Naris and beak malformation caused by the parasitic fly, *Philornis downsi* (Diptera: Muscidae), in Darwin’s small ground finch, *Geospiza fuliginosa* (Passeriformes: Emberizidae)

TOBY H. GALLIGAN and SONIA KLEINDORFER*

School of Biological Sciences, Flinders University, GPO Box 2100, Adelaide, SA 5001, Australia

Received 17 March 2009; accepted for publication 22 May 2009

Parasites induce phenotypic modifications in their hosts, which can compromise host fitness. For example, the parasitic fly *Philornis downsi*, which was recently introduced to the Galápagos Islands, causes severe naris and beak malformation in Darwin's finches. The fly larvae feed on tissues from the nares of developing finch nestlings, thereby altering the size and shape of the nares and beak. Although the parasitism is age-specific (adult finches are not parasitized), naris and beak malformations persist into adulthood as parasite-induced malformations. We systematically examined adult populations of Darwin's small ground finch, *Geospiza fuliginosa*, on the islands of Santa Cruz for *P. downsi*-induced malformation. We found that malformed birds had significantly longer nares, and shorter, shallower beaks, than birds considered to be normal (i.e. with no nares or beak malformation). In addition, normal birds showed an isometry between naris length and beak dimensions (beak length feather and beak depth), which was not found in malformed birds. These differences suggest that beak morphology was influenced by *P. downsi* parasitism. Interestingly, we did not find any evidence of developmental impairment (smaller body size) or reduced foraging efficiency (lower body condition) between normal and malformed birds. Our findings of *P. downsi*-induced malformation raise new questions about the evolutionary trajectory and conservation status for this group of birds. © 2009 The Linnean Society of London, Biological Journal of the Linnean Society, 2009, 98, 577–585.


INTRODUCTION

Parasites reduce host fitness through the acquisition of resources (Price, 1980; Loye & Zuk, 1991; Clayton & Moore, 1997). Long-term observational and experimental studies have shown the effects on host longevity and fecundity as a result of permanent parasitism (Hudson, Newborn & Dobson, 1992; Hudson, Dobson & Newborn, 1998) and temporary parasitism (Møller, 1990; Richner, Oppliger & Christe, 1993; Brown, Brown & Rannala, 1995). These fitness estimates have been largely derived from examples of recurrent parasitism. By contrast, age-specific parasitism, where the host–parasite interaction is confined to a stage of the host’s life-cycle (for example during immaturity), has rarely been analysed in terms of future host fitness. Yet age-specific parasitism is important for many species, especially species that produce nidicolous young that are often hosts for nest-based ectoparasites (Marshall, 1981). It is possible that future fitness costs of age-specific parasitism are under-reported in the literature because the finite duration of the interaction erroneously implies a finite duration of total host fitness costs. On the basis of this misunderstanding, combined with a lack of data on age-specific parasitism, studies may preferentially report on direct parasite-induced mortality and neglect...
the fitness costs for hosts that survive age-specific parasitism (Hudson & Dobson, 1997). Failure to consider post-parasitism fitness costs of age-specific parasitism therefore can have serious conservation implications for many species.

Survivors of age-specific parasitism often exhibit phenotypic modifications that are the pathological consequence of past host–parasite interactions (Poulin & Thomas, 1999; Möller, 2006). For mature hosts, phenotypic modifications arise solely from parasite resource acquisition. However, for immature hosts, tissue damage (i.e. deformation) can be combined with parasite-induced developmental instability (i.e. malformation) to generate phenotypic modifications (Möller, 2006). Notably, although deformation can vary in persistence from short-term to permanent, malformation is almost always permanent. Furthermore, parasite-induced malformation has the potential to completely compromise host fitness because it occurs before the first reproductive event in the host (Möller, 1997; Möller & Swaddle, 1997). For these reasons, phenotypic modification is particularly important for age-specific parasitism where the host is immature. Consequently, in the present study, we will focus on parasite-induced malformation.

Parasite-induced malformation is expected to impair an individual’s overall development, increasing its vulnerability to competitors, predators, and parasites, and thereby decreasing its longevity and reproductive output (Möller, 1997). A small number of studies have shown that malformation caused by parasites can alter host development (Möller, 1997, 2006); of these studies, only a few have shown reduced fitness as a consequence (Möller, 1992, 1996; Polak, 1993; Potti, 2008). For example, a recent study by Potti (2008) showed delayed effects of nestling parasitism (i.e. post-parasitism): specifically, female pied flycatchers, Ficedula hypoleuca, that were parasitized as nestlings by the blowfly, Protocalliphora azurea, had consistently smaller egg size as adults.

The nestlings of Darwin’s finches (Passeriformes: Emberizidae; Gould) on the Galápagos Islands are novel hosts to the parasitic larvae of an introduced fly, Philornis downsi (Diptera: Muscidae; Dodge and Aitken). Adult finches are not parasitized; therefore, the interaction is age-specific. Philornis downsi larvae reside by day in the base of finch nests and feed by night on the blood and tissues of the nidicolous young. The preferred feeding site for the first-larval instar is the nestling’s nares, a behaviour that can cause malformation of the surrounding tissue and keratin, and result in permanent enlargement of the nares (Fessl, Sinclair & Kleindorfer, 2006b) and a shape change in naris from teardrop-shaped to circular. Second- and third-larval instars can further increase malformation by using the nares to access internal feeding sites, which causes repeated ulceration and bone-reaabsorption (J. O’Connor, J. Robertson & S. Kleindorfer, in review). In addition, malformation of the beak can also occur. Grooves, cracks, and gouges in the beak keratin radiating from the nares are obvious evidence of P. downsi-induced beak malformation, but an overall reduction in beak size as a result of developmental instability is also expected. Accordingly, naris and beak malformation observed in adult Darwin finches are considered symptoms of past P. downsi parasitism.

Philornis downsi is identified as the most significant threat to Darwin's finches (Causton et al., 2006). Originating from the northern Neotropics (Dudaniec & Kleindorfer, 2006), P. downsi larvae were discovered in the nests of Galápagos birds in 1997 (Fessl, Couri & Tebbich, 2001), although adult specimens were collected from the islands in 1964 (Causton et al., 2006). Presently, P. downsi is known to affect nine of the 13 species of Darwin’s finch (Fessl et al., 2001; Fessl & Tebbich, 2002; J. O’Connor, F. J. Sulloway, J. Robertson, and S. Kleindorfer, unpubl. data; B. Fessl pers. com.) on 12 of the 18 major islands of the Galápagos (Wiedenfeld et al., 2007; P. Grant, pers. comm.). Previous studies have shown a high incidence and impact of P. downsi parasitism in Darwin’s finches: 100% parasite prevalence in nests (Dudaniec, Fessl & Kleindorfer, 2007); up to six infestation events per nest (Dudaniec, Gardner & Kleindorfer, 2009); up to 64 larvae per nestling (Fessl & Tebbich, 2002); and 95% nestling mortality as a result of parasitism in some years (Fessl, Kleindorfer & Tebbich, 2006a). To date, there are no data available on parasite-induced malformation or associated fitness consequences among surviving fledglings and adult finches. In the present study, we examine the extent and consequences of malformation caused by P. downsi in an adult population of Darwin’s small ground finch (Geospiza fuliginosa). We predict that malformed birds will have larger nares (specifically, longer naris length), and smaller beak size (specifically, smaller overall size, shorter beak length, and shallower beak depth) than birds considered normal with no obvious malformation. We also predict that malformed birds will have smaller body size and lower body condition than their normal counterparts as a result of developmental impairment and its effect on foraging efficiency and competitiveness.

MATERIAL AND METHODS

STUDY SITE
This study was conducted between January and May 2008. All data were collected from the central and elevated Galápagos island of Santa Cruz (986 km²; 0°37'S, 90°21'W).
Elevated islands of the Galápagos archipelago can be divided into three main ecological zones based on the annual level of precipitation each receives: arid lowlands (mean annual rainfall from 1999–2008 = 288 mm), transitional midlands, and humid highlands (mean annual rainfall from 1999–2008 = 1035 mm). Prevailing southern winds brings more precipitation to lower altitudes on the southern side of the island in comparison to the northern side; as a result, the midland and highland zones extend to lower altitudes on the southern slope (i.e. midlands begin ~100 m and ~600 m a.s.l. on the southern and northern side, respectively).

We sampled individuals along three transects (~15 km) that ran from the lowlands through the midlands to the highlands of Santa Cruz. Transects 1 and 2 were located on the southern side: T1, Bahia Academy (0°44’S, 90°18’W) – Los Gemelos (0°37’S, 90°20’W); T2, El Garrapatero (0°41’S, 90°13’W) – Cerro Crocker (0°38’S, 90°19’W). Transect 3 was located on the northern side: Mina Cerro Rojo (0°37’S, 90°22’W) – Itabaca Canal (0°30’S, 90°18’W). We sampled a total of 21 sites: ten sites in the lowlands; seven sites in the midlands; and four sites in the highlands. Sites were grouped into altitude categories for later analysis on a scale of 1–8, with elevation intervals of 100 m.

Arid lowlands were characterised by dry-deciduous open forest dominated by *Bursera graveolens* (Jackson, 1993; McMullen, 1999). Humid highlands consisted of remnant evergreen *Scalesia* closed forest, *Miconia* shrubland, and fern-sedge pampa (Jackson, 1993; McMullen, 1999). Transitional midlands have been largely modified for agriculture, with the introduction of a variety of exotic trees, shrubs and grasses; however, stands of endemic transitional open forest co-dominated by *Psidium galapageium*, *Pisonia floribunda*, and *Piscidia cathagenensis* persisted in the midlands (Jackson, 1993; McMullen, 1999).

**DATA COLLECTION**

Birds were sampled randomly using mist-nets. Only adult birds were processed; we distinguished juveniles based on their prominent yellow gape. We inspected birds for signs of naris and beak malformation caused by *P. downsi*. We categorized individuals as either: normal (no obvious naris or beak malformation; Fig. 1A), malformed (obvious naris or beak malformation; Fig. 1B, C, D, E), or aberrant (presumably genetically caused naris malformation; Fig. 1F). Individuals were considered to have malformed nares if at least one of the following conditions was met for one naris or both nares: (1) enlarged in size (deep and/or wide); (2) circular in shape; (3) asymmetrical in size or shape; and/or (4) without a septum. In addition, individuals with a malformed beak (grooves, cracks, and gouges in the beak keratin radiating from the naris; Fig. 1B, C, D) were considered malformed. Most birds considered malformed exhibited three or more of the above criteria. Birds considered normal or aberrant did not exhibit any of the above criteria. Aberrant birds differed from normal birds by the absence or near absence of a naris or both nares.

Despite grossly enlarged nares in some malformed birds, an assessment of nares malformation was performed qualitatively because considerable overlap can exist in naris length among malformed and normal birds. We did not assume that any one type of disfigurement or combination of disfigurements was more detrimental than another, which was supported by preliminary analysis. Therefore, we grouped all malformed birds together for analyses.

To examine the effect of *P. downsi* parasitism on nares and beak size, we measured naris length and four beak dimensions (mm): beak length feather (length of the culmen); beak length naris (length of the culmen to the anterior edge of the naris); beak depth (at the feather line); and beak width (at the feather line). To examine the possible correlation between body size and naris length, we measured two body size parameters (mm): tarsus length (length of the tarsometatarsus) and wing length (carpal joint to tip of seventh primary). All measurements were taken on the birds’ right side using dial callipers to an accuracy of 0.05 mm. We also recorded mass (g), sex, and the extent of black plumage in males on a scale of 0–4 as approximation for age (Grant & Grant, 1989).

Beak length feather, beak length naris, beak depth, and beak width were all positively correlated \( r > 0.3 \); as was tarsus and wing length \( r > 0.4 \). To avoid multicollinearity in regression analyses, we calculated two principle components using a varimax rotation method with Kaiser normalization: PC1 (beak size) and PC2 (body size). Together, these components explained 54.5 % of the total variance. The strength and direction of the factor loadings for each of the principle component are shown in Table 1.

To assess the effect of malformation on individual survival, we calculated body condition as the residual scores of a least squares linear regression of mass versus the derived body size variable PC2.

**STATISTICAL ANALYSIS**

All statistical analyses were performed using SPSS, version 16 for Windows (SPSS Inc.). The total data set was used to calculate the frequency of naris form categories, but aberrant individuals were removed before further analysis.

First, we examined the effect of covariates on naris form using the derived body size variable PC2.
Second, we examined whether naris form could be predicted by naris length, beak size, body size, or body condition using logistic regression analysis. We calculated the odds ratios (OR) and 95% confidence interval (CI) for the OR, to provide an effect size for the association between malformed and normal birds. Differences in morphology (analysis of variance) and associations between naris length and beak morphology (linear regression analysis) were also tested.

We did not control for body size in the above analyses of nares size and body condition because partial correlation analysis of naris length and beak morphology controlling for tarsus length and wing length revealed negligible differences compared to the zero order correlation ($r < 0.03$).

RESULTS

We collected data from a total of 623 individuals: 65.8% (410 of 623) were categorized as normal, 36.3% (226 of 623) as malformed, and 0.3% (two of 623) as aberrant.
The number of cases of malformation did not differ across sites ($\chi^2 = 24.45$, d.f. = 20, $P = 0.223$; $N = 621$), altitude categories ($\chi^2 = 11.33$, d.f. = 7, $P = 0.13$; $N = 621$), or ecological zones ($\chi^2 = 4.03$, d.f. = 3, $P = 258$; $N = 621$). We also found no significant difference in the frequency of malformation between the southern and northern sides of the island (Fisher’s exact test, $P = 0.912$; $N = 621$), the sexes (Fisher’s exact test, $P = 0.93$; $N = 621$), or across male age categories ($\chi^2 = 4.08$, d.f. = 4, $P = 0.395$; $N = 406$).

Our logistic regression model correctly classified 72.8% of individuals as either malformed or normal ( Hosmer and Lemeshow test: $\chi^2 = 15.58$, d.f. = 8, $P = 0.49$; $N = 592$). Naris length (Wald statistic = 52.78, $b = 4.03$, $P < 0.001$) and beak size (PC1; Wald statistic = 18.35, $b = -0.54$, $P < 0.001$) contributed significantly to the overall model. Neither body size (PC2; $P = 0.096$), nor body condition ($P = 0.314$) predicted naris formation. A malformed bird was more likely to have a greater naris length (OR = 56.66, CI = 19.07–168.37) and a smaller overall beak size (OR = 0.58, CI = 0.46–0.75) than a normal bird (Table 2). A significantly shorter beak length naris and beak length feather, as well as smaller beak depth, contributed significantly to a smaller beak size in malformed birds (Table 2).

Naris length for birds with normal nares was positively correlated with beak length feather ($r = 0.20$, $F_{1,396} = 16.55$, $P < 0.001$; Fig. 2A) and beak width ($r = 0.23$, $F_{1,396} = 21.00$, $P < 0.001$), and, to a lesser extent, beak depth ($r = 0.10$, $F_{1,394} = 3.87$, $P = 0.05$). We found no association between naris length and beak length naris for normal birds ($r = 0.03$, $F_{1,396} = 0.45$, $P = 0.502$; Fig. 2C). By contrast, naris length for malformed birds showed a large and significant negative correlation with beak length naris ($r = -0.32$, $F_{1,223} = 25.40$, $P < 0.001$; Fig. 2D), and, to a lesser extent, a significant positive correlation with beak width ($r = 0.16$, $F_{1,223} = 5.48$, $P = 0.020$). We found no association between naris length and either beak length feather ($r = 0.08$, $F_{1,223} = 1.59$, $P = 0.208$; Fig. 2B) or beak depth for malformed birds ($r = 0.09$, $F_{1,223} = 1.82$, $P = 0.179$). Comparing correlation coefficients between malformed and normal birds, we found a significant difference for naris length and beak length naris only: naris length explained more variance in beak length naris for malformed birds than normal birds ($z_{obs} = -3.59$).

### Table 1. Principal component analysis factor loadings (PC1 and PC2) calculated using a varimax rotation method with Kaiser normalization

<table>
<thead>
<tr>
<th>Variable</th>
<th>PC1</th>
<th>PC2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beak length feather</td>
<td>0.91</td>
<td></td>
</tr>
<tr>
<td>Beak length naris</td>
<td>0.68</td>
<td></td>
</tr>
<tr>
<td>Beak depth</td>
<td>0.50</td>
<td></td>
</tr>
<tr>
<td>Beak width</td>
<td>0.59</td>
<td></td>
</tr>
<tr>
<td>Tarsus length</td>
<td></td>
<td>0.49</td>
</tr>
<tr>
<td>Wing length</td>
<td></td>
<td>0.99</td>
</tr>
</tbody>
</table>

Note: Only loadings > 0.40 are shown.

### Table 2. Beak measurements (shown as means ± SD) and results of an analysis of variance comparison between beak category (normal, malformed) for Darwin’s small ground finch, Geospiza fuliginosa, on Santa Cruz, Galápagos Islands

<table>
<thead>
<tr>
<th>Variable (mm)</th>
<th>Category</th>
<th>$N$</th>
<th>Mean ± SD</th>
<th>$F$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naris length</td>
<td>Normal</td>
<td>396</td>
<td>1.78 ± 0.16</td>
<td>69.79</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Malformed</td>
<td>224</td>
<td>1.92 ± 0.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beak length naris</td>
<td>Normal</td>
<td>400</td>
<td>8.39 ± 0.42</td>
<td>50.21</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>Malformed</td>
<td>226</td>
<td>8.13 ± 0.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beak length feather</td>
<td>Normal</td>
<td>401</td>
<td>12.87 ± 0.65</td>
<td>5.47</td>
<td>0.020</td>
</tr>
<tr>
<td></td>
<td>Malformed</td>
<td>226</td>
<td>12.75 ± 0.64</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beak depth</td>
<td>Normal</td>
<td>398</td>
<td>7.60 ± 0.35</td>
<td>9.59</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>Malformed</td>
<td>226</td>
<td>7.51 ± 0.31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beak width</td>
<td>Normal</td>
<td>397</td>
<td>6.69 ± 0.38</td>
<td>3.09</td>
<td>0.079</td>
</tr>
<tr>
<td></td>
<td>Malformed</td>
<td>226</td>
<td>6.63 ± 0.36</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

DISCUSSION

In the present study, we provide quantitative evidence for naris and beak malformation in adult *G. fuliginosa* as a result of *P. downsi* parasitism. As predicted, malformed birds had greater naris lengths, shorter beak lengths (beak length feathers and beak length naris), and shallower beak depths than birds categorized as normal. Naris length was positively correlated with beak length feather in normal birds, whereas naris length was negatively correlated with beak length naris in malformed birds. Interestingly,
malformation had no measurable consequence for adult body size or body condition.

The present study also confirmed the wide and apparently comparable distribution of *P. downsi* across ecological zones on Santa Cruz (sensu Dudaniec et al., 2007) and revealed a lack of sex or age bias among malformed individuals affected by *P. downsi* as nestlings.

In sum, our results suggest *P. downsi* parasitism in Darwin’s finches: (1) is widespread, well established, and indiscriminate for host sex; (2) has led to measurable naris malformation in adult birds; (3) which is associated with smaller beak dimensions (as a result of beak malformation); (4) but does not have apparent effects for overall growth and adult body condition (fitness costs).

**GENERAL PATTERNS OF *P. DOWNSI*-INDUCED MALFORMATION IN *G. FULIGINOSA***

We found no difference in the number of malformed birds in relation to normal birds across 21 sites of varying vegetation, altitude, and latitude on Santa Cruz. This result concurs with those obtained in previous studies examining the prevalence of *P. downsi* in Darwin’s finches (Fessl & Tebbich, 2002; Dudaniec, Kleindorfer & Fessl, 2006; Dudaniec et al., 2007). Currently, we have little knowledge of finch subpopu-
lation movement within large islands of the Galápagos Archipelago. However, it is unlikely that all malformed birds that we sampled originated from one or a few locations and dispersed widely across the island. Indeed, during our long-term monitoring of Darwin’s finch populations on Santa Cruz subsequent to 2000, we have never recovered colour banded birds in sites other than the site of banding (Kleindorfer et al., 2006). The lack of sex and age bias in malformation corresponds with the high prevalence of P. downsi parasitism both at the time of its discovery in Darwin’s finches in 1997 (Fessl et al., 2001) and subsequently (Dudaniec & Kleindorfer, 2006; Fessl et al., 2006a, b; Dudaniec et al., 2007; Huber, 2008; Kleindorfer & Dudaniec, 2009). Furthermore, there is no evidence to suggest differences in parasite vulnerability among male and female nestlings at present.

**Philornis downsi-induced beak malformation in G. fuliginosa**

The positive relationship between naris length and beak length feather in normal birds provides evidence of isometry in these traits. By contrast, the negative correlation between naris length and beak length naris in malformed birds suggests a loss of this isometry as a result of malformation.

We acknowledge that naris length and beak length naris are not independent measurements, and a negative relationship between the two was possible. Thus, we addressed this problem by examining beak length feather in addition to beak length naris. Beak length naris and beak length feather are dependent measurements, but naris length and beak length feather are not. Because we found that beak length feather was also shorter in malformed birds, we conclude that a decrease in total beak length in malformed birds was not caused by the position or length of the naris (as could be expected for effects on beak length naris alone), but was rather explained by malformation of the beak. Furthermore, the correlation between naris length and beak length feather in malformed birds showed no association. These findings provide evidence of a loss of the isometry that exists between naris length and beak length feathers in normal birds and support the idea that beak malformation has lead to shorter beak length in malformed birds.

Our finding of significant beak malformation is further supported by the only other study that has measured differences in beak dimension between parasitized and nonparasitized birds (Huber, 2008). Using an experimental approach in Darwin’s medium ground finch, *Geospiza fortis*, Huber (2008) showed that nestlings in nests without *P. downsi* larvae had greater beak depth than nestlings in infested nests. Nestling beak length (equivalent to beak length naris) did not differ in nests with and without parasites (Huber, 2008); but, importantly, adult beak dimensions in Darwin’s finches are not reached until 8–9 weeks post-fledging (Grant, 1999). Thus, beak length malformation may only become measurable later in finch development.

**Variation in *P. downsi*-induced malformation in *G. fuliginosa***

The specific criteria used to assess *P. downsi*-induced malformation leaves little doubt that approximately one-third of all birds sampled showed evidence of malformation. So, why did the majority of birds sampled have no obvious disfigurement to the nares and/or beak despite the fact that all finch nests on Santa Cruz are likely to have had parasites (100% prevalence; Dudaniec et al., 2007)? One possible explanation for this discrepancy is the extreme variation in intensity of *P. downsi* per nest and per individual, resulting in variable fitness costs and nesting outcomes (Dudaniec et al., 2007). Another possibility is that not all cases of parasitism lead to long-lasting naris or beak disfigurement. A recent study analysing within-nest video recordings of interactions between fly larvae and finch nestlings (J. O’Connor, J. Robertson & S. Kleindorfer, unpublished data) showed a series of factors that can lead to variation in naris and beak malformation in Darwin’s finches. These factors can be summarized as the number of larvae that feed in the nares, the frequency and duration of these feeding events, the nestling’s ability to defend itself, and the amount of parental antiparasite behaviour per nestling. Therefore, variation in adult naris and beak formation is supported by variation in parasite intensity, and the behaviour of both parasite and host.

**Fitness costs of *P. downsi*-induced malformation in *G. fuliginosa***

We predicted that malformed birds would suffer fitness costs as a direct result of reduction in foraging efficiency and competitiveness. The standard indicators of growth (body size) and health (body condition) revealed no significant difference between malformed and normal birds in this respect. Indeed, malformed birds were observed as active members of the breeding population: malformed females had brood patches and malformed males held territories. Perhaps birds with severe beak malformations incur high survival costs and are not recruited into the breeding population, and hence were not measured here. However, numerous malformed individuals that we sampled were severely disfigured suggesting otherwise (Fig. 1B, D).
In a study examining fitness costs incurred by adult Darwin’s finches with physical disfigurement caused by avian poxvirus, Kleindorfer and Dudaniec (2006) also found no effect on adult body condition but significantly lower pairing success among disfigured males. Similarly, Potti (2008) found no difference in body size or mass between female F. hypoleuca in relation to nestling parasitism from P. azurea, but smaller egg size among females that had been parasitized as nestlings. Therefore, fitness costs associated with nares and bill malformation in adult G. fuliginosa (and with post-parasitism in hosts in general) may be less apparent and more varied than the standard indicators often used to measure fitness costs in hosts presently harbouring parasites.

Previous studies that have compared body condition among nestling Darwin’s finches in nests with and without P. downsi have yielded different results (Fessl et al., 2006a; Huber, 2008). Fessl et al. (2006a) found reduced mass gain in parasitized nestlings, whereas, Huber (2008) found no difference in mass gain, tarsus length, or wing length between parasitized and nonparasitized nestlings. The relationship between parasitism and body size is therefore complex both in space and time (Møller, 1997).

We believe that a phenotypic effect of parasitism in nares and beak dimensions was found in the present study because the nares comprise the physical location for larval feeding and development (Fessl et al., 2006b), which therefore undergo direct modification as a result of parasitism. By contrast, tarsus and wing length, and mass can be influenced by environmental factors (e.g. the level of parental care and food quality; Kruuk, Merilä & Sheldon, 2001).

CONCLUSIONS

Beak length naris (often referred to in the literature as beak length) and beak depth are standard morphological measurements in ecological, social, and evolutionary studies in Darwin’s finches, as well as other bird species. The results obtained in the present study highlight the role played by an introduced parasite as an agent of change for these key beak variables. These findings are significant given the available evidence indicating that beak dimensions are important for mate selection in Darwin’s finches (Christensen, Kleindorfer & Robertson, 2006; Christensen & Kleindorfer, 2007; T. H. Galligan & S. Kleindorfer, unpublished data). For example, beak dimensions are known to influence the production of song characteristics that are used to recognize mates and competitors (Podos, 2001; Christensen et al., 2006). Individuals with nares and beak disfigurement may produce altered and unrecognizable songs, which remains to be tested. An examination of the effects of P. downsi-induced malformation on song production and mate choice in Darwin’s finches may reveal fitness costs to malformed males. It is now apparent that, on islands affected by P. downsi parasitism, induced beak malformation could set Darwin’s finches on a slightly or fundamentally different evolutionary trajectory.

ACKNOWLEDGEMENTS

We thank the Galápagos National Park and the Charles Darwin Research Station for the opportunity to conduct this research; Max Planck Institute for Ornithology, American Bird Conservancy, Conservation International, Galápagos Conservation Fund, and Flinders University for their generous financial support; TAME airlines for assistance with the reduced airfare to and from the archipelago; Birgit Fessl, Zonnetje Auburn, Mari Cruz, Michael Dvorak, Jody O’Connor, Frank Solloway, Sarah Huber, Jennifer Koop, and Ryan Buss for their assistance in the field; and Rachael Dudaniec, Jody O’Connor, and two anonymous reviewers for their comments on this manuscript. This research was conducted under the ethics approval granted by Flinders University (E189).

REFERENCES


