to the small, panmietic population (Fig. 2), demonstrating that the internal genetic structure itself plays a disproportionate role. As the possibility of detecting migrants increases following fragmentation, so too does the possibility of statistically detecting the presence of the barrier itself. Before the populations were fragmented, individuals had genotypes that could be assigned to eith the same side of the barrier or the opposite side, with equal likelihood resulting in most comparisons being non-significant (Table 2). Shortly after fragmentation, however individuals' genotypes were much more likely on the side of the barrier from which the individual was sampled than from the other side, resulting in statistically significant Ven Astrutto incremed values for the paired t-test. In all populations, the number of simulations in which a barrier was defected (even if none were present; type I errors) was higher than expected given a for this test was either 0.05 or 0.0001. This was particularly so for structured independent. Type I errors (detecting a barrier when none were present) were redu increasing the separation between paired sampling sites. Increasing the separation biased the result against detecting the barrier, reducing the number of type I errors to 0 with a separation between sampling sites of only 1 neighborhood (Table 3). However, this came at the cost of increasing the time until barriers could be reliably detected to approximately 35 generations. We found that the proportion of Type I errors in structured populations could be reduced to approximately 5% if the critical value of the t-test was increased such that p < 0.0001 (Tables 2 and 3).