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EVOLUTIONARY POTENTIAL OF A LARGE MARINE VERTEBRATE: QUANTITATIVE GENETIC PARAMETERS IN A WILD POPULATION

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Estimating quantitative genetic parameters ideally takes place in natural populations, but relatively few studies have overcome the inherent logistical difficulties. For this reason, no estimates currently exist for the genetic basis of life-history traits in natural populations of large marine vertebrates. And yet such estimates are likely to be important given the exposure of this taxon to changing selection pressures, and the relevance of life-history traits to population productivity. We report such estimates from a long-term (1995–2007) study of lemon sharks (*Negaprion brevirostris*) conducted at Bimini, Bahamas. We obtained these estimates by genetically reconstructing a population pedigree (117 dams, 487 sires, and 1351 offspring) and then using an "animal model" approach to estimate quantitative genetic parameters. We find significant additive genetic (co)variance, and hence moderate heritability, for juvenile length and mass. We also find substantial maternal effects for these traits at age-0, but not age-1, confirming that genotype–phenotype interactions between mother and offspring are strongest at birth; although these effects could not be parsed into their genetic and nongenetic components. Our results suggest that human-imposed selection pressures (e.g., size-selective harvesting) might impose noteworthy evolutionary change even in large marine vertebrates. We therefore use our findings to explain how maternal effects may sometimes promote maladaptive juvenile traits, and how lemon sharks at different nursery sites may show "constrained local adaptation." We also show how single-generation pedigrees, and even simple marker-based regression methods, can provide accurate estimates of quantitative genetic parameters in at least some natural systems.

KEY WORDS: **Heritability, lemon shark, maternal effects, morphological traits, power and sensitivity analysis, sibship reconstruction.**

The rate and direction of evolution depends on a number of variables that include the strength of selection, genetic and environmental variances or covariances, and genotype–phenotype interactions between parents and offspring (maternal effects; Roff 1997; Lynch and Walsh 1998) or among individuals (indirect genetic effects [IGEs]; Wolf et al. 1998). These quantitative genetic parameters are thus critical to understanding phenotypic variation in the wild (Endler 1986) and for informing viable conservation strategies (see Stockwell et al. 2003; Ferrière et al. 2004; Carroll and Fox 2008). Most existing estimates of these parameters, however, come from studies of controlled populations (e.g., laboratory, greenhouse, domestic, hatchery) and may therefore bear little relation to the actual parameters in natural populations (Sgro and Hoffman 1998; Hermida et al. 2002; also see Charmantier and Garant 2005). For this reason alone, it is important to obtain estimates directly from the wild (Merilä et al. 2001; Kruuk 2004). In addition, estimates from controlled populations are not possible for some taxa, such as large marine vertebrates (sharks, pinnipeds, whales), and so estimates from wild populations are the only option here.

To date, relatively few quantitative genetic analyses have been performed for natural vertebrate populations—owing to the logistic constraints of incomplete sampling, unknown relatedness between individuals, and sometimes high vagility or even low philopatry. The few studies that have overcome these constraints, using methods to be described later, have focused mainly on passerine birds (e.g., Merilä et al. 2001; Sheldon et al. 2003; Garant et al. 2004) and insular ungulates (Réale et al. 1999; Kruuk et al. 2000; Coltman et al. 2005), as well as some small mammals (Réale et al. 2003), and freshwater fish (Thériault et al. 2007). These studies show that variance components can differ dramatically among populations, species, and even higher taxonomic levels. To date, however, we still do not have a single quantitative genetic estimate from a large marine vertebrate, and so predicting evolutionary change in the face of natural or anthropogenic influences is not possible. The present study begins to fill this gap by estimating quantitative genetic parameters in the lemon shark (*Negaprion brevirostris*), and integrating these estimates with our previous work on selection (DiBattista et al. 2007) and among-population phenotypic variation in this species (see Barker et al. 2005).

Trait heritability and genetic correlations have long been recognized as important predictors of evolutionary change. Only more recently, however, have we come to fully appreciate the importance of maternal effects in modifying evolutionary responses to natural selection (Mousseau and Fox 1998; Räsänen and Kruuk 2007), although animal breeders have earlier examined such effects in detail (e.g., Southwood and Kennedy 1990; Vanvleck et al. 1996). Maternal effects occur when the genotype (i.e., maternal genetic effects) or environment (i.e., maternal environmental effects) of the mother influences the phenotype of the offspring, independent of any genes she may pass on. Not all maternal effects have adaptive benefits for the offspring in a population, but will have evolutionary consequences whenever they drive phenotypic change (see Räsänen and Kruuk 2007). As one example, maternal effects may enable population persistence and thus facilitate future evolutionary change (see McAdam et al. 2002; Sheldon 2002; Räsänen et al. 2003; Badyaev 2005). As another, maladaptive maternal genetic effects can dampen selection on a trait and thereby slow its subsequent evolution (McAdam and Boutin 2003), or even drive populations away from fitness peaks (Kirkpatrick and Lande 1989). We are particularly interested in putative maternal effects in our study species because although the outcome is not obvious, it could have dramatic effects. On the one hand, sharks are placentally viviparous, which should enhance maternal effects (Mousseau and Fox 1998). On the other hand, sharks do not show parental care after birth (Pratt and Casey 1990), which should reduce maternal effects (see Reinhold 2002). Our results might therefore inform the current uncertainty regarding interactions between additive genetic and maternal effects in natural populations (see Hunt and Simmons 2002; Garant et al. 2003).

Estimating quantitative genetic parameters in the lemon shark is also important from a conservation perspective. Sharks are heavily harvested worldwide either by directed fisheries or as bycatch on pelagic longlines (Baum et al. 2003; Myers and Worm 2003). In many species, harvesting is known to reduce the abundance of large size classes, either through methods that target older and larger individuals, or simply through elevated mortality rates (for review see Fenberg and Roy 2008). These forms of selection may then cause evolutionary change in the exploited populations. For example, recent work on experimental (Conover and Munch 2002) and natural populations (Grift et al. 2003; Olsen et al. 2004; Hard et al. 2008) of bony fish has demonstrated apparently heritable changes in life-history traits, such as body size, growth, age-at-maturity, and fecundity. These changes may sometimes reduce population productivity and contribute to fishery collapses (see Olsen et al. 2004). For sharks, we know that life-history traits influence population dynamics (Stevens et al. 2000), and so understanding the potential to respond to these selective forces may be critical for the future persistence of such taxa.

EVOLUTIONARY DYNAMICS OF A LARGE MARINE VERTEBRATE

The lemon shark is a large, placentally viviparous coastal species mainly found in the western Atlantic from New Jersey to Brazil (Compagno 1984). At coastal nursery sites, females give birth to between 4 and 18 pups in a given year (Feldheim et al. 2002a, 2004). At our study site in particular (Bimini, Bahamas), juveniles remain highly site-attached for at least three years and have

daily home ranges of no more than a few hundred square meters (Morrissey and Gruber, 1993). The enclosed nature of the Bimini nursery lagoon allows us to sample 99% of the newborn sharks, and then resample a large proportion of these individuals in subsequent years (Gruber et al. 2001; DiBattista et al. 2007). Our recent work on this population has taken advantage of these properties to assess evolutionary processes in a large marine vertebrate (DiBattista et al. 2007, 2008a,b).

Of particular interest is the observation that some juvenile traits in the Bimini population are under strong viability selection—consistently favoring small size, low condition, and slow growth (DiBattista et al. 2007). These findings conflict with the conventional wisdom that size, condition, and growth are usually under positive selection (Sogard, 1997; Blanckenhorn, 2000; Kingsolver and Pfennig, 2004). Interestingly, however, they do fit with typical life-history traits observed at Bimini relative to those in another lemon shark population that we have intensively studied (Marquesas Key, Florida). In this comparison, Bimini sharks are much smaller at age (body length: 54 cm vs. 74 cm at age-1), and grow much slower than Florida sharks (6 cm vs. 20 cm between age-0 and age-1; Barker et al. 2005). This pattern is also consistent with juvenile lemon sharks sampled at other nursery sites, which are always larger and grow much faster (e.g., Atol das Rocas, Brazil; Freitas et al. 2006). Based on these results, we have argued that the Bimini population shows some adaptation to local conditions (DiBattista et al. 2007), which belies the common perception for large and mobile marine vertebrates. A missing piece of the puzzle, however, is the genetic basis for the traits involved, which is the overall goal of the present study.

Quantitative genetic studies have, until recently, been out of reach for such large, highly mobile, and cryptic marine organisms. Part of the problem is the confident assignment of genetic relationships among individuals—because mating or birthing events are only rarely observed (Pratt Jr. 1993; Pratt Jr. and Tanaka 1994). This constraint can now be overcome with molecular markers and statistical methods for reconstructing pedigrees in wild populations (for review see Blouin 2003). An additional problem for large marine vertebrates is that some species are so long-lived, and putative parents are so rarely sampled, that establishing multigeneration pedigrees is not feasible. We aim to overcome this by using 13 years of genetic data from sampled juveniles to establish detailed sibship arrays (Feldheim et al. 2002a, 2004; Strausberger and Ashley 2003).

Once a pedigree is obtained, quantitative genetic parameters can be inferred through restricted maximum likelihood (REML; Patterson and Thompson 1971) and the "animal model" (Henderson 1975). Animal model analyses have several benefits for our application (for review see Kruuk 2004; Garant and Kruuk 2005). First, they accommodate unbalanced datasets and make use of all known relationships in the pedigree. Second, they can be used to correct individual phenotypes for known sources of variation, such as age, sex, and cohort (Kruuk 2004). Third, they can be used to simultaneously evaluate multiple traits and thereby estimate genetic correlations. Fourth, they allow the estimation of maternal effects, thus avoiding the otherwise confounding nature of this additional source of variation (see Wilham 1972, 1980; also see Kruuk 2004). That said, few studies to date have used the animal model approach on pedigree data obtained with sibship-reconstruction (but see Thériault et al. 2007), and even fewer have applied it to biological systems that either: (1) lack generational data, or (2) have mostly unsampled parents (although genetically inferred in our case). The success of our analyses may therefore suggest applications to other systems in which few parents are sampled, or where multigenerational pedigrees are not currently available.

The objective of our study was to continue our assessment of evolutionary dynamics in the Bimini lemon shark population. We therefore performed simulation analyses that assess the potential power, bias, and sensitivity of datasets such as ours for quantitative genetic inference using the "animal model." We then use our 13 years of data to estimate heritability and maternal effects for juvenile length and mass. These results are used to further consider local adaptation, and to assess the potential for evolutionary responses to ongoing and future environmental change (e.g., climate change, selective harvesting, habitat loss, pollution).

Materials and Methods

STUDY AREA AND SAMPLING

Bimini, Bahamas, is a mangrove-fringed chain of islands located 85 km directly east of Miami, Florida (see Fig. 1). The Bimini islands enclose a 21 km^2 lagoon that is 0–120 cm deep at low tide, and that serves as a nursery area for approximately 300 juvenile and subadult lemon sharks (Morrissey and Gruber 1993). Each year since 1995, approximately 99% of the juveniles have been captured in the Bimini lagoon (Gruber et al. 2001). Sampling always takes place between 21 May and 25 June, which is just after pupping by females. During this sampling, newborn and juvenile sharks are captured in 180-m long and 2-m deep gill nets (Manire and Gruber 1991). In addition, some subadults (males: 70–175 cm; females: 70–185 cm) and adults (males >175 cm, females > 185 cm; Compagno 1984) are captured opportunistically by rod and reel or longline fishing gear.

All captured sharks are measured for precaudal length (PCL, tip of snout to precaudal pit in millimeters; Compagno 1984), and tagged intramuscularly with an individually coded passive integrated transponder (PIT) tag. All juvenile sharks are also weighed (kg). Each subsequent time a tagged shark is captured, its tag number, PCL, and mass (when feasible) are recorded. In addition, we

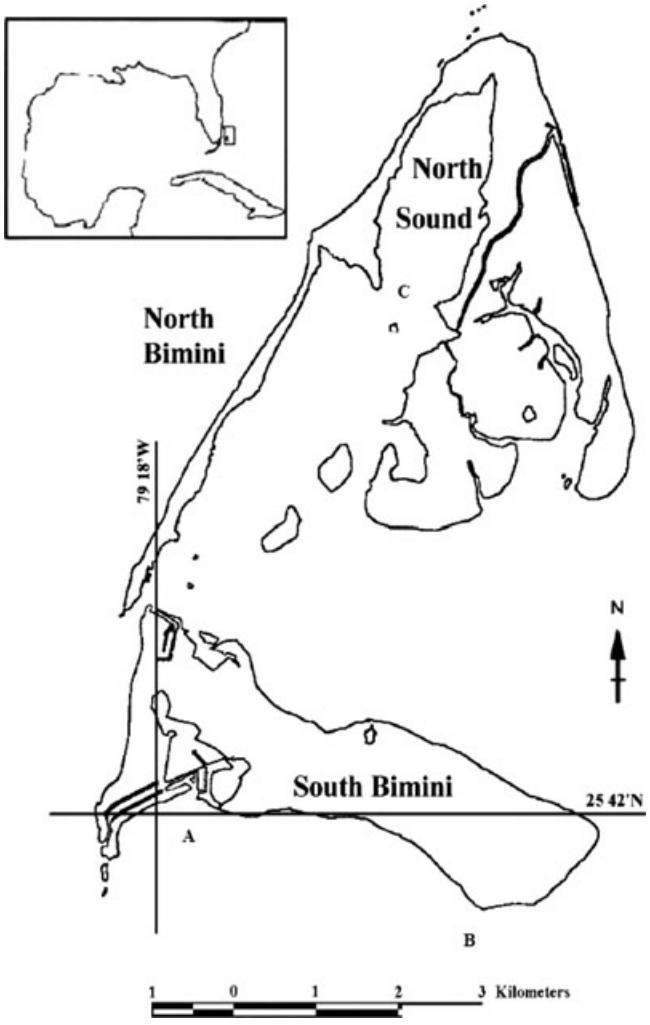

Figure 1. Map of Bimini, Bahamas (Courtesy of S. Newman).

were able to unambiguously assign birth year, and therefore age (Feldheim et al. 2004; DiBattista et al. 2008a,b) to 1364 of the 1509 captured juveniles. Adults are more difficult to catch however, and so nearly all are genetically inferred (see below) but not physically sampled. We therefore only have morphological data for the offspring generation, and not adult sharks.

GENOTYPES

A small piece of fin tissue (2 mm^2) was clipped from every captured shark, and genomic DNA was extracted with a salting-out protocol (Sunnucks and Hales 1996). Genotypes were then determined for 11 species-specific microsatellite loci (for details see Feldheim et al. 2002a,b, 2004; DiBattista et al. 2008a,b). Multilocus genotypes were obtained for a minimum of nine loci for all sharks. Our procedures lead to genotyping error rates (0.0018 PCR amplification or typing error per reaction; see DiBattista et al. 2008b) that are considered quite low for studies of natural populations (see Hoffman and Amos 2005).

The genotypes were first used to individually identify all captured sharks, thus allowing us to determine when a shark had shed their PIT tag. This analysis was done with the program IDENTITY, allowing for mismatches at one locus only (Allen et al. 1995). Our previous work has shown that the loci considered here are powerful enough to discriminate among potential unique or duplicate genotypes ($P_{ID} = 1.11 \times 10^{-15}$), and even to distinguish siblings with high confidence ($PI_{sib} = 1.40 \times 10^{-5}$) (DiBattista et al. 2008b). Feldheim et al. (2002b) performed the IDENTITY analyses for samples between 1995 and 2000, and we here did so for all samples since 2000. This procedure revealed that our dataset consisted of 1364 unique juveniles, which were considered in the subsequent analyses.

Analysis of microsatellite data from subsets of these samples has been previously used to characterize population genetics (Feldheim et al. 2001) and mating systems (Feldheim et al. 2002a, 2004; DiBattista et al. 2008a). Here we use them to reconstruct a pedigree in the interest of estimating quantitative genetic parameters. The present study focuses on pedigree data of newborn (i.e., age-0) and juvenile sharks (age-1 to age-4) sampled at Bimini from 1995 to 2007. These markers are highly polymorphic and unbiased (DiBattista et al. 2008a,b), making them appropriate for pedigree reconstruction (for review see Blouin 2003; Garant and Kruuk 2005) .

PEDIGREE RECONSTRUCTION

Pedigree reconstruction proceeded in several steps. We first used the maximum-likelihood program CERVUS version 3.0 (Marshall et al. 1998; also see Kalinowski et al. 2007) to assign individual offspring to: (1) the few candidate parents that we were able to catch (i.e., 19 mature females and 11 mature males), (2) the 175 captured subadult sharks that might have produced offspring in subsequent years, and (3) the 40 female and 81 male parents that were genetically inferred based on offspring analyzed between 1995 and 2000 (see Feldheim et al. 2004). Assignment to these potential parents (maternal and paternal analyses were separate) was done under a strict confidence level of 95%. Confidence limits were estimated in CERVUS via simulations; parameters included a 2% rate of typing error, 10,000 iterations, and 5–80% sampling of candidate mothers and fathers. As results under strict confidence were similar for all levels of candidate parents sampled, we used simulations corresponding to 33% sampling of candidate mothers and 5% sampling of candidate fathers, a situation likely typical for our study population (Feldheim et al. 2004; DiBattista et al. 2008a).

For the offspring not assigned with the above procedures (394 lacked mothers and 830 lacked fathers), we inferred sibling groups based on maximum-likelihood as implemented in COLONY version 1.2 (Wang 2004). This approach uses group-likelihood ratios to partition individuals into full- and half-sibling families based

on multilocus gene arrays, and also accounts for genotyping error (we assumed a 1% allelic dropout and typing error rate; see DiBattista et al. 2008b). In brief, we ran groups of age-0 sharks in COLONY, separated by year of birth (i.e., cohorts), to identify possible within-year sibling groups. Age-0 sharks from each cohort were also run separately with cohorts from every other year to identify potential between-year sibling groups. Each analysis was run up to three times using the same information, and converged on identical family structure in all cases. COLONY then automatically reconstructs the genotypes of unsampled parents assigned to sibling groups, which we accepted at greater than 95% confidence (see DiBattista et al. 2008b). Using all of the above procedures, our final dataset included 1304 offspring assigned to fathers, and 1351 offspring assigned to mothers (thus 1304 to both fathers and mothers).

The above analyses were facilitated by assuming that half-siblings through the mother were more likely than half-siblings through the father. This assumption is justified based on our previous work showing that the vast majority of half-siblings in our population are maternally related (Feldheim et al. 2004; DiBattista et al. 2008b). It should also be noted that our approach does not preclude the identification of paternal half-sibling groups, as several were identified during this study (see Supporting Table S1).

QUANTITATIVE GENETICS: POWER, BIAS, AND SENSITIVITY

PEDANTIX (Morrissey et al. 2007) was used to assess the likely performance of our lemon shark pedigree in estimating quantitative genetic parameters. First, we considered power and bias when estimating heritability (h^2) and maternal effects (V_M) for a single continuous trait. Second, we considered power and bias when estimating genetic correlations (r_G) between two continuous phenotypic traits. Third, we considered the robustness of our estimates to putative parental misassignment in our pedigree. It should be noted that the maternal variance could not be further partitioned into its genetic and environmental components owing to a lack of multigenerational data. A minimum of two generations are required to separate maternal genetic from maternal nongenetic effects (see Wilham 1972, 1980), but we only have morphological data for the offspring. Our study thus resembles that of a half-sibling design, and as expected, likelihood models with both individual maternal genetic and maternal nongenetic effects failed to converge.

The first two topics were addressed by simulating a single trait across our existing pedigree structure when assuming different user-defined variance components. For h^2, we ran 50 simulations for each value (0.1, 0.2, 0.3, 0.4, and 0.5), which we then analyzed in ASREML (ver. 2.0; VSN international Ltd.) following the procedures described below. Our findings were no different when we increased replicate simulations to 100 for a subset of the analyses (data not shown), and so we here present the complete results based on 50 simulations. Power was defined as the proportion of simulations that gave a significant h^2 estimate at $\alpha = 0.05$. Bias was defined as the deviation of the mean h^2 estimate from the user-defined h^2. An identical approach was employed for V_M, but in this case we used the ratio V_M / V_P (hereafter denoted m^2) as a direct measure for maternal effects, with h^2 held constant at 0.2 (as per Morrissey et al. 2007). For r_G, we simulated two traits, with a constant h^2 of 0.5 for each, and r_G values of 0, 0.2, 0.4, 0.6, 0.8, and 1.

The second topic (i.e., sensitivity to pedigree error) was addressed by adding error in maternal and paternal genetic assignment to our simulations in PEDANTIX. The exact rate of error in our lemon shark pedigree is not known (but likely less than 5% of the mothers or fathers are misassigned), and so for completeness we introduced between 10% and 50% maternal or paternal error into our simulations. That is, each offspring in the pedigree had a 0.1, 0.2, 0.3, 0.4, or 0.5 chance (in separate simulations) of being assigned to a false mother or father. False parents were selected (with equal probability) from a pool of "dummy" unsampled parental genotypes that we added to the pedigree ($N = 25$). We then compared the estimated quantitative genetic parameters (h^2, m^2, r_G; for details see below) to the user-defined values in the simulated datasets. We also reran the above analyses with false parents selected from the pool of existing parental genotypes identified in our study population. Results were similar to those for the "dummy" genotypes, and so the results using existing parental genotypes are not shown.

QUANTITATIVE GENETICS: PARAMETER ESTIMATION

The reconstructed pedigree, along with the measured phenotype of each juvenile shark at first capture, formed the basis of our quantitative genetic analyses. We began by estimating the heritability of PCL and mass. These traits influence juvenile survival at Bimini (DiBattista et al. 2007) and differ among lemon shark populations (Barker et al. 2005; Freitas et al. 2006). The basic dataset includes measurements of 1351 distinct individuals.

First, we evaluated fixed effects in univariate general linear models (SPSS ver. 10.1, SPSS, Chicago, IL). Age influenced both PCL ($F_{3,1348} = 600.51$, $P < 0.0001$) and mass ($F_{3,1348} = 320.21$, $P < 0.0001$), as did sex (PCL: $F_{1,1347} = 7.70$, $P = 0.005$; mass: $F_{1,1347} = 11.17$, $P < 0.0001$) and year of birth (YOB, PCL: $F_{14,1348} = 21.31$, $P < 0.0001$; mass: $F_{14,1348} = 28.64$, $P < 0.0001$). Subsequent analyses therefore included age and sex as fixed effects, thus removing their effects prior to the estimation of genetic parameters. YOB was instead included as a random effect, thus serving as a proxy for the effects of temporal environmental variability.

Second, we estimated the heritability of PCL and mass by using multivariate mixed model REML estimation in ASReml

version 2 (for more details see Kruuk 2004; Garant and Kruuk 2005). Four distinct models were tested: (1) the base model including fixed effects only; (2) an additive genetic random effect, with fixed effects; (3) additive genetic and YOB as random effects, with fixed effects; and (4) additive genetic, maternal identity, and YOB as random effects, with fixed effects. For each relevant trait and model, the total phenotypic variance (V_P) was partitioned into the additive genetic variance (V_A), residual variance (V_R), maternal variance (V_M), and temporal environmental variance (V_{YOB}). Again, the maternal variance could not be further partitioned into its genetic and nongenetic components due to the nature of our pedigree. Narrow-sense heritability (h^2) was estimated as the ratio of additive genetic variance to total phenotypic variance ($h^2 = V_A/V_P$). Statistical significance was assessed with likelihood-ratio tests that compared the full model to a reduced model that lacked the parameter in question. Given that most (i.e., 83%) of the individuals in this pedigree are newborns, our results may be biased toward effects experienced during an individual's first year. We therefore repeated the analyses with age-1 individuals only, which allowed us to assess the extent of ontogenetic shifts in quantitative genetic parameters.

Third, repeated measurements of the same individual in different years allowed us to quantify permanent between-individual environmental effects (V_{PE}; see Kruuk and Hadfield 2007). This parameter was estimated from an expanded dataset that also included all recapture events for each individual ($N = 2265$ measurements on the same 1351 individuals). All models outlined above were rerun with this larger dataset, in addition to a model that included additive genetic, maternal identity, YOB, and permanent environment as random effects, with age and sex as fixed effects.

Fourth, genetic correlations (r_G) between PCL and mass were calculated using pairwise multivariate animal models in ASREML. The same fixed and random effects used in heritability estimation (above) were also considered here. Genetic correlations were defined as

$$r_G = COV_{12}/SQRT(V_{A1} \times V_{A2})$$

where COV_{12} is the genetic covariance between the first and second trait, and V_{A1} and V_{A2} are the additive genetic variances for each of these traits. Significance of the genetic covariance was assessed using likelihood-ratio tests that compared the full model versus a reduced model with no genetic covariance (i.e., fixed at 0).

Finally, for comparative purposes, we re-analyzed our entire dataset, estimating heritability and genetic correlations for PCL and mass using marker-based regression methods (Ritland 1996, 2000). More details on the application of these methods to our data are provided in the Supporting Information.

Results

GENETIC MATING PATTERNS

For the newborn sharks in our analysis, we were able to genetically assign or reconstruct 100% of the mothers and 97% of the fathers; the outcome was 117 unique mothers and 487 unique fathers. Our inferred lemon shark family structure (see Supporting Table S1) confirms and extends previous genetic work at this site (Feldheim et al. 2001, 2002a, 2004). First, polyandry was evident in 88% of the 250 inferred litters. Second, of the 50 inferred mothers that produced offspring in more than one year, 56% did so on a strict two-year cycle—eight of them continuously for all 13 years of the study. In contrast, only 46 of 487 inferred fathers sired offspring in more than one litter—although 11 did so with the same female each time (possibly owing to sperm storage; Pratt and Tanaka 1994). As we will now show, these high levels of polyandry and philopatry provide suitable pedigrees for downstream quantitative genetic analyses—despite the lack of a multigeneration pedigree (i.e., lemon sharks mature at 12 years of age; Compagno 1984).

QUANTITATIVE GENETICS: POWER, BIAS, AND SENSITIVITY

Heritability: In the absence of pedigree error, power was approximately 100% at $h^2 >= 0.2$, and 82% when h^2 was 0.1 (see Fig. 2A,B). Adding paternal pedigree error caused no reduction in power for $h^2 \geq 0.2$, and only a slight reduction when h^2 was 0.1 (power was still 62% when half of the offspring were assigned to the wrong father; Fig. 2B). Adding maternal pedigree error caused minor decreases in power for $h^2 \geq 0.2$, but larger decreases when h^2 was 0.1 (Fig. 2A). Nevertheless, power remained greater than 68% as long as h^2 was greater than 0.1, or maternal error was less than 0.3. Error in the assignment of mothers and fathers both led to a downward bias in the heritability estimate (Fig. 3A,B), which was more pronounced with higher error rates (Fig. 3A, average downward bias from maternal error: 0.1 = 6.86%, 0.2 = 18.96%, 0.3 = 30.39%, 0.4 = 39.60%, 0.5 = 47.61%; Fig. 3B, average downward bias from paternal error: 0.1 = 6.69%, 0.2 = 16.35%, 0.3 = 21.26%, 0.4 = 26.95%, 0.5 = 31.60%).

Maternal effects: In the absence of pedigree error, power was approximately 100% when $m^2 \geq 0.2$, and 56% when m^2 was only 0.1 (Fig. 2C,D). Adding paternal pedigree error caused no reduction in power for all m^2 values (Fig. 2D). Adding maternal pedigree error, however, caused large reductions in power (Fig. 2C)—as would be expected for maternal effects. Nevertheless, power was still greater than 66% when m^2 was higher than 0.1, or when maternal error was less than 0.3. Error in the assignment of fathers led to little bias (Fig. 3B, average downward bias from paternal error: 0.1 = 4.16%, 0.2 = 12.0%, 0.3 = 13.15%, 0.4 = 13.34%, 0.5 = 16.46%), but error in the assignment of mothers led to a clear downward bias in maternal effect estimates, which increased with increasing error rates (Fig. 3A, average downward

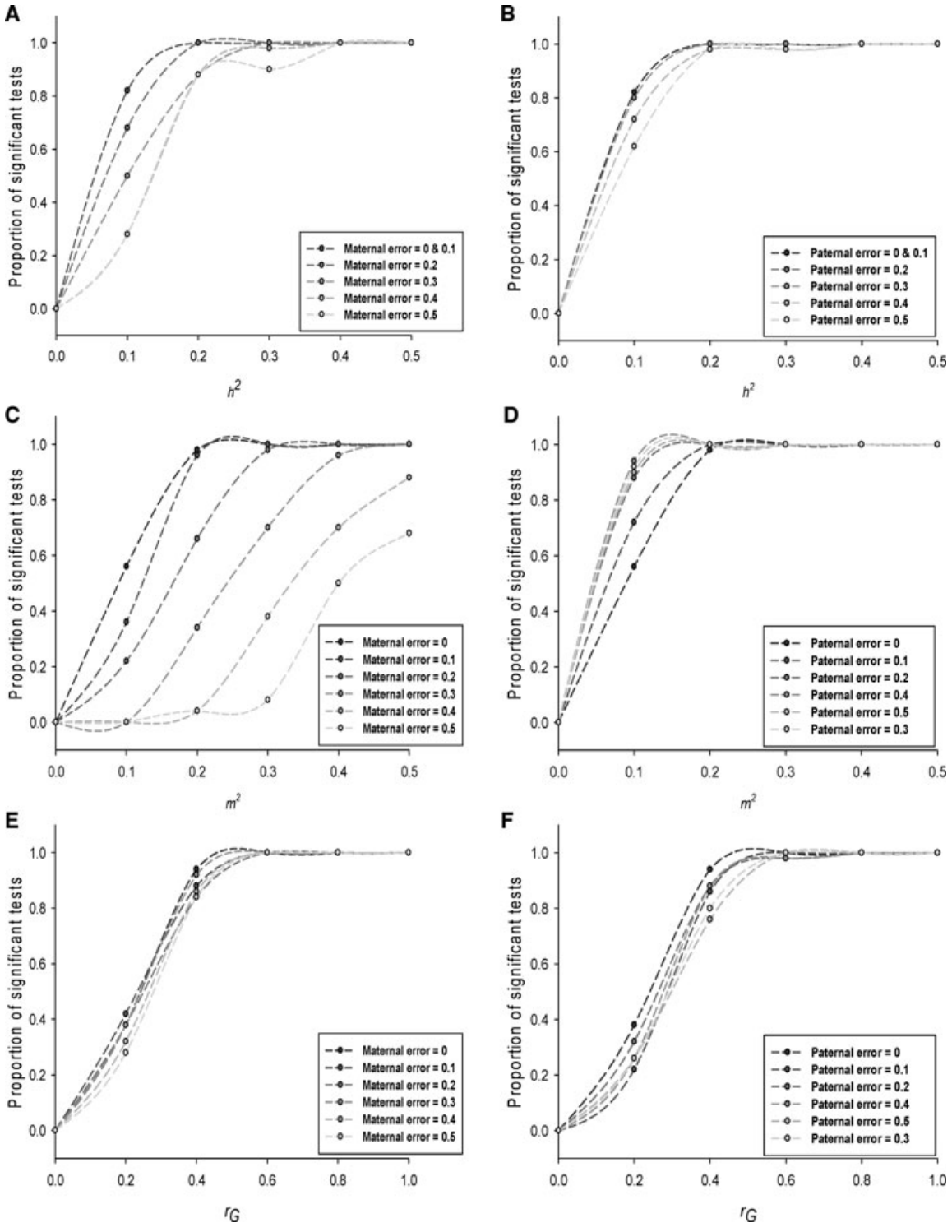

Figure 2. Power analysis, with and without pedigree error—a comparison of the power to detect heritability (h^2; or m^2, or r_G) based on the proportion of simulations that gave a significant genetic variance estimate. Simulations were performed with a range of maternal (A,C,E) and paternal (B,D,F) misassignment introduced into the lemon shark pedigree ($N = 1351$ offspring). In this case, all offspring in the pedigree had a 0.1, 0.2, 0.3, 0.4, or 0.5 chance of being assigned a false mother or father from a pool of "dummy" unsampled parental genotypes added to the pedigree (i.e., adults that we failed to sample directly or reconstruct). The x-axis represents the true level of heritability (or m^2, or r_G), whereas the y-axis represents the proportion of heritability (or m^2, or r_G) values that were significant. Fifty replicates (N) were run for each value of heritability (or m^2, or r_G) and its corresponding level of pedigree error was considered.

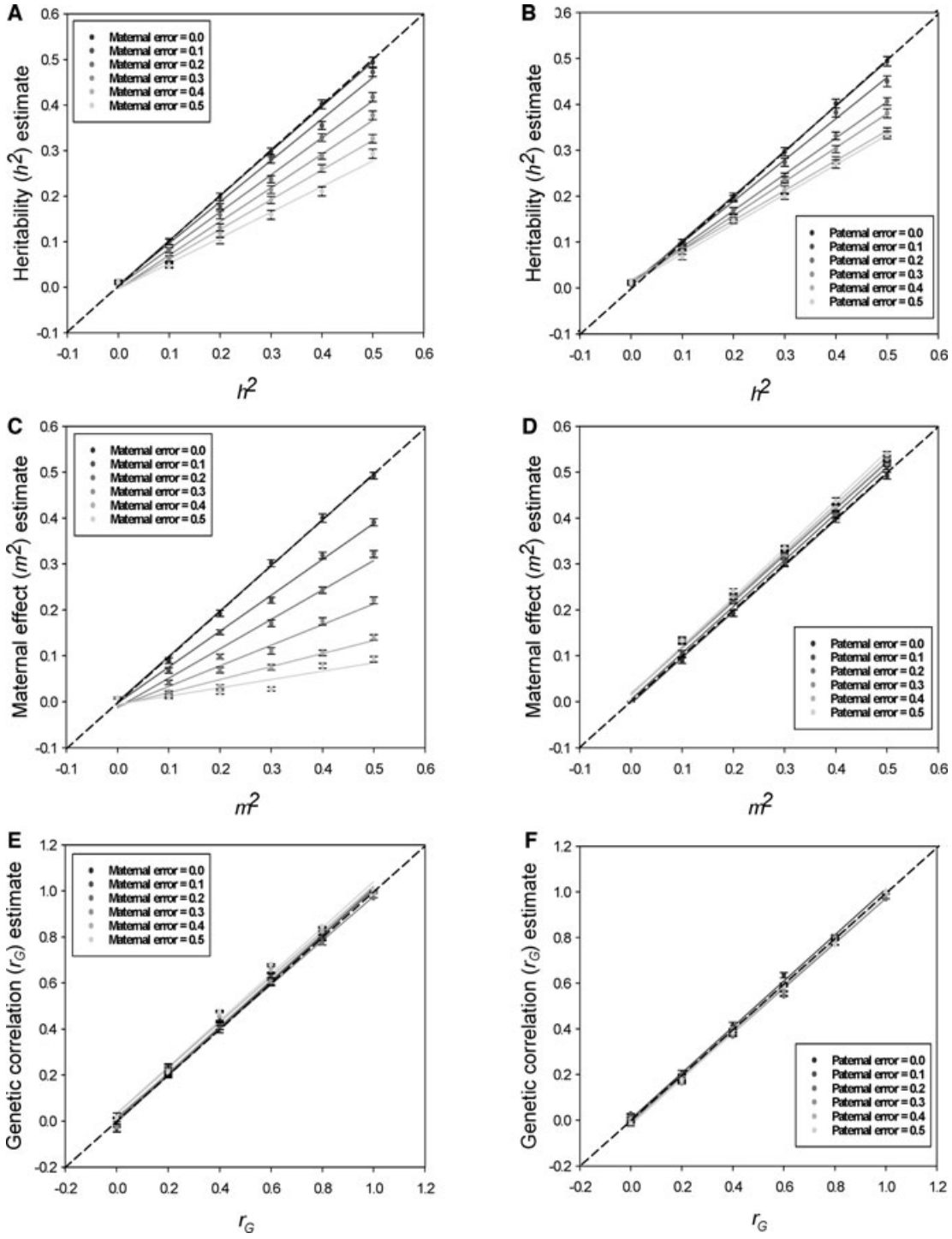

Figure 3. Sensitivity analysis—a comparison of heritability (h^2; A, B), maternal effects (m^2; C, D), or genetic correlations (r_G; E, F) estimated from phenotypic traits simulated in PEDANTIX using our lemon shark pedigree (N = 1351 offspring), with and without introduced pedigree error. In this case, all offspring in the pedigree had a 0.1, 0.2, 0.3, 0.4, or 0.5 chance of being assigned to a false mother (A,C,E) or father (B,D,F) from a pool of "dummy" unsampled parental genotypes added to the pedigree (i.e., adults that we failed to sample directly or reconstruct). The x-axis represents the true level of heritability (or m^2, or r_G), whereas the y-axis represents the heritability (or m^2, or r_G) recovered with the animal model approach from our simulated populations. The solid lines are separate regressions for each level of pedigree error considered, whereas the dashed black line is the ideal case in which one would find exactly the level of heritability that is present in the simulated population. The means and associated standard errors for each heritability (or m^2, or r_G) value, and its corresponding level of pedigree error, are presented here (N = 50). Abbreviations: Pat. err. = proportion of offspring per simulated dataset paternally misassigned; Mat. err.= proportion of offspring per simulated dataset maternally misassigned.

Table 1. Estimates of variance components (V_A, additive genetic variance; V_{YOB}, temporal environmental variance; V_M, maternal genetic and environmental variance; V_R, residual variance; V_P, phenotypic variance) and heritability (h^2) with their standard error, for morphological traits (PCL, precaudal length; Mass) in a natural lemon shark population based on the "animal model." na, not available or not calculated for that particular model.

Traits/Models[1]	N	V_A (SE)	V_{YOB} (SE)	V_M (SE)	V_R (SE)	V_P (SE)[4]	h^2 (SE)
Animal random effects model, age and sex as fixed effects (Likelihood=−463.47; df=1; LRT=223.24; P-value ≤0.001)[2]							
PCL	1351	6.89 (0.66)	na	na	1.25 (0.39)	8.14 (0.38)	0.85 (0.052)
Mass	1351	0.075 (0.01)	na	na	0.047 (0.007)	0.12 (0.006)	0.61 (0.065)
Animal/YOB random effects model, age and sex as fixed effects (Likelihood=−383.65; df=1; LRT=159.64; P-value ≤0.001)							
PCL	1351	6.64 (0.64)	0.29 (0.16)	na	1.29 (0.39)	8.22 (0.40)	0.81 (0.054)
Mass	1351	0.059 (0.008)	0.005 (0.002)	na	0.0056 (0.006)	0.12 (0.006)	0.49 (0.06)
Animal/YOB/Dam random effects model, age and sex as fixed effects (Likelihood=−368.16; df=1; LRT=30.98; P-value ≤0.001)[3]							
PCL	1351	3.95 (0.93)	0.39 (0.20)	3.49 (0.86)	2.51 (0.58)	10.35 (0.85)	0.38 (0.092)
Mass	1351	0.023 (0.011)	0.005 (0.003)	0.041 (0.009)	0.071 (0.008)	0.14 (0.0097)	0.17 (0.076)

[1]Values are the model likelihood, degrees of freedom, likelihood ratio test (LRT) score versus reduced models, and *P*-value for each model considered here.
[2]This particular model was compared to a basic model with only fixed effects for likelihood testing.
[3]This model includes "dam" as a random factor to account for possible maternal effects present in the population.
[4]Phenotypic variance estimates as calculated from raw data only were V_P=17.31 for PCL and V_P=0.214 for mass. These values can be used for standardizing our model-specific heritability estimates, and thus included in future meta-analyses (Wilson 2008).

bias from maternal error: 0.1 = 24.82%, 0.2 = 45.26%, 0.3 = 63.91%, 0.4 = 78.63%, 0.5 = 86.42%).

Genetic correlations: In the absence of pedigree error, power was greater than 90% when r_G was 0.4 or higher, but was only 38% when r_G was 0.2 (Fig. 2E,F). Adding parental pedigree error had little effect on power, except for modest reductions when r_G was 0.2 (see Fig. 2E,F). Moreover, error in the assignment of mothers and fathers caused almost no bias (Fig. 3E, average downward bias from maternal error: 0.1 = 2.76%, 0.2 = 4.22%, 0.3 = 5.76%, 0.4 = 7.22%, 0.5 = 8.62%; Fig. 3F, average downward bias from paternal error: 0.1 = 5.33%, 0.2 = 3.96%, 0.3 = 5.26%, 0.4 = 4.61%, 0.5 = 4.29%).

In summary, simulations suggest that we should have high power (>80%) to find significant quantitative genetic effects as long as they are not excessively small (i.e., $h^2 < 0.2$, $m^2 < 0.2$, or $r_G < 0.4$). This conclusion holds with pedigree error, except for the case of maternal effects (m^2) in the presence of extensive maternal pedigree error. Pedigree error also leads to downwardly biased h^2 and m^2 estimates—the extent of this bias appears to be contingent on the level of error inherent to the pedigree, but only an issue when 20% or more of the offspring were assigned incorrect parents.

QUANTITATIVE GENETICS: PARAMETER ESTIMATION

Table 1 shows genetic variance components for PCL and mass. Additive genetic components, and therefore heritability, were significant in all models and ranged from 0.38 to 0.85 for PCL, and from 0.17 and 0.61 for mass. Moreover, the inclusion of additional random effect terms significantly improved model fit for both traits (Table 1). First, YOB had a modest influence on model likelihood (Log-likelihood = −383.65, df = 1, likelihood-ratio test = 159.64, $P < 0.001$) and variance component estimates, suggesting that common environmental effects are present here. Second, maternal identity had very large effects, accounting for 34% and 29% of the phenotypic variance for PCL and mass, respectively (which is also referred to as m^2 for the purposes of this study), although we cannot be sure if these are genetic or nongenetic effects. As a result, heritability estimates were two to three times smaller after maternal effects were added to the model (Table 1).

Using the expanded dataset that included all recapture events for each individual (Table 2), we found that permanent between-individual environmental effects (V_{PE}) were also important (Log-likelihood = −1409.20, df = 1, likelihood-ratio test = 18.2, $P < 0.001$), but did not drastically change heritability. Maternal identity, however, had greater effects here. Indeed, heritability estimates for PCL and mass were two times smaller with the inclusion of the "dam" parameter in the model. Interestingly, when these (and earlier) analyses were repeated with age-1 individuals exclusively, maternal effects no longer accounted for any of the phenotypic variance in PCL and mass ($V_M \approx 0$, $P > 0.05$), whereas additive genetic variance now explained 89% for PCL ($h^2 = 0.89 \pm 0.032$; $P < 0.001$), and 23% for mass ($h^2 = 0.23 \pm 0.090$; $P = 0.015$).

The additive genetic covariance (COV) and correlation (r_G) between PCL and mass were positive (COV : 0.65 ± 0.074; r_G:

Table 2. Based on an expanded "repeated measures" dataset and "animal model" analyses—estimates of variance components (V_A, additive genetic variance; V_{YOB}, temporal environmental variance; V_M, maternal genetic and environmental variance; V_{PE}, permanent environmental variance; V_R, residual variance; V_P, phenotypic variance) and heritability (h^2), with their standard error, for morphological traits (PCL, precaudal length; Mass) in a natural lemon shark population are reported here. na, not available or not calculated for that particular model.

Traits/Models[1]	N	V_A (SE)	V_{YOB} (SE)	V_M (SE)	V_{PE} (SE)	V_R (SE)	V_P (SE)[4]	h^2 (SE)
Animal random effects model, age and sex as fixed effects (Likelihood=−1506.8; df=1; LRT: 615.62; P-value ≤0.001)[2]								
PCL	2265	6.86 (0.47)	na	na	na	4.93 (0.22)	11.79 (0.44)	0.58 (0.023)
Mass	2265	0.092 (0.008)	na	na	na	0.138 (0.006)	0.23 (0.008)	0.40 (0.027)
Animal/YOB random effects model, age and sex as fixed effects (Likelihood=−1418.3; df=1; LRT: 177; P-value ≤0.001)								
PCL	2265	6.77 (0.47)	0.06 (0.08)	na	na	4.94 (0.22)	11.78 (0.44)	0.58 (0.023)
Mass	2265	0.081 (0.008)	0.01 (0.005)	na	na	0.139 (0.005)	0.230 (0.009)	0.35 (0.027)
Animal/YOB/PE random effects model, age and sex as fixed effects (Likelihood=−1409.2; df=1; LRT: 18.2; P-value ≤0.001)[3]								
PCL	2265	5.33 (0.92)	0.05 (0.08)	na	1.08 (0.64)	4.89 (0.22)	11.35 (0.47)	0.47 (0.068)
Mass	2265	0.04 (0.010)	0.01 (0.005)	na	0.034 (0.009)	0.14 (0.006)	0.22 (0.008)	0.18 (0.045)
Animal/YOB/PE/DAM random effects model, age and sex as fixed effects (Likelihood=−1406.23; df=1; LRT: 5.94; P-value=0.015)								
PCL	2265	2.97 (1.08)	0.09 (0.09)	1.53 (0.61)	2.39 (0.73)	4.88 (0.21)	11.86 (0.60)	0.25 (0.090)
Mass	2265	0.018 (0.014)	0.009 (0.004)	0.014 (0.007)	0.046 (0.011)	0.14 (0.006)	0.22 (0.009)	0.084 (0.063)

[1]Values are the model likelihood, degrees of freedom, likelihood ratio test (LRT) score versus reduced models, and P-value for each model considered here.
[2]This particular model was compared to a basic model with only fixed effects for likelihood testing.
[3]This model includes permanent environment (PE) as a random factor to account for possible between-individual environmental effects on phenotype.
[4]Phenotypic variance estimates as calculated from raw data only were V_P=43.66 for PCL and V_P=0.617 for mass.

0.90 ± 0.023) and significant ($P < 0.001$ in all cases). Once again, including YOB in the model had only a modest influence on these estimates (COV: 0.61 ± 0.069; r_G: 0.98 ± 0.013; both $P < 0.001$), but adding maternal identity had larger effects ($COV = 0.30 \pm 0.093$; r_G: 0.99 ± 0.064; both $P < 0.001$). The additive genetic correlation between age-0 and age-1 was also strong (0.85 ± 0.084), and significant ($P < 0.001$).

Variance component and genetic correlation estimates from marker-based regression methods (Ritland 1996, 2000) were remarkably similar to those obtained with animal model analyses (see Table 3). This result suggests the possibility of obtaining reasonable estimates of quantitative genetic parameters in at least some shark populations in which reconstruction of pedigrees is impossible.

Discussion

No previous study of a large marine vertebrate has estimated heritability, genetic correlations, or maternal effects. We did so here for lemon sharks by combining the long-term monitoring of marked individuals with genetic pedigree reconstruction and animal model estimation. We found significant additive genetic (co)variance, and hence moderate heritability, for juvenile length and mass. We also found substantial maternal effects for these traits at age-0, but not age-1, confirming that genotype–phenotype interactions between mothers and offspring are strongest at birth. These results have important implications for evolutionary dynamics in large marine vertebrates, which may be particularly susceptible to ongoing environmental change. In the following sections we discuss (1) the power of our lemon shark pedigree for estimating quantitative genetic parameters, (2) parameter estimates obtained in this study, and (3) how evolutionary processes may differ in large marine vertebrates.

POWER, BIAS, SENSITIVITY, AND ALTERNATIVE METHODS

Nearly all previous animal model estimations of quantitative genetic parameters in wild populations have used multigenerational pedigrees (Kruuk 2004). This type of pedigree is not possible for long-lived organisms unless the population has been monitored for many years. Indeed, our dataset spans 13 years, but we have yet to detect a single parent that had been previously sampled as a juvenile at our study site; our pedigree more closely resembles those used in half-sibling designs in the laboratory (Tallamy et al. 2003) or field (Thériault et al. 2007). We therefore started our investigation by determining how a single-generation pedigree could be used effectively to estimate quantitative genetic parameters in the wild. Overall, we found that power was very high (> 80%) unless the true parameters were quite small ($h^2 < 0.2$, $m^2 < 0.2$,

Table 3. Heritability estimated for phenotypic traits in natural populations, but only considering studies that used marker-based regression methods (Ritland 1996); some papers mentioned here also used "animal model" approaches for comparison. na, information not available in the literature.

Species	Traits examined	Range of N	Range of h^2	P-value[10]	Range of var(r_{ij})[12]	Range of var(r_{ij}) SE	Comparison to animal model
Bighorn sheep[1]	Life history	107–186	na	Rit.: 2 of 15 sig. AM: 8 of 15 sig.	0.0063–0.0093	0.0021–0.0024	Downwardly biased by 42% to 76%
Japanese flounder[2]	Life history and morphological	50	−6.24 to 41.54	None sig.	0 to 0.0001	0.0001 to 0.0003	n/a
Soay sheep[3]	Body weight	529	−2.81 to 1.45	Rit.: 0 out of 6 sig. AM: 6 of 6 sig.	Always=0	na	Downwardly biased
Eucalyptus[4]	Physiological (chemical defense)	259	0.19 to 0.87	2 of 4 sig.	0.0015 to 0.003	0.001 to 0.0015	na
Yellow monkeyflower[5]	Life history and morphological	300	−0.09 to 2.04	8 of 17 sig.	0.027 to 0.044	na	na
Yellow monkeyflower[6]	Life history and morphological	230	0.01 to 0.24	6 of 7 sig.	Always=0.009	na	na
Turkey oak[7]	Unable to estimate heritability as per Ritland (1996) due to low variance in relatedness	na	na	na	na	na	na
Lemon shark[8,11]	Life history and morphological	644 to 1364	0.16 to 0.25	Rit.: 2 of 2 sig. AM: 2 of 2 sig.	0.002 to 0.011	0.001 to 0.002	Downwardly biased by 6% to 35%
Capricorn silvereye[9]	Morphological	479	V_A=−0.001 to 0.002	None sig.	−0.001 to 0.008	0.005 to 0.012	Downwardly biased by 98 to 100%

[1]Coltman (2005).
[2]Shikano (2008).
[3]Thomas et al. (2002).
[4]Andrew et al. (2005).
[5]Ritland and Ritland (1996).
[6]van Kleunen and Ritland (2005).
[7]Klaper et al. (2001).
[8]Present study.
[9]Frentiu et al. (2008).
[10]Number of significant heritability estimates for all traits considered. In cases in which Ritland's regression method (Rit.) and the animal model approach (AM) were used within the same study, both are presented.
[11]Estimates from the best fit "animal model" (i.e., animal/YOB/dam random effects model, see Table 1) are here considered for comparison.
[12] var(r_{ij})=variance in relatedness.

or $r_G < 0.4$), and bias was low (> 7%) unless pedigree error was quite high (≥ 20% of the offspring incorrectly assigned to parents). Our quantitative data thus support previous assertions that the value of a pedigree depends not only on its size and depth, but also on the number of relatives (Quinn et al. 2006). Our pedigree included many pairwise links, a number of which were full- or half-siblings, thus providing enough information for the robust estimation of quantitative genetic parameters.

The suitability of our pedigree structure should encourage the use of animal model estimation methods for other populations where multigeneration pedigrees are lacking—at least when many links between relatives are known. This conclusion will be good news for investigators seeking to infer parentage through molecular genotyping in long-lived and cryptic species. We do caution, however, that high pedigree error can be a problem when estimating some parameters (see also Hoffman and Amos 2005), particularly maternal effects (Figs. 2 and 3). Fortunately, this type of error can be overcome by using many polymorphic loci, carefully screening for genotyping errors, and using strict confidence (> 95%) in pedigree reconstruction—which was the case in our study (see also DiBattista et al. 2008b).

Remarkably, our animal model estimates of heritability and genetic (co)variance (see below) could be recovered quite well even with marker-based regression methods (Ritland 1996, 2000). This close correspondence in our study conflicts with most previous comparisons of animal model and marker-based regression methods (see Table 3). The reason for our success is not clear, but it does suggest that marker-based regression methods may be a useful and simpler alternative under specific conditions. If our study system is any guide, these conditions may include appropriate mating systems (i.e., polygamy), large sample sizes, numerous and variable markers, and an appropriate mix of relatives and nonrelatives. These conditions are often met for at least some marine vertebrates, particularly sharks, which may allow us to quickly obtain estimates for more populations. For instance, application of these methods to another shark population that we study (Marquesas Key), but where we lack the ability to reconstruct a pedigree (see DiBattista et al. 2008b), will further our comparative inferences regarding local adaptation and evolutionary dynamics (see below).

QUANTITATIVE GENETIC PARAMETERS

Juvenile body length and mass showed high levels of additive genetic variance in our study population (Table 1). Although significant heritability for body size has been shown in a number of vertebrate systems in the wild (e.g., Charmantier et al. 2004; Thériault et al. 2007; Wilson et al. 2007), this is the first instance for a large, strictly marine vertebrate. To compare our heritability estimates (h^2 for PCL = 0.38 and mass = 0.17) to those from other taxa, some standardization is required. Wilson et al. (2007) suggests scaling additive genetic variance by the raw phenotypic variance to remove any bias introduced by fixed effects considered in different studies. This provides heritability values of 0.23 for PCL and 0.11 for mass in our study. If considered in relation to heritability values estimated from other marine populations in the wild (i.e., all salmonids; Carlson and Seamons 2008), our values rank just as high (median $h^2 = 0.22$). Moreover, the genetic correlation we obtained between mass and length ($r_G = 0.99$) is slightly higher than those estimated for morphological traits in salmonid populations (median $r_G = 0.71$; see Carlson and Seamons 2008). Another standardization method involves using the trait mean to calculate CV_A (i.e., evolvability; see Houle 1992), which gives us values of 28.57 for PCL and 13.14 for mass. These values are higher than most representative (morphological, life-history, behavioral, physiological) traits estimated from *Drosophila melanogaster* populations (Houle 1992). Thus, heritability for body size in the lemon shark is not any lower than those estimated in other taxa, and may even be slightly higher.

We expected that maternal effects might be important in lemon sharks as well, given that they are viviparous (maternal-fetal connection) and thus give birth to live young. This expectation turned out to be correct given that maternal effects explained as much as 34% of the phenotypic variance for juvenile body length and mass. These maternal effects were largely restricted to age-0 individuals, as we failed to detect any for age-1 individuals. Thus, it appears that size at birth is strongly influenced by the mother's genotype or phenotype, but that the effects of the juvenile's own genotype become predominant thereafter (also see Heath et al. 1999). This pattern makes sense given that direct maternal contributions cease at birth in sharks (i.e., no parental care). It should be noted, however, that we only modeled a general maternal effect in our analysis, which includes some unknown combination of both maternal genetic and maternal environmental effects. The separation of these two effects requires multigenerational pedigrees, which were not available in this case. We therefore cannot be certain whether the effects of the mother on her offspring are due largely to the expression of her own genes, or the effects of the environment on her ability to provide nutrition for her developing embryos.

Maternal effects can therefore, on the one hand, be considered statistical artifacts that inflate estimates of heritability. Indeed, our heritability estimates decreased considerably after including maternal effects in the animal model (Tables 1 and 2). On the other hand, maternal genetic effects can make important contributions to adaptation and the evolution of life histories (e.g., Sheldon 2002). As one example, maternal genetic effects may accelerate evolution in sharks if large or small body size is favored in both the mother and their offspring. Indeed, this appears to be the case for some vertebrate systems (for review see Räsänen and Kruuk 2007). In this scenario, however, the targeted removal of adult

females from an exploited shark population can have unexpected consequences. Whereas fishing typically selects for small adult females by removing the oldest (largest) individuals (for review see Fenberg and Roy 2008), the size and quality of offspring for marine (e.g., Kjesbu et al. 1996; Vallin and Nissling 2000; Berkeley et al. 2004) and live-bearing fish (see Goodwin et al. 2002) are positively correlated with maternal length and age. Size-selective harvesting would therefore result in small, possibly less viable offspring. As another example, maternal genetic effects might also be maladaptive if adult females are selected for large (or small) body size, but their offspring are selected for small (or large) body size (i.e., life-history trade-offs). This fits a scenario of "constrained local adaptation," whereby large females are selected for, but in turn their large offspring are selected against, then the population continually pushes toward an "optimum" phenotype it may never reach.

Genetic correlations arise when the same genes influence multiple traits (i.e., pleiotropy), or when loci that influence different traits show linkage disequilibrium (Roff 1997). Negative genetic correlations are often interpreted as evidence for life-history trade-offs (e.g., egg size and fecundity, see Czesak and Fox 2003). Positive correlations, however, are often used to infer the possibility of trait coevolution, which might accelerate evolutionary change (e.g., Gutteling et al. 2007). Indeed, we found strong positive genetic correlations between mass and body length in this study, which suggests that selection acting on one trait would likely have an indirect effect on the other. The close relationship between these two traits is both obvious and inevitable; genetic correlations between mass and body length have been shown in birds (Jensen et al. 2003), mammals (Milner et al. 2000), and amphibians (Olsson and Uller 2002). Positive genetic correlations also appear to be common for traits under sexual selection (i.e., female preference for male ornaments; Bakker 1993), or those for parasite resistance (e.g., Coltman et al. 2001).

EVOLUTION AND ADAPTATION

Short-term evolutionary responses for traits can be predicted as $R = h^2 S$, where R is the single generation response to selection (change in the trait mean), h^2 is the trait heritability, and S is the selection differential. Based on our estimates of selection (DiBattista et al. 2007) and heritability (this study) for sharks at Bimini, we would expect a per-generation decrease of 0.7% for PCL and 1.1% for mass in age-0 sharks, or 26% for PCL and 5.3% for mass in age-1 sharks. Selection gradients are only available for PCL (and not mass) in this population (DiBattista et al. 2007), and so similar comparisons based on multivariate estimates are not possible. Moreover, testing these predictions will require many more years of data as we have yet to sample any individuals from a second generation. The outcome is also uncertain given that some studies do not find correspondence between observed and predicted evolutionary responses (Merilä et al. 2001). Possible reasons for such discrepancies include selection acting on nonheritable environmental components of a phenotype (Kruuk et al. 2002), correlations among traits (Sheldon et al. 2003), fluctuating selection pressures (e.g., rapid environmental change; Grant and Grant 2002), opposing selection at other life-history stages (Schluter et al. 1991), and even competition among individuals (for more details see below).

Regardless of the success of such quantitative predictions, the high heritability for juvenile size indicates that this trait has the potential to evolve in response to changing selection pressures. More subtly, this suggests that adaptation may proceed owing to changes in local (or global) conditions. Indeed, phenotypic changes certainly seem likely given that sharks are subject to commercial and recreational fishing (Baum et al. 2003; Myers and Worm 2003), and that fisheries are often size-selective (see Fenberg and Roy 2008). Whether such changes are rapid enough to have a measurable impact on time scales relevant to human interests remains to be seen however, owing to long generation times in such taxa.

Although we have previously discussed possible explanations for the evolution of small body size in our population (e.g., predation pressure; see DiBattista et al. 2007), we now consider an alternate explanation based on IGEs (for review see Wolf et al. 1998). Such effects occur when environmental influences on the phenotype of one individual are caused by the expression of genes in other members of the population. The existence and potential importance of IGEs is supported by a growing body of theoretical (Bijma et al. 2007a) and empirical (Muir 2005; Bijma et al. 2007b;) work. IGEs occur most frequently with intraspecific competition, so that individual phenotypes within a population with the greatest fitness survive, but at the detriment to the available resources for the entire population. This could conceivably occur in the lemon shark if competition was common and competitive ability was heritable. This might be particularly pronounced for size-related traits—we would expect to see positive selection for individual body size and growth rate based on relative fitness, but competitive effects would actually decrease size and growth at the population level. The largest individuals would therefore have the highest survival, but these large individuals would grow relatively large at the expense of growth for all remaining individuals; thus the population would "evolve" a smaller size.

Although the idea of IGEs is consistent with the observed phenotypes at Bimini, we feel this is unlikely for several reasons. First, we have previously shown individual level selection (i.e., relative fitness) for smaller size and slower growth (DiBattista et al. 2007). Second, although intraspecific resource competition is common in nature, the nursery area at Bimini is not resource limited, especially with respect to the primary prey item of the lemon shark (i.e., yellowfin mojarra, *Gerres cinereus*; Newman

and Gruber, 2002). Indeed, we have never witnessed aggressive interactions among juvenile sharks at Bimini (S. H. Gruber, pers. comm.), suggesting a lack of interference competition. Moreover, we found no relationship between juvenile shark density (a proxy for competition potential) and the strength of selection acting on morphological traits (DiBattista et al. 2007). As a final point, Bimini is not more likely to experience competition than other nursery sites, and so this leads us to believe that the small size and slow growth characteristic of Bimini sharks is largely due to the effects of "true" selection, and not IGEs.

As a final point, most studies of vagile marine organisms do not consider the possibility of local adaptation—but we have recently argued that this might be a mistake (DiBattista et al. 2007, 2008b). Here we incorporate quantitative genetic data into our hypothesized scenario for partial local adaptation by lemon sharks to local nursery sites. First, our data show that juvenile sharks are under strong selection for small body size at Bimini (DiBattista et al. 2007). Given their long residence in the nursery site (Morrissey and Gruber 1993), such selection should weed out many genotypes for large size. Second, females born at Bimini likely return there for breeding (Feldheim et al. 2004), bringing with them their genes for small size; this should lead to the evolution of small body size. Indeed, Bimini juveniles are always smaller than sharks sampled at other nursery sites (Barker et al. 2005; Freitas et al. 2006). Third, adult males move extensively among nursery sites (Feldheim et al. 2004), some of which house much larger juveniles (Barker et al. 2005; Freitas et al. 2006). This male dispersal should constrain adaptation at Bimini short of the optimum, which would explain observations of both continuing selection for small size (DiBattista et al. 2007) and high additive genetic variance for size (present study). We therefore suggest that local adaptation partially constrained by gene flow (e.g., Bolnick and Nosil 2007; Garant et al. 2007; Moore et al. 2007) may be a general phenomenon in marine systems.

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Supporting Information

The following supporting information is available for this article:

Table S1: Maternity (and paternity) reconstruction in the Bimini nursery using offspring genotypes from lemon sharks captured between 1995 and 2007.

Table S2: Estimates of trait heritability and genetic correlation (±SE) using a regression method based on juvenile sharks sampled from Bimini.

Supporting Information may be found in the online version of this article.
(This link will take you to the article abstract).