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Rapid effects of estradiol on aggression depend on genotype in the white-throated sparrow, a species with an estrogen receptor polymorphism

By

Jennifer Merritt

Master of Arts

Psychology: Neuroscience & Animal Behavior

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Date

Rapid effects of estradiol on aggression depend on genotype in the white-throated sparrow, a species with an estrogen receptor polymorphism

By

Jennifer R. Merritt

B.S., University of Illinois at Urbana-Champaign

Advisor: Donna L. Maney, Ph.D.

An abstract of

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**Abstract**

Rapid effects of estradiol on aggression depend on genotype in the white-throated sparrow, a species with an estrogen receptor polymorphism

by

Jennifer R. Merritt

The white-throated sparrow represents a powerful model in behavioral neuroendocrinology because it occurs in two plumage morphs that differ with respect to steroid-dependent social behaviors. Birds of the white-striped (WS) morph engage in more territorial aggression than birds of the tan-striped (TS) morph. This behavioral polymorphism is caused by a chromosomal inversion that has captured many genes, including estrogen receptor alpha (ERα). ERα expression depends on morph in a number of brain regions implicated in social behavior, including the rostral medial preoptic area (rPOM) and nucleus taeniae of the amygdala (TnA), suggesting that the behavioral polymorphism might be explained by differential sensitivity to sex steroids. In this study, we tested whether exogenous estradiol (E2) administration produces differential effects on behavior and the brain in the two morphs, as predicted by the ERα polymorphism. We administered a bolus dose of E2 and quantified aggression toward a conspecific 10 min later—a time point at which E2 is known to increase aggression in song sparrows (Experiment 1). E2 increased aggression in WS birds, but not TS birds. Thus, in this study we found that the rapid effects of E2 depended on morph. To map neural responses to E2, we administered an identical dose of E2 and quantified Egr-1 Expression in regions with known differential expression of ERα (Experiment 2). E2 treatment decreased Egr-1 immunoreactivity (IR) in both rPOM and TnA, but this effect did not depend on morph. We then tested whether morph differences in Egr-1-IR emerge after birds are treated with E2 for much longer (7 days; Experiment 3). We found an interaction between morph and treatment; E2 treatment increased Egr-1 in the TnA of WS birds, but decreased it in TS birds. Overall, our results suggest that the ERα polymorphism may contribute to morph differences in aggression *via* both nongenomic and genomic mechanisms.

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