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COMMENTARY

Population bottlenecks: the importance of looking beyond genetics

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Hale & Briskie's (2007) study addresses an age-old conservation question: to what extent do population bottlenecks influence fitness-relevant traits such as pathogen resistance? In the age of high-throughput molecular techniques, genetic signatures of population size reductions are unveiled with increasing frequency and ease. However, the question most relevant to conservation biology of whether reductions in genetic variation actually matter to fitness remains challenging to document even in the most opportune study systems.

From all standpoints, Hale & Briskie's (2007) New Zealand robin study system is ideally suited for this question. The demographic history of these island species has been unquestionably documented because individuals were physically translocated from one island to another. The two island populations compared in this study consist of a source population (Nukuwaiata island) and its introduced descendants (Motuara island) known to be founded by five individuals 33 years ago. This human-induced bottleneck created the ideal natural experiment to examine how a recent population bottleneck influences immunocompetence and pathogen susceptibility. The experiment is even in large part controlled: both populations live in a similar habitat and on small islands of comparable size (both <200 ha). Finally, the basic assumptions underlying the influence of a bottleneck on immune responses and parasite load have already been met: both neutral microsatellite variation and functional MHC genetic variation are lower in the recently founded Motuara island population (Miller & Lambert, 2004), and reduced reproductive success consistent with inbreeding effects has been documented on Motuara (Mackintosh & Briskie, 2005). Altogether, Hale & Briskie (2007) have a robust natural experiment for testing the effect of a recent bottleneck that reduced genetic variation on immune responses and parasite loads. Although the study system is straightforward, the results raise a number of interpretational challenges that highlight the difficulty of studies such as these in even the most ideal ecological systems.

One of the obvious strengths of Hale & Briskie's (2007) study is the inclusion of parasite loads in addition to simply measuring immunity via standardized assays. Immune assays provide one measure of overall 'health' or, more specifically, the likelihood of resisting a subset of parasites. However, the ultimate currency of fitness is parasite resistance *per se*, and immune assays more frequently than not show little direct correlation with resistance to relevant parasites (reviewed in Adamo, 2004). This discrepancy may explain why Hale & Briskie (2007) find some evidence for a reduced immune response in the bottlenecked population on Motuara, and yet no detectable differences in parasite load. However, there are several additional explanations for these inconsistent patterns that Hale & Briskie (2007) fail to consider. First, the detected immunosuppression on Motuara (which, by one measure, was only seasonal) simply may not be sufficiently strong to cause meaningful differences in parasite load. Second, the environmental context may have masked any effect on parasite load that otherwise would have been evident. Even if Motuara island robins are significantly more immunosuppressed than their source island counterparts, this effect will only be apparent in the presence of relevant parasite selection pressures. Islands are presumed to harbor lower parasite loads, and this is consistent with what Hale & Briskie (2007) find: both study islands were essentially depauperate of parasites (i.e. only four coccidial loads were detected across 146 fecal analyses). This left the authors with sufficient comparative data for ectoparasites alone, which are the subset of parasites least likely to respond to subtle differences in the immune system. The fitness effects of immunosuppression necessarily depend on the intensity of parasite threats in the environment, which may not be sufficient on these isolated islands in the south Pacific. Incorporating environmental context into studies of bottlenecks and fitness reductions is imperative. The question must not be simply *whether* population bottlenecks influence fitness-relevant traits such as parasite loads, but *when* and *where*? More studies such as Hale & Briskie's (2007) need to be carried out on demographically characterized systems in order to address this question within and

across taxa. Systems with multiple independent replicates of introduction events would be ideal in order to confirm the true generality of these effects. Until fitness-relevant effects as a result of bottlenecks can be robustly documented, conservation biologists would be well served by maintaining a healthy skepticism in their interpretation of genetic data alone.

Finally, as elegantly argued by Caro & Laurenson (1994), other explanations need to be considered for why endangered or small populations have reduced fitness, even if genetic differences have already been documented. In particular, the seasonal nature of the detected immunosuppression in Hale & Briskie's (2007) study calls into question whether genetic differences are the culprit, as the effects of reduced genetic variation should be evident year-round. Instead, the immunosuppression of Motuara island robins during autumn could result from lower general condition or food limitation due to the higher breeding densities on Motuara (Mackintosh & Briskie, 2005) rather than direct effects of genetic variability. Overall, direct links are sorely needed between population size reductions, loss of genetic variability and fitness-relevant traits such as immunocompetence and parasite resistance. Population bottlenecks will always remain a significant conservation concern, but we need to move beyond genetics in order to understand why.

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