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Letter

Comments on T. De Meeûs *et al.*'s Article

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Control programmes against vector-borne diseases often aim to stop pathogen transmission by increasing vector mortality. This may be compromised if other parameters governing population dynamics change as populations decline. Quantifying possible effects of reduced vector density, when populations are sparse, is however difficult because insufficient individuals can be sampled. As De Meeûs *et al.* [1] propose, population genetics could provide insights in this area. From an analysis of published data, they conclude that the dispersal of tsetse flies increases ~100-fold with population reduction, thereby threatening the success of trypanosomiasis-control operations.

Evidence for the authors' claim is presented in their Figure 1, involving a regression of dispersal distance against population density. These results do not, however, prove that reduced density will cause increased dispersal during control of any given tsetse species. The data are from multiple species and locations and from populations not subject to vector control.

Differences in dispersal rates between species exist, relating to fly size, ecological niche, and host-seeking strategies [2]. Analysis including multiple species would therefore need to consider these factors. Even if adequate data were available for a single species in a single location, we ask the authors to provide a plausible hypothesis for the mechanism behind an increase in dispersal with increased mortality rates in a control operation.

Under natural conditions, tsetse populations of lower density may have relatively high dispersal rates, but this would not be the case for a population reduced by control efforts. In areas of contiguous habitat and high density of hosts, flies will not have to move far to find food and a suitable microclimate, resulting in low dispersal and high density. The opposite will be true in areas of more fragmented habitat. Dispersal and density could therefore be correlated, explaining the observation for a given species in Figure 1, but there is no evidence that one causes the other and indeed, no evidence that artificially induced mortality would increase dispersal.

In Zimbabwe, the rate at which female *Glossina pallidipes* and *Glossina morsitans* moved from high-density areas into a block subject to aerial application of insecticide was ~800 m/day [3] – consistent with the high mobility of marked flies in uncontrolled populations nearby. If we use the regression presented in Figure 1, the daily displacement of a very sparse population would be ~100 times greater, at ~80 km/day, a rate incompatible with the fly's energy budget [4].

Van Sickle and Phelps [5] demonstrated that sustained and extensive killing of adult tsetse flies reduced the mean age of the residual population, especially by a several-fold enhancement of the proportion of flies that are around 1 week old. Given that the flight capacity of young tsetse flies is limited [6], daily dispersal will therefore tend to decline, not increase, when control reduces the population density. The poor mobility of young populations must decrease the efficacy with which they encounter killing devices, but this hardly matters because young populations cannot prosper, since females <2 weeks old do not reproduce.

The authors state that Box 3 shows that a negative density-dependent dispersal has long been known in tsetse flies. Box 3, however, argues that, as density declines, the flies can feed more readily, implying that it is not necessary to travel so far to find a host and thus tending to reduce dispersal. The authors also state that, in areas of high tsetse density, immigrants have low feeding success and high mortality, but no references are provided to support this claim. Moreover, incongruously, the authors also imply that immigrants become 'safely settled' in sparse populations sustained by invasion.

Even if we did allow that dispersal increases by two orders of magnitude as control proceeds, we could still not accept that this threatens the success of operations. Indeed, control would become more successful since the residual population would emigrate from the operational area at increased rates, and those flies that did not depart would have a greatly enhanced rate of encountering killing devices. Furthermore, if the proportion of emigrating flies was indeed relatively high in any residual population in the controlled area, the fact that the population density there has been reduced substantially would mean that the absolute number of flies going to the uncontrolled areas nearby would tend to increase but little, if at all, and hence would be of minor concern to vector-control managers.

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Letter

Response to the Comments of J.S. Lord

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We thank Dr Lord for her letter [1] that presents criticism on some of the ideas we proposed in [2]. In the following, we answer the different points raised by Dr Lord.

Saying that dispersal of tsetse flies increases ~100-fold with population reduction represents a distortion of what we demonstrated. We showed that populations of tsetse flies with high densities disperse at much smaller distances than populations with low densities. In our graph, the 100-fold dispersal increase raised by Dr Lord would mean a 10^5-fold density decrease, which would be hardly observable.

We agree with Dr Lord that dispersal is species-specific. In our figure, the same species is present more than once. Some species disperse more than others at the same density, or display different densities, but the correlation remains strong. From there, we predict that any other data set of tsetse flies should fit into our graphic. This is the case for a recent paper [3] (density = 169 flies/km^2 and dispersal = 1.12 km/generation, fitting in the middle of our graphic). The population genetics parameters we used were measured in populations in relative migration–mutation–drift equilibrium. Any evaluation undertaken just after treatment would either have provided the same parameters, if no immigration occurred (survivors represent a subsample of the same population), or a change in these parameters in case of recolonization from neighboring sites. The second case seems to describe best what occurred in [3]. Measuring an effect of treatment on tsetse dispersal (as we defined it) would require a follow up of the same sites during several years of continuous control to let the population adjust, or come back several years after a one-time treatment, to check if the population displays again the same parameters as before treatment. Such data seem unavailable to date.

Dr Lord asked us to provide ‘a plausible hypothesis for the mechanism behind an increase in dispersal with increased mortality rates’. We did not deal with mortality rate but with increased mortality rate of tsetse flies immigrating in sites with high tsetse densities. We provided arguments for this in our Box 3 and proposed a coevolution model in our main text.

Dr Lord insists on the idea that higher host densities may explain smaller dispersal and higher tsetse fly densities. According to [4], Serengeti National Park probably harbors one of the highest densities of big mammals. However, it displayed the smallest *Glossina* effective population density, and the largest dispersal of our study. At least for this site, host density does not allow a good prediction of tsetse density and dispersal. Besides hosts densities, tsetse presence and densities strongly rely on macroclimatic parameters such as temperature [5–7].

Applying the daily dispersal distance of [8] to our graph, as Dr Lord suggests, is inappropriate since our dispersal distances correspond to the average distance between reproducing adults and their parents of wild and unmanipulated flies, which was not the case in [7]. Tsetse flies both die and disperse faster when released in an unsuitable than in a suitable habitat (J. Bouyer, PhD thesis, Université Montpellier II, 2006, https://www.researchgate.net/publication/334684650_Ecologie_des_glossines_du_Mouhoun_au_Burkina_Faso_interet_pour_l’epidemiologie_et_le_controle_des_trypanosomoses_africaines).

The comment of Dr Lord on our Box 3, and lack of references, is inaccurate. Three references were provided where negative density dependence is discussed. The sentence ‘incongruously,…immigrants become ‘safely settled’ in sparse populations sustained by invasion’ is particularly unfair since we never stated such a thing. What we discussed was the possibility that, in sites with low tsetse densities, with untrained hosts, immigrants, even those coming from the most remote sites, can still have a good chance of a successful blood meal.

The last paragraph of Dr Lord’s letter confirms a misunderstanding of our paper. Our paper dealt with evolutionary ecology parameters that evolved in