COMMENTARY

Gregarious locusts down-regulate muscular catabolic capacities yet fly far

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Locust swarms continue to threaten food security on multiple continents, and our understanding of the basic biology and ecology of locusts remains a limiting factor in our capacities to develop new strategies for management. One of the most striking aspects of locust biology is the still poorly understood phenotypic transformation from a solitarious, nonswarming form to a gregarious, swarm-migrating form (1). Du et al.'s publication (2) in PNAS provides important insights into the mechanisms and consequences of this transformation, with insights relevant to anyone interested in performance trade-offs between speed and endurance.

Gregarious locusts are well known for their capacities to fly for many hours, enabling their dispersal across continents. However, solitarious locusts also fly, both to escape predators and to perform shorter-distance solitary migrations (3). Prior studies have shown that gregarious locusts are more prone to fly (4). We have known that these differences in flight behaviors are partially enabled by higher lipid reserves and hemolymph lipid levels in the gregarious forms and more rapid and stronger activation of adipokinetic hormone signaling (5). However, how the flight muscles of solitarious and gregarious locusts differ has been almost unstudied.

Based on studies of the flight muscles of migrating bats (6) and birds (7, 8), the obvious starting hypothesis is that the long-flight performances of gregarious locusts would be enabled by a higher capacity for oxidative energy metabolism. Du et al. (2) find the opposite. Gregarious forms have lower levels of transcripts and maximal activities for a large number of enzymes involved in oxidative catabolism. Also surprisingly, gregarious forms had slower maximal speeds in tethered flight and had lower average speeds during the first 15 min of flight; however, in a 10-h test, the gregarious forms spent much more time flying on the flight mill and traveled much farther. Thus, gregarious locusts have greater flight endurance, despite what is likely a lower maximal aerobic capacity.

The lower flight durations and distances of the solitarious locusts could be attributed to motivational differences. However, Du et al. (2) demonstrate that solitarious, but not gregarious, locusts show evidence of redox disruption during flight, a pattern increasingly linked with muscle fatigue. After 1 h of flight, solitarious, but not gregarious, locusts showed an increase in muscle hydrogen peroxide levels and decreases in the redox scavengers xanthine, hypoxanthine, uric acid, inosinic acid, and glutathione. These data provide strong evidence that some processes in solitarious flight muscle quickly lead to excessive reactive oxygen species (ROS) production, which is avoided in the flight muscles of gregarious locusts. Plausibly this elevated ROS production could be due to the higher maximal speeds exhibited by the solitarious locusts in the first 15 min of flight, as ROS production has been demonstrated to be positively correlated with aerobic exercise intensity in vertebrate muscle. Du et al. (2) hypothesize that the solitarious forms are adapted for high initial flight performance as they are likely to depend on rapid acceleration for escape from predators, perhaps a less strong selective force for gregarious forms.

These data provide interesting insights relevant to understanding trade-offs between velocity and endurance in the animal kingdom. In vertebrates, greater endurance is almost always correlated with higher maximal aerobic capacity of muscle, but burst performance is associated with higher anerobic capacities. Insect flight muscle is completely aerobic and so lacks an anaerobic-powered burst, which may explain this uncoupling of aerobic capacity and endurance.

Most of these experiments were conducted with locusts maintained for generations under crowded or solitary conditions, but Du et al. (2) also demonstrate that many of the same transcriptional, protein, ROS-scavenger, and behavioral patterns arose in gregarious and solitarious

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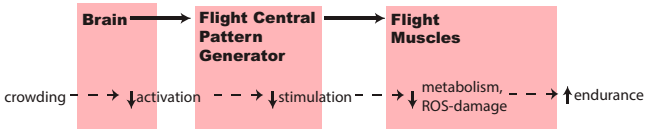

Fig. 1. Flight velocity depends on modulation of the central pattern generator for flight by the brain. Du et al. have demonstrated that gregarious locusts fly with a reduced velocity and flight muscles with lower capacities for catabolic catabolism, yielding reduced ROS disruption and better endurance (2).

locusts switched and reared in solitary or crowded conditions, respectively, for 2 wk as young adults. Prior research has shown that the transformation into the gregarious form is species specific but depends on subsets of mechanosensory, visual, and/or olfactory input (1, 9). These inputs are known to alter catecholamine signaling in the *Locusta* brain, with dopamine promoting gregariousness and serotonin activating solitariousness in this species (10). During flight the brain controls the flight central pattern generator, located in the thoracic ganglia, with muscarinic signaling, which can be modulated by octopamine, dopamine, tyramine, and histamine (11) (Fig. 1). Du et al.'s (2) data suggest that in gregarious forms flight is initiated and activated less strongly, perhaps with less stress-associated signaling, leading to reduced flight speeds. Acetylcholine signaling through nicotinic receptors has been postulated to be necessary for very-high-frequency flight in locusts, so plausibly such signaling is reduced in gregarious forms (11). Another important remaining question is how the transcriptional changes in flight muscles occur. Plausibly these could be controlled by circulating neurohormones such as the monoamines, neural activity patterns, or levels of juvenile hormone, which sometimes differ among locust phases (1).

Du et al. demonstrate that solitarious, but not gregarious, locusts show evidence of redox disruption during flight, a pattern increasingly linked with muscle fatigue.

Perhaps the most interesting question raised by the Du et al. (2) study is why down-regulation of oxidative catabolism reduces ROS damage and improves endurance. The reduced ROS damage may improve lifespan as well as endurance, as flight activity increases ROS damage and decreases lifespan in *Drosophila* (12, 13). Decreased ROS production could arise simply from lower flight intensities and metabolic rates, with the lower oxidative enzyme activities programmed in the flight muscles to match the reduced requirements for adenosine triphosphate generation. Alternatively, the reduced oxidative pathway activities might help ensure matching of oxygen supply to demand during flight. The observed ROS damage in solitarious fliers might arise from functional hypoxia occurring in the flight muscles due to oxygen demand outstripping supply, followed by ischemia-reperfusion injury (14). In the Glanville fritillary butterfly, prolonged, intense flight activity causes ROS damage, but such damage is reduced in butterflies with greater tracheation (15, 16). Oxidative damage also occurs in the flight muscle of migrating birds (17, 18) and in the leg muscles of undertrained human runners (19). Strategies to prevent ROS damage during locomotion may be key to successful endurance across the animal kingdom.

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