



Original Article

Maximum performance expression is affected by octopamine and antennae removal in *Acheta domesticus*

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Animals in nature seldom use their maximum performance abilities, likely in part due to context-dependent differences in performance motivation. Despite interest in the factors affecting performance expression, the physiological mechanisms underlying variation in performance motivation are poorly understood. We manipulated levels of the biogenic amine octopamine (OA) to test the hypothesis that OA drives motivation to express maximum bite force in male house crickets. We also tested the effect of antenna removal on bite force given prior evidence of potential links among antennaectomy, aggression, and OA. We found that administration of an OA antagonist, epinastine, significantly decreases realized maximum bite force, as does antenna removal. In addition, the performance decrement induced by antennaectomy is abolished by administration of excess OA, and that rescue effect is itself nullified by the simultaneous administration of epinastine. These data show that OA is an important mediator of performance in insects, and thus of performance motivation, and potentially a promising candidate for the short term manipulation of performance.

INTRODUCTION

The survival and fitness of animals is affected by their ability to conduct, on a daily basis, ecologically relevant, dynamic tasks such as running, flying, or biting (Arnold 1983; Irschick et al. 2008) that are referred to as whole-organism performance traits (Bennett and Huey 1990). Realized maximum performance is moderated, constrained, or otherwise influenced by a host of intrinsic and extrinsic factors ranging from sex (Cullum 1998; Lailvaux et al. 2003), age (Lailvaux et al. 2011; Hämäläinen et al. 2015), and immune status (Bonneaud et al. 2016; Husak et al. 2021), to season (Irschick et al. 2006), habitat characteristics (Irschick and Losos 1999), and predation risk (Bro-Jørgensen 2013). But while variation in the upper limit of performance capacities is well understood, our grasp of the extent to which animals choose to use those maximum capacities in certain ecological contexts is poor (Wilson and Husak 2015; Kasumovic and Seebacher 2018).

Performance researchers have recently realized that despite sometimes remarkable maximum capacities, animals in nature

seldom perform to their maximum performance limits (Wilson and Husak 2015). For example, Wilson et al. (2013) used accelerometry to show that free-ranging cheetahs in the wild never achieved their top speed during a series of recorded hunts. Potential factors driving the level of sub-maximal performance expression that animals do exhibit, termed ecological performance, include carryover effects into adulthood of exceptional juvenile performance capacities that compensate for small size (Carrier 1996); optimization strategies balancing the costs and benefits of maximum performance (Wilson et al. 2015); and buffering of selection which would otherwise act intensely on maximum performance if it were to be expressed at all times (Cespedes and Lailvaux 2015). However, few studies have considered the role of performance motivation in determining the context-sensitivity of performance expression.

Motivation in performance studies is seldom explicitly defined, but is generally used in the methodological sense of encouraging individuals to perform to their maximum limits (Losos et al. 2002). Performance motivation nonetheless likely has a biological basis, and there is mounting evidence that motivation can affect both the mean and variance of measured performance data. For example, sprint-trained *Anolis carolinensis* lizards exhibit clear physiological changes associated with enhanced sprinting but run no faster than

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untrained individuals in the laboratory, likely due to habituation to the researchers and training protocol and thus decreased motivation to perform maximally (Husak et al. 2015; Lailvaux et al. 2020). The behavioral state of an organism can also affect its level of expressed performance; male *Acheta domesticus* house crickets that lost a fight against another male cricket experienced a 20% decrease in measured maximum bite force compared to males that did not lose, despite no change in the underlying biting apparatus as a result of losing (Condon and Lailvaux 2016). Biogenic amines such as octopamine (hereafter OA), the invertebrate equivalent of norepinephrine, have been generally implicated in moderating aggressive behavior in invertebrates (Hoyer et al. 2008; Bubak et al. 2014) and depletion of OA specifically is associated with the decrease in aggression elicited by the recent experience of losing a fight in crickets (Stevenson et al. 2005). Collectively, these findings led Condon and Lailvaux (2016) to posit that motivation to perform maximally is affected by OA as well. Indeed, OA affects muscle contraction in other orthopteran species (Malamud et al. 1988; López-Pérez et al. 2021), and treatment with a synthetic OA receptor antagonist, epinastine, blocks flight in the cricket *Gryllus bimaculatus* (Stevenson et al. 2005). OA is thus a likely neuropharmacological candidate for mediating context-specific realized performance, and thus for affecting performance motivation.

We manipulated OA levels in male *A. domesticus* crickets to test the hypothesis that OA affects bite performance motivation. Specifically, we predicted that blocking OA signaling with epinastine will decrease maximum bite force. Furthermore, there is evidence from earlier studies that aggression is moderated by antenna removal in male crickets, such that antennectomized males are less likely to initiate fights compared to males with intact antenna (Sakura and Aonuma 2013). Previous studies have demonstrated a relationship between OA and the sensory neurons of insect antennae (Schendzielorz et al. 2015), suggesting that OA might also be diminished in antenna-less males. We therefore tested the secondary hypothesis that removing antenna will decrease maximum bite force in males, and the associated predictions that this effect can be abolished by supplementing antennectomized males with excess OA; and then restored again by supplementing antennectomized crickets with both OA and epinastine.

MATERIALS AND METHODS

All crickets used in this study were the second-generation offspring of *A. domesticus* crickets obtained from a commercial supplier (Fluker's). Males were raised separately to adulthood on a diet of dried cat food (Purina Cat Complete Chow). All experiments were conducted using crickets of similar ages (i.e. between 12 and 15 days post-eclosion). Following eclosion, we measured maximum bite force of all crickets using standard methods (Losos et al. 2002). Briefly, a Tekscan FlexiForce wireless ETF flexible force circuit was placed between the mandibles of a cricket which would then invariably bite down vigorously on the circuit. We measured bite force 5 times per individual and retained the largest of those five individual measures for analysis, consistent with both standard maximum performance methodology (Losos et al. 2002), and with comparable studies in crickets, including *A. domesticus* (Hall et al. 2010; Lailvaux et al. 2011; Condon and Lailvaux 2016). Following initial bite force measurement, we allocated crickets randomly to one of five groups: control; epinastine; antennectomized; antennectomized + OA; and antennectomized + OA + epinastine. Crickets then spent 2 h inside individual 5 × 5 × 5 cm plastic containers with either plain or

supplemented excess pureed sweetcorn (Bubak et al. 2013, 2014, 2015) under one of five conditions depending on their group membership. Control crickets ($n = 14$) were simply placed in a container with untreated pureed sweetcorn. The sweetcorn of epinastine crickets ($n = 15$) was supplemented with epinastine at a concentration of 15 mg/ml. Antennectomized crickets ($n = 18$) had both antennae removed with scissors following initial bite force measurement before being placed in a container with untreated pureed sweetcorn. Antennectomized + OA crickets ($n = 15$) had their antennae removed and were placed in a container containing pureed sweetcorn supplemented with OA at a concentration of 15 mg/ml. Antennectomized + OA + epinastine crickets ($n = 15$) had their antennae removed and were placed in a container with pureed sweetcorn supplemented with both OA and epinastine at the same concentrations as above. Following the 2-h treatments, we measured all crickets for maximum bite force again using exactly the same procedure as before.

All experimental methods complied with the national and institutional ethical guidelines where this work was conducted.

Statistical analysis

We used the *nlme* R package (Pinheiro et al. 2022) to fit a mixed-model with bite force as a dependent variable; treatment with the levels described above; measurement with the levels pre- and post-treatment as an independent variable; thorax size as a covariate to account for size effects; and cricket identity as a random factor because pre- and post-treatment bite force were measured on the same animals. We also included an interaction between treatment and measure to test our hypotheses which both pertain to significant differences between pre- and post-treatment bite force in certain treatment levels, but not others, and an interaction between size and measure to test for the possibility of size-dependent plasticity in bite force (Lailvaux et al. 2019). *P*-values associated with individual predictor variables are approximate due to penalty factors applied to random effects during calculation of the likelihood function. Consequently, we assessed the significance of predictors using log-likelihood ratio deletion tests to determine the minimum adequate model, which we then re-fit using restricted estimate maximum likelihood (REML; Silk et al. 2020). To test for specific differences, we used the *emmeans* package to extract contrasts comparing pre- and post-treatment bite force within each treatment level (Lenth 2019). We used R v 3.6.0 for all analyses (R Core Team 2019).

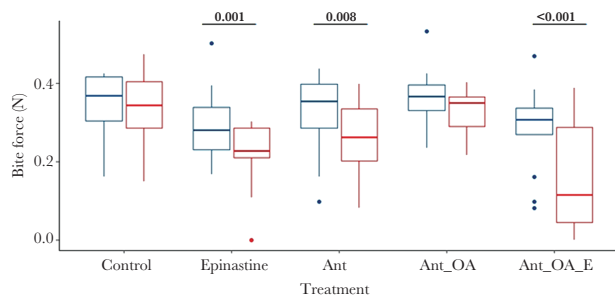
RESULTS

The minimum adequate model that best described bite force variation retained only the interaction between treatment and measure, as well as the respective main effects (see Engqvist 2005), such that some, but not all, treatment levels exhibited differences in pre- and post-treatment bite force (Table 1). The interaction between size and measure therefore did not explain a significant amount of variation in measured bite force. Contrasts showed that the significant differences corresponded to three of the five treatment levels: epinastine; antennectomized; and antennectomized but supplemented with both OA and epinastine (Figure 1). Thus, both epinastine and antennae removal significantly decrease maximum bite force measured in the same animals, but bite force is restored by administration of OA following antennae removal. However, this restoration effect is annulled by epinastine administration

Table 1

Best-fit model describing variation in bite force in male *A. domesticus*. The coefficients describe the estimated change in bite force between the baseline categories and the categories named in the table. The baseline category for treat is “antennae” (i.e. antennectomized), and the baseline category for measure is “post” (i.e. post-treatment).

d.f.	Coefficient	S.E.
Intercept	-0.43	0.17
Size	0.14	0.034
Treat (Antennae + OA)	0.062	0.031
Treat (Antennae + OA + E)	-0.089	0.031
Treat (control)	0.058	0.032
Treat (E)	-0.052	0.031
Measure (pre)	0.067	0.019
Treat (Antennae + OA): measure (pre)	-0.029	0.028
Treat (Antennae + OA + E): measure (Pre)	0.05	0.028
Treat (control): measure (pre)	-0.06	0.028
Treat (E): Measure (pre)	0.0013	0.028

**Figure 1**

Pre-treatment (blue) and post-treatment (red) bite force measures in male *A. domesticus* crickets. Solid lines indicate cases where bite force differed significantly from the initial measures following treatment administration, along with the associated *P*-values for the significant contrasts calculated from the mixed model using emmeans. “epinastine” = crickets treated with epinastine only; “ant” = antennectomized crickets; “ant_OA” = antennectomized crickets supplemented with OA; and “ant_OA_E” = antennectomized crickets supplemented with both OA and epinastine.

in antennae + OA + epinastine crickets. Collectively, these results point towards a prime role for OA depletion in particular in decreasing realized maximum bite force in male house crickets.

DISCUSSION

Variation in performance motivation is potentially linked to variation in the biogenic amine OA in crickets. We tested two different OA-linked interventions, namely blocking OA receptors and removing the antennae, to test the effect of OA depletion on maximum bite force in house crickets. Our first hypothesis, that OA affects bite performance motivation, was supported; epinastine treatment decreased maximum bite force by ~21% in the same animals compared to their maximum pre-treatment performance. This effect is due neither to the time course of the experimental design nor to fatigue associated with the earlier bite force measures, as untreated control animals subject to the same performance measurement regime exhibited no significant change in bite force. Our second hypothesis, that removing the antennae affects bite force, was also supported, and antennectomized males exhibited a significant decrease in bite force of similar magnitude to that of

the epinastine treated males in our dataset (~20%). Furthermore, performance decrement caused by antennae removal is abolished by OA supplementation, such that the pre- and post-treatment bite forces of antenna+ OA males were not significantly different, strongly suggesting that this performance-dampening effect of antennae removal is also driven by OA. Our final treatment provides additional evidence for this scenario, as the combination of antennae removal and epinastine administration caused an average bite force decrease of 50%, which was not ameliorated by the simultaneous application of OA. These striking changes in expressed maximum performance within the same individuals bearing the same underlying morphology implicates OA as a likely candidate underlying variation in performance motivation in invertebrates.

One potential mechanism driving the remarkably similar performance-decreasing effects of antennae removal and OA signaling blockage is the shared neural circuitry in the part of the insect brain called the mushroom body. Antennae are a vital part of the sensory apparatus in insects, and the antennal lobe in the cricket brain is closely associated with the mushroom body (Heisenberg 1998), which is itself linked with locomotor activity in insects (Huber 1960). In *Drosophila melanogaster*, for example, the mushroom bodies exert a suppressive effect on locomotion, such that ablation of the bodies increases locomotor activity (Martin et al. 1998). Direct connections between OA and the antennal sensory neurons exist in the hawkmoth *Manduca sexta* (Schendzielorz et al. 2015), and OA receptors are also expressed in the mushroom bodies of *A. domesticus* (Cayre et al. 1999), providing a direct, mechanistic link between both our neuropharmacological and physical interventions here. Sakura and Aonuma (2013) note that the lack of sensory input from antennectomized *G. bimaculatus* males could account for the reluctance of such males to initiate fights in their study, and Rillich and Stevenson (2015) showed that treatment with epinastine does not affect the efficacy of mechanical antennal stimulation to elicit aggressive mandible spreading in the same species. However, our results here indicate that both antennectomy and blockage of OA receptors with epinastine do affect expressed bite force in *A. domesticus*. Furthermore, an OA-mushroom body circuit has also been shown to modulate aversive memory formation in *Drosophila* (Wu et al. 2013), suggesting yet another possible link with the loser-effect-driven reduction in maximum bite performance reported by Condon and Lailvaux (2016). Although previous studies have focused on the locomotor implications of OA in particular, our results extend this purview to that of a non-locomotor performance trait, bite force. This suggests that any muscle-powered performance trait is potentially subject to the modulating effects of OA, and quite possibly also the mushroom bodies, in insects. Experiments that combine OA and/or antennae manipulation with measurement of bite force, a key predictor of fight outcomes in *A. domesticus* (Hall et al. 2010), within staged male combat trials would be useful for untangling the relationships among behavior, performance, and neuropharmacology in this species.

Despite considerable interest in manipulating levels of performance and performance motivation, researchers have lacked a general and reliable means of doing so. Our finding that bite performance can be reduced by altering levels of a biogenic amine therefore offers scope for diminishment of invertebrate performance, albeit in a somewhat coarse-grained manner. An important caveat is that OA, like other neurohormones, has multiple effects, and in crickets is also implicated in fat metabolism and other aspects of homeostasis (Fields and Woodring 1991; Roeder 2020). Consequently, manipulating OA likely has secondary effects as well (Husak and Lailvaux 2022). Finally, because we were mostly

interested in the mechanistic role of OA signaling in explaining sub-maximal performance variation, we did not implement a fully-factorial design that also tested whether the maximum bite performance of crickets is enhanced by OA supplementation alone, although we note that the potential to do so will be limited by the number of unbound OA receptors at any given time. In addition, there is likely to be an upper limit to maximum performance that is set by the existing underlying morphology and beyond which bite force does not increase further without changes to that morphology as well. Future research might test explicitly for such effects, as well as potential links among OA, the mushroom bodies, performance motivation, and flight experience.

In conclusion, we show that bite performance is significantly diminished by both an OA receptor antagonist and by antenna removal, implicating both OA and, potentially, the mushroom bodies of the insect brain as key elements of cricket performance motivation. These results hold promise for understanding not only why animals perform to the various levels that they do in different ecological contexts in nature, but also as a method for manipulating invertebrate performance expression.

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Data Availability Statement: Analyses reported in this article can be reproduced using the data provided by (Lailvaux et al. 2022).

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