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PUBLISHED: 3 APRIL 2017 | VOLUME: 1 | ARTICLE NUMBER: 0130

ECOLOGY

From genes to metapopulations

A tribute to Ilkka Hanski. Empirical data modelling shows that molecular variation at a candidate gene within populations has consequences for metapopulation size and persistence.

Michel Baguette, Radika Michniewicz and Virginie M. Stevens

Metapopulation theory[1,2] aims to predict if and how a species will either cope with the threats that follow habitat destruction and fragmentation, or will become extinct. Predictions such as these are difficult to make as dispersal between patches of fragmented habitat is under strong evolutionary pressure from factors such as landscape structure and configuration. In a recent study in *Nature Communications*[3], the late Ilkka Hanski and his colleagues of the Metapopulation Research Centre (University of Helsinki, Finland) provide empirical evidence that allelic variation in a gene associated with dispersal correlates with population colonization and extinction turnover within metapopulations.

Within a landscape, individuals of a species are restricted to areas of habitat where there are enough resources for them to complete their life cycle. This landscape may therefore be viewed as a patchwork of suitable habitats embedded within an area of unsuitable habitat, or matrix. If an individual is forced to leave its current habitat patch because of pressures such as competition, lack of resources or predation, it has to disperse across the matrix and is therefore exposed to risks that may result in death. However, the dispersing individual may have the reward of arriving at a new patch with better conditions. The overall result of this dispersal is that local populations are not isolated but are in fact connected. These groups of local populations that are connected by dispersal are called metapopulations. Metapopulations have properties that the local populations do not possess alone. First, the persistence of metapopulations is more stable than that of local populations. This is because extinction

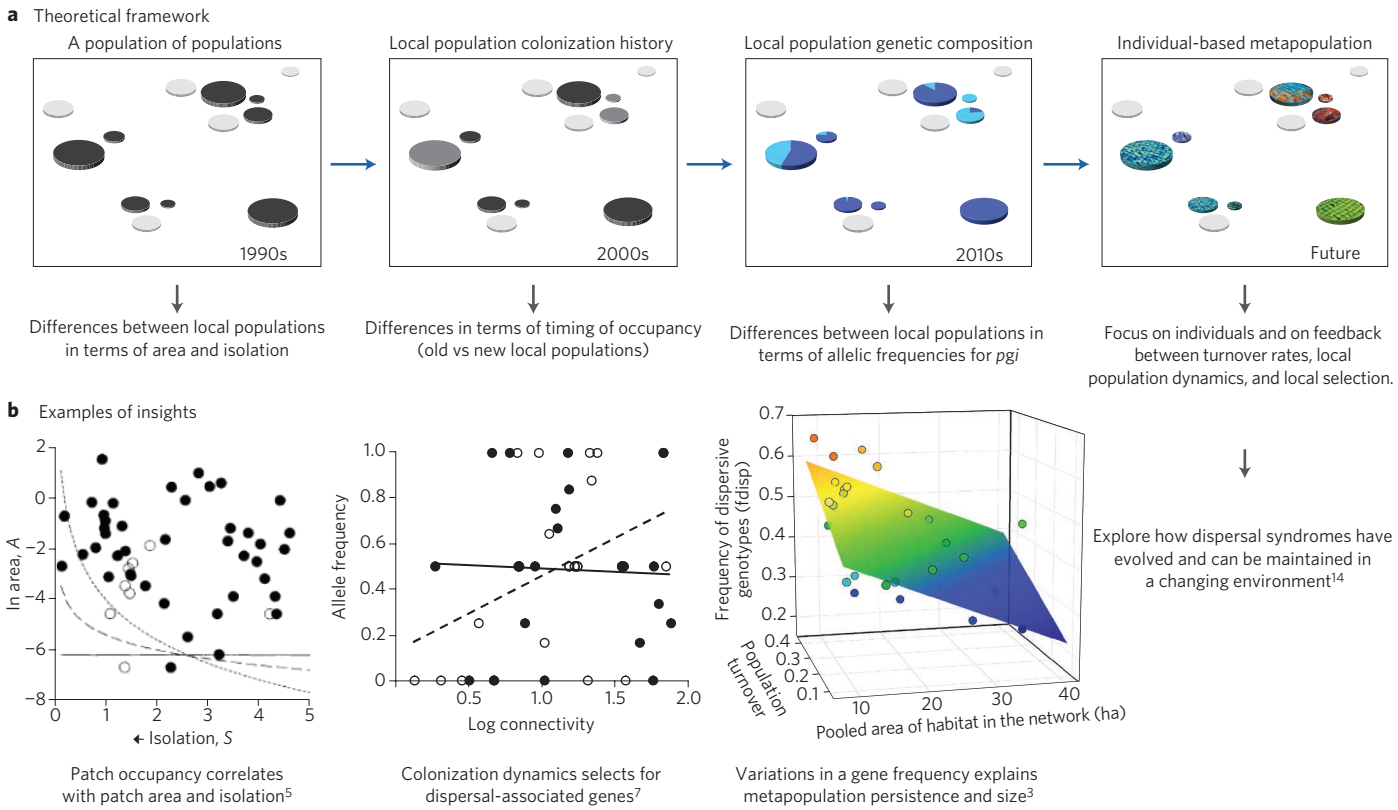

Figure 1 | Conceptual changes in metapopulation biology under the impulsion of Ilkka Hanski and some associated empirical insights. **a**, A metapopulation in which patch colours reflect the level of complexity considered: from a binary view in the 1990s (occupied versus unoccupied patches), through the inclusion of colonization history in the 2000s (new populations in grey; old in black) and of genetic variation in the 2010s (pie charts illustrate allelic frequencies for one gene), to the emergence of an individual-based metapopulation (phenotypic and genotypic differences among individuals are illustrated by colour scales). **b**, Associated empirical insights; for more detail see the original publications. Charts in panel **b** reproduced with permission from ref. [5], British Ecological Society (left); ref. [7], Royal Society (middle); and ref. [3], Macmillan Publishers Ltd (right).

of local populations can be counter-balanced by the creation of new ones (colonization) elsewhere in the landscape following successful dispersal events. Second, the dynamic nature of a metapopulation (that is, gene flow associated with dispersal and local population turnover) contributes to its genetic structure and diversity, and hence to its evolutionary trajectory. This idea was formalized for the first time by Richard Levins[4] in a spatially implicit model, and the metapopulation concept was expanded under the prominent influence of Ilkka Hanski[1,2] (Fig. 1).

The brilliant contribution of Hanski[5] was to model population turnover using 'incidence functions'[6]. These predict the fraction of occupied patches in a landscape from the properties that affect extinction and colonization probability, such as patch area and isolation. Small or isolated patches have higher extinction and lower colonization probabilities. The most emblematic empirical study of classical metapopulations is the long-term monitoring of the butterfly *Melitaea cinxia* metapopulations, studied by Hanski and colleagues on the Åland Islands in Finland. Besides the empirical validation of the classical metapopulation model, this system provided many insights into the mechanisms driving metapopulation dynamics. A crucial advance was to link variation in dispersal rates to allelic variation in a metabolic enzyme (for example, that responsible for phosphoglucose isomerase[7], Pgi), and to disentangle the selective processes that maintain this variation[8,9]. These studies suggest that extinction and colonization turnover generate molecular changes that might impact metapopulation dynamics by affecting dispersal[10].

In their paper in *Nature Communications*[3], Hanski and colleagues build on classical metapopulation theory to test the predictive power of a spatially realistic model aimed at predicting the capacity of landscapes to support viable metapopulations[11]. Their innovation is to add habitat quality and the effects of allelic variation of the *Pgi* gene on metapopulation size and dynamics. They test the model using an impressive 22-year empirical data set consisting of 66,527 butterfly presence/absence records in 4,415 habitat patches distributed throughout 125 metapopulations.

When habitat patch isolation, area and quality are considered, the model explained 40% of the variation in the annual weighted average of patch occupancies. Importantly, 33% of the metapopulations had colonization/extinction ratios above an extinction threshold and the model predicted the annual weighted average of patch occupancy reasonably well. Counter-intuitively, many of the remaining 67% of threatened metapopulations that were below the extinction threshold were densely populated, albeit transiently. This finding nicely demonstrated that dispersal plays a crucial role in metapopulation dynamics, not only locally but also globally.

The authors then turned to the molecular basis of dispersal. Investigating metapopulations above the extinction threshold, Hanski and colleagues found that the frequency of dispersive genotypes correlated positively with population turnover and negatively with the total habitat area in the patch network. This result is consistent with the idea that colonization selects for individuals with high dispersal capacity. Moreover, there was a positive relationship between the frequency of dispersive genotypes and both metapopulation size and persistence. Hanski *et al.*[3] emphasize that genetic and demographic changes are closely coupled: a high frequency of dispersive genotypes increases colonizations and decreases extinctions, while population turnover selects for increased frequency of dispersive genotypes within networks.

This elegant modelling approach provides a snapshot of the end product of the multiple and complex selective pressures acting on dispersal within metapopulations. However, it is likely that there is huge heterogeneity in dispersal behaviours across space and time, and among individuals. Individual dispersal strategies coevolve with variations in other phenotypic traits. Selective pressures acting on dispersal, like habitat fragmentation, could thus entail modifications of these other traits[12]. The accurate assessment, interpretation and extrapolation of such dispersal syndromes are currently hindered by the strong covariations among traits and their unresolved genetic architecture[13]. To fully consider covariation between dispersal and other traits, a recent research agenda has proposed an individual-based approach integrating genomic, transcriptomic, epigenomic, proteomic and metabolic data to focus on the mechanistic understanding of the generation, maintenance and evolution of dispersal syndromes in metapopulations[14], using a framework designed to uncover complex phenotype–genotype interactions.

Hanski *et al.*[3] highlight the role of eco-evolutionary dynamics of dispersal in metapopulation persistence by focusing on mean allelic frequencies in a single candidate gene within local populations. Even in the absence of its founder[15], and owing to the insightful instincts of Ilkka Hanski, metapopulation biology will continue to develop (Fig. 1) by adopting a shift from a population to an individual focus. ❐

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Acknowledgements

The authors acknowledge financial support from the French National Research Agency (ANR) programmes open call INDHET (ANR-12 -BSV7-0023-02) and young researcher GEMS (ANR-13-JSV7-0010-01). The authors are part of the 'Laboratoire d'Excellence' (LABEX) entitled TULIP (ANR-10-LABX-41).

Competing interests

The authors declare no competing financial interests.