Lateral plate asymmetry, diet and parasitism in threespine stickleback

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Abstract

Individuals with random left–right departures from bilateral symmetry are predicted to exhibit fitness reduction including increased parasitism. In an insular lake population of stickleback (*Gasterosteus aculeatus*) from the Queen Charlotte Islands, Western Canada, phenotypes with high or low number of lateral bony plates exhibited increased plate asymmetry relative to modal phenotypes. Asymmetric lateral plate phenotypes had increased prevalence and to a lesser extent intensity of parasitism relative to symmetric individuals, suggesting that differences in genetic resistance to pathogens contributed to unequal parasitism. The effect occurred mainly in the larger adults and during the warmest season, which may be due to the high metabolic costs incurred during the summer breeding season. Dietary differences between symmetric and asymmetric phenotypes were also detected and could contribute to unequal infection rates by mediating exposure to infected prey items. Our study, which is one of the first long-term field assessments of asymmetry and parasitism, yields results that are consistent with studies linking asymmetry to reduced fitness and indicate that lateral plate asymmetry can be an indicator of poor individual quality, despite its apparent directionality.

Introduction

Departures from bilateral symmetry occur at low frequency in many organisms and can result from environmental or genetic causes (Van Valen, 1962; Palmer & Strobeck, 1986; Palmer, 1994). Typically, the random left–right departures of fluctuating asymmetry (FA) are considered to result from stress during development (Parsons, 1990; Imasheva *et al*., 1999). Genotypes may differ in their ability to buffer development in the face of such stress and thus FA has been used as a measure of developmental stability and has been linked to reduced fitness (Møller, 1997, for review; Møller & Thornhill, 1997; Shykoff & Møller, 1999; but see Leamy, 1997; Clarke, 1998a; Bjorksten *et al*., 2000). Conversely, asymmetries exhibiting a consistent side bias (directional asymmetry or DA) are thought to contain a strong genetic component and to be poor predictors of exposure to stress, developmental stability and/or individual quality (Palmer *et al*., 1993; Palmer, 1994; but see Møller, 1994a; Leamy *et al*., 1997; Leamy, 1999).

For morphological characters under stabilizing selection, extreme phenotypes at the tails of the size distribution often have higher levels of FA than modal phenotypes (Soulé & Cuzin-Roudy, 1982; Møller & Pomiankowski, 1993; Møller, 1994b; Kodric-Brown, 1997). Increased FA may result from decreased heterozygosity in phenotypically extreme individuals as there is a positive association between heterozygosity and developmental stability (Soulé, 1982; Mitton, 1978; O'Brien & Evermann, 1988; Mitton, 1993; for review). Alternatively, increased FA in extreme phenotypes could result from genomic imbalance (Thoday, 1958; Clarke, 1993; for review). Both these explanations rely on genetic differences in developmental stability among individuals.

Numerous studies of asymmetry have documented inverse associations between asymmetry (usually FA) and fitness (Thornhill, 1992; Møller, 1994a, 1997; for

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review). One measurement of fitness is relative levels of infections from parasites as these can impose major metabolic costs on the host (Zuk, 1990; Tierney *et al.*, 1996). Recent evidence shows elevated parasitism of highly asymmetric individuals (Bonn *et al.*, 1996; Møller, 1996a, b, for review; Markusson & Folstad, 1997), potentially due to associations between asymmetry and genetic resistance to parasitic infection (Wakelin, 1978; Sage *et al.*, 1986; Herbert & Cohen, 1993; Møller, 1996b, 1999). Alternatively, increased parasitism of asymmetric individuals could occur if ecological differences between symmetric and asymmetric individuals result in unequal exposure to parasites (Bonn *et al.*, 1996; Møller, 1995; Reimchen & Nosil, 2001a,b) or if the stress caused by the costs of parasitism directly induces asymmetry (Møller, 1992; Polak, 1993; Folstad *et al.*, 1996; Markusson & Folstad, 1997).

Threespine stickleback (*Gasterosteus aculeatus*) have been used as a model system in a diversity of evolutionary and ecological studies (Wootton, 1984; Bell & Foster, 1994). One of the well-studied traits on these fish are the bony lateral plates. These serially arranged plates, which usually extend over the anterior trunk, are part of the antipredator defence apparatus and can vary in number among individuals and among populations according to local selective regimes (Hagen & Gilbertson, 1972; Moodie & Reimchen, 1976; Gross, 1978; Reimchen, 1983, 1994; for review). Bilateral asymmetry in number of plates also occurs among individuals and while this asymmetry appears to be heritable (Hagen, 1973), the frequency distribution of asymmetries is usually normally distributed and approximates ideal FA (Moodie & Reimchen, 1976). General FA theory predicts that asymmetric stickleback should exhibit reduced individual quality. Evidence for this is limited and contradictory. There is a lower incidence of asymmetries in populations exposed to extensive predation by trout, suggesting fitness reduction (Moodie & Reimchen, 1976; Bergstrom & Reimchen, 2000). Alternatively, asymmetric male stickleback appear to have increased success in nesting behaviour and care of young relative to symmetric males and this leads to higher fitness (Moodie & Moodie, 1996).

As part of a long-term evolutionary investigation of endemic stickleback populations from the Queen Charlotte Islands, off the west coast of Canada, a population was monitored over 15 years for several morphological and life history parameters, including spine morphology, parasites and dietary composition (Reimchen, 1980, 1982, 1997). Stickleback were also scored for numbers of lateral plates on both sides of the body and these data provide an opportunity to test several hypotheses on asymmetry: (1) extreme lateral plate phenotypes will exhibit increased asymmetry relative to modal phenotypes; (2) asymmetrical fish will exhibit increased parasitism relative to symmetrical fish and (3) differential parasitism is influenced by dietary variation.

Materials and methods

A total of 10 204 adult (≥ 45 mm standard length – SL) stickleback collected from 1970 to 1987 were scored for sex, number of left and right lateral plates and number of dorsal, pelvic and anal spines, and number of cestodes (*Schistocephalus solidus*, *Cyathocephalus truncatus*), nematodes (*Eustrongylides* spp.) and trematodes (*Bunodera* sp., years 1981–1987 only) (details in Reimchen, 1980, 1982, 1997). Stickleback in this population are all of the low-plated morph.

Absolute lateral plate asymmetry was calculated as R–L. To determine whether extreme phenotypes exhibit higher levels of absolute asymmetry than modal phenotypes, we used methods outlined in Soulé & Cuzin-Roudy (1982). Results were the same whether left or right lateral plate number was used as a measure of trait size and thus we report results from analyses using the number of left lateral plates only. The distributions of both trait size and absolute asymmetry for each sex within each year were standardized (*z* scores, Sokal & Rohlf, 1981). Trait size values within 1 standard deviation (SD) of the mean were designated modal phenotypes, while trait values greater than 1 SD above or below the mean were designated extreme phenotypes. ANOVA was used to test for variability in asymmetry levels between the extreme and modal phenotypes. Sex and year were included as factors in the ANOVA to test whether the effect was similar between genders and among years. Results were congruent when nonparametric analyses were performed comparing the presence, rather than the level, of asymmetry between the extreme and modal phenotypes (data not shown).

We examined the statistical characteristics of signed lateral plate asymmetry (R–L) (Palmer & Strobeck, 1986; Palmer, 1994). We tested for normality using one-sample Kolmogorov-Smirnov tests (K–S), for directional asymmetry (DA) using one sample *t*-tests, binomial tests and measures of skewness (g_1, Sokal & Rohlf, 1981) and for antisymmetry using measures of kurtosis (g_2). Absolute lateral plate asymmetry was used in all subsequent analyses.

Stickleback from Boulton Lake exhibit size- and gender-related variability in the number of dorsal (1–3) and pelvic (0–2) spines and in levels of parasitism (Reimchen, 1980, 1997). However, the presence or absence of plate asymmetry was independent of spine morph and size class in males (morph, $\chi^2_8 = 8.87$, $P = 0.35$, $n = 6059$; size class, $\chi^2_2 = 1.95$, $P = 0.38$, $n = 6059$) and in females (morph, $\chi^2_8 = 13.29$, $P = 0.10$, $n = 4145$; size class, $\chi^2_2 = 0.03$, $P = 0.99$, $n = 4145$).

We compared prevalence (% infected) and intensity (numbers of parasites per individual) of parasitic infection between symmetric and asymmetric stickleback. Patterns of parasitism in Boulton Lake vary seasonally (Reimchen, 1982) and, consequently, we performed all analyses involving parasite prevalence or intensity in four seasons (Winter, Jan.–Mar.; Spring, Apr.–Jun.; Summer, Jul.–Sept.; Fall, Oct.–Dec.). For the three parasite species scored throughout the entire sample period (*S. solidus*, *C. truncatus*, nematodes), we analysed individual and total parasitism (all spp.). For the single species analyses with *S. solidus*, *C. truncatus* and nematodes we exclude fish that were also infected with one or both of the other two species. For analyses with *Bunodera* sp., which was only scored systematically from years 1981–1987, we exclude all multiple species infections.

We tested for associations between lateral plate asymmetry (yes–no) and the prevalence of parasitism using log-likelihood *G*-tests (Sokal & Rohlf, 1981) and loglinear multiway contingency analyses (LL). The loglinear model included five factors: lateral plate asymmetry (symmetric/asymmetric), parasite status (uninfected/infected), gender (male/female), size (<57 mm SL, $= 57$ mm SL) and sample year. To test for associations between asymmetry and parasite intensity we excluded uninfected stickleback and assigned infected fish from each species into one of two infection categories (1 parasite; > 1 parasite). Interactions were tested with *G*-tests and loglinear analyses, replacing parasite status with parasite intensity. To test whether the effect of an interaction in the loglinear analysis was zero we used partial chi-square values (Norusis, 1993). We present two-way interactions between asymmetry and parasitism as well as all significant higher-order interactions involving asymmetry and parasitism. The directionality of trends among years (% asymmetrical infected, % symmetrical infected) was tested using Wilcoxon's Signed Rank tests (WR).

We also compared the diet of symmetric and asymmetric lateral plate phenotypes. The stomach contents of uninfected adult stickleback ($n = 3753$), previously scored for the presence or absence of various food items (Reimchen, 1982), were grouped into two major categories: (1) zooplankton (cladocerans, copepods); (2) macrobenthos (chironomids, amphipods, zygopterans and trichopterans). We used *G*-tests to compare the proportion of these food types between symmetric and asymmetric stickleback overall and partitioned for gender and season. As pelagic copepods (*Diaptomus* spp.) and benthic amphipods (*Gammerus* spp.) act as primary hosts for *S. solidus* and *C. truncatus*, respectively (Clarke, 1954; Vik, 1958), we also compared the consumption of these specific food items. The primary hosts of nematodes are benthic oligochaetes (Hoffman, 1967) and hosts for *Bunodera* are variable, including molluscs and ostracods (Cannon, 1971; Skorping, 1981), but these items were not detected in stickleback stomachs. All statistical analyses were done using SPSS (v.9.0) and all significance levels are two-tailed.

Results

Lateral plate number and general character asymmetry

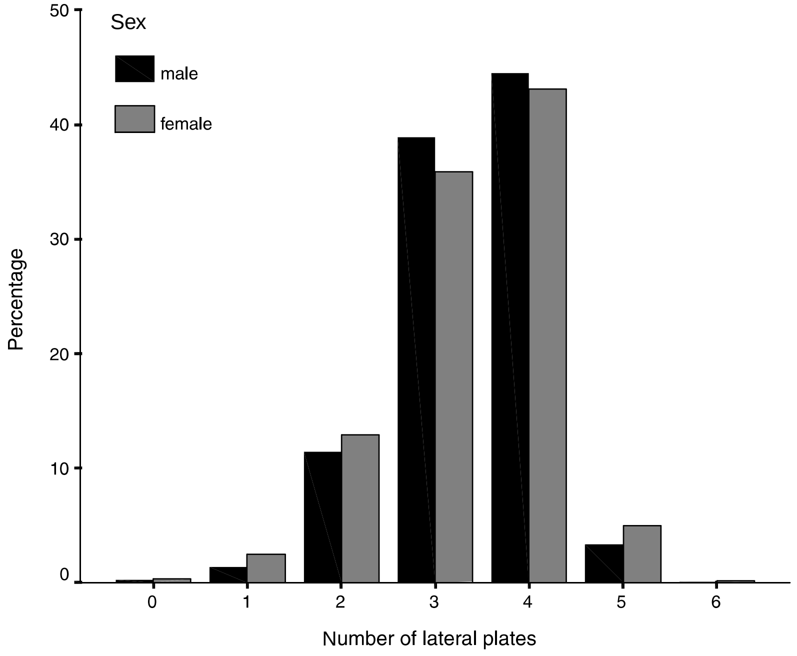
Lateral plate number varied from 0 to 6 among individuals with a mode at four plates (Fig. 1A). Stickleback with low or high plate number exhibited much higher levels of asymmetry than modal phenotypes (Fig. 1B, $F_{6,10198} = 149.72$, $P < 0.001$) and this occurred in all 15 years of sampling ($P < 0.001$; Table 1). Differences between the extreme and modal phenotypes were accentuated in males (0.59, –0.12, respectively) relative to females (0.47, –0.12, respectively).

The frequency distribution of right and left asymmetry was approximately normal (Fig. 2; Table 2) but signed (R–L) asymmetry exhibited a slight left-side bias in males and females (0.55, 0.57 left side departures, respectively, both $P < 0.001$, Binomial tests). Signed lateral plate asymmetry was significantly different from zero (both sexes, $t_{10203} = -9.04$, $P < 0.001$; males, $t_{6058} = -5.70$, $P < 0.001$; females, $t_{4144} = -7.19$, $P < 0.001$, one-sample *t*-test). This left-side bias was greater in females than in males ($t_{10202} = 2.28$, $P < 0.05$), as was absolute asymmetry ($Z = -4.78$, $P < 0.001$, Mann–Whitney *U*-test; Fig. 3). The extent of the directionality and of the differences between the genders fluctuated among years (Fig. 3), with lateral plate asymmetry in some years often approximating ideal FA (i.e. mean signed asymmetry = 0). We detected no significant skewness when the sexes were combined ($t_\infty = 1.17$, $P > 0.20$) or when males and females were treated separately (males, $t_\infty = 1.32$, $P > 0.10$; females, $t_\infty = 0.05$, $P > 0.80$). Although statistical departures from normality were detected (overall, $Z = 24.35$, $P < 0.001$; males, $Z = 19.36$, $P < 0.001$; females, $Z = 14.75$, $P < 0.001$, K–S), the distribution of unsigned asymmetry was leptokurtic (overall, $t_\infty = 12.77$, $P < 0.001$; males, $t_\infty = 11.16$, $P < 0.001$; females, $t_\infty = 6.32$, $P < 0.001$), indicating no evidence of antisymmetry.

Parasite prevalence

We detected increased parasitism in asymmetric stickleback but such associations were dependent on season and size class. Symmetric and asymmetric stickleback exhibited similar infections in each of the four seasons and we detected no two-way interactions between lateral plate symmetry and parasite status (all partial $\chi^2_1 < 2.00$, all $P > 0.05$). However, there was a three-way interaction between parasitism, asymmetry and size class during summer (partial $\chi^2_1 = 3.91$, $n = 3263$, $P < 0.05$, LL; Fig. 4). In the smaller fish (<57 mm), symmetric and asymmetric stickleback exhibited similar infection in both sexes (males, 21.1%, 20.7%, respectively,

(a) Distribution of lateral plate number



(b) Lateral plate number and asymmetry

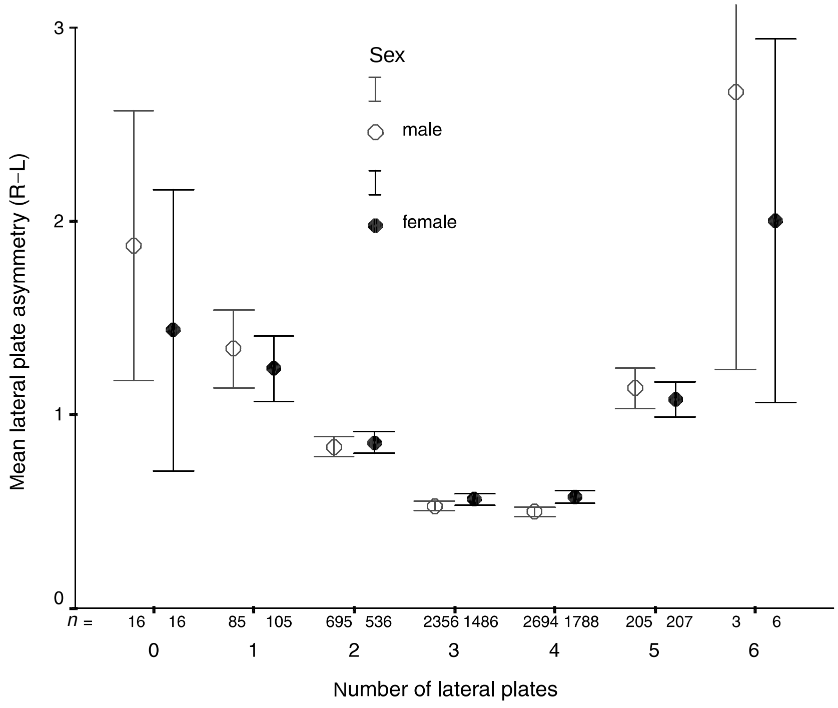


Fig. 1 Distribution of lateral plate number and asymmetry for stickleback from Boulton Lake. (A) Frequency distribution of lateral plate number for male and female stickleback. (B) Absolute | R–L | lateral plate asymmetry (mean ± 95% C.I.) for male and female stickleback with different numbers of left lateral plates. Individuals with relatively high or low plate number exhibit increased asymmetry ($P < 0.001$; ANOVA).

$G = 0.04$, d.f. $= 1$, $P = 0.83$, $n = 1743$; females, 28.0%, 28.2%, respectively, $G = 0.00$, d.f. $= 1$, $P = 0.95$, $n = 1100$, G-tests). In the larger fish (≥57 mm), asymmetric stickleback exhibited increased parasite prevalence relative to symmetric individuals (63.6%, 52.5%, respectively, $G = 5.35$, d.f. $= 1$, $P < 0.05$, $n = 420$). This differential was accentuated in males (58.4%, 44.0%, respectively, $G = 5.61$, d.f. $= 1$, $P < 0.05$, $n = 271$) compared with females (72.3%, 69.7%, respectively, $G = 0.12$, d.f. $= 1$, $P = 0.73$, $n = 149$, G-tests).

Table 1 Results of three-factor ANOVA testing for significant variability in standardized absolute lateral plate asymmetry between extreme and modal lateral plate phenotypes in Boulton Lake stickleback (classification of phenotypes according to procedures outlined in Soulé & Cuzin-Roudy, 1982; see Methods). There was significant variability in lateral plate asymmetry between extreme and modal phenotypes, with extreme phenotypes exhibiting higher levels of asymmetry (see Fig. 1B). This was accentuated in males but was not dependent on sample year.

Source	d.f.	*F*-ratio	*P*
Extreme-Modals	1	345.21	<0.001
Sex	1	4.27	<0.05
Year	14	0.71	0.77
Extreme-Modals × Sex	1	3.75	0.053
Extreme-Modals × Year	4	1.21	0.26
Sex × Year	14	0.58	0.88
Extreme-Modals × Sex × Year	14	1.09	0.36
Total	10198		

Individuals with high or low lateral plate number exhibited increased asymmetry and, consequently, we tested whether overall parasite prevalence or the increased parasitism of large asymmetric fish (all spp.) during summer was influenced by lateral plate number. A loglinear analysis with lateral plate number (1–2, 3–4, 5–6) as a factor revealed parasite prevalence was independent of lateral plate number (all interactions with plate number, $P > 0.15$). In fact, the three-way interaction between parasitism, asymmetry and size class was improved (partial $\chi^2_1 = 5.07$, $P < 0.05$, $n = 3263$, LL).

Trends in single species parasites comparisons were congruent with those from total parasitism. Among pooled size classes, no significant interactions between lateral plate symmetry and parasitic infection were detected in any season (all partial $\chi^2 < 3.00$, all $P > 0.05$). When partitioned for size, asymmetric stickleback exhibited increased parasite prevalence relative to symmetric fish in large but not in small fish (Table 3). None of the three–way interactions between lateral plate condition, parasite status and size class during summer reach statistical significance (*S. solidus*, partial $\chi^2_1 = 2.04$, $P = 0.15$, $n = 3016$; *C. truncatus*, partial $\chi^2_1 = 3.28$, $P = 0.07$, $n = 2370$; nematodes, partial $\chi^2_1 = 1.65$, $P = 0.20$, $n = 2467$; *Bunodera* sp., partial $\chi^2_1 = 0.35$, $P = 0.55$, $n = 684$) but the differential occurred for all four species of parasite (combined probabilities, $\chi^2_8 = 15.70$, $P < 0.05$, Sokal & Rohlf, 1981). Partitioning these data for gender suggests an interaction, as pronounced differences in infection in the large fish occurred for all four parasite species in males but only for nematodes and *Bunodera* sp. in females (Fig. 4). However, a higher order statistical interaction with sex was not detected for any of the parasite species in the loglinear analyses (all $P > 0.05$).

The differential parasitism observed in large fish during summer showed temporal consistency as asymmetric fish had increased parasite prevalence in 8 of 10 years ($Z = -1.785$, $P = 0.07$, WR; heterogeneity among years – partial $\chi^2_{15} = 5.83$, $P = 0.98$, $n = 3263$; Fig. 5). Among smaller fish, trends were less consistent among years ($Z = -0.94$, $P = 0.35$, WR).

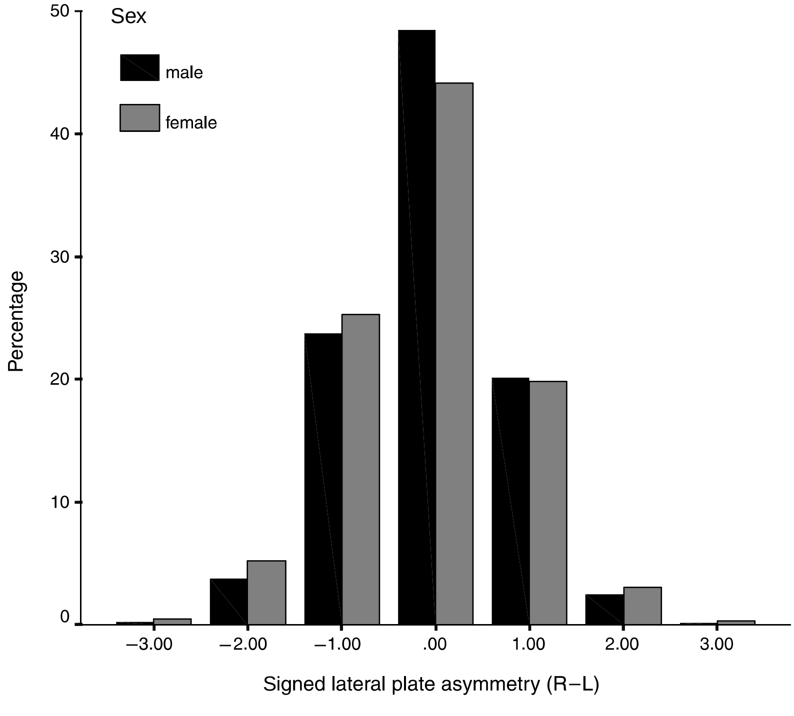


Fig. 2 Frequency distributions of R–L signed lateral plate asymmetry for male and female stickleback from Boulton Lake. $n = 10\ 204$.

Table 2 Descriptive statistics for the number of right (rlp) and left (llp) lateral plates and for signed (R-L) and absolute |R − L| asymmetry in the Boulton Lake stickleback. Values in parentheses are SD except for skew and kurtosis where values in parentheses are SE. Results are given for the population as a whole (n = 10 204) and for each gender separately (males, n = 6059; females, n = 4145). See text for statistical tests concerning asymmetry and Fig. 2 for yearly means.

	No. of lp					
	rlp mean	llp mean	Signed asymmetry mean	Skewness G_1	Kurtosis G_2	Absolute asymmetry mean
Population	3.28 (0.83)	3.36 (0.84)	−0.08 (0.89)	−0.03 (0.02)	0.61 (0.05)	0.61 (0.65)
Males	3.30 (0.79)	3.36 (0.80)	−0.06 (0.86)	−0.04 (0.03)	0.70 (0.06)	0.59 (0.64)
Females	3.24 (0.88)	3.34 (0.89)	−0.10 (0.93)	−0.002 (0.04)	0.48 (0.08)	0.65 (0.67)

Parasite intensity

Among infected stickleback ($n = 3270$), asymmetric individuals exhibited increased parasite intensity, but this was dependent on species of parasite, season and sex. For *S. solidus,* we did not detect any overall association between lateral plate asymmetry and parasite intensity in any of the four seasons (all partial $\chi^2 < 1.55$, all $P > 0.05$). However, in summer increased parasitism of asymmetric stickleback was dependent on sex (partial $\chi^2_1 = 7.44$, $P < 0.05$, $n = 672$) and on year (partial $\chi^2_{15} = 27.10$, $P < 0.05$, $n = 672$). There was no statistical effect in males ($G = 0.88$, d.f. = 1, $P = 0.35$, $n = 386$) but, in females, asymmetric fish exhibited increased frequencies of multiple parasite infection relative to symmetric individuals (57.3%, 44.2%, respectively, $G = 4.91$, d.f. = 1, $P < 0.05$, $n = 286$) and this occurred in 7 of 10 years.

For *C. truncatus,* we detected an association between lateral plate asymmetry and parasite intensity in winter (partial $\chi^2_1 = 9.47$, $P < 0.01$, $n = 362$), but not in the other three seasons (all partial $\chi^2 < 2.00$, all $P > 0.10$). Unexpectedly, symmetric fish had higher levels of multiple parasite infections than asymmetric fish (50.6%, 37.5%, respectively). There were few infected females during winter and these exhibited similar infection intensities between symmetric and asymmetric individuals (25.0%, 25.8%, respectively, $G = 0.01$, d.f. = 1, $P = 0.94$, $n = 63$, G-test). Thus the interaction detected in the loglinear analysis was essentially due to increased levels of multiple parasite infections in symmetric vs. asymmetric male stickleback. This occurred in both size classes (< 57 mm, 51.1%, 38.6%, respectively, $G = 3.39$, d.f. = 1, $P = 0.07$, $n = 219$; ≥ 57 mm, 71.1%, 42.9%, respectively, $G = 6.56$, d.f. = 1, $P < 0.01$, $n = 80$, G-tests) and was consistent across years (lateral plate asymmetry × parasite intensity–year interaction, partial $\chi^2_7 = 8.25$, d.f. = 1, $P = 0.41$).

Asymmetrical females exhibited increased multiple nematode infections relative to symmetric females in the seasons where these parasites were consistently detected in (Fig. 6). Partitioning the data by gender and pooling the results across seasons revealed a significant excess of multiple parasite infections in asymmetric vs. symmetric females (51.4%, 32.8%, respectively, $G = 4.56$, d.f. = 1, $P < 0.05$, $n = 128$, G-test) and no significant differences in males (36.2%, 44.3%, $G = 0.87$, d.f. = 1, $P = 0.35$, $n = 130$, G-test).

We detected increased *Bunodera* infection among asymmetric fish during winter (80.4%, 69.7%, respectively, partial $\chi^2_1 = 3.88$, $P < 0.05$, $n = 300$) but not during the remaining seasons (partial $\chi^2 < 2.80$, $P > 0.05$). The effect was most expressed in females (Fig. 6). We partitioned the data by gender, pooled the results across seasons and introduced season in a five-factor loglinear analysis. In females, this revealed increased parasite intensity in asymmetric vs. symmetric individuals independent of other interactions (74.2%, 60.7%, $n = 336$, partial $\chi^2_1 = 6.77$, $P < 0.01$, $n = 336$). Yearly trends were fairly consistent as asymmetric fish exhibited increased levels of multiple parasite infection in 6 of 7 years ($Z = -2.03$, $P < 0.05$, WR). In males, we did not detect any differences in multiple infections between asymmetric and symmetric fish (62.9%, 64.3%, respectively, partial $\chi^2_1 = 0.31$, $P = 0.72$, $n = 530$).

Diet of uninfected stickleback

Among pooled samples, asymmetric stickleback exhibited increased consumption of macrobenthos relative to that of symmetric phenotypes (63.6%, 58.3%, $G = 6.23$, d.f. = 1, $P < 0.05$, $n = 3753$) but there was similar consumption of zooplankton, copepods and amphipods (all $P > 0.15$). Partitioning the data shows that the amount of association was dependent on season and gender. Although macrobenthos was more common in asymmetric fish, this effect was greatest in males during summer (62.2%, 54.0%, respectively, $G = 4.19$, d.f. = 1, $P < 0.05$, $n = 601$) and in females during winter (84.9%, 71.2%, respectively, $G = 3.94$, d.f. = 1, $P < 0.05$, $n = 145$). During summer, zooplankton was more common in asymmetric males (57.5%, 49.3%, respectively, $G = 4.05$, d.f. = 1, $P < 0.05$, $n = 601$) but, in females, it was less common (32.5%, 45.0%, respectively, $G = 4.42$, d.f. = 1, $P < 0.05$, $n = 277$). Amphipods were less common in asymmetric females than symmetric females

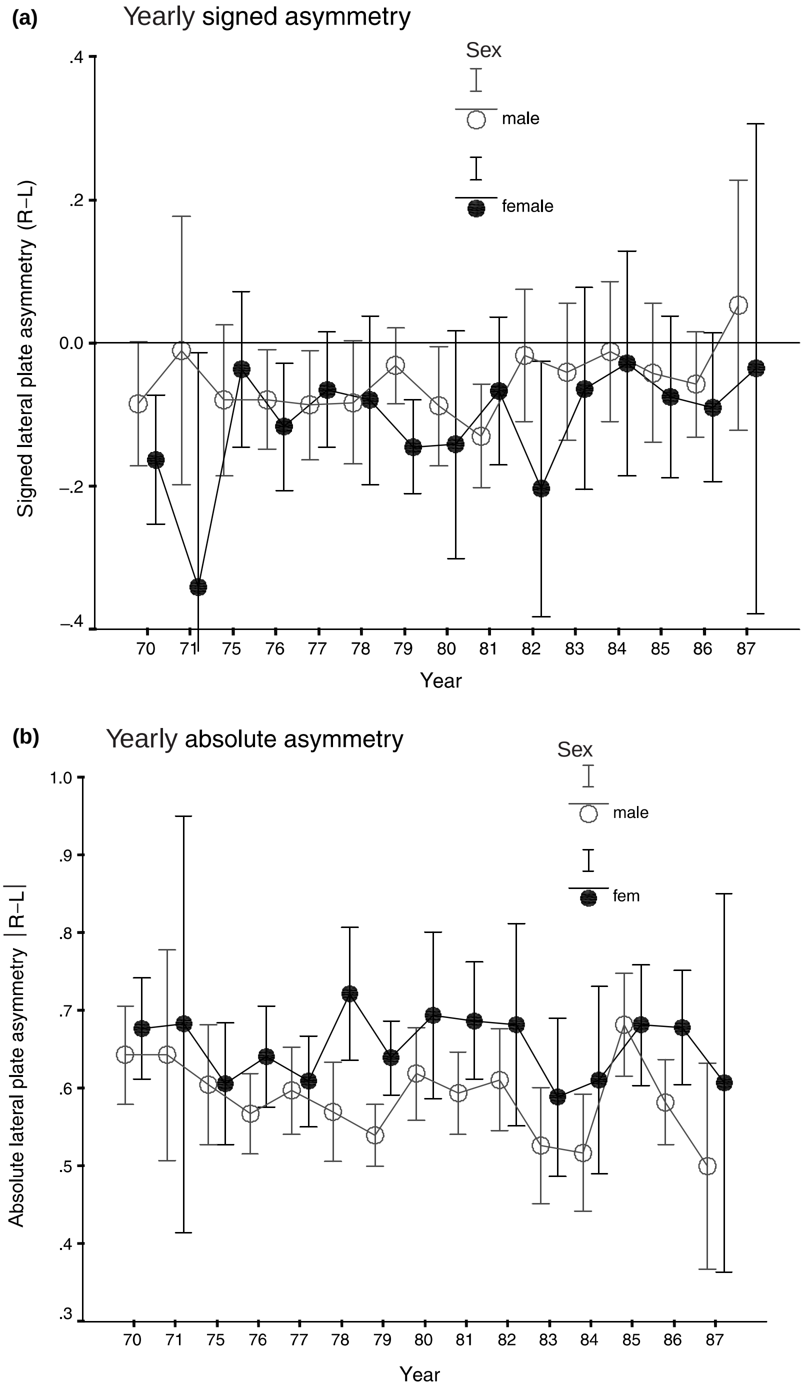


Fig. 3 Yearly levels of lateral plate asymmetry. (A) Signed (R–L) lateral plate asymmetry (mean ± 95% C.I.) in male and female stickleback. Lateral plate asymmetry was slightly directional, with increased left-sided asymmetry evident in most years. (B) Yearly absolute |R–L| lateral plate asymmetry (mean ± 95% C.I.) in male and female stickleback.

during summer (4.2%, 11.7%, respectively, $G = 5.45$, d.f. = 1, $P < 0.05$, $n = 277$). All other seasonal comparisons of diet between symmetric and asymmetric fish were nonsignificant (all $P > 0.05$, G-tests). Including size and year in multiway contingency analyses did not reveal higher order interactions with either factor for any seasonal dietary comparison (all $P > 0.05$).

Discussion

Elevated FA has been linked to increased parasitism in numerous taxa, including mammals, insects, birds and fish (Møller, 1996b, for review). Lateral plate asymmetry approximates FA in most stickleback populations (Moodie & Reimchen, 1976) as did some yearly

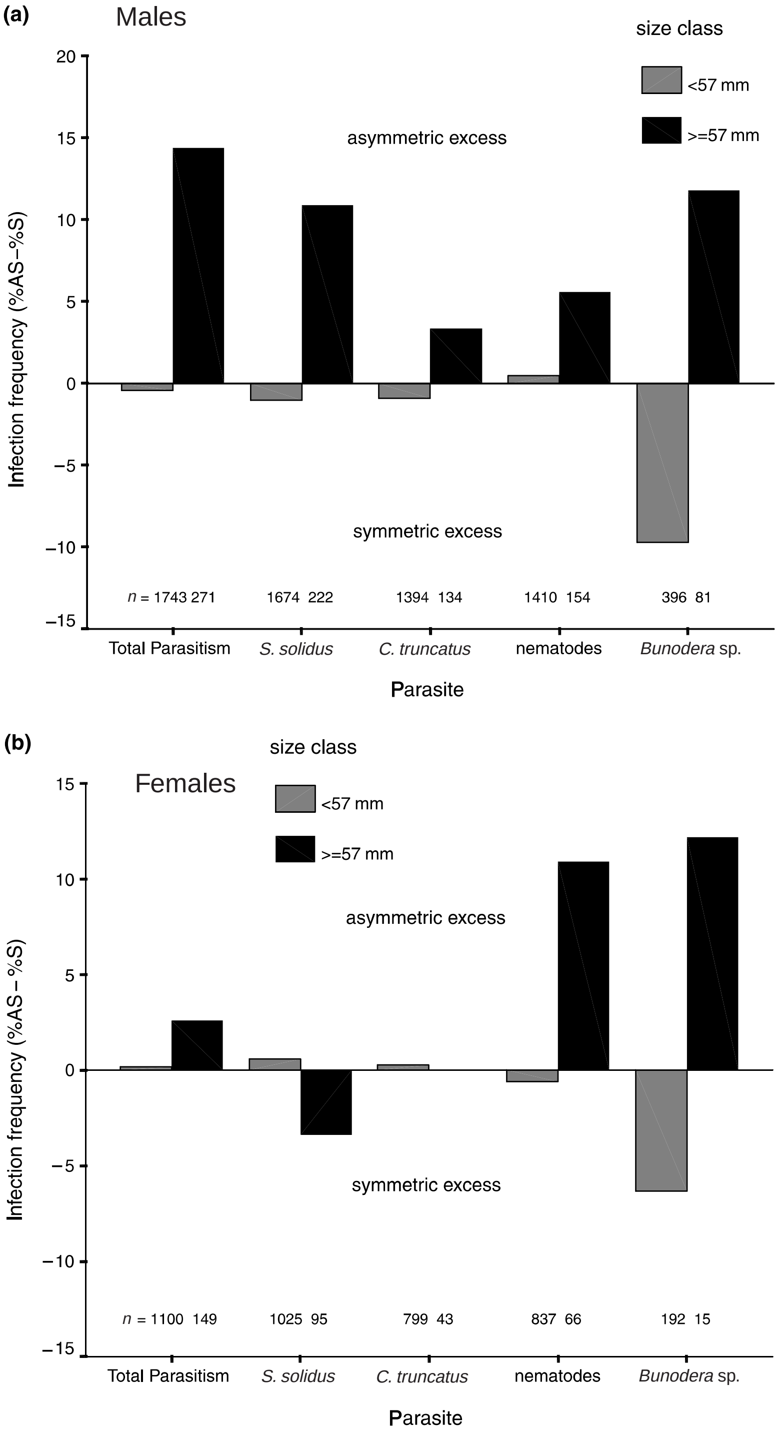


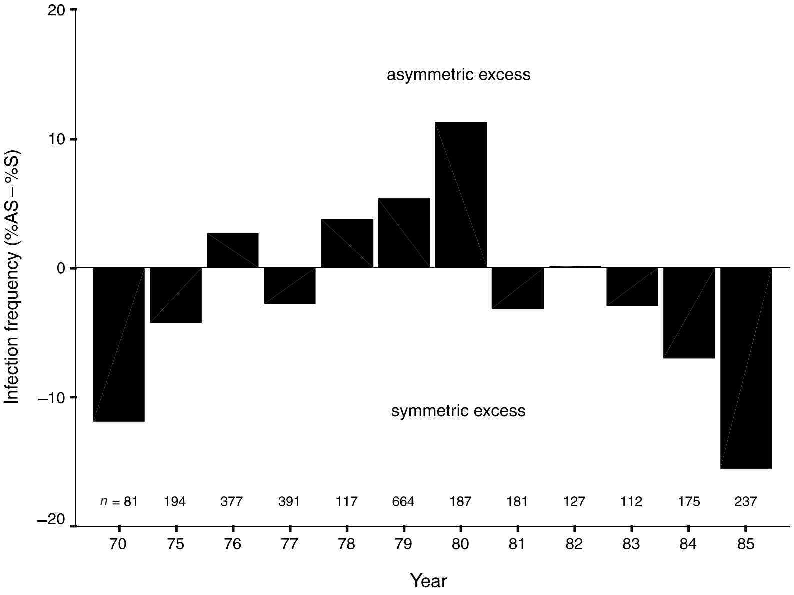
Fig. 4 Signed differences in infection frequency between asymmetric (AS) and symmetric (S) stickleback (%AS –%S) during summer (Jul.–Sept.). Results are shown for total parasitism (all spp.) and for each species separately as well as for two size classes (small, <57 mm; large ≥57 mm). Infection frequencies tend toward equality in the smaller fish while asymmetric fish generally show increased parasite prevalence in the larger fish. (A) Males. (B) Females.

samples within Boulton Lake. Also, associations between lateral plate asymmetry and trait size met the predictions of FA theory (cf. Soulé & Cuzin-Roudy, 1982; Møller & Pomiankowski, 1993). These results, coupled with the increased parasitism of asymmetric individuals, which was independent of plate number, suggest lateral plate asymmetry is a measure of individual quality, despite its slight directionality (Palmer *et al.*, 1993; Palmer, 1994). Indeed, several recent studies suggest traits exhibiting DA may be under very weak genetic control (Leamy *et al.*, 1997; Leamy, 1999; see also Coyne, 1987), can arise from developmental stress

Table 3 Parasite prevalence for symmetric (S) and asymmetric (AS) stickleback collected during the summer (Jul.–Sept.) season. Infection frequencies tend towards equality in the smaller (<57 mm) fish but are reduced in symmetric vs. asymmetric stickleback for the larger fish (≥57 mm, combined probabilities, $P < 0.05$). G-values shown are from log-likelihood G-tests for each parasite species and size class.

	<57 mm					≥57 mm				
	%S	%AS	*n*	*G*	*P*	%S	%AS	*n*	*G*	*P*
S. solidus	19.7	19.6	2699	0.01	0.92	41.4	48.4	317	1.58	0.21
C. truncatus	1.3	0.9	2193	1.12	0.29	0.0	2.4	177	3.10	0.21
Nematodes	3.4	3.6	2247	0.05	0.82	15.9	25.2	220	2.94	0.09
Bunodera sp.	53.4	44.7	588	4.38	0.04	43.1	60.0	96	2.73	0.10

(a) Total parasitism (fish <57 mm)



(b) Total parasitism (fish >=57 mm)

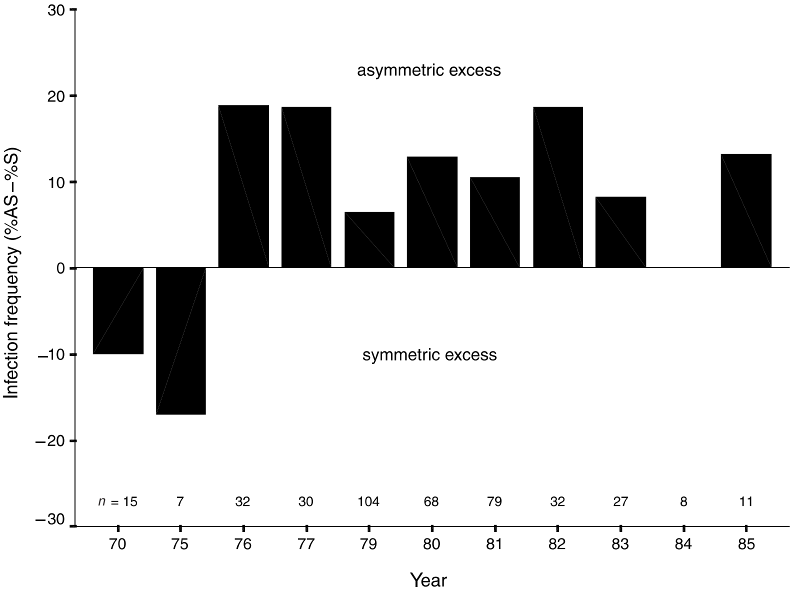


Fig. 5 Yearly signed differences in total parasite prevalence (all spp.) between asymmetric (AS) and symmetric (S) stickleback (%AS – %S) during summer (Jul.–Sept.). Results are shown for two size classes. (A) <57 mm; (B) ≥57 mm.

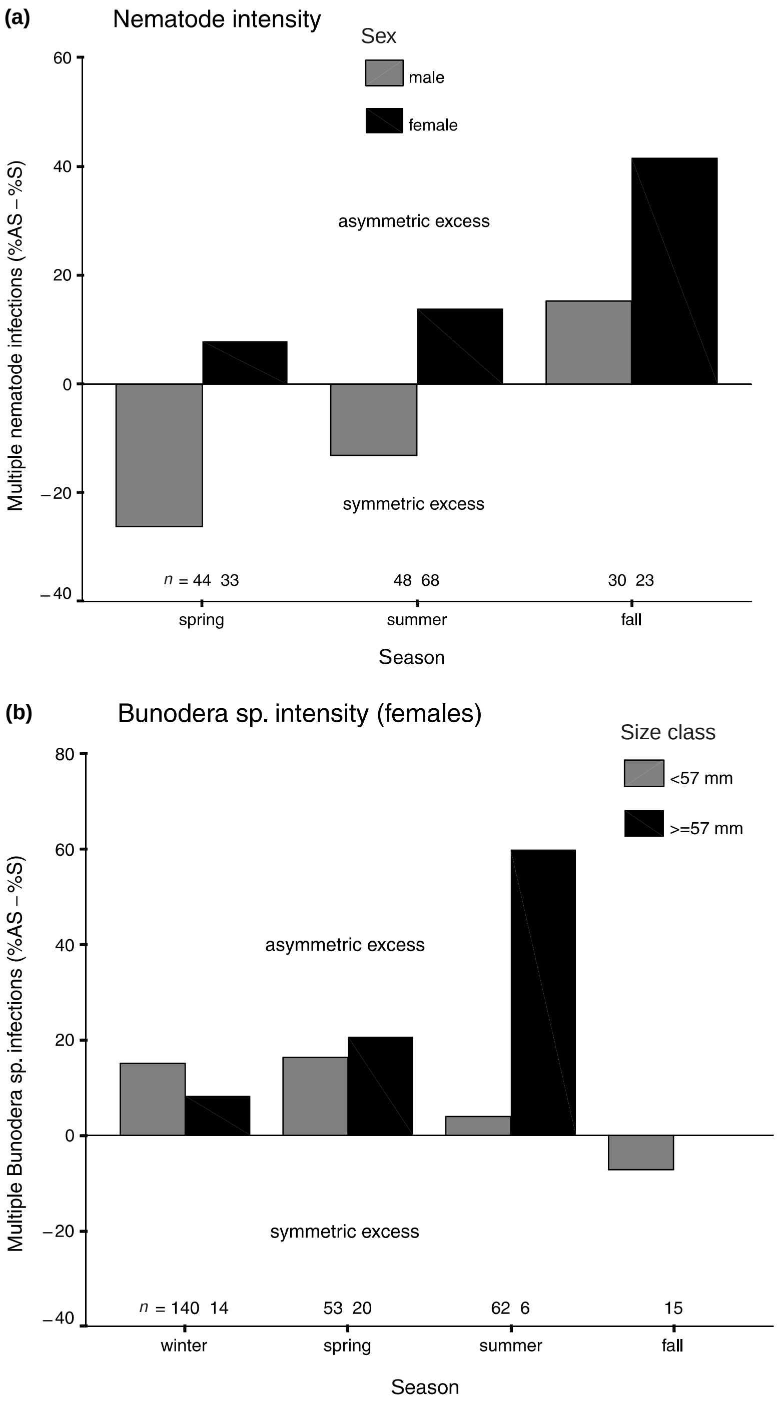


Fig. 6 Seasonal variation in signed differences in multiple (>1 parasite) nematode and *Bunodera* sp. infections between asymmetric (AS) and symmetric (S) stickleback (%AS – %S). (A) Seasonal differences in nematode intensity. Differences pooled across seasons are significant in females ($P > 0.05$, *G*-test) but not in males ($P = 0.35$). (B) Seasonal differences in *Bunodera* sp. intensity in females for two size classes (<57 mm, ≥57 mm). Differences pooled across seasons are significant ($P > 0.05$, LL).

(Graham *et al.*, 1993) and are linked to individual quality (Møller, 1994a; Simmons & Ritchie, 1996; but see Kimball *et al.*, 1997).

Four mechanisms, which are not mutually exclusive, could account for the increased parasitism of asymmetric individuals. Firstly, differential infection by parasites or disease could reflect genetic variability in immunocompetence (Wakelin, 1978; Sage *et al.*, 1986; Møller, 1999). Such genetic differences between phenotypes may be particularly feasible for plate asymmetry, which is heritable in *Gasterosteus* (Hagen, 1973). Associations between plate asymmetry and plate number also support this hypothesis. As reduction in lateral plate number or symmetry may have functional implications (i.e. reduced defence against predators, Bergstrom & Reimchen, 2000; Reimchen, 1983, 2000), we predicted associations between asymmetry and trait size (number of plates) would be indicative of traits under stabilizing selection, with extreme phenotypes exhibiting increased asymmetry (Møller, 1991; Balmford *et al.*, 1993; Møller & Pomiankowski, 1993; Crespi & Vanderkist, 1997; Clarke, 1998b). Consistent with theory and prediction, stickleback with relatively low or high lateral plate number had higher levels of asymmetry than modal phenotypes, as has been documented in other species of fish and insects (Soulé & Cuzin-Roudy, 1982; Kodric-Brown, 1997; but see Woods *et al.*, 1999). Homozygosity has been linked to inferior developmental stability (Leary *et al.*, 1984; Mitton, 1993; for review; but see Clarke, 1993) and Soulé (1982) suggested homozygotes could be partitioned into the tails of the size distribution for some morphological characters. Immunological defences against infection are accentuated by enzyme heterozygosity (Tooby, 1982; Thornhill & Gangestad, 1993; Mitton, 1995). If stickleback at the edge of the frequency distribution for lateral plates, which exhibit high levels of asymmetry, are highly homozygous relative to modal phenotypes, they may have reduced resistance to parasites and such associations provide one plausible mechanism to account for the increased parasitism of asymmetric stickleback observed in our study.

However, alternative explanations for the associations between trait size and asymmetry exist. Genotypes with extreme phenotypes may contain poorly balanced or poorly integrated genes (Thoday, 1958) and exhibit weaker developmental stability as a result of such genetic imbalance (Clarke *et al.*, 1992; Clarke, 1993; for review). Also, extreme phenotypes may exhibit higher FA if individuals with extremely large or small morphological structures suffer greater stress during development as a result of bearing traits of suboptimal size (Møller & Pomiankowski, 1993). If asymmetrical structures result in reduced performance (e.g. Møller, 1991; Balmford *et al.*, 1993), this may cause even greater stress during subsequent development. Conceivably, elevated FA in extreme phenotypes results from a combination of these factors, each of which may influence relative parasitism of asymmetric phenotypes.

Secondly, increased infection of highly asymmetric phenotypes could simply reflect increased exposure to infectious agents (Bonn *et al.*, 1996). Consistent with prediction, we detected some dietary differences between symmetric and asymmetric phenotypes that could influence encounter rates with infected prey items. For example, zooplankton consumption in summer was biased towards asymmetric plate phenotypes in large males but towards symmetric phenotypes in large females. Only asymmetric males exhibited increased prevalence of *S. solidus* and this could occur if the relatively more pelagic diet of asymmetric males, but not asymmetric females, results in high encounter rates between asymmetric males and the pelagic copepod that acts as primary host for *S. solidus*. Also, asymmetric adult males, but not asymmetric females, had increased *C. truncatus* infections relative to symmetric fish during summer. Reduced consumption of amphipods, the primary host of *C. truncatus*, by asymmetric females relative to symmetric females could contribute to the lack of excess parasite prevalence observed in asymmetric females.

Such associations between diet and parasitism are consistent with results for symmetric and asymmetric pelvic spine phenotypes in subadult stickleback and for differential parasitism between the sexes, where dietary variation appears to contribute to differential parasitism (Reimchen, 1997; Reimchen & Nosil, 2001a,b). Competition for resources is one mechanism that could lead to ecological differences among individuals differing in levels of asymmetry (e.g. Møller, 1995; Nosil & Reimchen, 2001). Our results suggest ecological sources of differential parasitism or fitness among individuals varying in levels of asymmetry may be of greater importance than currently appreciated. If so, this may account for the increased *C. truncatus* intensity in symmetric vs. asymmetric males, a trend opposite to that predicted by theory (see also Reimchen, 1997).

Thirdly, asymmetry may itself be a signal of increased environmental stress during development and/or poor phenotypic condition, and both these factors can result in decreased immunocompetence (Gershwin *et al.*, 1985; Parsons, 1990; for review; Herbert & Cohen, 1993; Badyaev *et al.*, 2000; Nosil & Reimchen, 2001). For example, we detected increased parasite prevalence in asymmetric stickleback only during summer. Metabolic costs for ectotherms are accentuated during warm weather (Wootton, 1984) and stickleback in poor phenotypic condition may be unable to mount an efficient immune response. A similar process could account for why increased parasite prevalence in asymmetric stickleback was restricted to large adult fish, which bear the costs of reproduction (Wootton & Evans, 1976; Fitzgerald *et al.*, 1989; Bakker, 1994). Costs of reproduction may also explain why differences were more pronounced in males than females, as male stickleback provide all the parental care and exhibit all the territorial defence (De-Fraipont *et al.*, 1992; Bakker, 1994).

Fourthly, the costs of parasitism could result in increased physiological stress during development and thus directly induce high levels of asymmetry (Møller, 1992; Polak, 1993; Folstad *et al.*, 1996; Markusson & Folstad, 1997). This could occur in stickleback as initial *S. solidus* infections can occur before and during the expression of the lateral plates. However, the general importance of this is unknown as two species of parasites (*C. truncatus* and *Eustrongylides*) occur predominantly on fish larger than 40 mm SL that have completed plate development.

In summary, stickleback with lateral plate numbers that departed most from the population mean had increased asymmetry and these asymmetrical phenotypes tended to exhibit higher prevalence and to a lesser extent higher intensity of parasitism than symmetric phenotypes. Such associations between asymmetry, trait size and parasitism suggest differences in genetic resistance to parasitic infection, possibly mediated by enzyme heterozygosity. Our results represent one of the few long-term studies of the fitness consequences of departures from symmetry and have allowed the detection of slight seasonal and age-related trends in differential parasitism between symmetric and asymmetric phenotypes. Furthermore, ecological factors influencing parasitism are rarely addressed in studies of bilateral asymmetry and our data suggest that niche and dietary differences among individuals varying in levels of asymmetry can contribute to unequal infection rates. The findings have additional implications to the expanding body of literature on asymmetry as they demonstrate that fitness reduction is not restricted to FA but can occur with traits that exhibit slight directionality.

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