Independent and Additive Contributions of Postvictory Testosterone and Social Experience to the Development of the Winner Effect

Matthew J. Fuxjager, Temitayo O. Oyegbile, and Catherine A. Marler

Departments of Zoology (M.J.F., C.A.M.) and Psychology (T.O.O., C.A.M.), University of Wisconsin—Madison, Madison, Wisconsin 53706

The processes through which salient social experiences influence future behavior are not well understood. Winning fights, for example, can increase the odds of future victory, yet little is known about the internal mechanisms that underlie such winner effects. Here, we use the territorial California mouse (*Peromyscus californicus*) to investigate how the effects of postvictory testosterone (T) release and winning experience individually mediate positive changes in future winning ability and antagonistic behavior. Male mice were castrated and implanted with T capsules to maintain basal levels of this hormone. We found that males form a robust winner effect if they win three separate territorial disputes and experience a single T surge roughly 45 min after each encounter. Meanwhile, males exhibit only an intermediate winner effect if they either 1) acquire three previous wins but do not experience a change in postvictory T or 2) acquire no previous wins but experience three separate T pulses. The results indicate that the effect of postvictory T must be coupled with that of winning experience to trigger the maximum positive shift in winning ability, which highlights the importance of social context in the development of the winner effect. At the same time, however, postvictory T and winning experience are each capable of increasing future winning ability independently, and this finding suggests that these two factors drive plasticity in antagonistic behavior via distinct mechanistic channels. More broadly, our data offer insight into the possible ways in which various species might be able to adjust their behavioral repertoire in response to social interactions through mechanisms that are unlinked from the effects of gonadal steroid action. **(*Endocrinology* 152: 3422–3429, 2011)**

Social experiences that individuals acquire throughout their lives can shape future behavior (1–3). These effects may persist for a relatively short amount of time or for days to weeks. For long-term changes to occur, animals must first integrate social information and then use this information to modify physiological substrates that govern behavioral output (4). Only a handful of studies have investigated this process, and they focused on a few species that exhibit unique and highly derived abilities to adjust to social encounters [rapid sex-changing (5), rapid growth-changing, (6)].

The winner effect is an ideal behavioral phenomenon for investigating the mechanisms through which social interactions control behavior. It is defined as an increased ability to win fights after previous victories (7), and it occurs in diverse taxa (8), including humans (9). Moreover, the winner effect can persist for a relatively long time (10, 11), suggesting that the effect of winning reorganizes the mechanisms that regulate aggression.

To date, most research suggests that androgens are the key hormonal mediators of the winner effect, which is a compelling model because of the role that androgens play in many species. For example, androgens are often released from the gonads in response to social competitions (12–15) and are known to regulate the output of aggression and the motivation to engage in antagonistic compe-

ISSN Print 0013-7227 ISSN Online 1945-7170
Printed in U.S.A.

doi: 10.1210/en.2011-1099 Received April 13, 2011. Accepted June 24, 2011.
First Published Online June 19, 2011

Abbreviation: T, testosterone.

titions (16–18). This specific hormone-behavior relationship is encompassed by the well-accepted Challenge Hypothesis, which proposes that the ability to mount a testosterone (T) response to aggressive conflicts is related to the evolution of social traits (19–21). However, the role that social context plays in mediating changes in future winning ability is often overlooked by studies examining the physiological framework of the winner effect. In other words, do experiential effects associated with fighting gate the role that postencounter androgens have on mediating the winner effect? Or, alternatively, do experiential or contextual effects of winning by themselves impact future winning behavior, regardless of whether postencounter androgens are released from the gonads?

We explore these questions by investigating the interactive effects that postencounter T and winning experience have on the winner effect. We use the territorial and monogamous California mouse (*Peromyscus californicus*) as the model system (22, 23), because the winner effect and its mechanisms have been studied extensively in this species of New World rodent. For example, males are known to release T from the gonads roughly 45 min after winning a fight in their home territory (24–26), and studies suggest that this hormonal response enhances antagonistic performance in the future (27, 28). However, it is unknown whether this postencounter T pulse is solely responsible for mediating plasticity in winning ability. One study shows that California mice given T pulses outside of the context of a winning experience fail to robustly improve their ability to win fights later on (28). Although these results imply that postvictory T surges are linked to contextual cues associated with the experience of winning to drive a full and complete winner effect, this study itself did not test this idea exclusively. In particular, this experiment did not simultaneously control and manipulate 1) basal androgen levels, 2) gonadal androgen responsiveness to winning, and 3) the experience of winning a fight to adequately separate and measure the relative influences of both gonadal T and winning experience on future winning behavior. Strong evidence for an effect of winning experience alone on future behavior would add new complexity to the Challenge Hypothesis, which often places greater emphasis on how socially induced fluctuation in androgens modulate behavior. Thus, to our knowledge, the avian literature has largely ignored the concept that experience by itself mediates adaptive behavioral plasticity.

Here, we tease apart for the first time the effects that postencounter T and winning experience have on the winner effect. Based on the studies described above, we predict that both of these factors cause a positive change in future winning behavior, but that both factors are needed to induce the maximum winner effect.

Materials and Methods

Animals

We obtained virgin California mice from our laboratory colony at the University of Wisconsin at Madison. All mice were group-housed (three to four per cage; 48 × 27 × 16 cm) and fed Purina mouse chow 5001 and water *ad libitum*. The colony was kept on a reversed 14-h light, 10-h dark cycle and maintained according to the National Institutes of Health *Guide for the Care and Use of Laboratory Animals*, and the appropriate institutional authorities at University of Wisconsin—Madison approved of this study.

One week before the study, 160 male mice were moved from the colony to a separate room used for testing. Of these mice, males were assigned at random to be focal males (n = 65), training intruders (n = 40), or testing intruders (n = 65). Behavioral manipulations always occurred at least 1 h after the onset of the dark cycle and under dim red light.

Experimental design and timeline

On d 1, focal males were surgically castrated and sc implanted with a SILASTIC (Dow Corning Corp., Midland, MI) tube containing crystalline T (inner diameter = 0.04 in., outer diameter = 0.085 in., containing 1 mm T; see Ref. 27). Research in California mice shows that implanting mice with this T dose maintains circulating T at levels typical of adult males and, in effect, prevents males from altering plasma T in response to social or environmental cues (27).

On d 11, focal males were randomly assigned to one of four treatment groups (Table 1). In each group, males were subjected to a test encounter after receiving either 1) three separate wins each followed by a single T injection (W+T, n = 18); 2) three separate wins each followed by a saline injection (W+S, n = 14); 3) three separate handling experiences each followed by a T injection (H+T, n = 17); or 4) three separate handling experiences each followed by a saline injection (H+S, n = 16).

Once assigned to one of the groups, each focal male was placed in its own polycarbonate observation cage (30 × 50 × 30 cm) with ample food, water, and nesting material. On d 13, 14, and 15, focal males received winning and handling experiences. Winning experiences were administered using a resident-intruder paradigm that was slightly modified from that which was described previously (26). In short, a male training intruder was

TABLE 1. Treatment groups to which males were randomly assigned

Group	Experience on d 13, 14, and 15	Postexperience hormone treatment
W+T	Win	T injection
W+S	Win	Saline injection
H+T	Handle	T injection
H+S	Handle	Saline injection

Treatment groups are 1) three wins followed by T injections (W+T), 2) three wins followed by saline injections (W+S), 3) three handles followed by T injections (H+T), and 4) three handles followed by saline injections (H+S).

placed into the focal mouse's home in the side furthest from the focal mouse at that particular moment. The two mice were given 7 min to interact freely with each other, after which the intruder was removed. An observer watched the encounter and noted which individual won the fight. The winner was defined as the mouse that initiated three consecutive attacks toward its opponent that each elicited losing behavior (definitions of attack and losing behavior below) (26). Intruders were assigned at random to focal males. Each intruder was used only twice in the study and never encountered the same focal individual more than once. All encounters during this phase of the study were biased in favor of focal males; thus, intruders were always smaller and had lost at least one previous fight. The two focal mice that did not win all three encounters on d 13, 14, and 15 were removed from the study.

Handling experiences were administered using the same protocol described above (*i.e.* open cage top, insert hand, *etc.*), except that an intruder was never placed inside the focal mouse's cage. In effect, this means that focal mice that received handling served as no-fight controls, which is very common in studies that investigate the role that aggressive experiences have on behavior and physiology (29–32). Other types of social interactions, such as losing experiences or nonaggressive social encounters, are often deemed inadequate because either 1) psychological or subtle aggressive events are still not controlled for or 2) these encounters induce their own unique set of changes to physiology and behavior (33–35).

Intraperitoneal saline and T (36 μg/kg) injections were given 30 min after the conclusion of the winning or handling experience. We used T-cyclodextrin inclusion complex because it quickly delivers T to the bloodstream and is metabolized rapidly, resulting in a short-lived pulse of circulating T (36) that closely resembles the natural T pulse that California mice experience after a fight (25). Studies by Trainor and colleagues (27) have confirmed this in male California mice and have shown that this preparation of T described above sufficiently increases circulating T above baseline levels roughly 40–45 min after a fight, yet in a manner that is still within the species' physiological range. For delivery, T was suspended in saline because T-cyclodextrin inclusion complex is water-soluble, explaining why control animals were given injections of just saline solution.

On d 16, focal males were subjected to test encounters to assess whether previous winning experience and hormone treatments changed overall winning ability and other antagonistic behavior. Test encounters were staged using the same resident-intruder paradigm described above, but test intruders, rather than training intruders, were used in these fights. Test intruders held a competitive advantage over the focal male, which decreased the probability that focal males would win test encounters at random (26); thus, test intruders were larger than focal males (3.0 ± 0.4 g; mean ± SEM) and had won a single fight on the day before the test encounter. During the study, test intruders were housed identically to focal males, in that test intruders lived by themselves in a standard cage from d 1–10 and in an observation cage from d 11–16. Test intruders were assigned to focal males at random and were used only once. All test encounters were videotaped.

Quantification of behavior

An observer who was blind to the treatment groups analyzed the test encounter videotapes. For each male, the observer recorded attack latency (*i.e.* time between test encounter onset and individual's first attack), total attacks (*i.e.* sum of bites, chases, and wrestling bouts), and total losing behavior (*i.e.* sum of jumps away, retreats, and freezes). The observer then computed a ratio of attack efficiency (*i.e.* the ratio of intruder losing behavior to focal mouse total attacks). This ratio reflects the relative amount of losing behavior focal mice elicit from opponents per attack, and a higher ratio indicates that focal mice are able to elicit more losing behavior with each attack they direct at their opponent. The observer also determined which male won the test encounter by using the definition of a winner described above. For eight of the test encounters, a malfunction of the recording equipment prevented the test encounters from being videotaped; thus, measurements of aggressive behavior were not available for these males, and only contest outcome was recorded by the individual who observed the encounter in real time. This observer was also blind with respect to the mouse's treatment. Accuracy between determining correct contest outcome in real time and in video was ~95%.

Statistical analyses

Group differences in winning ability were examined using the Fisher exact test modified for data in a 2×4 table (37). Between-group differences in winning ability were subsequently analyzed using standard 2×2 Fisher exact tests, controlling for type I error with procedures outlined by Holm (38). A series of one-way ANOVA were used to test for group differences among antagonistic behavior, with any significant effects being followed by Student-Newman-Keuls *post hoc* tests. Attack latency, total attack, and total losing behavior data were natural log transformed [$\ln(x + 1)$], and attack efficiency data were cube root transformed ($x^{1/3}$), because Q-Q plots revealed that these transformations yielded more normally distributed data (39).

Results

Winning ability

The ability of males to win the test encounter varied significantly as a function of previous winning experience and postencounter T injections (Fig. 1; Fisher exact, $P < 0.001$). Mice given previous wins and postvictory T injections won a significantly higher proportion of test en-

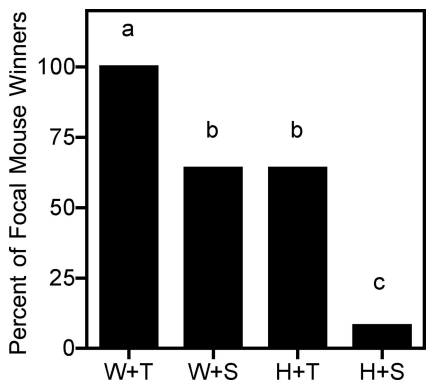

FIG. 1. Proportion of focal males in each treatment group that won the test encounter. Differences in the *letters above the bars* denote significant differences between groups (Fisher exact tests).

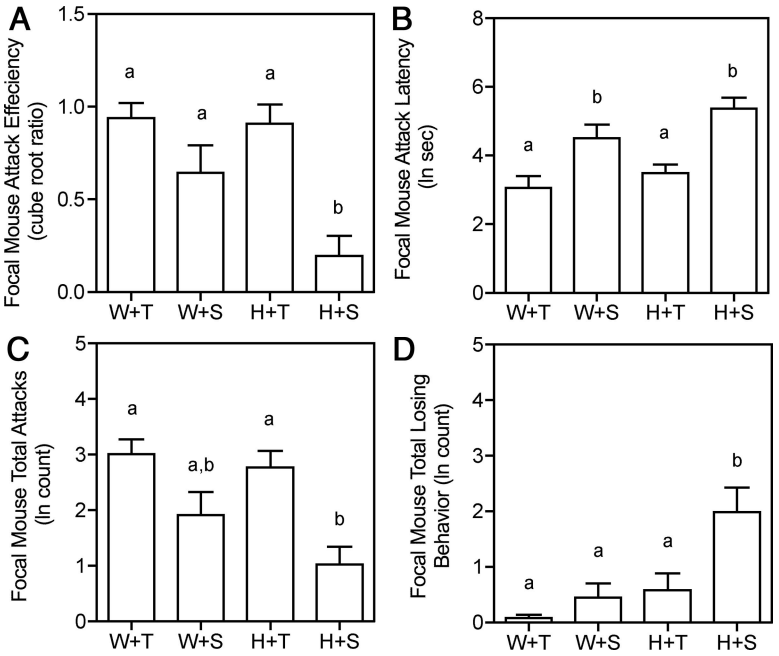

FIG. 2. Antagonistic behavior of focal mice during the test encounter: A, attack efficiency; B, attack latency; C, total attacks; D, total losing behavior. Treatment groups are depicted on the *horizontal axis* [three wins followed by T injections (W+T), three wins followed by saline injections (W+S), three handles followed by T injections (H+T), three handles followed by saline injections (H+S)]. Data represent means ± 1 SEM. For each behavioral measure, differences in the *letters above the bars* denote significant differences between groups (Student-Newman-Keuls, $P < 0.05$).

counters than mice given handling experience and posthandling saline injections (Fisher exact, $P < 0.001$). However, compared with both of these groups, mice given either previous wins and postvictory saline injections or handling experience and posthandling T injections won an intermediate proportion of test encounters. In other words, mice given only past wins or only T injections won significantly more test encounters than handled control mice that received saline injections (Fisher exact, $P < 0.01$), but significantly fewer test encounters than mice that received winning experience and postencounter T (Fisher exact, $P < 0.01$).

Antagonistic behavior

There was also significant variation among groups with respect to the contest behavior of focal mice (Fig. 2; attack efficiency: ANOVA, $F_{(3,54)} = 10.34$, $P < 0.001$; attack latency: ANOVA, $F_{(3,42)} = 10.23$, $P < 0.001$; total attacks: ANOVA, $F_{(3,54)} = 8.19$, $P < 0.001$; total losing behavior: ANOVA, $F_{(3,54)} = 8.43$, $P < 0.001$). Males given any combination of postencounter T injections and past winning experience displayed significantly higher attack efficiency ratios and significantly lower levels of losing behavior than control mice that received handling experience and saline injections (Student-Newman-Keuls *post hoc*, $P < 0.05$). Moreover, mice that received T injections, regardless of past winning experience, showed significantly lower attack latencies compared with controls (Student-Newman-Keuls *post hoc*, $P < 0.05$). Finally, mice that received T injections exhibited a higher number of total attacks than handled and saline-injected controls (Student-Newman-Keuls *post hoc*, $P < 0.001$); however, the total attacks of mice that received winning experiences and postencounter saline injections was statistically indistinguishable from any of the other groups (Student-Newman-Keuls *post hoc*, $P > 0.05$).

Discussion

Our study reveals that two main factors underlie the winner effect in California mice: gonadal release of androgens after a fight and cues associated with the social experience of winning. Mice form a full and robust winner effect when they accumulate three separate victories in their home territory and receive a T injection after each of these contests. At the same time, mice form an intermediate winner effect when they either 1) accumulate the same number and type of victories and receive postencounter saline injections or 2) accrue three separate handling experiences (*i.e.* no-fight controls) and received posthandling T injections. Thus, the relative effects of postencounter T and winning experience on plasticity in winning ability appear to be independent in nature and likely combine additively to induce a maximum winner effect. This result is intriguing because it suggests that the winner effect itself is not the result of a permissive interaction between postencounter T and winning experience. In other words, neither of these factors appears to gate the effect that the other factor has on enhancing winning ability.

Gonadal androgens, the experience of victory, and the winner effect

Past studies that explore the mechanistic underpinnings of the winner effect have suggested that the pulsatile release of androgens from the gonads is primarily responsible for driving changes to contest behavior (18, 27). Our data, however, indicate that such postencounter surges of T must be coupled with the social experience of victory to fully enhance future winning. To our knowledge, this is the first demonstration that experiential factors can appreciably influence plasticity in winning behavior independently of gonadal androgen titers, even though this

idea has been hinted at by empirical work in both California mice and a species of hermaphroditic territorial fish (28, 40). Both of these prior studies, however, were limited by the fact that they did not experimentally manipulate both winning experience and postvictory androgen responsiveness. Thus, the findings reported here are highly significant because they underscore the importance of factors relating to social context in mediating internal processes that govern adaptive behavioral flexibility.

From a more conceptual point of view, our study suggests that there is variation in the extent to which postencounter androgen titers drive the winner effect, and this idea has novel implications for the Challenge Hypothesis (19, 20). John Wingfield and colleagues (20) first developed this hypothesis to describe why species differ in their ability to mount a robust androgen response to fights and/or periods of social instability. In particular, the Challenge Hypothesis proposes that animals are more likely to evolve the ability to increase androgens after a social dispute when such a response helps facilitate sexual behavior and/or improve future fighting ability during instances of intense male-male competition (41, 42). On one hand, our data are consistent with the Challenge Hypothesis because they show that postencounter gonadal T has a profound (and necessary) effect on maximizing behavioral changes (*i.e.* the winner effect) later in life. On the other hand, our data add to the Challenge Hypothesis by showing that some changes to behavior that transpire in response to an aggressive encounter need not depend on release of T from the gonads. As such, this is consistent with the notion that some species might evolve mechanisms to enhance their own sexual or aggressive behavior independently of androgen responsiveness to male-male conflicts. This, of course, means that species with little flexibility in plasma androgen levels after fights or across seasons (43–48) may still be able to modify their own behavior after experiencing antagonistic interactions with other competing males.

The precise cues allied with winning experience that trigger changes in behavior are not yet clear in California mice. Any number of stimuli may be responsible for such changes, including the expression of aggressive behavior or somatic feedback from muscle tissues during a fight (49). Past research in fish and birds indicates that the primary cue during conflicts that trigger changes in physiology and/or subsequent behavior is the perception of oneself as the winner (50, 51). We speculate that this also applies to California mice because the robustness of the winner effect in this species appears to be influenced more by the saliency of acquired victories than by levels of aggressive behavior displayed during those victories (see below) (24, 26).

Gonadal androgens, the experience of victory, and plasticity in antagonistic behavior

Our findings are also compelling because they illustrate how postencounter T and the experience of winning affects different types of contest behavior. For example, T injections by themselves caused males to increase attack efficiency and total attacks, as well as decrease total losing behavior and attack latency. On the other hand, winning experience by itself increases attack efficiency and decreases total losing behavior but has no measurable effect on total attacks or attack latency. Thus, these data imply that the effects of postencounter T and winning experiences differentially impact metrics of aggression, possibly because postvictory T and winning experience influence distinct physiological and/or neural substrates responsible for controlling the disparate elements of antagonistic performance (52).

In California mice, however, it is unclear how these metrics of aggression relate to winning ability *per se* (24, 26). For example, mice that receive winning experience and postvictory T are able to win a significantly higher proportion of test encounters than mice that receive T injections alone. Yet, these two groups are indistinguishable with respect to their attack latencies, total attacks, attack efficiencies, and total losing behavior. This result suggests that the measures of aggression we documented in this study do not necessarily predict an individual's ability to win a fight. It is possible that the winner effect results from other changes in behavior linked to contest performance, such as olfactory and acoustic communication (53, 54) or the temporal patterning of attacks (55).

Endocrine mechanisms of plasticity in winning and aggressive behavior

Our results raise the question of how postencounter T and winning experience influence future winning ability and aggressive behavior. Given that a fully developed winner effect in California mice persists at least 2 d after the third aggressive encounter (26), it is likely that accumulating multiple wins affects future aggressive and winning behavior in a lasting manner by changing the brain. Although the type and extent of these changes to neuronal substrates are still unclear, we recently found that winning multiple fights increases the expression of androgen receptors in key brain areas that control antagonistic motivation and performance (56). Moreover, these changes to neural androgen sensitivity are positively associated with winning. Thus, it seems possible that postencounter T, winning experience, or both of these factors increases the expression of androgen receptors in select brain nuclei as a way of facilitating the winner effect.

However, we cannot rule out the possibility that other steroidal mechanisms also contribute to the winner effect. For example, postencounter gonadal T may be converted to estrogen by the enzyme aromatase and subsequently act on estrogen receptors in the brain. Activation of these receptors has pronounced effects on aggression (57, 58). Yet, this idea is made more complex because previous work in California mice has suggested that aromatase activity in select regions of the hypothalamus and limbic system is not necessarily affected by previous antagonistic behavior (27). This experiment did not measure winning ability *per se*, so it is unclear whether aromatase activity is associated with other aspects of aggression and winning.

Winning experience may also cause the adrenal glands to emit glucocorticoids, sex steroid precursors, or both, and this in turn might modulate plasticity in aggression. Experiments have found that these hormonal substances either themselves or via conversion to other bioactive substances (*e.g.* dehydroepiandrosterone) can affect antagonistic behavior in many mammals and birds (59–62). Nevertheless, despite the absence of stress hormone release after fights in California mice (24, 26), there has not been adequate exploration of the role that adrenals and other glands that secrete glucocorticoids play in mediating the winner effect. For example, recent work shows that the lack of a stress hormone response to fighting in California mice may be seasonally dependent (63), suggesting the possibility that glucocorticoids may affect aggression in this species under some circumstances.

There are, of course, many other plausible endocrine mechanisms that might guide the formation of the winner effect, ranging from shifts in neurochemical synthesis and activity (64–66) to *de novo* production of steroids in the brain (67, 68). Another intriguing possibility is that natural variation in the ability to mount an androgen response to winning (69, 70) is somehow associated with the ability to rapidly develop the winner effect. Future experiments will be needed to investigate these possibilities to better assess how endocrine pathways are integrated to guide not only the winner effect but also potentially other types of adaptive behavioral plasticity.

Conclusion

In sum, we show that both postvictory T and winning experience contribute to the winner effect in an independent and additive manner. These factors also have different influences on separate measures of antagonistic behavior, suggesting that they modify different substrates that control social aggression. From a broader functional perspective, these results suggest postvictory androgens increase the probability that the winner effect will be expressed but that species may be capable of evolving the ability to adjust their behavior in response to social interactions via mechanisms that are independent of direct gonadal androgen action. Future work should address how winning experience affects the neuroendocrine mechanisms of the winner effect, particularly the androgen system.

Acknowledgments

We thank Jon Montgomery and Brenna Knaebe for experimental assistance as well as Kyla Davidoff, Janice Davidoff, Charles Snowdon, Ben Pawlisch, Erin Gleason, and Brook Schmeichel for comments on the manuscript. We thank Lauren Riters and Benjamin Pawlisch for assistance and materials to make T-implants.

Address all correspondence and requests for reprints to: Matthew Fuxjager, Ph.D., University of Wisconsin—Madison, Department of Zoology, 250 North Mills Street, Madison, Wisconsin 53706. E-mail: mfoxhunter@gmail.com.

This work was funded by a National Science Foundation (NSF) Graduate Research Fellowship (to M.J.F.), NSF Grant IOS-0620042 (to C.A.M.), and a Wisconsin Alumni Research Foundation grant (to C.A.M.).

Disclosure Summary: The authors have nothing to disclose.

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