

The social environment shapes fitness through indirect genetic effects in spotted hyenas

Kasha Strickland¹, Matthew J. Silk¹, Oliver P. Höner^{2,3}, Eve Davidian³, Loeske E.B. Kruuk¹, Joel L.

Pick^{1*}

1. Institute for Ecology and Evolution, School of Biological Sciences, University of Edinburgh, UK
2. Department of Evolutionary Ecology, Leibniz Institute for Zoo and Wildlife Research, Berlin, Germany
3. Evolutionary Anthropology Team, Institute of Evolutionary Science of Montpellier (ISEM), University of Montpellier, CNRS, IRD, Montpellier, France

* corresponding author: joel.pick@ed.ac.uk

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Abstract

Additive genetic variance in fitness determines the rate of adaptive evolution. Yet, estimates from wild populations often exceed observed rates of contemporary evolution and realised population growth rates, suggesting that important sources of variation may remain unaccounted for. One important source of variation rarely fully accounted for is the social environment, because social interactions are known to influence survival and reproduction across taxa. Here, we combined social network analysis with quantitative genetic animal models in a wild population of spotted hyenas (*Crocuta crocuta*) inhabiting the Ngorongoro Crater, Tanzania, to explore how estimates of genetic variance in fitness are affected by the inclusion of social environment effects. In line with previous research, standard animal models recovered significant additive genetic variance in lifetime reproductive success. However, this variance substantially reduced to near zero when we accounted for shared social and spatial environments, indicating that previous estimates likely overestimated direct additive genetic variance. Instead, we found strong indirect genetic effects from social associations on fitness. Fine-scale spatial overlap also explained substantial variation in fitness whereas non-genetic indirect effects from social associations were comparatively small. Our results demonstrate that heritable variation in fitness may be expressed predominantly through social interactions rather than individuals' own genotypes in this population. More generally, our results suggest that incorporating realistic social environments into quantitative genetic models may be essential in understanding and predicting adaptive evolution in natural populations.

Keywords: Fitness, social evolution, quantitative genetics, social interactions, social networks, adaptive potential

22 **Introduction**

23 Evolution by natural selection depends on there being heritable variation in fitness. Accordingly,
24 Fisher's fundamental theorem equates additive genetic variance in fitness with the expected per-
25 generation increase in mean fitness caused by selection (R. A. Fisher, 1930), making it one of the
26 key parameters in determining a population's capacity for adaptive evolution. Estimates from wild
27 populations have demonstrated substantial additive genetic variance in lifetime fitness across
28 diverse taxa (Bonnet et al., 2022; Hendry et al., 2018; L. E. B. Kruuk et al., 2000; Tuliozi et al., 2026).
29 Yet, current estimates may overlook important ecological sources of variation in fitness that
30 quantitative genetics models seldom fully account for. Identifying these additional sources of
31 variation may therefore be needed to accurately predict the evolutionary dynamics of natural
32 populations.

33 Although fitness is often treated as an emergent property of an individual's own traits, it is
34 also shaped by the individuals it interacts with. The social environment, which describes the sum of
35 an individual's social interactions, is among the most pervasive environments experienced by
36 animals and has long been recognised as a major determinant of fitness (Alexander, 1974; Clutton-
37 Brock, 2021). For example, social relationships can determine access to food and breeding
38 opportunities, social partners can facilitate information transfer, and group composition can
39 influence exposure to predation, disease or other environmental risks (Snyder-Mackler et al., 2020).
40 Consequently, the social environment shapes the conditions under which individuals survive and
41 reproduce, generating variation in health, survival and reproductive success. Although the
42 importance of social environments for determining fitness is well recognised in behavioural ecology

(Krause & Ruxton, 2002; Rubenstein & Abbot, 2017), its contribution to fitness variation is seldom fully incorporated into quantitative genetic models used to estimate additive genetic variance in fitness and predict the response to selection. This omission may cause additive genetic variance in fitness to be misestimated, which could contribute to any observed discrepancy between predicted and realised evolutionary change (Danchin et al., 2011; Pujol et al., 2018).

A key mechanism through which social environments may contribute to variation in fitness is through indirect genetic effects (IGEs), which describe how the genes in one individual influence the fitness of another as a result of their social interactions (Falconer, 1965; Griffing, 1967; Moore et al., 1997). Importantly, because social partners are themselves genetically variable, part of the variation in an individual's social environment is also heritable, allowing IGEs to contribute to heritable variation in fitness alongside direct genetic effects (Bijma, 2010b; McGlothlin et al., 2010; Moore et al., 1997; Wolf, 2003). Consequently, failing to account for IGEs may lead to inaccurate estimates of adaptive potential (D. N. Fisher & McAdam, 2019). Importantly, the genetic covariance between direct and indirect effects, which describes whether genotypes that increase an individual's own fitness also tend to increase or decrease the fitness of its social partners, can influence both the magnitude and direction of evolutionary responses to selection (Bailey et al., 2018; Falconer, 1965; Moore et al., 1997; Wolf et al., 1998, 1999). In competitive social environments, theory predicts that this covariance may be negative because genotypes that increase an individual's own fitness may reduce the fitness of social partners through competition for limited resources (Moore et al., 1997; Wolf et al., 1998, 1999). Thus, ignoring IGEs can obscure an important source of variance in fitness and lead to misleading predictions of evolutionary change. Although empirical evidence for IGEs in behavioural, morphological, and life-history traits has

65 accumulated in laboratory, agricultural, and wild populations (Santostefano et al., 2025), estimates
66 of IGEs on fitness remain extremely rare and restricted to domesticated species (D. N. Fisher et al.,
67 2019; Santostefano et al., 2025).

68 Estimating social effects on fitness, including the estimation of IGEs, requires an explicit
69 representation of each individual's realised social environment. Yet social interactions are dynamic
70 and heterogeneous in strength, making it difficult to define a biologically meaningful and statistically
71 tractable measure (De Moor & Brent, 2025). Most empirical approaches have relied on dyadic
72 interaction data, treating social partners as independent and equally weighted, providing a
73 simplified representation of the broader social environment (Baud et al., 2022; D. N. Fisher et al.,
74 2019; Wilson et al., 2011). Social network analysis provides a natural solution because it explicitly
75 captures both the identity and strength of interactions among individuals (De Moor & Brent, 2025;
76 D. N. Fisher & McAdam, 2017). By integrating social network structure into quantitative genetic
77 animal models, the contribution of each social partner can be weighted by empirically estimated
78 association strengths, allowing social effects on fitness to be estimated under natural conditions
79 (D. N. Fisher & McAdam, 2017; Frère et al., 2010; Thomson et al., 2018).

80 Here we combined social network analysis with quantitative genetic animal models to test
81 whether the social environment contributes to lifetime fitness and whether explicitly accounting for
82 shared social environments alters estimates of additive genetic variance. We did so using data
83 collected as part of a longitudinal study of spotted hyenas (*Crocuta crocuta*) in the Ngorongoro
84 Crater, Tanzania. Spotted hyenas provide an exceptional opportunity to estimate the effect of social
85 environments on fitness as they live in complex fission–fusion societies with female philopatry

(Höner et al., 2005; H. Kruuk, 1966), nepotistic dominance hierarchies (Holekamp & Smale, 1991; Vulloud et al., 2019), and pronounced reproductive skew in both sexes (Engh et al., 2002; Mosna et al., 2026), generating persistent social structures that affect individual fitness across hyenas' lifetimes (Holekamp et al., 1996; Höner et al., 2010). The long-term study of the Ngorongoro Crater population combines over three decades of behavioural observations with detailed demographic records with a multigenerational pedigree, allowing reconstruction of social environments and genetic relationships for thousands of individuals. Additionally, previous work in this population has demonstrated substantial additive genetic variance in lifetime fitness (Bonnet et al., 2022), providing evidence of contemporary adaptive evolution and an opportunity to test whether those estimates are influenced by social environment effects. Due to the extremely social and nepotistic nature of hyena groups, we expected that there should be strong social effects on fitness, and that accounting for these would reduce the direct additive genetic variation in fitness. We used empirically derived social association strengths to define each individual's realised social environment to test the extent to which accounting for social environments impacts our estimates of additive genetic variance in fitness using a multi-matrix animal model design (Frère et al., 2010; Thomson et al., 2018). We then used social associations within an indirect-effects framework to partition variation in fitness into direct genetic effects, indirect genetic effects from social partners, and shared environmental effects. In doing so, we provide one of the first empirical tests of indirect genetic effects on fitness in a wild population.

Methods

Study population and data collection

We used data collected during a long-term individual-based study of spotted hyenas in the Ngorongoro Crater in Tanzania (approx. 250 km², 3°11' S, 35°34' E). The population consists of eight social groups (called clans) and is part of the wider hyena metapopulation of the Greater Serengeti-Mara ecosystem (Davidian et al., 2016) and has been continuously monitored since 1996. All hyenas in the population are individually identified by their spot pattern and colouration of their coat, as well as other uniquely identifiable features (e.g., scars and ear notches, described in (Davidian et al., 2016; Höner et al., 2007)). Data were collected via surveys of the Crater conducted on five to seven days a week, during which observations were made from vehicles to which hyenas are habituated from birth (Davidian et al., 2016, 2021). Surveys consisted of systematic searches of the Crater during which the GPS coordinates of all encountered individuals sighted are recorded as well as demographic (e.g., births, deaths), life-history (e.g., dispersal) and behavioural data (e.g., social, mating and competitive interactions). Birth and death dates are rarely observed directly in the wild but are estimated using a combination of resighting records, behavioural observations, and morphology (Davidian et al., 2016; Dheer et al., 2022). Social ranks of all individuals are determined via established social conventions of maternal inheritance and coalitionary support, and verified via behavioural interaction data (Davidian et al., 2021).

DNA was extracted from tissue, faecal, skin, or hair samples collected opportunistically. Parentage was assigned using a combination of nursing observations and genetic parentage analyses based on nine polymorphic microsatellite loci implemented in CERVUS 3.0, which has been validated extensively in this population (Höner et al., 2010; Kalinowski et al., 2007). Here we used records collected between April 1996 and February 2025. The full pedigree used contained 3,265 individuals sampled across a maximum depth of nine generations. Of these 3,265 individuals,

1,843 had both parents identified, 966 had only the mother identified, and four had only the father identified, leaving 452 with no known parentage. Data were compiled into a centralized database and processed using the hyenaR package version 0.10.0.9000 (Bailey et al., 2025).

Field data collection and transport was permitted by the Vice President's office of the United Republic of Tanzania (Ref. No. BA 78/130/01/42), the Tanzania Wildlife Research Institute (TAWIRI), the Tanzania Commission for Science and Technology (COSTECH; Permit No. 2021-380-NA-1990), and the Ngorongoro Conservation Area Authority (NCAA). All study procedures were performed in compliance with the ethical regulations of these institutions and the Internal Committee for Ethics and Animal Welfare of the Leibniz Institute for Zoo and Wildlife Research Berlin (No. 2020-06-02).

Estimation of social networks and covariance of social environment

We calculated a social association network using repeated observations of individuals collected between 12 April 1996 and 1 September 2025. Individuals were assigned to the same social group using a chain rule whereby observations occurring within 50 m and 60 min of one another were considered part of the same grouping event. This threshold was selected based on previous association thresholds used in this population based on extensive field experience and focal observations of grouping behaviour (Davidian et al., 2021). Individuals varied substantially in their number of observations across their lifetime, ranging from one to 700 (median = 24). To reduce uncertainty in estimated association strengths associated with heterogeneous sightings records while also minimising bias arising from the exclusion of individuals with low fitness (Croft et al., 2011), we restricted the network to only include individuals observed at least five times. Higher thresholds would disproportionately exclude individuals that died young and therefore had both

fewer observations and lower lifetime reproductive success (see *Statistical analysis*). The dataset used for calculating a social association network included a total of 176,171 observations of 2,570 individuals, of which 2,354 had a known mother and 1,810 had a known father (observations per individual: median = 36, range = 5 - 700, inter-quartile range = 16 - 87).

We estimated the strength of dyadic social associations from observations of group membership using the simple ratio index (SRI), estimated as $SRI = N_{ab} / (N_{ab} + N_a + N_b)$ where N_{ab} is the number of times individuals a and b are seen associating in the same group, and N_a and N_b are the number of observations individuals a and b are seen without the other, respectively (Whitehead, 2008). Association strengths were used to weight the extent to which individual a exerts an indirect effect on the fitness of individual b (see below) and the equation for SRI presented above results in a symmetric adjacency matrix that assumes that the effect pairs of individuals have on each other is symmetric. However, this assumption may be inappropriate when two individuals overlap only partially in time. In such cases, the potential influence of individual b on individual a should be scaled relative to the period during which a was alive and available to interact. For example, individual a lives for 1 year, during which time it is always observed with individual b , but individual b lives for 10 years. The relative effect of individual b on individual a is likely to be much larger than the effect of individual a on individual b , as individual b experienced much of its life in the absence of individual a . We therefore calculated the SRI matrix after truncating the observation dataset used for the calculation of each row of the matrix (each individual) to the period spanning that individual's lifespan. This produced an asymmetric association matrix in which association strengths were expressed relative to the observation period for the individual in that row. Social association

networks were calculated using the *asnipe* R package (Farine, 2013) and custom R scripts (see data availability statement).

To estimate similarity in social environments experienced by individuals, we calculated weighted Jaccard dissimilarity calculated from the asymmetric social association network described above (hereafter, social similarity) where 1 describes pairs of individuals that share the exact same social environment, and zero is where they share none. We selected to calculate social similarity in the asymmetric social network (rather than, for instance, recalculating a symmetric one) in order to account for heterogeneous temporal overlap between individuals, as described above. Specifically, social similarity of the social network allowed us to estimate similarity in realised social environments, assuming that some elements of the social environment may not be encoded in the social network. The social similarity matrix was estimated using custom R scripts, following equations for weighted Jaccard dissimilarity set out in (Harbert, 2026).

Home range overlap

We calculated home range overlap (HRO) between individuals to account for shared environment effects that may bias the estimation of both additive genetic effects and indirect genetic effects when social partners and relatives share common environments (L. E. B. Kruuk & Hadfield, 2007; Stopher et al., 2012; Thomson et al., 2018). We estimated individual home ranges for all individuals with at least five relocations (as above) using kernel density estimation implemented in the R package *adehabitatHR* (Calenge, 2006). We used repeated location observations to estimate an individual utilisation distribution (UD), representing the probability of observing an individual at a given location (Worton, 1995). UDs were estimated on a grid with a 100 m pixel size using the ad hoc

method to determine individual-specific smoothing parameters (Calenge, 2006). We added 50 m to each smoothing parameter to match the spatial threshold used in social network construction and to avoid restricting UD to a single grid cell. Home range overlap was then estimated using the volume of the intersection (VI) of 95% of UD to represent that majority of individuals' home ranges (Fieberg & Kochanny, 2005). Home range overlap matrices were symmetric and ranged from 0 (complete segregation of UD) to 1 (identical UD) (see (Fieberg & Kochanny, 2005) for more detail). We acknowledge that five sightings may not be sufficient to estimate highly precise UD for individuals with lower number of sightings, but as above, restricting home range analyses to more sightings would systematically remove individuals with low fitness (who died young and had few relocations). Nonetheless, we believe that five sightings should be sufficient to estimate an approximate home range per individual that can be used to account for shared environment effects.

Measuring fitness

We measured fitness as lifetime reproductive success (LRS), defined as the total number of offspring produced by each known individual. LRS was calculated using the pedigree described above and includes offspring identified after emerging from the birth den (at a mean age $\pm$ standard deviation of 84.1 ± 73.5 days (White et al., 2025)). To ensure complete lifetime records, we included only individuals born after the start of the study (1996) and known to have died by February 2025. In order to avoid only including shorter lived individuals from more recent cohorts, we excluded individuals born after 2015. We chose 2015 because fewer than 10% of hyenas survive beyond 10 years of age. The final dataset comprised 1,338 Crater-born individuals for which maternal identity

and social rank at birth were known and were also contained within the social network and home range overlap estimations described above.

Statistical modelling

We analysed LRS using Bayesian generalised linear mixed effects models with a negative binomial error distribution and a log link, chosen to account for overdispersion in LRS. All models included sex, ordinal social rank at birth, clan size at birth (number of individuals), inbreeding coefficient (F) and proportion of immigrant genetic ancestry as fixed effects. F was estimated from the pedigree using the *gggroups* R package (Nilforooshan & Saavedra-Jiménez, 2020). Proportion of ancestry to immigrants was estimated with the *nadiv* R package (Wolak, 2012) and was included to account for the positive effect of immigrant ancestry on fitness in hyenas (Strickland et al., 2025; Wolak & Reid, 2017). Models also included maternal identity, birth year and birth clan as random effects to account for shared maternal, temporal and social-group environments (Gicquel et al., 2022; Höner et al., 2010). Additive genetic effects were modelled using an animal model parameterised directly from the population pedigree and Mendelian sampling variances (Damgaard, 2007; Quaas, 1989). For all models, fixed effects were assigned Gaussian priors with mean 0 and standard deviation 10, standard deviations for random effects and the negative binomial overdispersion parameter were assigned half-Student-t priors with 3 degrees of freedom, location 0 and scale 10 ($\sigma \sim \text{Student-t}+(3,0,10)$) and a LKJ prior on the direct-indirect genetic correlation. All models were fitted in Stan using the *rstan* R package (Stan Development, 2025). The first model was run for 30,000 iterations, while subsequent models were run for 10,000 iterations; all models used 2,000 warm-up iterations across three chains. Convergence was assessed using trace plots, effective sample sizes, and Rhat

values (< 1.01). Posterior distributions were summarised using posterior means and 95% credible intervals. Models were compared by examining changes in estimated variance components. Random effect standard deviations and variance estimates presented are on the latent (log) scale, but variance estimates across draws of the posterior were also converted to data scale using QGglmm R package (de Villemereuil et al., 2016).

The first model ('baseline' model) we fitted was a standard animal model including only direct additive genetic effects in addition to maternal, birth-year and birth-clan random effects. This model provided a baseline estimate of direct additive genetic variance (V_A) in fitness without estimating any social or other environmental effects.

Second, to assess whether social environments contribute to variation in fitness and bias estimates of additive genetic variance, we fitted a model in which social effects were represented as the covariance among individuals experiencing similar social environments (the 'social similarity' model), using the social similarity matrix described above (Frère et al., 2010; Thomson et al., 2018). This tested whether individuals with similar social associations also have more similar fitness. We fitted this model as a multi-matrix animal model incorporating covariance structures defined by both the social similarity matrix and the home-range overlap matrix explained above (Thomson et al., 2018). Inclusion of the home-range overlap matrix accounted for the possibility that socially associated individuals experience similar ecological conditions because they occupy similar areas of the landscape (Webber & Vander Wal, 2018). This model therefore tested whether variance previously attributed to additive genetic effects could instead be explained by shared social and spatial environments.

254 Third, we tested whether the mechanisms associated with social environment effects could
255 be attributed to indirect effects from social associations (i.e., whether individuals have a consistent
256 effect on the individuals they associate with, the 'indirect effects' model). Here, indirect effects were
257 estimated by specifying the design matrix for the indirect-effects random effect as the asymmetric
258 social association matrix. Under this framework, the fitness of each focal individual depended on
259 the sum of the weighted contributions of its social partners, with association strengths determining
260 the relative contribution of each partner (Fisher & McAdam, 2019; Moore et al., 1997). The social
261 association matrix included as a random effect design matrix included the $N = 1,337$ individuals
262 included in analyses (see above) as rows with $N = 2,456$ potential social partners as columns which
263 can exert an indirect effect on the focal individuals fitness when they associated for at least part of
264 the time. As above, the model also included the home-range overlap matrix to account for shared
265 environmental effects. This model partitioned variation in fitness into direct additive genetic effects,
266 indirect social effects arising from interaction partners, and shared environmental effects.

267 Finally, we extended the indirect-effects framework to decompose indirect effects from
268 social associations into environmental and genetic components (the 'IGE' model). In this model,
269 indirect genetic effects were modelled as additive genetic effects expressed through social
270 partners, allowing estimation of direct additive genetic variance, indirect additive genetic variance,
271 and the covariance between them. By partitioning indirect effects into genetic and environmental
272 components, this model quantified the extent to which heritable variation in fitness may be
273 mediated through social interactions.

To evaluate the ability of the variance-partitioning model to recover direct additive genetic, indirect genetic, indirect environmental, maternal, temporal, clan and spatial variance components, we also conducted simulation-based power analyses. We generated 100 datasets using the observed pedigree, social association matrix, home-range overlap matrix, fixed effects and sample size, with parameters set from randomly drawn posterior samples of the fitted empirical models. Each simulated dataset was refitted with the corresponding model, and coverage was assessed from the proportion of simulations in which the true parameter values were recovered within the 95% posterior credible intervals.

Results

The median number of associates per individual in the full social network of 2,570 individuals was 79 (min = 0, max = 468) and 96 among individuals included in phenotypic analyses (i.e., excluding those with under 5 observations: min = 8, max = 464, Figure 1). We found weak to moderate positive correlations among home range overlap, social association, social similarity, and pairwise relatedness (Figure 1). In particular, social similarity showed the strongest association with home range overlap ($r = 0.634$), whereas the remaining relationships were weaker ($r = 0.245\text{--}0.337$).

Most of the fixed effects estimates were qualitatively similar across our different models (Table 1). Clan size at birth always negatively predicted LRS; higher social rank was always associated with higher LRS; males always had slightly fewer offspring than females, reflecting the slightly lower rate of paternities assigned than maternities in the pedigree (see methods); and the estimated effect of the proportion of immigrant ancestry on LRS was negative in the baseline model, although the 95% credible interval overlapped zero, and was substantially attenuated in the

remaining models. The effect of inbreeding (F) on LRS, however, differed between models that did and did not include social and shared environment effects. In the baseline model that excluded social similarity, home-range overlap and indirect effects, F negatively predicted LRS, consistent with previously reported inbreeding depression in this population (Chan et al., 2026). In models incorporating social similarity or indirect effects alongside home-range overlap, the negative association between F and LRS weakened, with 95% credible intervals overlapping zero. This suggests that the estimated effect of inbreeding was sensitive to social and spatial covariance structures, although the reasons for this remain unclear. One possibility, which remains to be tested, is that individuals with similar levels of inbreeding share aspects of their social or spatial environments.

In the baseline model that estimated V_A without accounting for social-environment effects via either a social similarity matrix or an indirect-effects framework, the posterior for V_A in LRS on the latent scale was clearly distinct from zero, with a posterior median of 0.118 (95% CI = 0.001–0.362; Table 1, Figure 2). However, in models that included either a social similarity matrix or indirect effects (together with home-range overlap), estimates of V_A collapsed (Figure 2). In each model that accounted for social and shared environment effects, the posterior mean for V_A declined by more than an order of magnitude, providing limited evidence for V_A in LRS after accounting for social and spatial environmental effects. These results indicate that much of the variation previously attributed to additive genetic effects in the baseline animal model is instead explained by shared social and spatial environments.

In the ‘social similarity’ model, substantial variance was attributed to both social similarity and home-range overlap. Variation associated with social similarity (V_{ss}) had a posterior median of

0.903 [0.715–1.105], while variance explained by home-range overlap (V_{HRO}) was even larger, with a posterior median of 1.810 [1.292–2.406] (Table 1, Figure 2). Both variance components were clearly distinct from zero, indicating that individuals experiencing similar social and spatial environments exhibited more similar LRS, and that social and fine-scale spatial environments contributed substantially to fitness variation in the population. Simulation-based posterior predictive analyses showed that this model generally recovered the fixed and random effects with good accuracy, with coverage above 90% for all parameters.

In the ‘indirect-effects’ model, in which the social association matrix defined the contribution of social partners to each individual's phenotype, indirect effects arising from social associations explained substantial variation in LRS. The variance attributable to indirect effects (V_{IE}) was very similar to that explained by social similarity, with a posterior median of 0.896 [0.486–1.394], whereas the variance explained by home-range overlap was substantially larger (posterior median = 4.518 [3.660–5.457], Table 1, Figure 2).

Finally, decomposing indirect effects arising from social associations into environmental and genetic components (the ‘IGE’ model) revealed that the majority of indirect-effect variance was attributable to indirect genetic effects. The posterior median for indirect genetic variance (V_{IGE}) was 0.736 [0.420–1.114], whereas the environmental component of indirect effects (V_{IEE}) was comparatively small (posterior median = 0.080 [0.000–0.301]; Table 1, Figure 2). Together, these results suggest that the heritable component of variation in LRS is expressed predominantly through the genotypes of social partners rather than through individuals' own direct genetic effects. There was evidence for a small positive covariance between V_A and V_{IGE} . The posterior mean covariance was 0.01 [0.001 - 0.033] which translated into a correlation between V_A and V_{IGE} of 0.21 [0.068 -

0.348]. Simulation-based posterior predictive analyses indicated that the full variance-partitioning model generally recovered the true fixed and random effects well, with particularly strong recovery of indirect genetic variance (94%), direct-indirect genetic covariance (100%), and shared environmental effects such as cohort (93%) and clan variance (91%). Recovery was lower for direct additive genetic (68%) and maternal variance (60%), which were empirically very small and had posterior distributions close to zero; thus, although these components were less precisely recoverable, the analyses provide reassurance that the data are sufficient to rule out large direct genetic or maternal contributions to LRS.

All variance estimates presented above are on the latent (log) scale, but variance estimates on the observed data scale can be found in Table 2. Although variance estimates on the latent scale quantify variation in the linear predictor attributable to each random effect, the observed-data scale estimates provide a representation of how such variance translates into differences in LRS, thereby facilitating a more biologically interpretable scale of variance associated with each random effect. Despite differences in absolute variance estimates between scales, the relative contributions of random effects appear broadly consistent between scales. Among other random effects, variance attributable to maternal identity remained consistently low across all models (Table 1, Figure 2). Estimates of cohort and clan variance changed markedly among models as additional social and spatial covariance structures were introduced, indicating that these variance components were sensitive to how shared social and environmental effects were partitioned (Table 1, Figure 2). This may reflect similarities in social networks and home ranges among clan members, or age-related structure in social associations, although these possibilities remain speculative. Home-range overlap consistently accounted for the largest non-genetic source of variation in LRS in models that

included shared environmental effects (Table 1, Figure 2), emphasising that fine-scale spatial heterogeneity in environments contributes substantially to variation in fitness.

Discussion

Our analyses show that incorporating social interactions into quantitative genetic models of fitness fundamentally changes the interpretation of heritable variation in fitness in wild spotted hyenas, measured here as lifetime reproductive success. A standard animal model recovered significant direct additive genetic variance in fitness, consistent with previous work in this population (Bonnet et al., 2022). However, this variance approached zero once shared social and spatial environments were accounted for. When we modelled these social effects directly as indirect effects via social associations, most of the heritable component of fitness variation was explained by indirect genetic effects rather than by direct additive genetic effects, whereas non-genetic indirect effects were comparatively small. These findings suggest that, in this population of a nepotistic group-living carnivore, heritable variation in fitness is expressed predominantly through social interactions and that conventional animal models likely overestimate direct additive genetic variance when realistic social and spatial environments are omitted.

As expected, incorporating the social and spatial environments experienced by individuals largely removes the direct additive genetic variance in lifetime reproductive success, suggesting that previous estimates of substantial additive genetic variance in this population (Bonnet et al., 2022) may have captured variation attributable to unmodelled environmental variation (Frère et al., 2010; Stopher et al., 2012; Thomson et al., 2018). Accounting for social and spatial environments is particularly important in kin-structured populations, in which relatives tend to share social and

spatial environments, potentially inflating estimates of additive genetic variance if these effects are not accounted for (Hamilton, 1964; L. E. B. Kruuk & Hadfield, 2007; Lukas & Clutton-Brock, 2018; Pereira et al., 2023). Importantly, our simulations suggested that the full indirect-effects model had relatively limited ability to recover very small direct additive genetic variance. However, the social-similarity model recovered variance components fairly well, and both modelling approaches converged on the conclusion that direct additive genetic variance in fitness is close to zero. Together, these results suggest that the low estimate of direct additive genetic variance is unlikely to be a statistical artefact and instead reflects a genuine feature of fitness variation in this population.

Interestingly, the social-similarity and indirect-effects frameworks produced very similar estimates of variance in fitness associated with the social environment. Biologically, the close correspondence between the two approaches might suggest that social effects on fitness are captured primarily by indirect effects arising from social associations, rather than by broader group-level social conditions that may influence fitness beyond specific social associations. Such conditions could include group size, the age, sex and relatedness composition of groups, and clan-wide dominance structure, which may affect fitness through processes not fully captured by individual social associations (Araya-Ajoy, Westneat, and Wright 2020; Silk 2002; Vulliamy et al. 2019). However, similar estimates of variance do not necessarily indicate that the two approaches capture the same underlying processes, particularly given the discrepancy in the shared-environment effect between otherwise identical models.

From a statistical perspective, our results suggest that when the primary objective is to obtain accurate estimates of direct additive genetic variance while accounting for social effects, a

multi-matrix approach based on the estimation of social covariance between individuals may account for social environment effects without requiring the more complex indirect-effects parameterisation (Frère et al., 2010; Stopher et al., 2012; Thomson et al., 2018). Previous studies have suggested using the social network directly as a measure of the social environment (Frère et al., 2010; Thomson et al., 2018), assuming that similarity between individuals in their social environment can be represented by their direct interaction. We instead derive a social similarity matrix from the social network, reflecting the similarity between two individuals in their associations with others, thereby more directly capturing how similar two individuals should be as a consequence of the indirect effects acting on them. Importantly, the social network and social similarity matrices will estimate a similar amount of variance only if there is a positive covariance between direct and indirect effects, because this will cause interacting individuals to experience similar effects. However, the social network would not detect variance if there was zero or a negative covariance, in which case interacting individuals would be dissimilar (Fisher & McAdam 2017). We therefore suggest that deriving social similarity from the social network, rather than using the social network itself, may provide a more suitable representation of social similarity.

A substantial proportion of indirect effect variance in fitness had a genetic basis. Although there is considerable theory that suggests that such effects should be common, estimates of IGEs on fitness remain very rare in wild populations (Santostefano et al., 2025). Our results show that the effects that social partners have on one another's reproductive success are themselves genetically variable. Given the stable and structured social relationships characteristic of spotted hyena societies (H. Kruuk, 1972), genetic variation in the effects individuals have on one another through social associations could itself be a target of selection or other evolutionary processes. In this way,

IGEs on fitness provide a mechanism through which genetic variation can be expressed both on individuals' own fitness, and on the fitness consequences they generate for their social partners, potentially driving evolutionary change in sociality (Martin et al., 2025).

The presence of substantial IGEs, and a positive covariance between direct and indirect genetic effects, alters our inference about the population's evolutionary potential. If the rate of adaptive evolution was predicted from direct additive genetic variance alone, the expected response would be very small. However, including indirect genetic effect variance indicates that the total amount of genetic variation contributing to fitness is likely considerably larger (Bijma, 2010b). Importantly, this indirect genetic variance cannot simply be interpreted as additional additive genetic variance: because it is expressed through social interactions, its contribution to evolutionary change would depend on the social environment and on the covariance between direct and indirect genetic effects (Bijma & Wade, 2008). In our population, the positive covariance between direct and indirect genetic effects suggests that genotypes associated with higher individual fitness also tend to confer positive fitness effects on social partners. Theory predicts that a positive covariance will increase the rate of adaptive evolution (Bijma & Wade, 2008; McGlothlin et al., 2010; Wolf et al., 1998). In spotted hyenas, this covariance could plausibly arise because social relationships are closely tied to rank and access to resources: offspring inherit a rank immediately below their mothers at birth (East et al., 2009), and close maternal and sibling alliances can provide priority access to kills, benefiting both partners (Holekamp et al., 1996; Ilany & Akçay, 2016). Similar mechanisms may operate in other socially structured mammals, including some primates, where maternal inheritance of social status likewise structures social relationships and access to resources. Our results are therefore consistent with the possibility that social interactions

contribute to accelerating evolutionary change in this population, although further work is needed to determine how these predictions translate into realised genetic evolution.

It is important to note that our estimation of social environment effects represents a fairly broad overview of possible effects from social associations which may miss interesting complexity or nuances in how social environments influence individual fitness. In particular, estimating social environment effects and IGEs on lifetime fitness is challenging because social environments are highly dynamic across both time and ontogeny (Pinter-Wollman et al., 2013; Wild et al., 2024; Woodman et al., 2024) and the strength of social effects is unlikely to be constant across developmental stages. In species with prolonged maternal care and extended development, such as spotted hyenas, social interactions experienced early in life may have stronger indirect effects on later fitness than interactions occurring during adulthood. In addition, the influence exerted by any given social partner may be diluted as the number of social associates or group size increases (Bijma, 2010c, 2010a; Heidaritabar et al., 2019). We did not model such an effect explicitly because the manner in which dilution operates is unlikely to be constant through time, and implementing a biologically realistic formulation would require strong assumptions about how social influence scales with changing group composition and association structure across hyenas' lifetimes. Instead, we used an asymmetric social association network that standardized association strengths relative to each individual's lifetime and included group size, social rank, and maternal identity as covariates or random effects to account more broadly for variation associated with social structure and development. Nevertheless, the appropriate representation of social environments is likely to be highly system- and dataset-specific, and caution should be applied when applying similar approaches to other empirical systems, with careful consideration of species biology and sampling

structure. Finally, we note that indirect genetic effects were relatively strong in this population, and our data had good power to detect effects of the magnitude observed. When indirect genetic effects are weaker, substantially larger datasets will likely be required to estimate them reliably, as illustrated by our supporting simulations.

In conclusion, our study shows that incorporating realistic social environments can alter estimates of heritable variation in fitness in a wild population. In spotted hyenas, the additive genetic variance in lifetime reproductive success inferred from a conventional animal model largely disappeared once shared social and spatial environments were accounted for and we found substantial indirect genetic effects associated with social associations. These findings suggest that a significant component of evolutionary potential may reside in the heritable effects individuals have on one another rather than in direct genetic effects alone. More broadly, our results highlight that predicting adaptive evolution in natural populations requires moving beyond models that treat individuals as socially independent and instead integrating the social structure through which fitness is expressed and inherited.

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Data and code availability: R code and data needed to reproduce analyses and results, including all tables and figures, can be found at (<https://github.com/kashastrickland/hyenaindirecteffects>)

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742

Tables and figures

743 **Figure 1.** Plots showing the relationship between relatedness, social associations and shared space
744 in 1,338 individual spotted hyenas included in analyses. (A) The social association network where
745 nodes are individuals that are coloured based on their clan of birth, (B) the relationship between the
746 extent of home range overlap (HRO) between pairs and their association strength, (C) the
747 relationship between pairwise relatedness estimated from the pedigree and their association
748 strength, and (D) the relationship between relatedness and home range overlap. In plots C - D,
749 points are pairs of individuals and they are coloured based on the sex of those individuals grouped
750 into three categories (female-female, female-male and male-male).

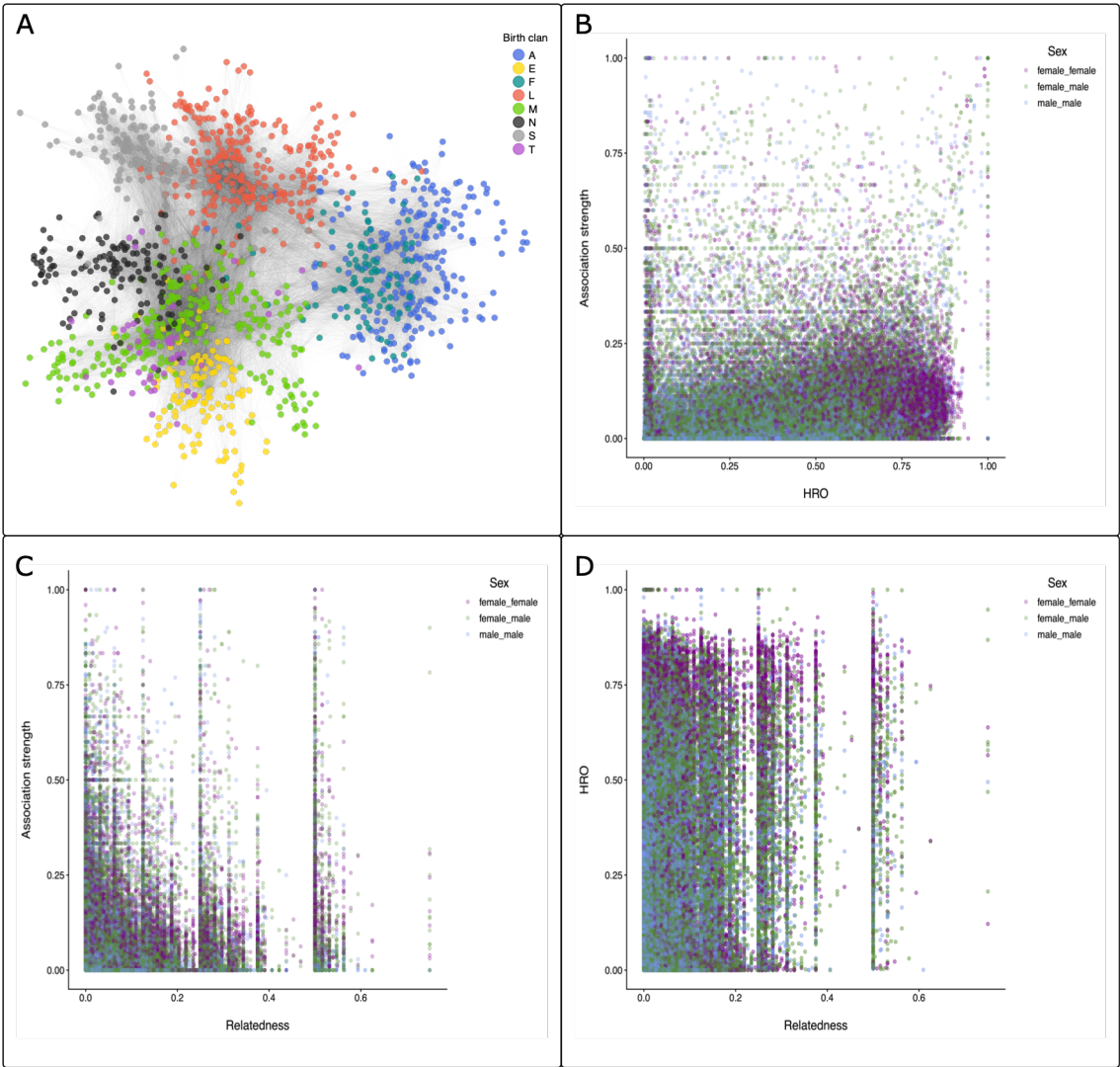
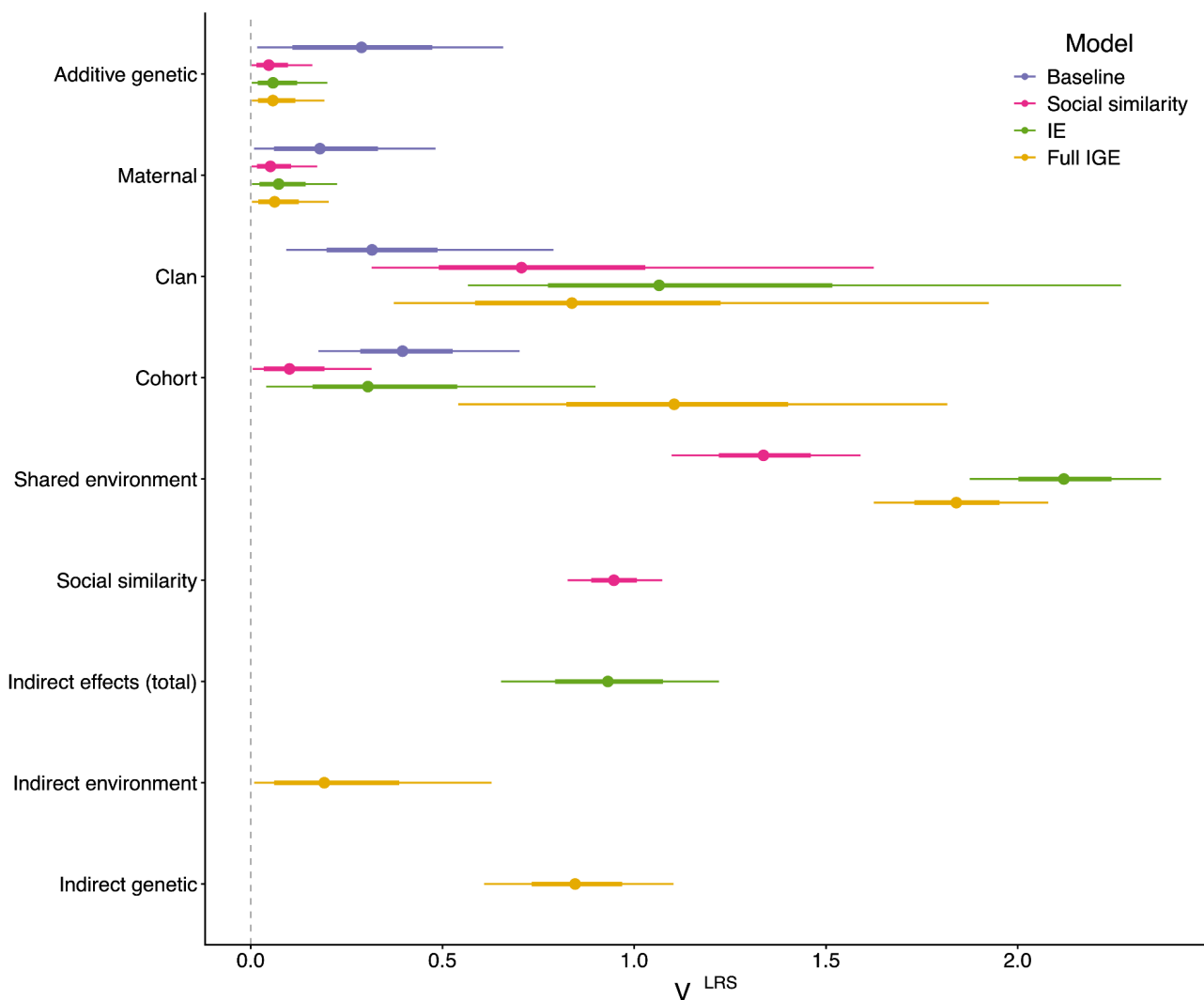


Figure 2. Posterior distributions of variance in LRS attributed to random effects components estimated in four animal models of different random effects structure. All models include maternal, clan and cohort level variance and colours then represent which additional variance components were included in the model: purple = just additive genetic variance; pink = additive genetic variance, shared environment effects via a home range overlap, and social covariance via a social similarity matrix describing similarity between individuals in their social associations; green = additive genetic variance, shared environment effects via a home range overlap, and indirect effects from social associations; gold = additive genetic variance, shared environment effects via a home range overlap, and indirect effects from social associations partitioned between environment and genetic effects. Points are medians of the posterior distribution, thick bars are 75% credible intervals and thin lines are 95% credible intervals.



764 **Table 1.** Table of results including fixed effects estimates and random effects variances for four models that differed in their random effects
765 structures. All models run with a total of 1,338 individual hyenas born into the Crater. Parameter estimates are all presented on latent (log)
766 scale and are posterior medians with 95% CI's in parentheses.

Parameter	Baseline	Social similarity	Indirect effects	IGE
Intercept	1.708 (1.169; 2.243)	-2.109 (-2.989; -1.244)	-3.004 (-4.226; -1.776)	-3.391 (-4.610; -2.193)
Clan size	-0.011 (-0.02; -0.003)	-0.024 (-0.032; -0.017)	-0.035 (-0.044; -0.024)	-0.025 (-0.037; -0.014)
F	-9.226 (-15.208; -3.174)	-4.059 (-9.271; 1.012)	-4.262 (-9.428; 0.67)	-3.124 (-7.961; 1.536)
Social rank	-0.035 (-0.045; -0.026)	-0.021 (-0.029; -0.012)	-0.022 (-0.03; -0.014)	-0.013 (-0.022; -0.005)
Sex _M	-0.553 (-0.764; -0.344)	-1.276 (-1.500; -1.058)	-1.405 (-1.635; -1.182)	-1.354 (-1.573; -1.138)
Immigrant ancestry	0.478 (-0.061; 1.051)	-0.041 (-0.423; 0.334)	-0.028 (-0.389; 0.335)	0.11 (-0.249; 0.469)
Overdispersion parameter (θ)	0.301 (0.264; 0.346)	63.947 (21.812; 150.478)	45.718 (15.723; 110.618)	44.402 (15.628; 104.32)
V_A	0.118 (0.001; 0.362)	0.005 (0.001; 0.019)	0.008 (0.001; 0.029)	0.007 (0.001; 0.028)
V_M	0.056 (0.001; 0.191)	0.006 (0.001; 0.023)	0.011 (0.001; 0.042)	0.008 (0.001; 0.031)
V_{Clan}	0.154 (0.016; 0.457)	0.712 (0.132; 1.955)	1.566 (0.386; 4.091)	0.995 (0.195; 2.700)
V_{Cohort}	0.184 (0.045; 0.410)	0.021 (0.001; 0.075)	0.173 (0.005; 0.614)	1.357 (0.405; 2.804)
V_{HRO}	NA	1.810 (1.292; 2.406)	4.518 (3.660; 5.457)	3.41 (2.746; 4.160)
V_{SS}	NA	0.903 (0.715; 1.105)	NA	NA
V_{IE}	NA	NA	0.896 (0.486; 1.394)	NA
V_{IEE}	NA	NA	NA	0.080 (0.001; 0.301)

	V_{IGE}	NA	NA	NA	0.736 (0.420; 1.114)
767	V_A = additive genetic variance; V_{Cohort} = between birth-year variance; V_{Clan} = between birth-clan variance; V_M = maternal effect variance; V_{HRO}				
768	= variance attributed to shared environments described in home range overlap matrix; V_{SS} = variance attributed to shared social				
769	environment via a social similarity matrix describing similarity between individuals social associations; V_{IE} = total indirect variance from				
770	social associations; V_{IEE} = indirect environmental effect variance from social associations; V_{IGE} = indirect genetic effect variance from social				
771	associations.				

Table 2. Random effects variance estimates on the observed data scale for four models that differed in their random effects structures. Parameters are summarised as posterior medians with 95% CI's in parentheses. Variance estimates across draws of the posterior were converted to data scale using QGglmm R package (de Villemereuil et al., 2016).

Parameter	Baseline	Social similarity	Indirect effects	IGE
V_A	3.692 (0.011, 51.720)	0.001 (0.000, 0.047)	0.008 (0.000, 1.981)	0.002 (0.000, 0.324)
V_M	1.508 (0.003, 21.938)	0.001 (0.000, 0.055)	0.011 (0.000, 2.884)	0.002 (0.000, 0.352)
V_{Clan}	4.748 (0.295, 72.510)	0.203 (0.009, 13.986)	2.820 (0.052, 1349.531)	0.492 (0.010, 98.186)
V_{Cohort}	7.599 (0.858, 59.467)	0.004 (0.000, 0.195)	0.237 (0.002, 45.198)	0.847 (0.018, 88.325)
V_{HRO}	NA	0.773 (0.064, 15.582)	11.900 (0.354, 1591.393)	2.502 (0.083, 168.017)
V_{SS}	NA	0.392 (0.037, 6.860)	NA	NA
V_{IE}	NA	NA	2.256 (0.064, 319.722)	NA
V_{IEE}	NA	NA	NA	0.024 (0.000, 3.577)
V_{IGE}	NA	NA	NA	0.521 (0.015, 38.865)