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Heritability of acoustic signalling time in the Texas field cricket, *Gryllus texensis*

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ABSTRACT

Hypothesis: Heritability estimates of total nightly signalling time will be equal for a contemporary data set in Austin (Texas) and an earlier data set collected 127 km away in San Antonio (Texas).

Organism: Texas field cricket, *Gryllus texensis* (Gryllidae: Orthoptera).

Background: A corpus of work has been done on *Gryllus texensis* in Austin, Texas with the underlying assumption that heritability values from San Antonio, Texas apply.

Methods: Previous study – realized heritability estimated using an artificial selection experiment. Current study – narrow sense heritability estimated using parent–offspring, full-sib/half-sib, and restricted maximum likelihood methods.

Results: Heritability of total nightly signalling time was previously computed as 0.50 and 0.53 for the San Antonio crickets. However, heritability of total nightly signalling time in the Austin crickets is estimated at only 0.006 ± 0.045. In Austin, only a small portion ($1.5 \pm 11\%$) of the genetic variance in total signalling time is additive.

Keywords: calling time, dominance, environmental variance, Gryllidae, heritability, maternal effects, Texas field cricket.

INTRODUCTION

Trait heritability determines to what extent the trait can respond to selection. Heritability values are population parameters and are unique to the environment in which they are measured. Therefore, extreme care should be taken when applying heritability values to other populations (Falconer and Mackay, 1996). However, possibly because obtaining a powerful heritability estimate is extremely labour-intensive, biologists often use the heritability value from one population to draw conclusions about the evolution of the trait in a nearby population.

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Heritability values have been estimated in two populations for several morphological and life-history traits. These results often show that heritability estimates differ across populations. Ali and Johnson (2000), for example, estimated heritability of winter hardiness of lentils in two Pakistani populations. Heritability was significantly higher in Kalat ($h^2 = 0.71$) than in Quetta ($h^2 = 0.32$–0.51). Stearns (1984) measured heritability of length at maturity of mosquito fish in two Hawaiian populations. Heritability was significantly higher in Reservoir 33 (male $h^2 = 0.68$, $P < 0.05$; female $h^2 = 0.91$, $P < 0.05$) than in Twin Reservoir (male $h^2 = -0.92$, non-significant; female $h^2 = -0.07$, non-significant). Avery (2005) estimated heritability of the percent dormant eggs produced by a planktonic marine copepod in Maine and Rhode Island. While the heritability values did not differ between the populations, the estimates were greatly dependent upon temperature (at 12–13.5°C: Maine, $h^2 = 0.91 \pm 0.20$; Rhode Island, $h^2 = 0.95 \pm 0.28$; at 17–17.5°C: Maine, $h^2 = 0.08 \pm 0.21$; Rhode Island, $h^2 = 0.25 \pm 0.38$). These studies suggest researchers should exercise extreme caution when assuming that the heritability value for one population is the same as that for a nearby population, because heritability values depend greatly on the population studied and the environmental conditions maintained.

Here we estimate the heritability of a behavioural trait and compare it with a 26-year-old estimate obtained from 127 km away. In 1981, William Cade conducted one of the first studies to quantify the genetic basis of a sexually selected trait. He estimated heritability of total nightly signalling time in the Texas field cricket, *Gryllus texensis* [formerly *G. integer* (Otte and Cade, 2000)]. Male Texas field crickets use two alternative mating behaviours to attract females: callers rub their forewings together to produce conspicuous long-distance acoustic signals to attract receptive females, whereas satellites silently attempt to intercept females attracted by the signaller's signals (Cade, 1975). To demonstrate whether field crickets differ genetically with respect to their total nightly signalling time, Cade (1981) selected on nightly signalling time over four generations by mating males from each end of the signalling time distribution with non-sister virgins. Using the ratio of selection response to the selection differential, he estimated realized heritability at 0.50 and 0.53 for the high and low lines, respectively (Cade, 1981). The close concordance in estimates from the high and low lines suggests that signalling time has an important genetic component (Cade, 1981). The realized heritability estimates also suggest that callers and satellites might be separate genetic strategies whose expression is partly dependent on the environment (Cade, 1981). Texas field crickets' signalling time and mating strategy have become widely cited examples of sexual behaviours exhibiting high heritability (116 citations in ISI Web of Science as of July 2007).

Cade's (1981) heritability study was conducted on crickets whose parents were collected in San Antonio, Texas. Since publication of Cade's 1981 study, most behavioural and evolutionary research on Texas field crickets has been conducted on crickets from Austin, which lies 127 km northeast of San Antonio. Many of the studies on *G. texensis* published since 1981 have drawn conclusions about the evolution of total nightly signalling time in Austin, referencing the San Antonio heritability study as evidence. Cade and Cade (1992), for example, conducted a field study in Austin, which revealed that cricket density influences both signalling time and mating success. They suggested density-dependent selection might maintain nightly signalling time's high heritability. Furthermore, Bertram (2002) observed significant shifts in nightly signalling time across several mating seasons, interpreting these shifts as an evolutionary response to seasonal changes in the selection regime. These are examples of the many studies that assume that the heritability value for San Antonio's nightly signalling time applies to crickets from Austin, Texas. This assumption has never

been tested. Here we estimate heritability of total nightly signalling time in Texas field crickets from Austin, Texas. Unexpectedly, our results reveal that signalling time in Austin exhibits minimal heritability. A substantial portion of the variance appears to be influenced by dominance and/or common environment effects, while the remainder appears to be environmental in origin.

METHODS

We collected 4744 crickets from the lights of a golf course driving range in the northern outskirts of Austin, Texas, USA during September 2002. All crickets were macropterous (winged and flight capable). Collected females mated multiply with males, and laid their eggs in moist soil. Offspring of these wild-caught adults were reared under standardized conditions (temperature and photoperiod: 26 ± 2°C, 14 h/10 h light/dark; uncontrolled humidity) in 36-litre plastic containers ($36 \times 28 \times 23$ cm). All containers were provided with food (Harland's Tekland Rodent diet 8604), water, and shelter. Food and water were replenished twice weekly, and containers were cleaned as necessary. Juvenile crickets were checked weekly for individuals that had reached nymphal stadium four. Crickets at nymphal stadium four and beyond were housed individually in 500-ml plastic coated paper bowls (7×11 cm). Individuals were checked daily to obtain the date they moulted to adulthood. These adults comprised the parental (P) generation for our quantitative genetic experiment.

We used parent–offspring and full-sib/half-sib experimental designs to estimate the variance components for total nightly signalling time. We monitored each sire's long-distance mate attraction signals from 10 to 17 days after final moult using electronic acoustic recorders (EARs – see below). Each sire was then mated to a randomly selected virgin dam. A subset was also mated to a second virgin dam. In total, 484 sires produced 1294 adult sons; 61 of these sires produced adult sons from *both* dams.

The F_1s were reared in the same standardized environmental conditions as their parents. Newly hatched individuals were reared in 500-ml plastic-coated paper bowls together with their full-siblings. When the full-siblings reached nymphal stadium two to three, they were sub-divided into identical containers so that family group size was no more than six individuals per container. Food and water was changed twice weekly. All containers were checked weekly for individuals that had reached nymphal stadium four. F_1s at nymphal stadium four and beyond were housed individually in 500-ml plastic coated paper bowls. These individuals were checked daily to obtain the date they moulted to adulthood. Like their sires (P generation), the nightly signalling time of the adult sons (F_1 generation) was monitored electronically from 10 to 17 days after final moult.

Electronic acoustic recorders

Male crickets had their long-distance acoustic mate attraction signals monitored electronically for 16 h each night (from 18.00 to 10.00 hours). We used four EARs to monitor cricket signalling behaviour. Each EAR can monitor the acoustic signalling behaviour of up to 128 individuals simultaneously by sampling the acoustic environment surrounding each individual cricket (for details, see Bertram and Johnson, 1998; Bertram *et al.*, 2004). Seven centimetres of acoustic foam separated each cricket from his nearest neighbours. The acoustic foam reduced each cricket's ability to hear the acoustic signals of his nearest neighbours, and made the close

quarters of the monitoring arena (nearest neighbours are separated by 13–20 cm) more similar to natural conditions. In this acoustic set-up, the acoustic signals of nearest neighbours sound like they are 1–2 m away. A microphone was hung within each container, approximately 5 cm above the cricket. Microphones sample the acoustic environment of their container. Our previous research confirmed that each microphone only detects the acoustic signals of the male that resides in the container, not any of its neighbours (Bertram *et al.*, 2004). Each microphone was sampled eight times a second, and the resultant data were summarized into a second-by-second description of each individual's acoustic signalling behaviour throughout the course of the monitoring period. Using these data, each male's total nightly signalling time was quantified as the total number of minutes spent signalling over the 16-h monitoring period.

Data analysis

Statistical analyses were conducted using JMP 6.0.3 (SAS Institute, Inc.) and ASReml (Gilmour *et al.*, 2002). A key assumption of estimating heritability and genetic correlations is that the data are normally distributed. We used a log-transformation on the signalling time data set because the resulting transformed data most closely approximated a normal distribution.

Genetic components of variance and heritability values were estimated using both the entire data set and a subset that included only the males that signalled for more than 1 min a night (the calling subset). We used a 1-min cut-off time to eliminate small background noises (e.g. door closing) that unintentionally is recorded as signalling time. This 1-min cut-off also reduced the chances of inadvertently including a non-calling satellite male as a caller. Research by Cade (1991) has shown that non-calling satellite males occasional signal when they are placed in a acoustic recording apparatus, albeit with significantly lower total nightly signalling times. For the calling subset, 371 of 484 sires were included in the analysis, 73 of the 113 excluded males did not signal, and the other 40 signalled for less than 1 min; 847 of 1294 F_1 males were included in the analysis, 257 of the 447 excluded males did not signal, and the other 190 signalled for less than 1 min. The exclusion of males from the calling subset did not dramatically alter the distributions (see Fig. 1).

Each male's mean total signalling time was used to calculate the genetic components. We estimated the additive and non-additive genetic components of log signalling time using two separate but related approaches: a full-sib/half-sib analysis and an animal model analysis (Knott *et al.*, 1995). We used restricted maximum likelihood (REML) parameter estimation for both analyses because this is a more accurate approach than least squares when designs are unbalanced (Falconer and Mackay, 1996). The full-sib/half-sib analysis is equivalent to the standard ANOVA approach whereby the sire variance component equals ¼ V_A and the dam (nested within sire) component equals ¼ $V_D + V_{Ec}$ (ignoring epistasis). For the animal model analysis, the phenotype of each individual is written in terms of its additive genetic merit, other random effects (dominance, epistasis, common environment and maternal effects) and the residual. ASReml constructs an additive genetic relationship matrix on the basis of the relationships among parents and offspring and uses this to estimate V_A (Gilmour *et al.*, 2002).

Because the signalling time data set was not normally distributed, we checked the robustness of both of our heritability estimates using Roff's (2001) threshold model. The threshold model produces a generalized transformation that provides unbiased heritability

estimates (Roff, 2001). We used this approach to estimate the heritability of signalling time for the entire data set and the subset that included only the callers.

We estimated the repeatability of signalling time by partitioning the phenotypic variance of each trait into the variance within males and the variance between males using a nested analysis of variance [log(total signalling time) = id + age(id)]. The variance within males is the intra-individual correlation coefficient and is associated with changes due to age or temporary differences of environment between successive nights. The variance between males is the interclass component and is associated with environmental and genetic components that affect each male permanently. We used these partitioned variances to estimate the repeatability of signalling time throughout the monitoring period. Repeatability expresses the proportion of the variance that is due to permanent differences between individuals and is given by

$$r_i = \frac{\sigma_b^2}{\left(\sigma_b^2 + \sigma_w^2\right)}$$

where σ_b^2 is the mean square between males and σ_w^2 is the mean square within males. The mean square between males σ_b^2 is composed of $\sigma_w^2 + m\sigma_b^2$, where m is the number of nights on which the males signalled (Falconer and Mackay, 1996; Lynch and Walsh, 1998). When an individual did not signal on a particular night, that night was not included in the analysis.

RESULTS

Total nightly signalling time was highly repeatable within individuals ($r_i = 0.69$, based on all males monitored over 7 nights; $F = 5.572$, $P < 0.0001$, $R^2_{\text{adj}} = 0.65$, d.f. = 6695). Age explained none of the variation (ANOVA: sires, $F = 0.32$, $P = 0.94$; sons, $F = 1.31$, $P = 0.23$). The full-sib/half-sib analysis revealed an extremely low heritability value for signalling time when all individuals were included in the analysis ($n = 1284$ males including callers and non-calling individuals; $h^2 \pm$ standard error $= -0.1082 \pm 0.180$). When only callers were included (males that signalled for more than 1 min), the father–son regression also revealed a heritability value that was very low and not significantly different from zero ($h^2 \pm$ standard error $= 0.076 \pm 0.11$, $F = 0.47$, $P = 0.494$, d.f. = 231) (Table 1). This value corroborates those obtained via the full-sib/half-sib and animal model analyses on callers (full-sib/half-sib: $h^2 \pm$ standard error $= -0.12 \pm 0.225$, d.f. = 846; animal model: $h^2 \pm$ standard

Table 1. Variance component estimates for signalling time (results from full-sib/half-sib and animal model analyses)

	Full-sib/half-sib model		Animal model	
Sire/animal component	-0.069 ± 0.131	$\frac{1}{4}V_A$	0.015 ± 0.110	V_A
Dam component	0.350 ± 0.162	$\frac{1}{4}V_A + \frac{1}{4}V_D + V_{Ec}$	0.238 ± 0.088	$\frac{1}{4}V_D + V_{Ec}$
Residual	2.032 ± 0.115	V_E	2.192 ± 0.133	V_E
Total	2.313 ± 0.115	V_Z	2.445 ± 0.107	V_Z
V_A	-0.277 ± 0.522	sire*4	0.015 ± 0.110	animal
$V_D + 4V_{Ec}$	1.677 ± 1.125	(dam – sire)*4	0.951 ± 0.352	dam*4
$V_D + 4V_{Ec} / V_P$	0.752 ± 0.481		0.389 ± 0.137	

error = 0.006 ± 0.045, d.f. = 1227) (Table 1). We evaluated the robustness of these estimates by estimating the heritability using Roff's (2001) threshold model, which corroborated our results. The threshold trait heritability estimate for signalling time (based on additive variance) was –0.152 ± 0.084 (h^2 ± standard error) when all individuals were included in the analysis. When only callers were included in the analysis, the threshold trait heritability estimate was –0.163 ± 0.108.

The additive genetic variance was estimated to be 1.5%. The inclusion of dams allowed calculation of the variance attributable to dominance and/or common environment. The animal model indicated that 39% of the phenotypic variation could be attributed to dominance and/or common environment. Given that signalling time appears to be influenced by minimal additive genetic variance, the remaining 60% of the variance appears environmental in origin.

Total nightly signalling times of sires and F_1 sons were not normally distributed. Instead, they were highly skewed towards low signalling times (Fig. 1). Most males signalled for less than an hour a night (57% sires and 64% F_1s), some signalled for 1–2 h a night (17% sires and 17% F_1s), fewer for 2–4 h a night (15% sires and 13% F_1s), and even fewer for more than 4 h a night (12% sires and 6% F_1s). The distributions of sires and F_1 sons differed significantly. Sires had higher mean nightly signalling times (mean ± standard deviation: sires = 71 ± 119 min, F_1 = 44 ± 90 min; Tukey-Kramer HSD means comparison $q = 1.96$, $P = 0.02$) and different variances (ANOVA: $F = 25.33$, $P < 0.0001$, $R^2_{adj} = 0.02$, d.f. = 1456).

Sires and sons were the same age when they were monitored and reared in an identical manner, so age and rearing environment cannot explain why sons called significantly less often than fathers. The only component of the experimental protocol that differed between sires and sons was the number of males having their acoustic signals monitored

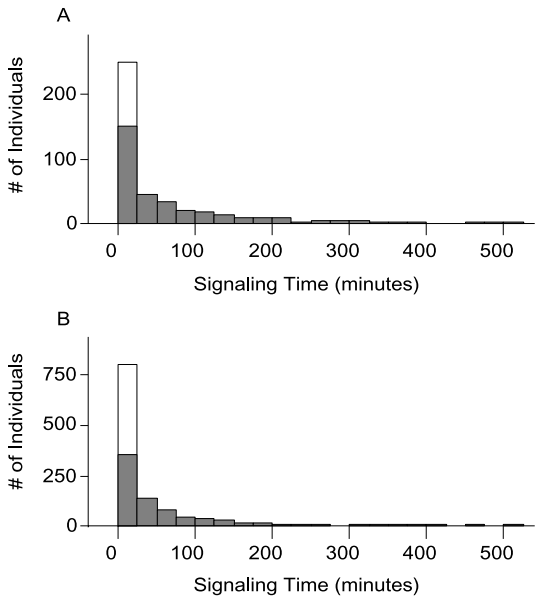

Fig. 1. Frequency distribution of signalling times among (A) sires and (B) F_1 sons. *Note*: The grey bars represent individuals used in the heritability analysis. The white bars represent individuals eliminated from the analysis because they signalled for less than 1 min a night.

simultaneously. The number of sires being monitored each night ranged from 4 to 150 (mean ± standard error = 65 ± 11). These males were each separated by acoustic foam. The number of F_1 sons being monitored each night ranged from 1 to 285 (95 ± 8), also each separated by acoustic foam. Therefore, approximately 50 more sons were monitored over almost any given night than sires. Because of the difference in acoustic monitoring environment, we examined how monitoring number influenced signalling time. Neither signalling time nor the proportion of individuals signalling was affected by the number monitored (Regression: signalling time = $59.17 - 0.05 \times$ number monitored, $F = 0.62$, $P = 0.43$, $R^2_{adj} = -0.002$, d.f. = 135; proportion signalling = $0.45 + 0.0004 \times$ number monitored, $F = 2.12$, $P = 0.15$, $R^2_{adj} = 0.008$, d.f. = 135). Overall, these statistics reveal that the differences in nightly signalling time between sires and sons cannot be explained by the number of individuals being monitored simultaneously.

DISCUSSION

Total nightly signalling time in Austin field crickets is repeatable within individuals ($r_i = 0.69$) and variable among them, but appears to exhibit minimal heritability (mean $h^2 = -0.012$). Based on our quantitative genetic animal model, $V_P = V_A + (V_M + V_D + V_{Ec}) + V_E$, 1.5% of the variance in nightly signalling time appears to be influenced by additive genetic variance (V_A), and 39% of the variance appears to be influenced by dominance (V_D), maternal and other indirect genetic effects (V_M), common environment effects (V_{Ec}), and possibly other forms of non-additive genetic variance (e.g. epistasis). Variance attributable to dominance has the potential to be high because dominance is expected to be strong in traits that evolve under selection (Fisher, 1958; Mather and Jinks, 1977; Crnokrak and Roff, 1995). Crossing designs developed by Mather and Jinks (1977) could be used to quantify the dominance component but these are non-trivial to conduct. Variance attributable to parental effects may also influence sexually selected traits (Qvarnström and Price, 2001). Dams could influence offspring phenotypes with egg provisions, which represent the complete energy supply for cricket embryonic development (Mousseau and Dingle, 1991; Mousseau and Fox, 1998; Weigensberg *et al.*, 1998). Egg size correlates with initial offspring size, growth rate, and survival (Mousseau and Dingle, 1991; Mousseau and Fox, 1998; Weigensberg *et al.*, 1998; Roff and Sokolovska, 2004). Furthermore, parental effects appear to account for 10–30% of the phenotypic variance in growth rate and development time in the sand cricket, *G. firmus* (Roff and Sokolovska, 2004). Because parental effects may be important throughout the cricket's life cycle (Roff and Sokolovska, 2004), they should be quantified.

The Austin cricket's low additive genetic variance estimate (1.5%), coupled with their dominance and common environment estimate (39%), suggest that the remaining 60% of the variance in total nightly signalling time may be attributable to the environment (V_E). Crickets were reared in the same temperature, photoperiod, and density and were each provided with unlimited water and high-quality food. Males only differed in the environment in which they acoustically signalled (crickets could hear each other during acoustic monitoring; see below for our rationale for why we allowed males to hear each other). If male signalling time is influenced by the behaviour of nearby neighbours, it would account for the high environmental variance. This hypothesis requires formal testing.

Our quantitative genetic model had an omitted variable bias because it did not incorporate the interaction term $V_{G \times E}$. Our variance estimates could, therefore, be biased (Falconer and Mackay, 1996; Moore *et al.*, 1997; Gorelick, 2005). The interaction term $V_{G \times E}$ should,

therefore, be incorporated in future work. Rearing temperature may be an appropriate environmental parameter to use in estimating $V_{G\times E}$ because Texas field crickets are bivoltine; offspring of spring breeders develop in the warm summer, while offspring of fall breeders over-winter and develop in the cool spring. Diet may also be an appropriate environmental parameter to estimate $V_{G\times E}$ because several studies have shown that it influences signalling. Wagner and Hoback (1999) placed full-sibling adult *G. lineaticeps* brothers on high- and low-nutrition diets. Males on the high-nutrition diet signalled three times as much as their brothers on the low-nutrition diet (Wagner and Hoback, 1999). Hedrick (2005) fasted *G. integer* males and then compared their mean signalling bout durations [a highly heritable trait (Hedrick, 1988)] with bout duration of fed males. Fasted males lost 6% of their mass on average and dramatically decreased their signalling bout durations. Weight loss and decreased signalling bout duration were also positively correlated (Hedrick, 2005). Scheuber *et al.* (2003) limited the food intake of *G. campestris* and found that well-fed adults signalled twice as much those fed limited rations. Holzer *et al.* (2003) revealed that protein-supplemented males signalled more than controls. Mallard and Barnard (2004) fed *G. bimaculatus* and *Grylloides sigillatus* diets that differed in protein and fat; crickets reared on higher quality diets stridulated at a higher rate. Diet may therefore be a good way to quantify how $V_{G\times E}$ influences other variance components. Quantifying condition along with nightly signalling time would also enable us to quantify (1) whether these sexually selected characters are condition dependent and (2) the additive genetic variance of condition.

Our low heritability estimate suggests nightly signalling time in Austin field crickets cannot respond to selection across seasons. Bertram (2002) observed seasonal shifts in signalling time and suggested that they result from seasonal changes in selection. Instead, it is likely that seasonal shifts result from environmental changes, genotype × environment interactions, or genetic correlations with other traits that respond to the seasonal changes in selection (Bertram, 2002).

Our low heritability estimate might suggest that female Texas field crickets cannot benefit from good genes, given that all good genes models require elevated heritabilities. However, females use other signalling components to distinguish between potential mates and many of these traits have been shown to be highly heritable. Females, for example, select mates using the number of pulses in a trill (Wagner *et al.*, 1995), and number of pulses has been shown to be significantly heritable ($h^2 = 0.40$) in Austin crickets (Gray and Cade, 1999). Similarly, females select potential mates using pulse rate. Pulse rate has also been shown to be highly heritable ($h^2 = 0.40$) in Austin crickets (Gray and Cade, 2000). Female Texas field crickets from Austin have the potential, therefore, to benefit from good genes.

Why might the San Antonio and Austin heritability values differ?

The San Antonio and Austin studies exhibited similar statistical moments. Male signalling time ranged from zero to around 9 h a night in both studies. Both studies' frequency distributions were also highly skewed towards low signalling (prior to selection). Furthermore, mean nightly signalling time of the two studies did not differ dramatically (~2 h for the Austin study and ~3 h for the San Antonio study). However, the Austin crickets' heritability value differed markedly from the San Antonio crickets' heritability value [Austin: $h^2 = -0.012$; San Antonio: $h^2 = 0.51$ (Cade, 1981)]. Why might these heritability values be so different?

The heritability values might differ because the two studies used different experimental protocols. The heritability value for San Antonio was estimated using an artificial selection experiment, while the heritability value for Austin was estimated using parent–offspring and sib analyses. The two studies also differed in the length of time males were monitored. The San Antonio study calculated average nightly signalling time over eleven nights (from 7 to 16 days after final moult), whereas the Austin study calculated average nightly signalling time over seven nights (from 10 to 16 days after final moult). There are two reasons why we believe that the different protocols are unlikely to explain the differences in the two estimates. First, mean signalling times are likely to be unaffected by adding more monitoring nights. Second, empirical evidence suggests that realized heritability estimates from a few generations of selection usually show concordance with heritability measures obtained using relatives (Roff, 1997).

The low heritability estimates for signalling time in the Austin crickets do not result from a bias caused by not meeting the assumption of normality. We log-transformed the signalling time data to approximately normalize the data set before analysing the signalling time data set. We also used the threshold model to produce a generalized transformation to create unbiased and independent heritability estimates. Both of these methods resulted in heritability estimates that were also very low and not significantly different from zero.

The low heritability estimate for signalling time in the Austin population also does not result from using only a subset of the data because the heritability estimates for the entire data set and the calling subset were both extremely low and not significantly different from zero.

It is, however, possible that the low heritability estimate for Austin crickets could result from not controlling the acoustic environment during monitoring. We did not control the acoustic environment for two reasons. First, crickets can hear each other in nature. We therefore felt that signalling time heritabilities should be estimated without controlling the acoustic environment to allow us to approximate the natural heritability. Second, the acoustic environment was not controlled in the San Antonio study (Cade, 1981). We wanted to monitor signalling time in a similar manner as the San Antonio study to allow comparisons between estimates. We surrounded each male's monitoring container with acoustic foam so that neighbours' signals would sound like they were 1–2 m away (as in Cade, 1981), but the crickets could still hear each other. Exposure to different social environments could therefore have artificially reduced the heritability estimate by increasing the phenotypic variance. A subset of males will have resided next to neighbours that signalled with higher times. If male signalling is stimulated by neighbours' trilling, then not controlling the acoustic environment could have increased phenotypic variance. Since the magnitude of all variance components influences the heritability value (Falconer and Mackay, 1996), an increase in phenotypic variance would reduce the heritability estimate.

While some readers might find our uncontrolled acoustic environment problematic, we have three counter-points that deserve mention. First and foremost, we found signalling time to be highly repeatable within males ($r_i = 0.69$), suggesting that the social environment does not have a strong influence on signalling time. Second, two or more crickets were almost always signalling throughout each monitoring period. Crickets will, therefore, have heard continuous signalling throughout the night and not just the sporadic signalling of nearest neighbours. Third, average nightly signalling time was unaffected by the number of males monitored. Combined, these results suggest that the social environment may not have reduced the heritability estimates.

The high heritability estimate for the San Antonio crickets could also have resulted from methodological problems. Both lines in the San Antonio study were artificially selected to alter signalling time (one line was selected to increase signalling time and the other to decrease it). A control without selection was not utilized. A control line would have allowed the effects of environmental change and inbreeding depression to be quantified. The San Antonio study also did not use replicates, which would have enabled quantification of changes due to genetic drift (Falconer and Mackay, 1996; Roff, 1997). Because only 2–4 males of the 24–51 monitored each generation were mated, these low numbers of mating males could have allowed for high genetic drift and inbreeding. That said, we concur with Cade's (1981) conclusion that signalling time in the San Antonio crickets appears to be highly heritable. We concur because both lines responded to selection in the appropriate direction. The high selected line responded immediately to selection, bouncing from a mean signalling time of 3 h in the parental generation to one of 6 h in the first generation. It then remained virtually fixed at 6 h for the following three selection events. The low selection line also responded to negative selection, changing from 3 h of signalling in the parental generation to around 1 h of signalling by the fourth generation.

The disparity in the heritability values between the two studies could result from differences in geographic location and/or time. San Antonio and Austin are 127 km apart. Furthermore, the two experiments were conducted 25 years apart. Location and time could affect gene frequencies. Gene frequency differences would influence all of the genetic components of variance and could, therefore, affect the heritability estimates (Falconer and Mackay, 1996).

Overall, the large differences in the heritability values observed between Texas field crickets in San Antonio and Austin should encourage researchers to carefully evaluate heritabilities in their study populations. If this is impossible, extreme care should be taken before assuming that the heritability value of a nearby population is representative of the population being studied.

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