Supplementary Material for "Dispersal syndrome and landscape fragmentation in the salt-marsh specialist spider *Erigone longipalpis*"

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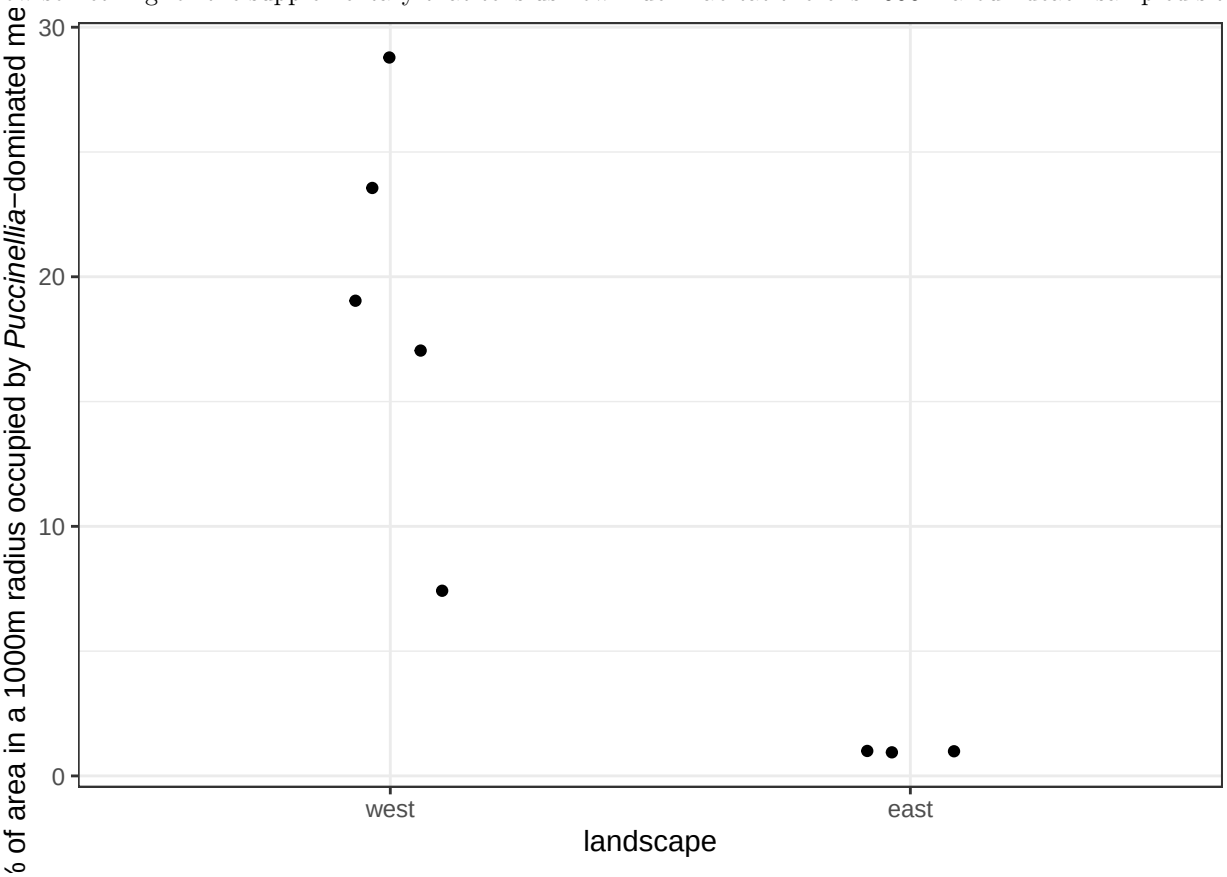
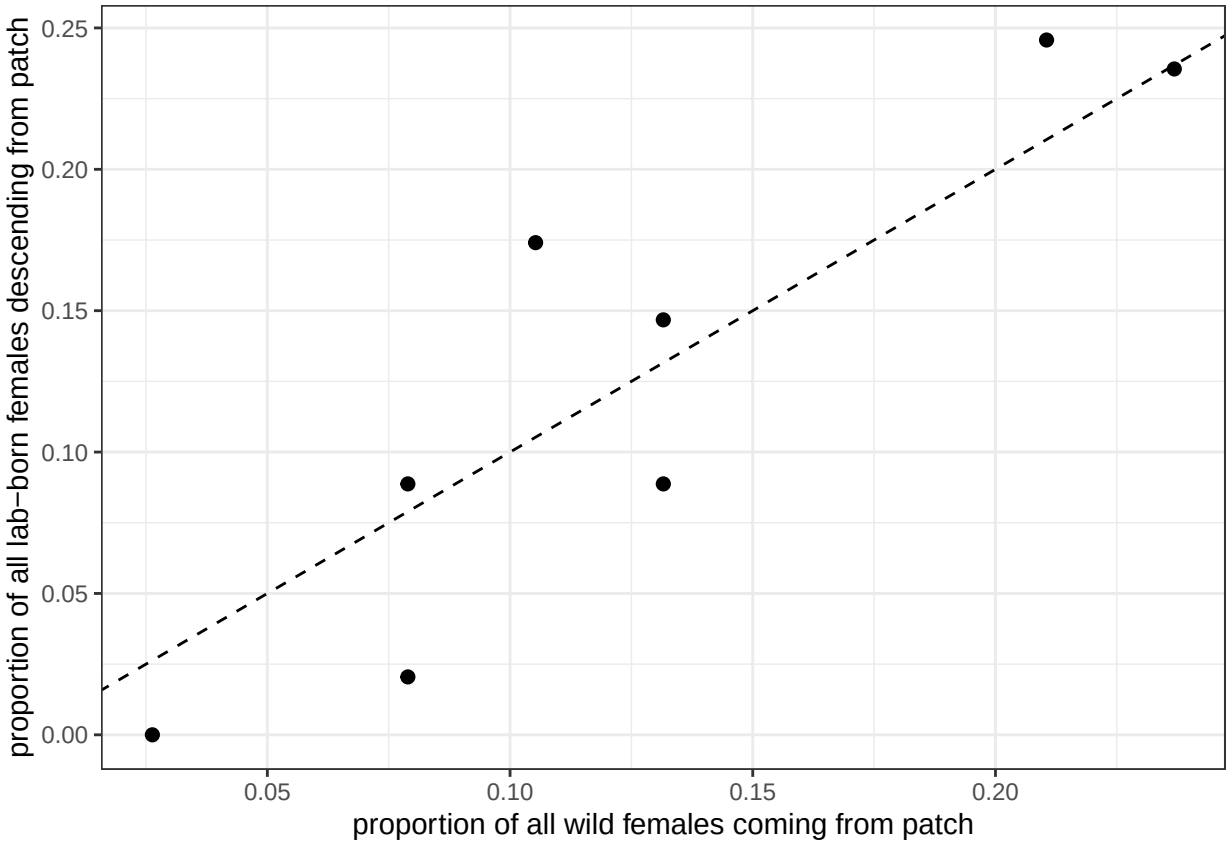
Supplementary 1: % of habitat in neighbourhood of patches

something something

now something for the supplementary that tells us how much habitat there is 1000m aroun deach sampled site



Supplementary 2: correlation between number of spider caught and number of spiders in lab



Supplementary 3: formal description of models

Spider abundance

The number of spiders N_p caught in patch p was analysed using the following model:

$$N_p \sim \text{Poisson}(\lambda_{[N]} \times t_p),$$

$$\log(\lambda_{[N]}) = \beta_{0[N]} + \beta_{1[N]} \times x_p,$$

with t_p being an offset corresponding to the patch-specific sampling effort (in person-hours), and x_p a binary variable denoting the landscape to which the patch p belongs (0: the western, more continuous landscape; 1: the eastern, more fragmented landscape). We used weakly informative priors as suggested by , namely Normal$(0, 1)$ for both the intercept β_0 and the landscape effect β_1.

Spider phenotype

Let $M_{i,p}$, $D_{i,p}$, $F_{i,p}$, $L_{i,p}$ be the *recorded* ages at maturity, dispersal propensity (number of rappelling attempts), fecundity and adult longevity of individual i whose (grand)mother was caught in patch p. In addition, let $S_{i,p,o}$ be the observation/measure o of individual i's body size (here cephalothorax width), *after* standardisation to mean 0 and SD 1. Then we can assume these traits are distributed as follows:

$$
\begin{aligned}
S_{i,p,o} &\sim \text{Normal}(\mu_{i,p}, \sigma_r), \\
M_{i,p} &\sim \text{Poisson}(\lambda_{[M]i,p}), \\
D_{i,p} &\sim \text{Poisson}(\lambda_{[D]i,p}), \\
F_{i,p} &\sim \text{Poisson}(\lambda_{[F]i,p} \times d_{i,p}),
\end{aligned}
$$

where $d_{i,p}$ is an offset based on the number of potential egg-laying days this individual was observed, and

$$
\begin{aligned}
L_{i,p}|C_{i,p} = 0 &\sim \text{Poisson}(\lambda_{[L]i,p}), \\
L_{i,p}|C_{i,p} = 1 &\sim \text{Poisson-CCDF}(\lambda_{[L]i,p}),
\end{aligned}
$$

where $C_{i,p}$ is a censoring indicator = 0 if natural death was recorded during the experiment, or = 1 if individuals outlived the experiment or died accidentally.

The models for the corresponding μ and λ are all pretty similar to each other:

$$
\begin{aligned}
\mu_{i,p} &= \beta_{0[S]} + \beta_{1[S]} \times x_p + \alpha_{[S]p} + \gamma_{[S]i}, \\
\log(\lambda_{[M]i,p}) &= \beta_{0[M]} + \beta_{1[M]} \times x_p + \beta_{2[M]} \times y_{[M]p} + \alpha_{[M]p} + \gamma_{[M]i}, \\
\log(\lambda_{[D]i,p}) &= \beta_{0[D]} + \beta_{1[D]} \times x_p + \alpha_{[D]p} + \gamma_{[D]i}, \\
\log(\lambda_{[F]i,p}) &= \beta_{0[F]} + \beta_{1[F]} \times x_p + \alpha_{[F]p} + \gamma_{[F]i}, \\
\log(\lambda_{[L]i,p}) &= \beta_{0[L]} + \beta_{1[L]} \times x_p + \beta_{2[L]} \times y_{[L]p} + \alpha_{[L]p} + \gamma_{[L]i},
\end{aligned}
$$

with y a binary variable denoting whether the time-to-event response (time to maturity or longevity) is based on records with gaps (i.e. maturity recorded after a week-end) and thus potentially biased. The random effects of patch of origin and individual identity are denoted by α and γ respectively. These random effects are distributed as follows:

$$
\begin{aligned}
\alpha_{[S]p} &\sim \text{Normal}(0, \sigma_{\alpha[S]}), \\
\alpha_{[M]p} &\sim \text{Normal}(0, \sigma_{\alpha[M]}), \\
\alpha_{[D]p} &\sim \text{Normal}(0, \sigma_{\alpha[D]}), \\
\alpha_{[F]p} &\sim \text{Normal}(0, \sigma_{\alpha[F]}), \\
\alpha_{[L]p} &\sim \text{Normal}(0, \sigma_{\alpha[L]}),
\end{aligned}
$$

$$
\begin{bmatrix} \gamma_{[S]i} \\ \gamma_{[M]i} \\ \gamma_{[D]i} \\ \gamma_{[F]i} \\ \gamma_{[L]i} \end{bmatrix} \sim \text{MVNormal} \left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \boldsymbol{\Omega} \right),
$$

where $\boldsymbol{\Omega}$ is the individual-level covariance matrix, which can be decomposed into its constituent standard deviations and correlation matrix $\boldsymbol{R}$ as follows:

$$
\boldsymbol{\Omega} = \begin{bmatrix} \sigma_{\gamma[S]} & 0 & 0 & 0 & 0 \\ 0 & \sigma_{\gamma[M]} & 0 & 0 & 0 \\ 0 & 0 & \sigma_{\gamma[D]} & 0 & 0 \\ 0 & 0 & 0 & \sigma_{\gamma[F]} & 0 \\ 0 & 0 & 0 & 0 & \sigma_{\gamma[L]} \end{bmatrix} \boldsymbol{R} \begin{bmatrix} \sigma_{\gamma[S]} & 0 & 0 & 0 & 0 \\ 0 & \sigma_{\gamma[M]} & 0 & 0 & 0 \\ 0 & 0 & \sigma_{\gamma[D]} & 0 & 0 \\ 0 & 0 & 0 & \sigma_{\gamma[F]} & 0 \\ 0 & 0 & 0 & 0 & \sigma_{\gamma[L]} \end{bmatrix}.
$$

Priors for fixed effects β are the same as in the abundance model (Normal$(0, 1)$) except for the intercepts of the time to maturity and longevity submodels. For these, priors were shifted to Normal$(3.4, 1)$ based on knowledge that typical development times and adult longevity in *Erigone* are on the order of 30 days (i.e. $\simeq \exp(3.4)$) . We used Half $-$ Normal$(0, 1)$ priors for all standard deviations σ (including the residual SD σ_r for the size submodel), and a LKJCorr(2) prior for the correlation matrix R of individual-level random effects.

Splitting among- and within-family correlations

In a second time, we refitted the above model, this time splitting the individual-level variation into its within- and among-family components. The model is largely as above, with two exceptions:

- first, individuals i are not only indexed by their patch of origin p, but also by their mother m (so the dispersal propensity $D_{i,p}$ is now written $D_{i,m,p}$)
- second, the individual-level random effects γ, and the corresponding covariance, are decomposed into a sum of family-level random effects η and the remaining within-family individual effects ν as follows:

$$\gamma_{[S]i,m,p} = \eta_{[S]m,p} + \nu_{[S]i,m,p},$$
$$\gamma_{[M]i,m,p} = \eta_{[M]m,p} + \nu_{[M]i,m,p},$$
$$\gamma_{[D]i,m,p} = \eta_{[D]m,p} + \nu_{[D]i,m,p},$$
$$\gamma_{[F]i,m,p} = \eta_{[F]m,p} + \nu_{[F]i,m,p},$$
$$\gamma_{[L]i,m,p} = \eta_{[L]m,p} + \nu_{[L]i,m,p},$$

$$\begin{bmatrix} \eta_{[S]i} \\ \eta_{[M]i} \\ \eta_{[D]i} \\ \eta_{[F]i} \\ \eta_{[L]i} \end{bmatrix} \sim \text{MVNormal}\left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \boldsymbol{\Omega_\eta} \right),$$

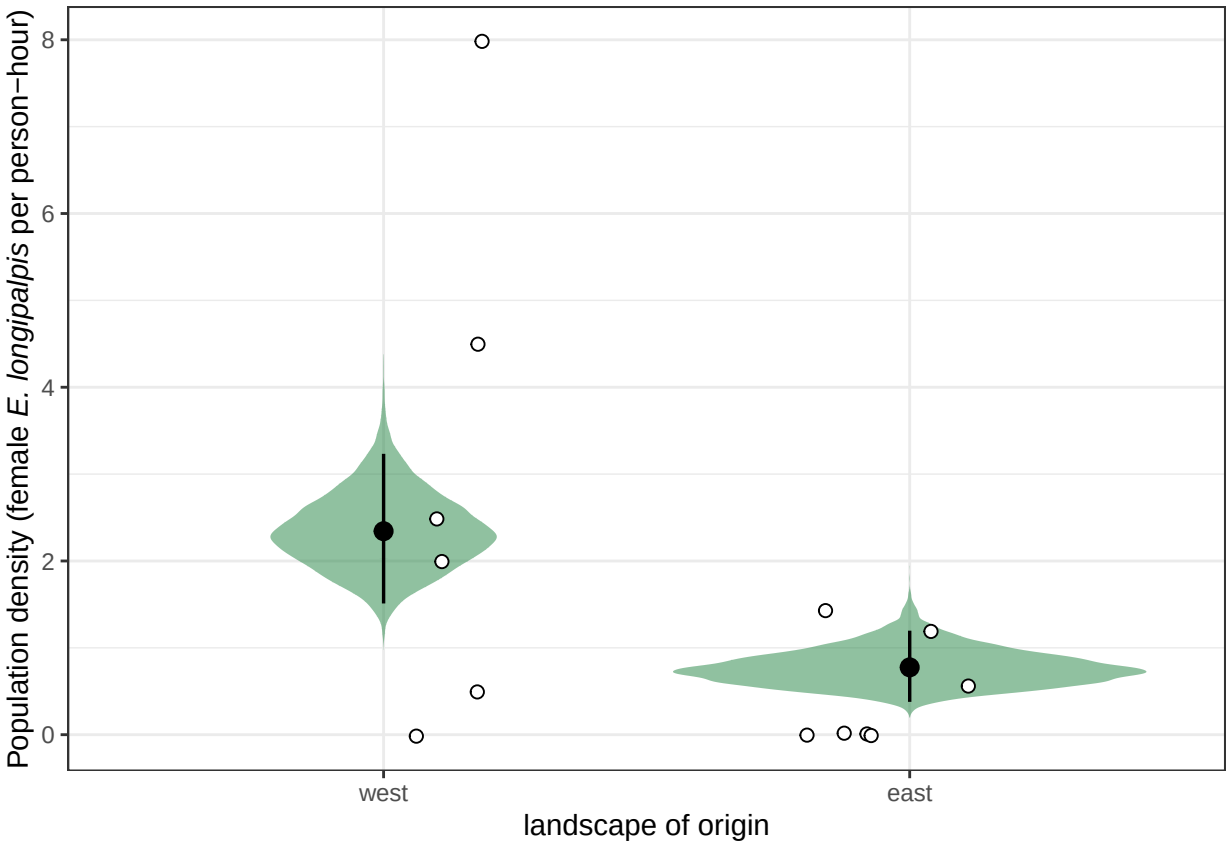
$$\boldsymbol{\Omega_\eta} = \begin{bmatrix} \sigma_{\eta[S]} & 0 & 0 & 0 & 0 \\ 0 & \sigma_{\eta[M]} & 0 & 0 & 0 \\ 0 & 0 & \sigma_{\eta[D]} & 0 & 0 \\ 0 & 0 & 0 & \sigma_{\eta[F]} & 0 \\ 0 & 0 & 0 & 0 & \sigma_{\eta[L]} \end{bmatrix} \boldsymbol{R_\eta} \begin{bmatrix} \sigma_{\eta[S]} & 0 & 0 & 0 & 0 \\ 0 & \sigma_{\eta[M]} & 0 & 0 & 0 \\ 0 & 0 & \sigma_{\eta[D]} & 0 & 0 \\ 0 & 0 & 0 & \sigma_{\eta[F]} & 0 \\ 0 & 0 & 0 & 0 & \sigma_{\eta[L]} \end{bmatrix},$$

$$\begin{bmatrix} \nu_{[S]i} \\ \nu_{[M]i} \\ \nu_{[D]i} \\ \nu_{[F]i} \\ \nu_{[L]i} \end{bmatrix} \sim \text{MVNormal}\left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \boldsymbol{\Omega_\nu} \right),$$

$$\boldsymbol{\Omega_\nu} = \begin{bmatrix} \sigma_{\nu[S]} & 0 & 0 & 0 & 0 \\ 0 & \sigma_{\nu[M]} & 0 & 0 & 0 \\ 0 & 0 & \sigma_{\nu[D]} & 0 & 0 \\ 0 & 0 & 0 & \sigma_{\nu[F]} & 0 \\ 0 & 0 & 0 & 0 & \sigma_{\nu[L]} \end{bmatrix} \boldsymbol{R_\nu} \begin{bmatrix} \sigma_{\nu[S]} & 0 & 0 & 0 & 0 \\ 0 & \sigma_{\nu[M]} & 0 & 0 & 0 \\ 0 & 0 & \sigma_{\nu[D]} & 0 & 0 \\ 0 & 0 & 0 & \sigma_{\nu[F]} & 0 \\ 0 & 0 & 0 & 0 & \sigma_{\nu[L]} \end{bmatrix}.$$

Supplementary 4: abundance results with all patches even the bad ones

something something



Supplementary 5: observation day biases? none

something something

Supplementary 6: phenotypic traits, but for the split variance model

something something

Supplementary 7: table 1a, but using the split model

something something

Supplementary 8: full posteriors

something something, may end up not including it (just need to delete a sentence in legend of Table1)