Hardie and W.W. Weisser, pers. comm.). An alternative explanation is that EBF in effect causes 'pseudo-crowding', and that mechanical stimulation between individuals 'induces groups of aphids but not single individuals to produce a higher proportion of winged forms among their offspring' (Kunert *et al.*, 2005). Lastly, as a further example of transgenerational influences, when highly resistant *M. persicae* are no longer subject to intense chemical insecticide selective pressure, they revert to a lower level of esterase (E4) expression (Sawicki *et al.*, 1980; French-Constant *et al.*, 1988), even though their genome is unaffected in terms of E4 amplicon number, as far as is known (Hick *et al.*, 1996; Field *et al.*, 1999). This reversion, due to epigenetic changes of the genome

(unlike mammals where DNA methylation switches genes off, here the E4 genes are switched on by such changes; Hick *et al.*, 1996), occurs within *just a few generations* (Sawicki *et al.*, 1980). However, for these generations at least, the highly resistant offspring express the phenotype of their forebears.

There is recent additional evidence in eukaryotes that stress on one generation may have transgenerational effects, such as ageing, increased incidence of cancers and other diseases, involving chromosome telomeres and associated phenomena (Monaghan & Haussmann, 2006; see also Pembrey *et al.*, 2006 in the case of humans) This may be equated with a kind of 'near-Lamarckism' at least, if *not* actually the 'real thing' (Lushai *et al.*, 2003). What is clear is that aphids prove to be exceptionally good experimental animals on which to study the effects of life cycle strategy on population genetics, indeed genetics in general, and this is likely to be true for transgenerational phenomena also. I am confident that

as the new century further unfolds, many more examples of such transgenerational changes will be recorded, both via the parthenogenetic (apomictic egg) as well as sexual (sperm/egg) germ lines, causing a revision of our view on the different mechanisms of evolution. Thus direct selection, although undoubtedly the major driving force of organic evolution and change, is not the only force in terms of genotypic/phenotypic change between generations, certainly in the short term; that is, a few generations.

### Concluding remarks

I hope that I have demonstrated that even a single female aphid is an evolutionary force to be reckoned with and that besides defying many of the current beliefs and concerns about the importance of inbreeding, mutational meltdown, and lack of sex in many species, not to mention

definitions of 'what constitutes a population', nevertheless causes us to rethink our position on these issues. All that can be said is that regardless of our textbooks, aphids 'do it their way' and as such, continue to be a thriving and successful group of organisms, having conquered most of the biomes, including the arctic (Strathdee *et al.*, 1993) and sub-antarctic regions (e.g. Hullé *et al.*, 2003), being found far out at sea (Hardy & Cheng, 1986) and on mountain tops (Bauer, 2002) and infesting a huge range of plants - over 40 plant families worldwide in the case of the ubiquitous and highly polyphagous *M. persicae* (Blackman & Eastop, 2000). They are a truly ancient group and from their past and current performance, are highly likely to be around for a very long time to come, despite our best efforts, and using our most sophisticated technology, to combat them. The recent evolution of insecticide

resistant forms in several species worldwide, notably *M. persicae* and involving cross-resistance mechanisms, shows that, contrary to what people would like to believe, evolution via natural selection – and perhaps to a much lesser extent, other mechanisms too – doesn't stand still. Whilst the great race of life continues, living organisms, including aphids, pick up the challenge and run – or fly – with it for all it is worth. They must; they have no choice otherwise.... except extinction.

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