Mesoscale spatiotemporal variability in a complex host-parasite system influenced by intermediate host body size

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Background. Parasites are essential components of natural communities, but the factors that generate skewed distributions of parasite occurrences and abundances across host populations are not well understood. **Methods.** Here, we analyse at a seascape scale the spatiotemporal relationships of parasite exposure and host body-size with the proportion of infected hosts (i.e. prevalence) and aggregation of parasite burden across ca. 150 km of the coast and over 22 months. We predicted that the effects of parasite exposure on prevalence and aggregation are dependent on host body-sizes. We used an indirect host-parasite interaction in which migratory seagulls, sandy-shore molecrabs, and an acanthocephalan worm constitute the definitive hosts, intermediate hosts, and endoparasite, respectively. In such complex systems, increments in the abundance of definitive hosts imply increments in intermediate hosts' exposure to the parasite's dispersive stages. **Results.** Linear mixed-effects models showed a significant, albeit highly variable, positive relationship between seagull density and prevalence. This relationship was stronger for small (cephalothorax length > 15 mm) than large molecrabs (< 15 mm). Independently of seagull density, large molecrabs carried significantly more parasites than small molecrabs. The analysis of the variance-to-mean ratio of per capita parasite burden showed no relationship between seagull density and mean parasite aggregation across host populations. However, the amount of unexplained variability in aggregation was strikingly higher in large that small intermediate hosts. This unexplained variability was driven by a decrease in the mean-variance scaling in heavily infected large molecrabs. **Conclusions.** These results show complex interdependencies between extrinsic and intrinsic population attributes on the structure of host-parasite interactions. We suggest that parasite accumulation—a characteristic of indirect host-parasite interactions—and subsequent increasing mortality rates over ontogeny underpin size-dependent host-parasite dynamics.

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18   **Abstract**

19   **Background.** Parasites are essential components of natural communities, but the factors that

20   generate skewed distributions of parasite occurrences and abundances across host populations

21   are not well understood.

22   **Methods.** Here, we analyse at a seascape scale the spatiotemporal relationships of parasite

23   exposure and host body-size with the proportion of infected hosts (i.e. prevalence) and

24   aggregation of parasite burden across ca. 150 km of the coast and over 22 months. We predicted

25   that the effects of parasite exposure on prevalence and aggregation are dependent on host body-

26   sizes. We used an indirect host-parasite interaction in which migratory seagulls, sandy-shore

27   molecrabs, and an acanthocephalan worm constitute the definitive hosts, intermediate hosts, and

28   endoparasite, respectively. In such complex systems, increments in the abundance of definitive

29   hosts imply increments in intermediate hosts' exposure to the parasite's dispersive stages.

30   **Results.** Linear mixed-effects models showed a significant, albeit highly variable, positive

31   relationship between seagull density and prevalence. This relationship was stronger for small

32   (cephalothorax length > 15 mm) than large molecrabs (< 15 mm). Independently of seagull

33   density, large molecrabs carried significantly more parasites than small molecrabs. The analysis

34   of the variance-to-mean ratio of per capita parasite burden showed no relationship between

35   seagull density and mean parasite aggregation across host populations. However, the amount of

36   unexplained variability in aggregation was strikingly higher in large that small intermediate

37   hosts. This unexplained variability was driven by a decrease in the mean-variance scaling in

38   heavily infected large molecrabs.

39   **Conclusions.** These results show complex interdependencies between extrinsic and intrinsic

40   population attributes on the structure of host-parasite interactions. We suggest that parasite

41   accumulation—a characteristic of indirect host-parasite interactions—and subsequent increasing

42   mortality rates over ontogeny underpin size-dependent host-parasite dynamics.

44   **Introduction**

45   Parasites commonly show aggregated distributions across host populations. The degree of

46   parasite aggregation—i.e., few hosts concentrate most of the parasites—can have consequences

47   for the stability of parasite-host interactions (Wilson et al., 2001). For example, seminal

48   theoretical work predicts that parasite aggregation stabilises host-parasite dynamics (Anderson &

49   May, 1978; May & Anderson, 1978). However, extreme aggregation in host populations with

50   low prevalence, such that a very small proportion of hosts carry a high parasite burden, might

51   reduce the ability of the parasite to regulate the host population (Anderson & May, 1978).

52   Variations in "extrinsic" factors such as exposure to infective stages of parasites (hereafter

53   referred to as "parasite exposure") can generate parasite aggregation across hosts (McCoy et al.,

54   2016). However, "intrinsic" host-population factors, such as age and body-size structure, can

55   significantly influence the links between variation in parasite exposure and aggregation

56   (Anderson & May, 1978). Determining the links of these extrinsic and intrinsic factors with

57   patterns of prevalence and aggregation across host populations will improve our understanding

58   of complex host-parasite dynamics (Shaw, Grenfell & Dobson, 1998; Wilson et al., 2001;

59   Morand & Krasnov, 2008).

60   Variations in parasite exposure can affect infection rates and thus parasite aggregation

61   across host populations. Assuming a certain, basal level of parasite aggregation (Wilson et al.,

62   2001), an increase in parasite exposure will lead to an increase in the proportion of infected hosts

63   (i.e. prevalence) and the parasite burden of the already-infected individual; this is because

64   infection increases host susceptibility to subsequent infections when there is no further increase

65   in host's immunity (Wilson et al., 2001). For example, the Pacific chorus frog *Pseudacris regilla*

66   (Baird & Girard, 1852) shows increased susceptibility to the trematode *Ribeiroa ondatrae* Looss,

67   1907 after being infected, because the previous infection impairs hosts' immunological system

68   (Johnson & Hoverman, 2014). Therefore, it can be hypothesised that increased parasite exposure

69   will increase the infection rate of already-infected hosts, increasing parasite burden and therefore

70   the aggregation across the population.

71   The effect of parasite exposure on aggregation can depend on host size structure.

72   Assuming that body size is correlated with host age (Ebert, 1999), it can be suggested that small

73   hosts have been exposed for a shorter time to parasite infection than larger conspecifics (Muñoz

74   & George-Nascimento, 2008)—this in turn should result in smaller parasite burdens in the

75   former than the latter (Grutter & Poulin, 1998; Poulin, 2013). For example, subpopulations of

76   fish composed by larger individual have a higher prevalence and per capita parasite burden than

77   subpopulation composed by smaller individuals (Lo, Morand & Galzin, 1998; Poulin, 2000;

78   Muñoz, Valdebenito & George-Nascimento, 2002). Since infection is positively related with host

79   susceptibility (e.g. Johnson & Hoverman, 2014), then it can be suggested that larger individuals

80   will have larger infections rates than smaller individuals (see Muñoz, Valdebenito & George-

81   Nascimento, 2002; Muñoz & George-Nascimento, 2008 for an example in fish hosts). In

82   complex parasite-host interactions, intermediate hosts are usually unable to expel the parasites

83   and thus they accumulate parasites over the lifetime (Muñoz & George-Nascimento, 2008;

84   Campião et al., 2015). In addition, the probability of mortality can be larger for heavily infected

85   hosts, leading to a decrease in prevalence and aggregation over ontogeny (Rousset et al., 1996;

86   Duerr et al., 2003). We can expect that in such complex systems, therefore, parasite exposure

87   should have differential effects on the parasitosis of small- and large-sized intermediate hosts.

88   Migratory seagulls and their parasites constitute a model system to investigate how

89   parasite exposure and intermediate host size-structure relate with prevalence and aggregation.

90   Migratory Charadriiformes are usually involved in indirect parasite-host interactions, in which

91   the birds are the definitive host and there is at least one intermediate host, usually invertebrates

92   that accumulate parasites over their ontogeny (e.g. Rodríguez, D'Elía & Valdivia, 2016). In these

93   interactions, parasite infective stages are trophically transmitted between definitive and

94   intermediate hosts, so that variations in the abundance of the former imply significant variations

95   in parasite exposure of the latter (Latham & Poulin, 2003; Smith, 2007). For example, local

96   increments in the abundance of migratory birds have been linked to increases in the prevalence

97   of infectious diseases in intermediate hosts (Altizer, Bartel & Han, 2011; McCoy et al., 2016). In

98   addition, increases in seagull abundances can be followed by concomitant increments in per

99   capita parasite loads in the intermediate host populations (Latham & Poulin, 2003; Smith, 2007).

100 Accordingly, it can be expected that increments in the abundance of migratory seagulls should

101 result in an increase in parasite exposure. If increasing exposure positively affects the mean

102 infection rate, then the concomitant increase in the spatial variance in parasite infection can

103 increase parasite prevalence and aggregation across intermediate host populations. Migratory

104 Charadriiform species are under severe threats worldwide, owing to human-induced habitat

105 destruction, hunting, and climate change-related impacts on migratory timing, among other

106 factors (Munro, 2017). Understanding how migratory Charadriiformes take part in complex host-

107 parasite systems can have therefore important implications for both, fundamental and applied

108 ecology.

109   In this study we test the hypothesis that, if the variability in parasite exposure and host

110 body size generate intra-population variability in host infection rates, both factors

111 interdependently influence prevalence and aggregation across host populations. We predicted

112 that increases in migratory seagull densities should be linked to increases in parasite prevalence

113   and aggregation in intermediate large-, but not small-sized, intermediate hosts. An indirect host-

114   parasite interaction, composed by migratory seagulls (definitive hosts), molecrabs (intermediate

115   hosts), and an acanthocephalan worm (parasite), was analysed. In this complex system, infected

116   seagulls release the infective stage of *Profilicollis altmani* Meyer, 1931 in their faeces. These

117   faeces are then ingested by the molecrab *Emerita analoga* Stimpson, 1857, and the cycle is

118   completed after predation on molecrabs by the seagulls *Larus dominicanus* (Lichtenstein, 1823),

119   *Chroicocephalus maculipennis* Lichtenstein, 1823, *Leucopheus modestus* (Tschudi, 1843), and

120   *Leucopheus pipixcan* (Wagler, 1831; Rodríguez, D'Elía & Valdivia, 2016).

122   **Materials and methods**

123   This study complies with the law Nº 19.473 of Servicio Agrícola y Ganadero of Chile,

124   which permits observational studies on any open access coastal area. We sampled four sandy

125   beaches located in the central-southern Chilean shore. The sites were Cheuque, Curiñanco,

126   Calfuco, and Chaihuín, and spanned ca. 150 km of the southeast Pacific shore (Fig. 1). Three of

127   the four seagulls analysed, namely *C. maculipennis, L. modestus* and *L. pipixcan*, use these

128   shores as feeding habitats during the austral summer. *Larus dominicanus* can be observed in the

129   area throughout the year. These populations display large proportions of individuals infected

130   with *P. altmani*. On average, the individuals of the four species show comparatively large

131   numbers of *P. altmani* parasites in the intestine (see Table 1 in Rodríguez, D'Elía & Valdivia,

132   2016). Previous records studies have described *Profilicollis* larvae in intestines of other

133   vertebrate hosts, such as sea otters (Hennessy & Morejohn, 1977; Margolis et al., 1997; Mayer,

134   Dailey & Miller, 2003). However, no adult parasites have been recorded in these hosts (Mayer,

135   Dailey & Miller, 2003), so the probability that sea otters encompass an alternative vector of

136   infection is sill low.

137   Densities of seagulls and *E. analoga* were quantified trimonthly between January 2014

138   and October 2015 (i.e. 22 months). This sampling period allowed us to analyse almost two full

139   seasonal cycles of seagull migrations. In each bird census, each sandy beach was sampled in full

140   during low tide—samplers walked parallel on effluent line to avoid disturbing the seagulls. A

141   prismatic lens, with a spotting scope between 20 and 60x, was used to quantify the number of

142   individuals of the four seagull species (Navedo et al., 2015). The length of each beach was

143   determined using a GPS and the Google Earth freeware software. The abundance of seagulls was

144   expressed as the kilometric abundance index (ind km^{-1}). Additionally, we quantified the number

145   of people, dogs, and motorised vehicles (i.e. "visitors") in each site and sample time as a

146   potential factor influencing bird abundance. Preliminary regression analyses revealed no

147   significant relationship between the total number of visitors and bird abundance across sites and

148   over time ($R^2 = 0.002$; $P > 0.05$. Fig. S1 in the Supplementary Material).

149   To survey the density of molecrabs, in each site and sampling time we deployed four

150   transects randomly and perpendicularly to the shoreline. In each transect, we placed four

151   sampling stations located ca. 2 m apart from each other from the effluent line to the swash line.

152   Plastic corers ($0.03\ \text{m}^{-2}$) were buried to a depth of 20 cm (equating a volume of $0.006\ \text{m}^{-3}$) and

153   the sand was sieved through a 1-mm mesh sieve. Cores were combined into transects in order to

154   integrate all molecrab sizes in each sampling unit. Molecrabs were transferred to the laboratory

155   for parasite identification and sorting under binocular microscope. In the laboratory,

156   cephalothorax length (mm) of each individual was recorded and then they were dissected in

157   order to extract the *P. altmani* cystacanths (i.e. larval stage) from the haemocoele. The host

158   population was composed by two subgroups of body sizes, one ranging from 0.65 to 15 mm and

159   a second group ranging from 15 to 32 mm (Fig. 2A). For brevity, the former and the latter will be

160   referred to as "small" and "large" hosts, respectively.

161   For each size group, site, and sampling time, prevalence and parasite burden were

162   separately estimated as the proportion of infected hosts and the per capita number of parasites,

163   respectively (Bush et al., 1997). Parasite aggregation was estimated in two ways, as the variance-

164   to-mean ratio and the scalar exponent of Taylor's power law (Taylor, 1961). The variance-to-

165   mean ratio was estimated as the quotient between the variance (s^2) and mean (m) of the per

166   capita parasite burden. This ratio varies from zero (indicating that parasites are uniformly

167   distributed among hosts) through one (indicating a random distribution of parasites), to a

168   maximum bounded by the number of parasites in the sample (indicating an aggregated

169   distribution of parasites; reviewed in Wilson et al., 2001). The scalar exponent b of Taylor's

170   power law was estimated from $s^2 = am^b$ (Taylor, 1961); where a is the variance when $m = 1$ and

171   b is the mean-variance scalar exponent. The b parameter was estimated as the slope of the natural

172   log-log regression between s^2 and m (Figs. S2 and S3 in the supplementary material). The scalar

173   exponent varies from zero (indicating a uniform distribution of parasites) through one (for a

174   random, Poisson, distribution of parasite burden), to infinity (highly aggregated distribution of

175   parasite counts). Since the intercept (a) of this regression can vary significantly from zero with

176   small m values, we took into account the former by estimating b at a standard mean of $\log_{10} m =$

177   1. This estimator is called I_{10} and is useful to disentangle the patterns of aggregation from those

178   of mean parasite load (Lester, 2012; Lester & McVinish, 2016). We estimated bias-corrected

179   confidence intervals for each site after 1000 bootstrapped estimations of I_{10}. To further assess the

180   relationship between mean parasite load and b, the fits were repeated for two subpopulations of

181    $m = [0.9, 1.59]$ and $[1.6, 6.1]$. Prevalence and aggregation were estimated as attributes of each

182   site and sampling time, over transects within each site. These estimations were conducted

183   separately for each size group. The b and I_{10} indexes were estimated as attributes of each site.

184

185   *Statistical Analyses*

186   The prediction of size-dependent relationships of parasite prevalence and aggregation

187   with seagull density was tested by means of linear mixed-effects models (LMM). In these

188   models, molecrab body size group and bird density were fixed and crossed variables, and

189   parasite prevalence and aggregation (variance-to-mean ratio) were the dependent variables. Site

190   and sampling time (nested in site) were included in the models as random factors. For each

191   LMM model, we estimated pseudo-R^2 according to Nakagawa & Schielzeth (2013) and Johnson

192   (2014). Two types of pseudo-R^2 were calculated for each model: the marginal R^2, which

193   represents the variance explained by the fixed factors, and the conditional R^2, which represents

194   the variance explained by both, fixed and random factors. The models were fit through Restricted

195   Maximum Likelihood (REML). The degrees of freedom of each fixed effect term (bird density,

196   molecrab size class, and interaction) were estimated through the Satterthwaite approximation,

197   and then hypothesis test was done on the basis of conditional t-tests (Pinheiro & Bates, 2004).

198   The treatment contrast was used to estimate effect coefficients. Standardised residual-vs.-fitted

199   values plots were used to check homogeneity of variances in LMMs. Variance-to-mean ratio data

200   were $\log_{10}$-transformed to homogenise the variances between both size groups (see Results). The

201   visual analysis of residuals suggested that a Gaussian model was the most appropriate for both,

202   prevalence and $\log_{10}$ variance-to-mean ratio. Autocorrelation functions were used to check the

203   temporal autocorrelation of residuals.

204   In order to determine if the potential relationships of bird density with parasite prevalence

205   and aggregation were lagged over time (for example, the increase in bird density would result in

206   an increase of parasitosis three months later), we computed cross-correlation functions (CCF)

207   between bird density (x) and prevalence (y) and between the former and the variance-to-mean

208   ratio (y)—we additionally cross-correlated bird and molecrab abundances. All statistical analyses

209   were conducted in R 3.3.0 (R Core Team, 2016). Linear mixed-effects models, pseudo-R^2, and

210   CCF were computed with the *lme4, MuMIn,* and *stats* libraries, respectively (Bates et al., 2015;

211   Bartón, 2016; R Core Team, 2016).

212

214 **Results**

215   A total of 1,780 individuals of *E. analoga* were captured throughout the study. Molecrab

216 cephalothorax length varied from 0.65 to 32.1 mm, with two modes located at 11.6 and 23.7 mm

217 respectively (Fig. 2A). Parasite burden—i.e. the per capita number of parasites—ranged between

218 zero and 22 ind. per molecrab (Fig. 2B). In addition, parasite burden tended to increase with

219 increasing molecrab' cephalothorax length (Fig. 2B; Table 1).

220   Seagull abundance (i.e. kilometric abundance index) varied between 11 ind km^{-1} in

221 winter, to 906 ind km^{-1} in summer and 922 ind km^{-1} in autumn (Fig. 3A). Molecrab density

222 ranged between 14 ind. 0.006 m^{-3} and 167 ind. 0.006 m^{-3} in summer and spring, respectively

223 (Fig. 3B). Bird and small-sized molecrab abundances showed instantaneous (i.e. no lagged)

224 correlations in Cheuque (Fig. S4 in the Supplementary Material). In addition, bird abundance

225 was correlated with both, small and large molecrabs at three-month lag in Calfuco (Fig. S4 in the

226 Supplementary Material).

227   Site-level prevalence values were 44 ± 18 % (mean ± standard deviation) and 79 ± 15 %

228 for small and large molecrabs, respectively (Fig. 4A and B). For both groups, prevalence was

229 highly variable across sites and over time, with maximum values occurring in summer 2015

230 (small molecrabs, Fig. 4A) and winter 2014 (large molecrabs, Fig. 4B). Despite these variable

231 patterns, the analysis of LMM detected a significant relationship between bird abundance and

232 prevalence (Fig. 4C, Table 2). In addition, large molecrabs showed ca. 40 % higher prevalence

233 than the small molecrabs (Fig. 4C, Table 2). The slope between bird density and prevalence

234 tended to be steeper for small than large molecrabs (Fig. 4C), albeit this difference was not

235 statistically significant at alpha = 0.05 (Table 2). The fixed factors in the model accounted for ca.

236 54 % of the variability in prevalence, while the entire model accounted for ca. 76 % (Table 2).

237   Parasite aggregation tended to be greater in large than small hosts across sites and over

238   time (Figs. 5A and 5B). In addition, the uncertainty was also greater in large than small hosts,

239   evidenced by the broader ranges in aggregation observed in the former (Figs. 5A and 5B). This

240   wide and size-dependent variation led to a statistically non-significant relationship of

241   aggregation with bird density and molecrab body size (Table 2). The analysis of aggregation at a

242   fixed mean parasite load of $m = 1$ (I_{10}) suggested, however, that the apparent greater aggregation

243   and stochasticity in large molecrabs was due to heterogeneities in mean parasite load, as I_{10}

244   scores remained similar across sites (Figs. 5C and 5D). The small-sized molecrab subpopulations

245   showed I_{10} scores between 1 and 2 across sites (Fig. 5C). The large molecrabs showed slightly

246   larger I_{10} scores in Cheuque and Chaihuín (Fig. 5D). These results point to a general Poisson

247   distribution of parasites in the study region.

248   In addition, b depended on mean parasite load, as large-sized molecrabs showed a

249   statistically significant $b = 1.1$ (0.4) when $m < 1.6$, but non-significant $b = 0.9$ (0.7) when $m \geq 1.6$

250   (standard errors bracketed). Prevalence and aggregation (variance-to-mean ratio) did not show

251   lagged correlations with bird density or molecrab abundance across host sizes—the lack of

252   correlation of molecrab abundance with prevalence indicates that the former values are

253   comparable between sites, times, and size groups (Figs. S5-S8 in the Supplementary Material).

254

Discussion

256

257   This study showed complex relationships of parasite exposure, represented by definitive

258   host abundances, with prevalence and aggregation across intermediate host population.

259   Acanthocephalan prevalence across molecrab populations was significantly related with bird

260   abundance, suggesting a link between parasitosis and parasite exposure. However, this

261   relationship was relatively inconsistent, as random effects of temporal surveys and sites

262   accounted for ca. 20 % of the variation in prevalence. In addition, we observed that prevalence in

263   large (> 15 mm) molecrabs was significantly larger than in small (< 15 mm) molecrabs. Over

264   seasonal surveys and across sites, parasite aggregation remained relatively constant for small, but

265   highly variable and unpredictable for large molecrabs. However, these latter patterns were

266   largely driven by variations in mean parasite load, rather than actual aggregation. Moreover,

267   further analyses of Taylor's power law showed that greater mean parasite loads were associated

268   with slower increases in the load variance with the mean of large molecrabs. Below, we discuss

269   how parasite accumulation over the ontogeny and differential mortality rates across host

270   populations can influence the links among parasite exposure, intermediate-host size structure,

271   and parasitosis.

272   We observed a significant positive relationship between prevalence and body size of the

273   intermediate hosts. Similarly, the analyses indicate heavier parasite load in large individuals.

274   Parasite accumulation over time could explain these trends. The analysed host-parasite

275   interaction is characterised by the inability of hosts to expel the parasites once they are allocated

276   in the haemocoele (Latham & Poulin, 2003). Accordingly, larger molecrabs, which have been

277   exposed for longer time to successive infections, accumulate parasites over the ontogeny. Similar

278   processes have been described for other endoparasites, like *Profilicollis antarcticus*

279 *Zdzitowiecki*, 1985 and *P. novaezelandensis* Brockerhoff & Smales, 2002, which accumulate

280 over the ontogeny of Ocipodidae and Grapsidae crabs (Latham & Poulin, 2003). Similarity, the

281 prevalence, intensity, and taxonomic richness of a number of endoparasite groups can increase

282 during the ontogeny of fish hosts (Muñoz, Valdebenito & George-Nascimento, 2002). Moreover,

283 body size of anuran hosts can be a strong predictor of the richness of helminthic fauna—this

284 pattern is consistent across hosts' geographic ranges and phylogenetic correlations, in addition to

285 sampling effort (Campião et al., 2015). Accordingly, parasite accumulation seems to be a

286 widespread characteristic of indirect host-parasite interactions.

287 In our study, size-dependent mortality might explain the significant decrease in the scalar

288 coefficient of Taylor power law with increasing mean parasite load in the large-sized molecrab

289 subpopulation. Latham & Poulin (2002) indicate that high parasite burden of two

290 acanthocephalan species increase the mortality rate in largest crab size-classes. Acanthocephalan

291 species are non-lethal parasites, but they can alter host's behaviour and increase their

292 susceptibility to predation (Latham & Poulin, 2002; Beisel & Médoc, 2010). For example,

293 molecrab' burrowing velocity is negatively correlated with parasite burden (Jerez & George-

294 Nascimento, 2010). In addition, egg mortality during dispersion could also have led to the non-

295 significant relationship between bird density (i.e. exposure to parasite infection) and the

296 comparatively low levels of aggregation observed in this study. Abiotic environmental variables

297 related with emersion, such as desiccation and heat stress can affect negatively parasite' egg

298 viability (Herrman & Poulin, 2011; Studer & Poulin, 2012; Studer, Lamare & Poulin, 2012).

299 Molecrab life history would offer a third explanation for the uncertainty in parasite load of large-

300 sized host. We observed that molecrab recruitment strongly varied across geographic locations,

301 with site-dependent peaks occurring in summer, autumn, and spring (but see Contreras, Defeo &

302   Jaramillo, 1999). An increase in non-infected recruits can have negative (positive) effects on

303   prevalence (aggregation) across the host population (e.g. Zambrano & George-Nascimento,

304   2010), which might decouple the relationship between exposure and the parasitosis in the

305   intermediate host. Further field-based comparative experiments would be useful to tease apart

306   the effects of exposure, egg mortality, and recruitment timing on parasite aggregation.

307   To what degree these results could be generalised to other host-parasite systems? The life

308   cycles of almost all acanthocephalan and digenetic species, as well as some nematodes and

309   cestodes, include at least one intermediate host that accumulates parasites over time (Thomas,

310   Guégan & Renaud, 2009). In addition, size-dependent responses of prevalence to parasite

311   exposure have been demonstrated in other complex host-parasite interactions involving

312   migratory birds and crustaceans (Latham & Poulin, 2003; Smith, 2007; Zambrano & George-

313   Nascimento, 2010). In the same line, parasite burden increases with body size of the toadfish

314   *Aphos porosus* Valenciennes, 1837, an intermediate host of cestodes, nematodes, trematodes, and

315   acantocephalans (Cortés & Muñoz, 2008). These examples suggest that accumulation of

316   endoparasites in intermediate hosts may be a common phenomenon. Contrarily, ectoparasites,

317   which can be removed by hosts, can show different patterns of prevalence and aggregation. In

318   such systems, the smaller or younger hosts accumulate greater parasite burdens than older hosts,

319   as a likely consequence of higher susceptibility to infections of the former than the latter (McCoy

320   et al., 2016). Therefore, significant relationships between parasite exposure and prevalence, in

321   addition with size-dependent patterns of stochasticity in parasite load could be observed in other

322   complex host-parasite systems, at least those involving endoparasites that accumulate over host'

323   ontogeny.

324   In summary, our results showed that parasitosis could be related with exogenous

325   (exposure) and endogenous (host body size) factors in a complex fashion. Parasite exposure was

326   positively related with prevalence in the intermediate host population. Prevalence and parasite

327   load were dependent of intermediate host body size, which may be explained by parasitosis-

328   related increasing mortality rates over host' ontogeny. Also, these results would be generalised to

329   other complex parasite with an intermediate host that accumulates parasite over time. These

330   results suggest that intrinsic host-population characteristic can have stronger effects on the

331   dynamic of host-parasite interactions than extrinsic factors. Overall, our study contributes to the

332   understanding of the processes that underpin the heterogeneity in parasite burden across host

333   populations.

334

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336

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341

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506   **Figure legend**

507   **Figure 1.** Study sites. Site codes are CHEU = Cheuque, CURI = Curiñanco, CALF = Calfuco,

508   and CHAI = Chaihuín.

509

510   **Figure 2.** (A) Size-structure of the molecrab *Emerita analoga* and (B) relationship between body

511   size (mm) and parasite burden (number of parasites ind^{-1}).

512

513   **Figure 3.** Mesoscale spatiotemporal patterns of (A) bird and (B) molecrab abundances during

514   2014 and 2015. Su = summer, Au = autumn, Wi = winter, Sp = spring. Symbols indicate the

515   study sites.

516

517   **Figure 4.** Patterns of parasite prevalence in population fractions composed by (A) small and (B)

518   large hosts during 2014 and 2015. Su = summer, Au = autumn, Wi = winter, Sp = spring. (C)

519   Relationship between bird abundance and prevalence; the statistical fits between both variables is

520   shown adjusted for host body size (see coefficients in Table 2).

521

522   **Figure 5.** Spatial patterns of parasite aggregation estimated as (A, B) the variance-to-mean ratio

523   and (C, D) the scalar coefficient of Taylor's power law for small (A, C) and large (B, D)

524   molecrabs.

525

527   Table 1: Number of small and large crabs of *E. analoga*, number of parasites in small and large

528   molecrabs (SM and LM) and number of seagulls for each sample seasons during 2014 and 2015.

529   Su = Summer, Au = Autumm, Wi = Winter, Sp = Spring.

530

	Su-2014	Au-2014	Wi-2014	Sp-2014	Su-2015	Au-2015	Wi-2015	Sp-2015
Nº small crabs	212	121	133	72	132	217	81	73
Nº parasites SM	117	102	83	83	89	157	49	56
Nº large crabs	131	100	76	49	74	168	72	69
Nº parasites LM	245	244	141	165	217	513	211	257
Nº seagulls	867	1,564	394	485	903	472	110	71

532 Table 2. Results of LMM for the relationships of parasitosis (prevalence and variance-to-mean

533 ratio of parasite burden) with bird abundance and molecrab body size. Estimate effects, their

534 standard errors (S.E.), degrees of freedom (D.F.), and t statistics are provided for each fixed

535 effect term. Degrees of freedom were estimated through Satterthwaite approximation. R^2m and

536 R^2c are the marginal and critical pseudo-R^2, which represent the variability accounted for fixed

537 and the entire model, respectively.

538

	Term	Estimate	S.E.	D.F.	t-value	P-value	R^2m	R^2c
Prevalence	Intercept	0.3871	0.0624	4.54	6.203	0.002	0.538	0.757
	Bird density	0.0002	0.0001	53.36	2.150	0.036		
	Effect of large							
	molecrabs	0.4120	0.0416	29.39	9.913	<0.001		
	Interaction	-0.0002	0.0001	28.93	-1.896	0.068		
Variance-to-mean	Intercept	0.1843	0.0538	18.32	3.4287	0.0029	0.138	0.154
ratio ($\log_{10}$)	Bird density	-0.0001	0.0002	58.32	-0.7427	0.4607		
	Effect of large							
	molecrabs	0.0726	0.0719	56.04	1.0096	0.3170		
	Interaction	0.0004	0.0002	55.98	1.5970	0.1159		

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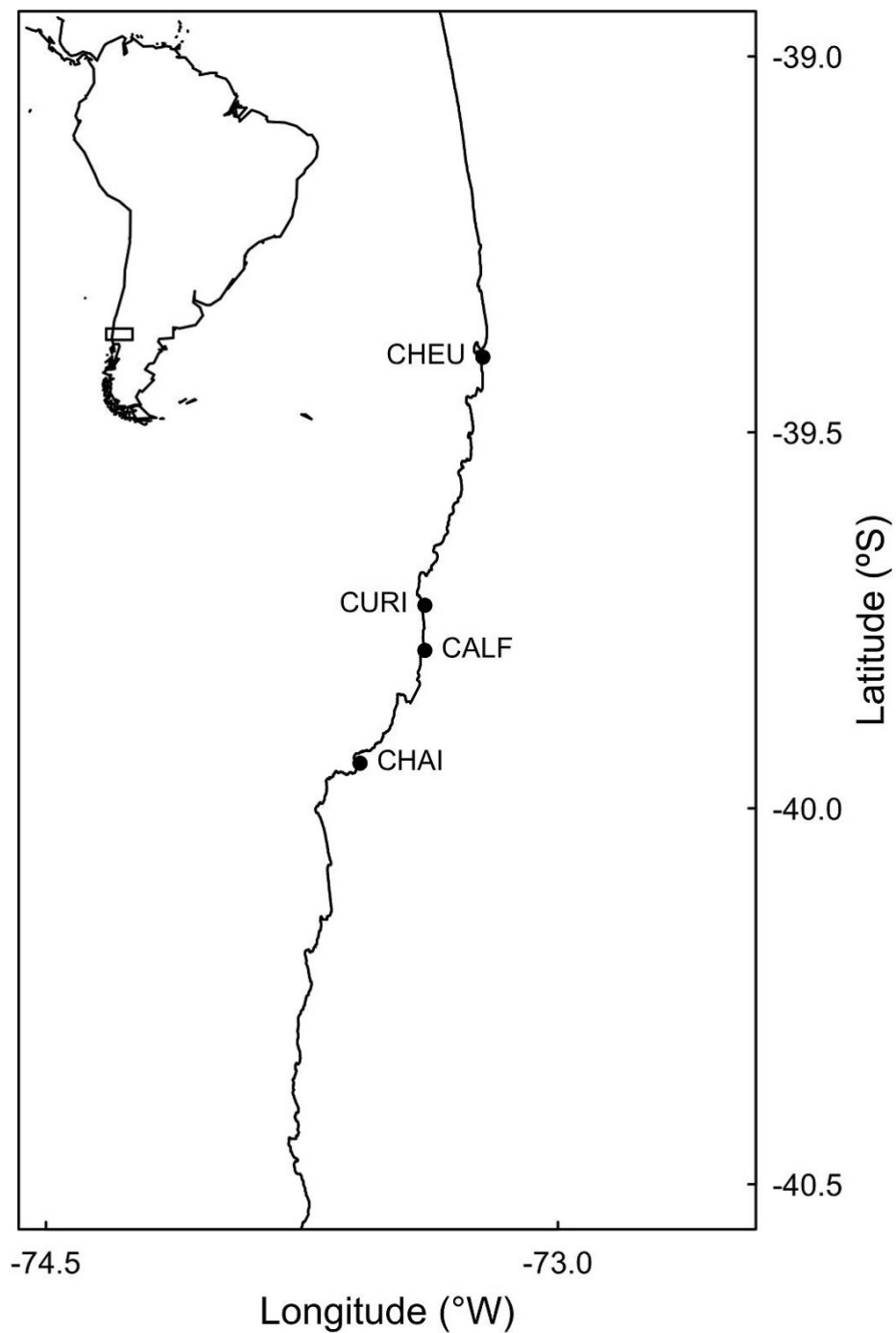
541   *Figure 1: Rodríguez & Valdivia*

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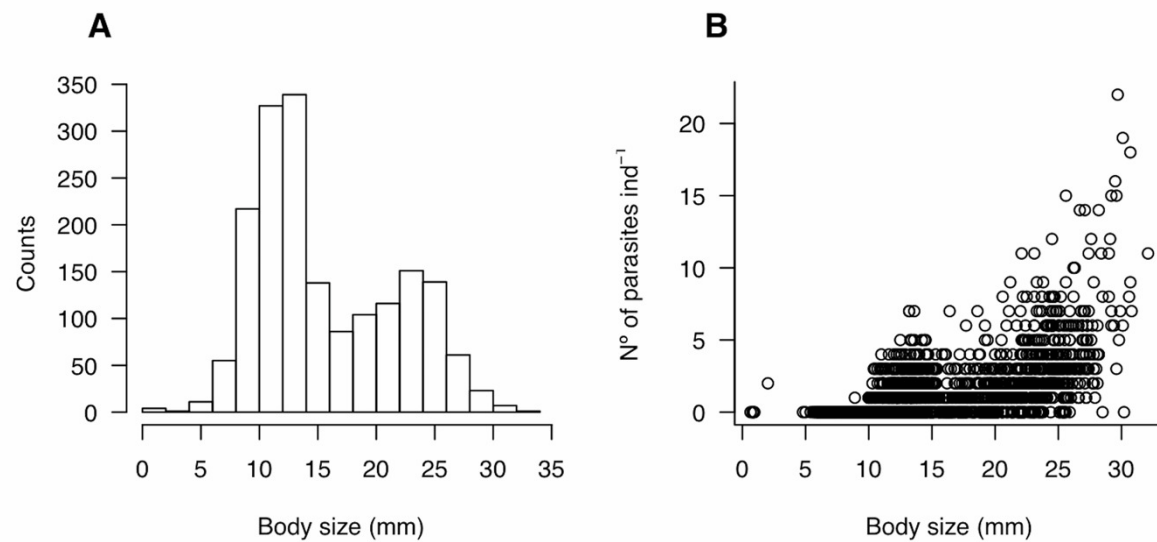
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546   *Figure 2: Rodríguez & Valdivia.*

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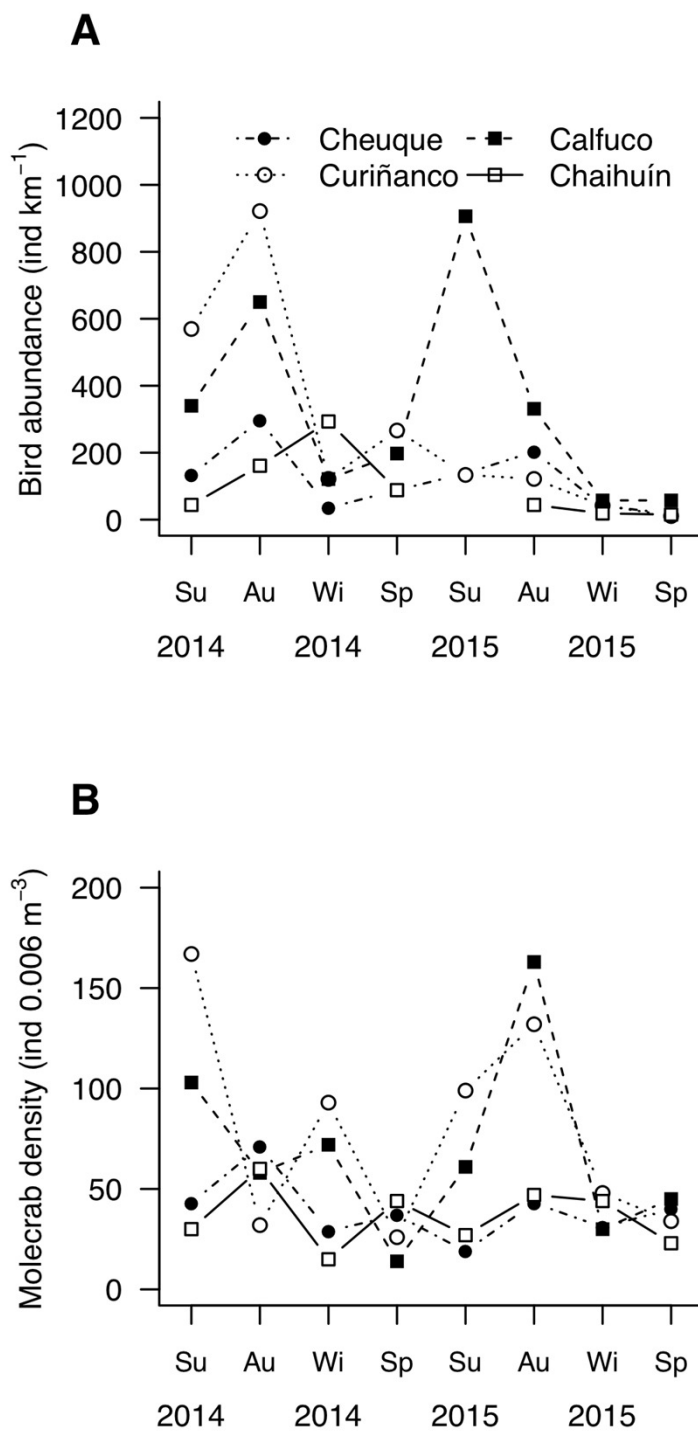
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550   *Figure 3: Rodríguez & Valdivia.*

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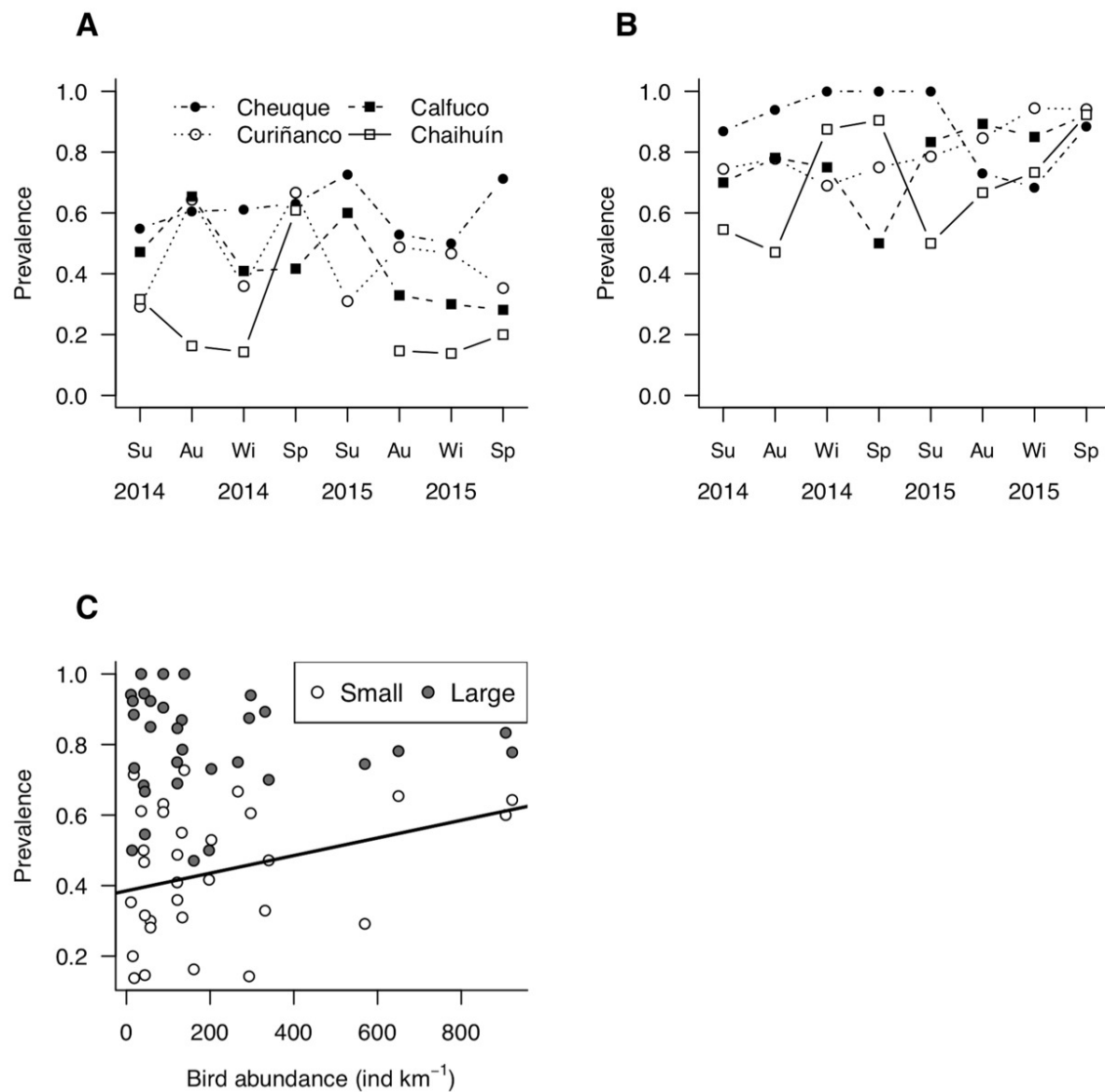
Figure 4: Rodríguez & Valdivia.

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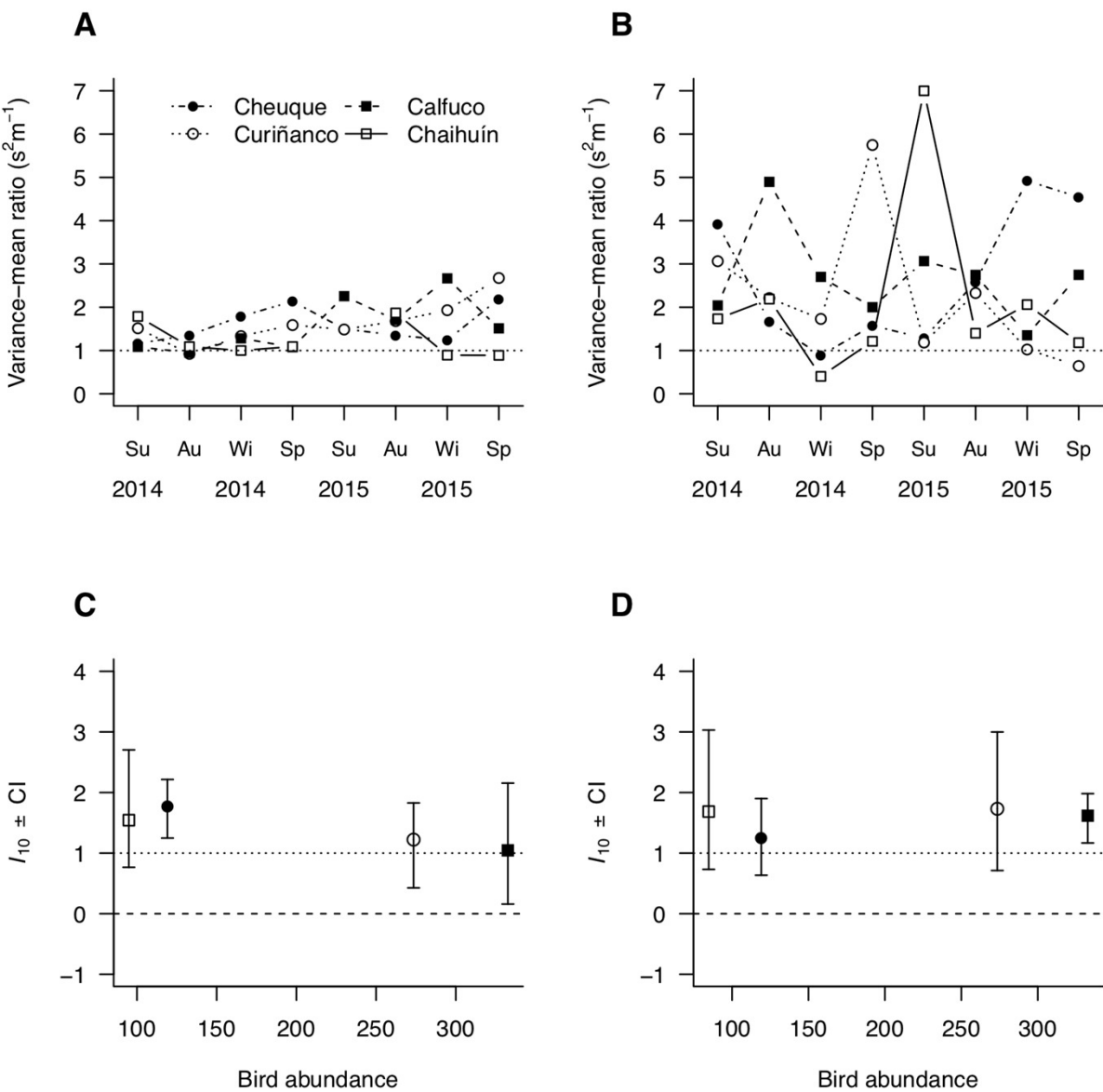
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557   *Figure 5: Rodríguez & Valdivia*

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