Supporting Information

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1. Mathematical details for crossover analysis

1.1 Derivation of the maximum likelihood estimate of crossover frequencies

Let n_0, n_1, and n_2 be the numbers of haplotypes having zero, one, or two recombination breakpoints on a given chromosome, respectively. Analogously, let c_0, c_1, and c_2 be the probabilities of having zero, one, or two crossovers on that chromosome. We assume at most two crossovers per chromosome. The likelihood of data given parameters is

$$\mathbb{P}(n_0, n_1, n_2 | c_0, c_1, c_2) = \frac{(n_0 + n_1 + n_2)!}{n_0! n_1! n_2!} p_0^{n_0} p_1^{n_1} p_2^{n_2} \tag{S1}$$

where

$$\begin{aligned} p_0 &= c_0 + c_1/2 + c_2/4 \\ p_1 &= c_1/2 + c_2/2 \\ p_2 &= c_2/2 \end{aligned} \tag{S2}$$

The log-likelihood $L(c_1, c_2, c_3) = \ln \mathbb{P}$ is thus given by (the difference is up to a constant):

$$L \sim n_0 \ln \left(1 - \frac{c_1}{2} - \frac{3c_2}{4}\right) + n_1 \ln \left(\frac{c_1}{2} + \frac{c_2}{2}\right) + n_2 \ln \left(\frac{c_2}{4}\right) \tag{S3}$$

Maximizing this function yields

$$\begin{aligned} c_0 &= (n_0 - n_1 + n_2)/(n_0 + n_1 + n_2) \\ c_1 &= (2n_1 - 4n_2)/(n_0 + n_1 + n_2) \\ c_2 &= 4n_2/(n_0 + n_1 + n_2) \end{aligned} \tag{S4}$$

However, the data sometimes contained too many recombined individuals (e.g., due to stochastic sampling error) and c_0 became negative. Then we maximized L along the boundary of the region $c_1 + c_2 \leq 1$. Since $c_1 > 0$ (no crossover is very unlikely), we substituted $c_1 = 1 - c_2$ into the above likelihood and maximized it within $0 \leq c_2 \leq 1$. This approach produced the following adjustment to the estimate:

$$\begin{aligned} c_0^* &= 0 \\ c_1^* &= (n_0 - n_2)/(n_0 + n_2) \\ c_2^* &= 2n_2/(n_0 + n_2) \end{aligned} \tag{S5}$$

2. Mathematical details for analyzing pupal weight on the Z chromosome

2.1 Derivation of $R^2_{n\text{-marker}}/R^2_{\text{Z-ancestry}}$ for different architectures of pupal weight

First, let's clarify our notations. The introgressed ancestry fraction on a chromosome (in this case, the Z chromosome) is denoted as f. Marker ancestry at relative position l ($0 \leq l \leq 1$) is p_l, and it takes binary values:

$$\begin{cases} 1, & \text{if introgressed} \\ 0, & \text{if not introgressed} \end{cases} \tag{S6}$$

This setup is sufficient for analyzing the single Z chromosome in backcross females.

Theorem 1 (Statistics under the crossover model)**.** *Crossover on the Z chromosome can be approximated by randomly selecting a position as the only crossover. With this model, we have the following statistics:*

$$\begin{aligned} \mathbb{E}[f] &= 1/2 \\ \mathrm{Var}[f] &= 1/6 \\ \mathbb{E}[p_l] &= 1/2 \\ \mathrm{Var}[p_l] &= 1/4 \\ \mathrm{Cov}(f, p_l) &= \frac{1}{8}\Big[1 + 2l(1-l)\Big] \\ \mathrm{Cov}(p_{l_1}, p_{l_2}) &= \frac{1}{4}\Big(1 - |l_1 - l_2|\Big) \end{aligned} \tag{S7}$$

Proof. For these statistics, it is helpful to think about the following probabilities on a backcross female's Z chromosome ($\mathrm{d}f$ is the differential in f):

$$\begin{aligned} &\mathbb{P}(f = 0) = \mathbb{P}(f = 1) = \frac{1}{4} \quad \text{(Non-recombined)} \\ &\mathbb{P}(\text{Introgressed from the right-hand-side with a fraction } f) = \frac{1}{4}\,\mathrm{d}f \quad \text{(Recombined)} \\ &\mathbb{P}(\text{Introgressed from the left-hand-side with a fraction } f) = \frac{1}{4}\,\mathrm{d}f \quad \text{(Recombined)} \end{aligned} \tag{S8}$$

We immediately have $\mathbb{E}[f]$, $\mathbb{E}[p_l]$, $\mathrm{Var}[f]$, and $\mathrm{Var}[p_l]$ from such probabilities. For covariances,

$$\begin{aligned} \mathbb{E}[p_{l_1} p_{l_2}] &= \frac{1}{4} + \frac{1}{4}(1 - |l_1 - l_2|) \\ \mathbb{E}[f p_l] &= \frac{1}{4} + \frac{1}{4}\int_l^1 f\,\mathrm{d}f + \frac{1}{4}\int_0^l (1-f)\,\mathrm{d}f = \frac{1}{8}\left[3 + 2l(1-l)\right] \end{aligned} \tag{S9}$$

These quantities are sufficient to derive the covariances between variables. □

Definition 1 (The linear polygenic model)**.** *For pupal weight W, we define its polygenic model as a linear function of average introgressed ancestry (f) on the Z chromosome:*

$$W = \alpha f + w \tag{S10}$$

where α and w are slope and intercept, respectively.

Note: This linear polygenic model will be generalized to nonlinear functions of f when analyzing ovary dysgenesis (discussed in section 2.3). All results in this section must be understood as the joint consequences of:

- A specific genotype-phenotype map (e.g., a linear polygenic model or other QTL models)
- A particular crossover process occurring on these butterflies' Z chromosome
- A backcross brood

With these assumptions in mind, we can predict expected patterns of marker-phenotype association.

Theorem 2 (Polygenic model & 1-marker scans)**.** *Conditioning on the polygenic model of pupal weight, we have the following relationship for 1-marker scans:*

$$\left.\frac{R^2_{1-\text{marker}}}{R^2_{\text{Z}-\text{ancestry}}}\right|_{\text{Polygenic}} = \frac{3}{8}\Big[1 + 2l(1-l)\Big]^2 \tag{S11}$$

Proof. The polygenic model of pupal weight posits that phenotype W is a linear function of f. Thus, the regression power of 1-marker scans $R^2_{\text{1-marker}}$ at position l, relative to the power of regression using Z-ancestry $R^2_{\text{Z-ancestry}}$, is simply the squared correlation coefficient between f and p_l:

$$\frac{R^2_{\text{1-marker}}}{R^2_{\text{Z-ancestry}}} = \rho^2_{p_l,f} = \frac{\text{Cov}^2(p_l, f)}{\text{Var}[p_l]\text{Var}[f]} = \frac{3}{8}\Big[1 + 2l(1-l)\Big]^2 \tag{S12}$$

□

The above equation shows an artifactual QTL at the center of the Z chromosome ($l = 0.5$) that maximizes $R^2_{1-\text{marker}}$.

Theorem 3 (1-QTL model & 1-marker scans). *Conditioning on a 1-QTL model of pupal weight, where the single QTL is at position x, we have the following relationship for 1-marker scans:*

$$\left.\frac{R^2_{1-\text{marker}}}{R^2_{\text{Z}-\text{ancestry}}}\right|_{1-\text{QTL}} = \frac{8}{3}\left[\frac{1-|l-x|}{1+2x(1-x)}\right]^2 \tag{S13}$$

Proof. Since the marker at position x contains all phenotypic information, the regression power using another marker at position l is the squared correlation coefficient between p_x and p_l, (i.e., $\rho^2_{p_x,p_l}$). Similarly, the regression power using the Z-ancestry is $\rho^2_{p_x,f}$. Thus,

$$\frac{R^2_{1-\text{marker}}}{R^2_{\text{Z}-\text{ancestry}}} = \frac{\rho^2_{p_x,p_l}}{\rho^2_{p_x,f}} = \frac{\text{Cov}^2(p_x,p_l)}{\rho^2_{p_x,f}\text{Var}[p_x]\text{Var}[p_l]} = \frac{8}{3}\left[\frac{1-|l-x|}{1+2x(1-x)}\right]^2 \tag{S14}$$

□

Theorem 4 (2-QTL model & 1-marker scans). *Conditioning on a 2-QTL model of pupal weight, where the two QTLs are at positions x_1 and x_2 ($x_1 < x_2$) with equal additive effects, we have the following relationship for 1-marker scans:*

$$\left.\frac{R^2_{1-\text{marker}}}{R^2_{\text{Z}-\text{ancestry}}}\right|_{2-\text{QTL}} = \frac{2}{3}\left[\frac{2-|l-x_1|-|l-x_2|}{1+x_1(1-x_1)+x_2(1-x_2)}\right]^2 \tag{S15}$$

Proof. Using the same logic as above theorem, this ratio between the two regression powers is

$$\begin{aligned}
\frac{R^2_{1-\text{marker}}}{R^2_{\text{Z}-\text{ancestry}}} &= \frac{\rho^2_{p_{x_1}+p_{x_2},p_l}}{\rho^2_{p_{x_1}+p_{x_2},f}} \\
&= \frac{[\text{Cov}(p_{x_1},p_l)+\text{Cov}(p_{x_2},p_l)]^2}{\text{Var}[p_l]\text{Var}[p_{x_1}+p_{x_2}]} \times \frac{\text{Var}[f]\text{Var}[p_{x_1}+p_{x_2}]}{[\text{Cov}(p_{x_1},f)+\text{Cov}(p_{x_2},f)]^2} \\
&= \frac{\text{Var}[f]}{\text{Var}[p_l]}\left[\frac{\text{Cov}(p_{x_1},p_l)+\text{Cov}(p_{x_2},p_l)}{\text{Cov}(p_{x_1},f)+\text{Cov}(p_{x_2},f)}\right]^2 \\
&= \frac{2}{3}\left[\frac{2-|l-x_1|-|l-x_2|}{1+x_1(1-x_1)+x_2(1-x_2)}\right]^2
\end{aligned} \tag{S16}$$

□

The above 2-QTL/1-marker relationship shows that markers between x_1 and x_2 all have the same predictive power, because $2-|l-x_1|-|l-x_2| = 2+x_1-x_2$ is independent of l when $x_1 < l < x_2$.

Below, we derive additional results when more than one markers are used.

Theorem 5 (Polygenic model & n-marker scans)**.** *Conditioning on the polygenic model of pupal weight, and assume that n markers with additive effects are used to fit the genotype-phenotype map, the relationship between regression powers is:*

$$\left.\frac{R^2_{n-\text{marker}}}{R^2_{\text{Z}-\text{ancestry}}}\right|_{\text{Polygenic}} = \begin{bmatrix} \rho_{p_{l_1},f} \\ \rho_{p_{l_2},f} \\ \vdots \\ \rho_{p_{l_n},f} \end{bmatrix}^{\top} \begin{bmatrix} 1 & \rho_{p_{l_1},p_{l_2}} & \cdots & \rho_{p_{l_1},p_{l_n}} \\ \rho_{p_{l_2},p_{l_1}} & 1 & \cdots & \rho_{p_{l_2},p_{l_n}} \\ \vdots & \vdots & \ddots & \vdots \\ \rho_{p_{l_n},p_{l_1}} & \rho_{p_{l_n},p_{l_2}} & \cdots & 1 \end{bmatrix}^{-1} \begin{bmatrix} \rho_{p_{l_1},f} \\ \rho_{p_{l_2},f} \\ \vdots \\ \rho_{p_{l_n},f} \end{bmatrix} \tag{S17}$$

Proof. This relationship is by definition the formula for the coefficient of multiple correlation between f and $p_{l_1}, \cdots, p_{l_n}$. □

Corollary 1 (Polygenic model & 2-marker scans)**.** *This is the explicit formula for Theorem 5 using two additive markers ($n = 2$):*

$$\left.\frac{R^2_{2-\text{marker}}}{R^2_{\text{Z}-\text{ancestry}}}\right|_{\text{Polygenic}} = \frac{6|l_1 - l_2|(l_1 + l_2 - 1)^2 + 3[1 + 2l_1(1 - l_1)][1 + 2l_2(1 - l_2)]}{8 - 4|l_1 - l_2|} \tag{S18}$$

Equation S18 shows that the two most informative markers under the polygenic model and 2-marker scans are located near $l_1 \approx 0.27$ and $l_2 \approx 0.73$—about a quarter into the chromosome from both ends.

3. Mathematical details for analyzing ovary dysgenesis on the Z chromosome

The same notation near Equation S6 is used throughout this subsection. In pupal weight analysis, the polygenic model posits that weight is a linear function of introgressed ancestry fraction on the Z chromosome (i.e., W and f are perfectly linearly correlated, ignoring noise). Since the expected ovary phenotypes in D(DB) females and *Heliconius* females are nonlinear with respect to f, we now consider a generalized polygenic model, where the expected phenotype V is a continuous function of f:

$$V = g(f) \tag{S19}$$

When g is a linear function, we recover the polygenic model for pupal weight. If g is a nonlinear function, it corresponds to global epistasis on Z-linked introgression. Moreover, we assume more generally that crossover positions are distributed along the chromosomal axis following a probability density function:

$$c(l) \quad \text{(The distribution of single crossover positions)}, \tag{S20}$$

Apart from this general assumption, we still assume that each chromosome pair per meiosis has one and only one crossover.

3.1 Artifactual QTL in 1-marker scans when the architecture is polygenic

The regression power of a 1-marker scan using the marker at position l against phenotype V is

$$R^2_{1-\text{marker}} = \frac{\text{Cov}^2(p_l, V)}{\text{Var}[p_l]\text{Var}[V]} \tag{S21}$$

Since $\text{Var}[p_l] = 1/4$ and $\text{Var}[V]$ is independent of l, the magnitude of $R^2_{1-\text{marker}}$ on different markers depends only on $\text{Cov}(p_l, V)$.

Theorem 6 (Covariance between p_l and V)**.** *Let $h(f) = g(1-f) - g(f)$. The covariance between p_l and V is given by the following formula:*

$$\text{Cov}(p_l, V) = \frac{1}{8}\left[h(0) + \int_0^{1-l} h(f)c(1-f)\,\mathrm{d}f + \int_0^{l} h(f)c(f)\,\mathrm{d}f\right] \tag{S22}$$

Proof. First, we have

$$\mathbb{E}[p_l] = \frac{1}{2}, \quad \mathbb{E}[V] = \frac{1}{4}g(0) + \frac{1}{4}g(1) + \frac{1}{4}\int_0^1 g(f)c(f)\,\mathrm{d}f + \frac{1}{4}\int_0^1 g(1-f)c(f)\,\mathrm{d}f \tag{S23}$$

The expectation of the product variable $p_l V$ is

$$\mathbb{E}[p_l V] = \frac{1}{4}g(1) + \frac{1}{4}\int_l^1 g(f)c(f)\,\mathrm{d}f + \frac{1}{4}\int_0^l g(1-f)c(f)\,\mathrm{d}f \tag{S24}$$

Thus,

$$\text{Cov}(p_l, V) = \frac{1}{8}\left\{g(1) - g(0) + \int_0^{1-l}[g(1-f) - g(f)]c(1-f)\,\mathrm{d}f + \int_0^{l}[g(1-f) - g(f)]c(f)\,\mathrm{d}f\right\} \tag{S25}$$

It is thus natural to define $h(f) = g(1-f) - g(f)$, which measures the level of asymmetry of the function $g(f)$ with respect to $f = 1/2$. This yields the final result. □

Theorem 7 (The existence of artifactual QTL in 1-marker scans)**.** *Suppose the polygenic model is true, and crossover positions are distributed according to $c(l)$. In that case, the necessary and sufficient condition for a non-zero association between a marker and a trait in a backcross brood is that $g(f)$ is a reflectionally asymmetric function with respect to $f = 1/2$. (Example: Figure S12A-F)*

Note. For simplicity, "with respect to" is written as "w.r.t."

Proof. The equivalent statement of the theorem is:

$$\text{i) } \mathrm{Cov}(p_l, V) = 0 \text{ for all } l \quad \Leftrightarrow \quad \text{ii) } g(f) \text{ is symmetric w.r.t. } f = 1/2.$$

Proving ii) $\Rightarrow$ i) is straightforward because a symmetric $g(f)$ means $h(f) \equiv 0$, so $\mathrm{Cov}(p_l, V) \equiv 0$.

To prove i) $\Rightarrow$ ii), since $\mathrm{Cov}(p_l, V) \equiv 0$, we have

$$0 \equiv \partial_l \mathrm{Cov}(p_l, V) = \frac{1}{8}\{-[g(l) - g(1-l)]c(l) + [g(1-l) - g(l)]c(l)\} \tag{S26}$$

Thus, $g(l) \equiv g(1-l)$, and g is symmetric w.r.t. position $f = 0.5$.

Finally, take the contrapositive statement to get the original theorem:

$$\text{i) } \mathrm{Cov}(p_l, V) \neq 0 \text{ for some } l \quad \Leftrightarrow \quad \text{ii) } g(f) \text{ is asymmetric w.r.t. } f = 1/2.$$

□

If crossover positions are uniformly distributed along the chromosome (e.g., *Papilio* males), we have $c(l) \equiv 1$. Then, the expected regression power $R^2_{1-\mathrm{marker}}$ will always be a symmetric function w.r.t. $l = 0.5$, because $\mathrm{Cov}(p_l, V) \equiv \mathrm{Cov}(p_{1-l}, V)$. Thus, all properties of $R^2_{1-\mathrm{marker}}$ can be discussed assuming that $l \leq 1/2$. Next, we give a sufficient condition for the existence of a unique peak of $R^2_{1-\mathrm{marker}}$ at the chromosome center under uniform crossovers.

Theorem 8 (A sufficient condition for a unique peak of $R^2_{1-\mathrm{marker}}$ at the chromosome center for uniform crossovers)**.** *If $h(f)$ is a continuous function and has no zeros in $0 < f < 1/2$, then there is a unique peak for $R^2_{1-\mathrm{marker}}$ at the chromosome center ($l = 1/2$). (Examples: Figure S12C,D)*

Proof. Again, note that $R^2_{1-\mathrm{marker}}$ is proportional to $\mathrm{Cov}^2(p_l, V)$ by a constant factor, so we only need to prove the existence of a unique peak for $\mathrm{Cov}^2(p_l, V)$ at $l = 1/2$. Second, $h(f)$ is anti-symmetric w.r.t. $f = 1/2$. If $r < 1/2$, the first integral in Equation S22 is:

$$\int_0^{1-l} h(f)\,\mathrm{d}f = \int_0^{l} h(f)\,\mathrm{d}f \tag{S27}$$

Thus,

$$\mathrm{Cov}^2(p_l, V) = \frac{1}{64}\left[h(0) + 2\int_0^l h(f)\,\mathrm{d}f\right]^2 \tag{S28}$$

By anti-symmetry, $h(1/2) = 0$. Since $h(f)$ is continuous and has no zeros in $0 < f < 1/2$, $h(f)$ does not switch sign in $0 < f < 1/2$. Thus, if $h(0) > 0$, the integrand in the previous equation will be positive, and $\left[h(0) + 2\int_0^l h(f)\,\mathrm{d}f\right]^2$ is an increasing function of l up to $l = 1/2$. If $h(0) < 0$, the integrand will be negative, and $\left[h(0) + 2\int_0^l h(f)\,\mathrm{d}f\right]^2$ is still an increasing function of l. If $h(0) = 0$, $h(f)$ will always be positive or negative, and the same result holds. Thus, $\mathrm{Cov}^2(p_l, V)$ is an increasing function of l when $0 \leq l \leq 1/2$, and by symmetry of $\mathrm{Cov}(p_l, V)$, $\mathrm{Cov}^2(p_l, V)$ has a unique maximum at $l = 1/2$. □

For ovary dysgenesis in *Papilio* D(DB) females, since more normal phenotypes are suppressed in backcrosses when the Z chromosome is not recombined in ancestry, we may assume that $h(0) = g(1) - g(0) = 0$. Then,

$$\begin{aligned} \mathrm{Cov}^2(p_l, V) &= \frac{1}{16}\left[\int_0^l h(f)\,\mathrm{d}f\right]^2 \\ \mathrm{Var}[p_l] &= \frac{1}{4} \\ \mathrm{Var}[V] &= \frac{1}{2}\int_0^1 [g(f) - g(0)]^2\,\mathrm{d}f - \frac{1}{4}\left[\int_0^1 [g(f) - g(0)]\,\mathrm{d}f\right]^2 \end{aligned} \tag{S29}$$

Without loss of generality, define $\widetilde{g}(f) = g(f) - g(0)$, and so $\widetilde{h}(f) = h(f)$. The regression power can thus be expressed as

$$R^2_{1-\mathrm{marker}}(l) = \left[\int_0^l \widetilde{h}(f)\,\mathrm{d}f\right]^2 \Big/ \left\{2\int_0^1 \widetilde{g}^2(f)\,\mathrm{d}f - \left[\int_0^1 \widetilde{g}(f)\,\mathrm{d}f\right]^2\right\} \tag{S30}$$

3.2 Artifactual epistatic QTL in 2-marker scans when the architecture is polygenic

To investigate the statistical interaction between a pair of markers to predict a trait, it is conventional to work with binary ancestry defined as

$$\begin{cases} 1, & \text{if introgressed} \\ -1, & \text{if not introgressed} \end{cases} \tag{S31}$$

Note that this representation does not change any prior results assuming additivity among markers. For two markers at positions l_1 and l_2, we assume that $l_1 \leq l_2$. The following statistics are associated with the crossover model with the new ancestry representation:

$$\begin{aligned} \mathbb{E}[p_{l_1} p_{l_2}] &= 1 - \int_{l_1}^{l_2} c(f)\,\mathrm{d}f \\ \mathrm{Var}[p_{l_1} p_{l_2}] &= 2\int_{l_1}^{l_2} c(f)\,\mathrm{d}f - \left[\int_{l_1}^{l_2} c(f)\,\mathrm{d}f\right]^2 \end{aligned} \tag{S32}$$

Let the average magnitude of $g(f)$ be:

$$\overline{g} = \int_0^1 g(f)\,\mathrm{d}f \tag{S33}$$

Then, we have the covariance between $p_{l_1} p_{l_2}$ and V as follows.

Theorem 9 (Covariance between $p_{l_1}p_{l_2}$ and V)**.** *Let $H(f) = g(f) + g(1-f) - 2\overline{g}$. The covariance between $p_{l_1}p_{l_2}$ and V is given by the following formula:*

$$\mathrm{Cov}(p_{l_1}p_{l_2}, V) = \frac{1}{4}\int_{l_1}^{l_2}\left[H(0) + \int_0^1 H(x)c(x)\,\mathrm{d}x - 2H(f)\right]c(f)\,\mathrm{d}f \tag{S34}$$

Proof. First, we arrange terms into the form of $g(f) + g(1-f)$:

$$\begin{aligned}
\mathbb{E}[p_{l_1}p_{l_2}V] &= \frac{1}{4}g(1) + \frac{1}{4}g(0) + \frac{1}{4}\left(\int_0^{l_1} + \int_{l_2}^1 - \int_{l_1}^{l_2}\right)g(f)c(f)\,\mathrm{d}f + \frac{1}{4}\left(\int_{l_2}^1 + \int_0^{l_1} - \int_{l_1}^{l_2}\right)g(1-f)c(f)\,\mathrm{d}f \\
&= \frac{1}{4}\left[g(1) + g(0) + \left(\int_0^{l_1} + \int_{l_2}^1 - \int_{l_1}^{l_2}\right)\left[g(f) + g(1-f)\right]c(f)\,\mathrm{d}f\right] \\
&= \frac{1}{4}\left[H(0) + \left(\int_0^1 - 2\int_{l_1}^{l_2}\right)H(f)c(f)\,\mathrm{d}f\right] + \left(1 - \int_{l_1}^{l_2}c(f)\,\mathrm{d}f\right)\overline{g} \\
\mathbb{E}[p_{l_1}p_{l_2}]\mathbb{E}[V] &= \left[1 - \int_{l_1}^{l_2}c(f)\,\mathrm{d}f\right]\left[\frac{1}{4}H(0) + \overline{g} + \frac{1}{4}\int_0^1 H(f)c(f)\,\mathrm{d}f\right]
\end{aligned} \tag{S35}$$

Thus,

$$\begin{aligned}
\mathrm{Cov}(p_{l_1}p_{l_2}, V) &= \mathbb{E}[p_{l_1}p_{l_2}V] - \mathbb{E}[p_{l_1}p_{l_2}]\mathbb{E}[V] \\
&= \frac{1}{4}H(0)\int_{l_1}^{l_2}c(f)\,\mathrm{d}f + \frac{1}{4}\int_0^1 H(f)c(f)\,\mathrm{d}f\int_{l_1}^{l_2}c(f)\,\mathrm{d}f - \frac{1}{2}\int_{l_1}^{l_2}H(f)c(f)\,\mathrm{d}f \\
&= \frac{1}{4}\int_{l_1}^{l_2}\left[H(0) + \int_0^1 H(x)c(x)\,\mathrm{d}x - 2H(f)\right]c(f)\,\mathrm{d}f
\end{aligned} \tag{S36}$$

□

Theorem 10 (The existence of artifactual interacting QTL pairs in 2-marker scans)**.** *Suppose the polygenic model is true, and crossover positions are distributed according to $c(l)$. In that case, the necessary and sufficient condition for a non-zero interaction between a pair of markers is that $g(f)$ is a rotationally asymmetric function with respect to the point $(1/2, f(1/2))$ by a degree of 180°. Examples: Figure S14.*

Proof. The logic is similar to the proof of Theorem 7. Take the derivative w.r.t. either l_1 or l_2 of the above covariance, we get:

$$0 \equiv \partial_{l_2}\mathrm{Cov}[p_{l_1}p_{l_2}, V] = \frac{1}{4}\left[H(0) + \int_0^1 H(x)c(x)\,\mathrm{d}x - 2H(l_2)\right]c(l_2) \tag{S37}$$

Since $c(l_2)$ cannot be zero for all l_2, we have

$$H(l_2) \equiv \frac{1}{2}H(0) + \frac{1}{2}\int_0^1 H(x)c(x)\,\mathrm{d}x \equiv \mathrm{Const.} \tag{S38}$$

This implies that $g(f) + g(1-f) \equiv \text{Const}$. This relationship indicates that g is a rotationally symmetric function w.r.t. the point $(1/2, g(1/2))$ by a degree of 180°. Conversely, if g is rotationally symmetric, $H(f) \equiv 0$, so the integrand becomes zero, and covariance is globally zero. □

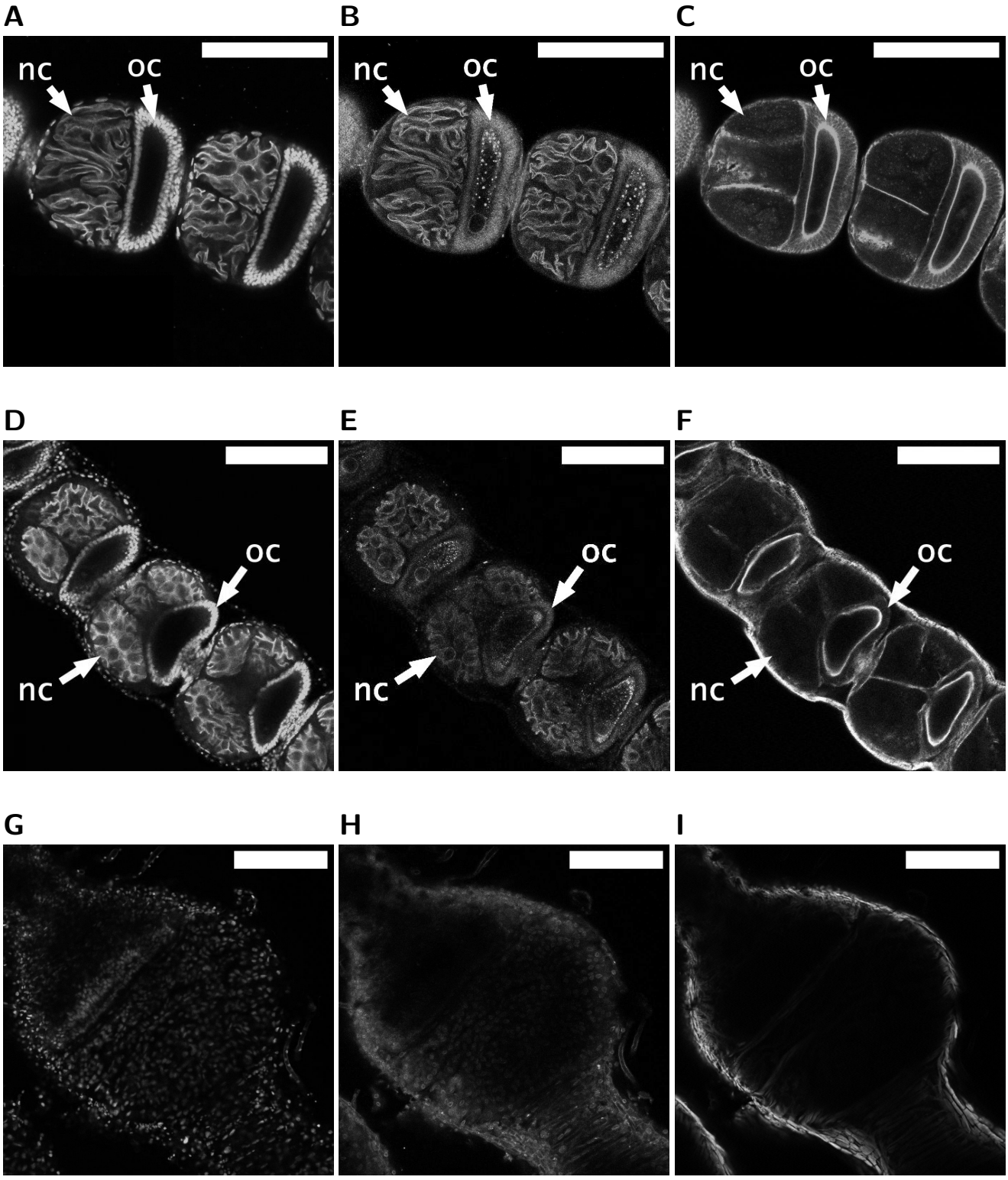


Figure S1: Confocal imaging of ovary phenotypes in *Papilio*. Scale bar=200μm. Left column: Hoechst (DNA); Middle column: WGA (membrane); Right column: Phalloidin (actin filaments). **(A-C)** Phenotype Normal in pure individuals. **(D-F)** Phenotype Normal in F_1 DB hybrids. **(G-I)** Phenotype Empty in F_1 BD hybrids.

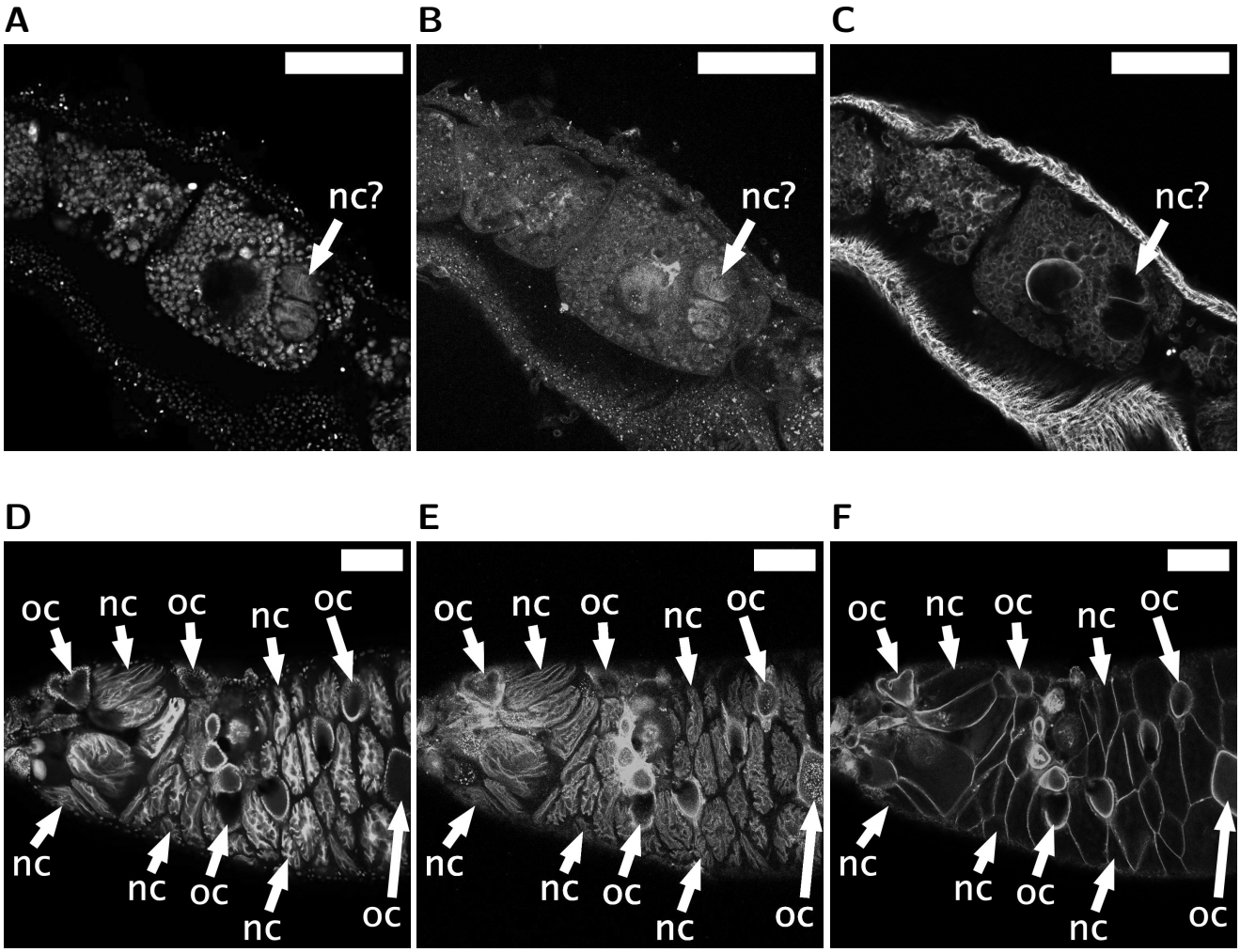


Figure S2: Confocal imaging of ovary phenotypes in *Papilio* (continued). Scale bar=200μm. Left column: Hoechst (DNA); Middle column: WGA (membrane); Right column: Phalloidin (actin filaments). **(A-C)** Phenotype Diminished (only in backcross individuals). **(D-F)** Phenotype Jammed (only in backcross individuals).

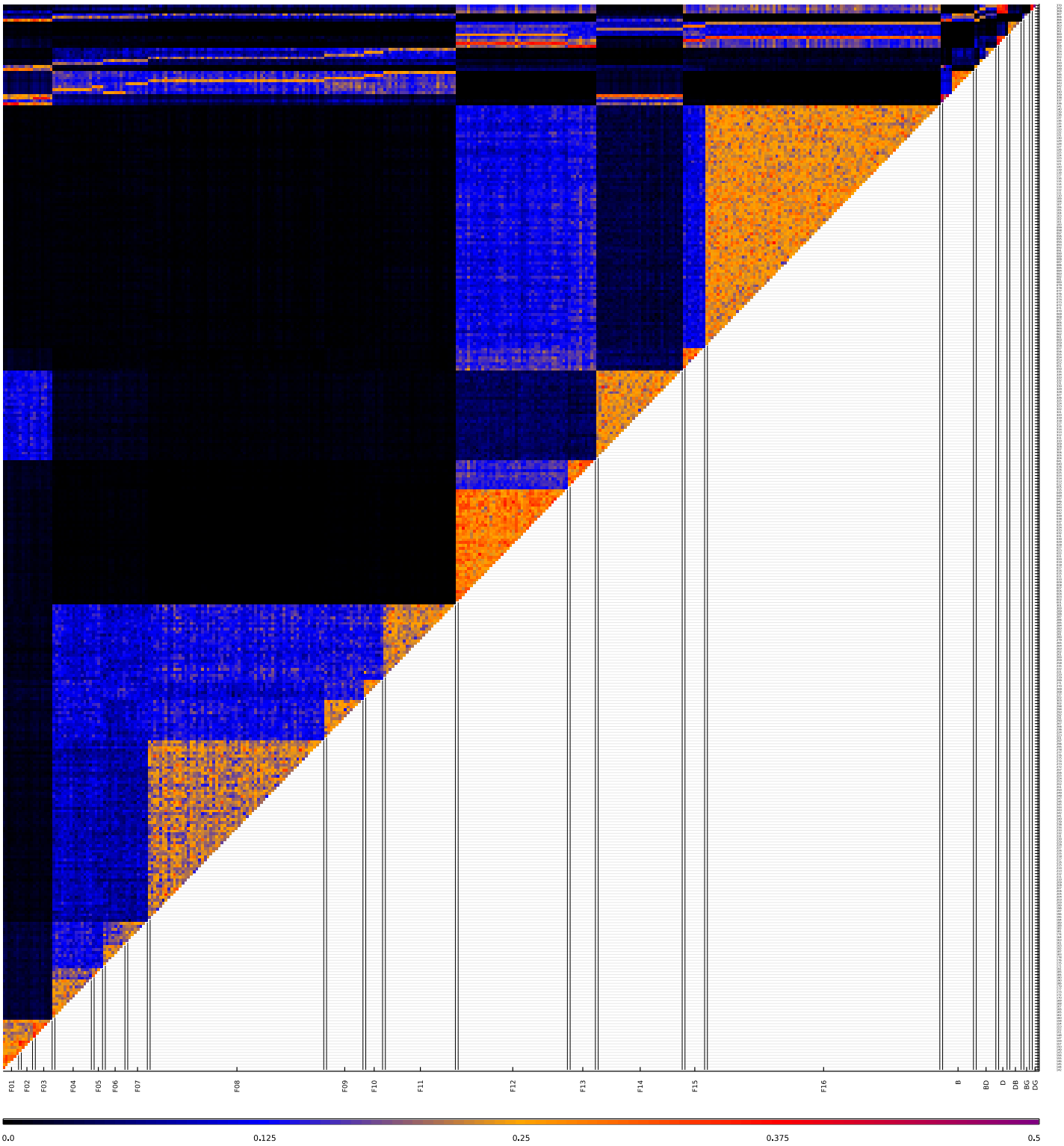


Figure S3: Kinship among individuals in *Papilio* after correcting for misplaced individuals in the pedigree. The horizontal axis contains family information (each "FXX" is a single family), and the vertical axis shows individual identifiers.

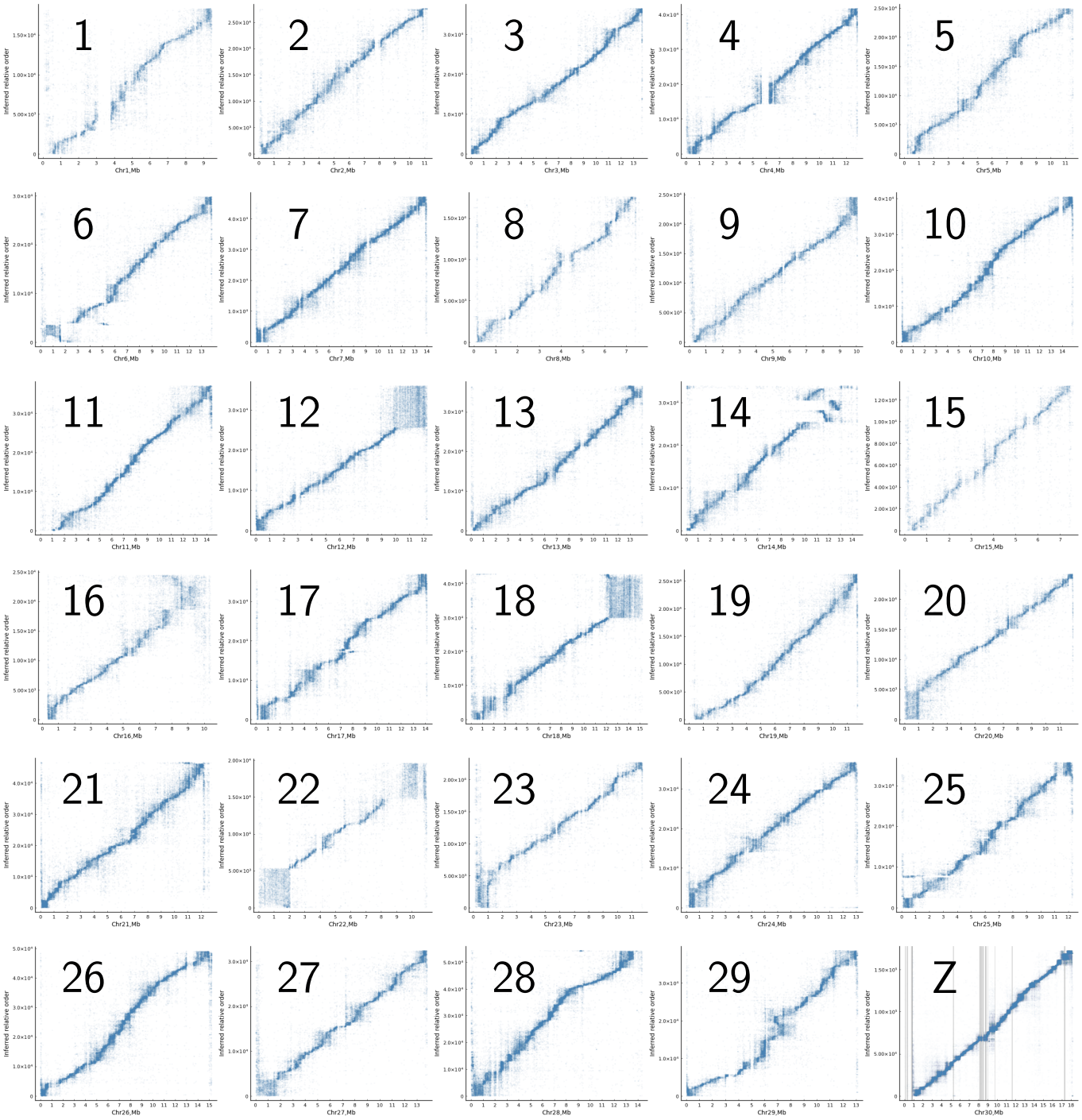


Figure S4: Inferred de novo marker order against the corrected reference genome of *Papilio bianor* shows good collinearity except for chromosome 14. Vertical lines in gray represent boundaries between PacBio scaffolds. Some chromosomal ends appear to have recombination suppressed (large blocks of unordered markers).

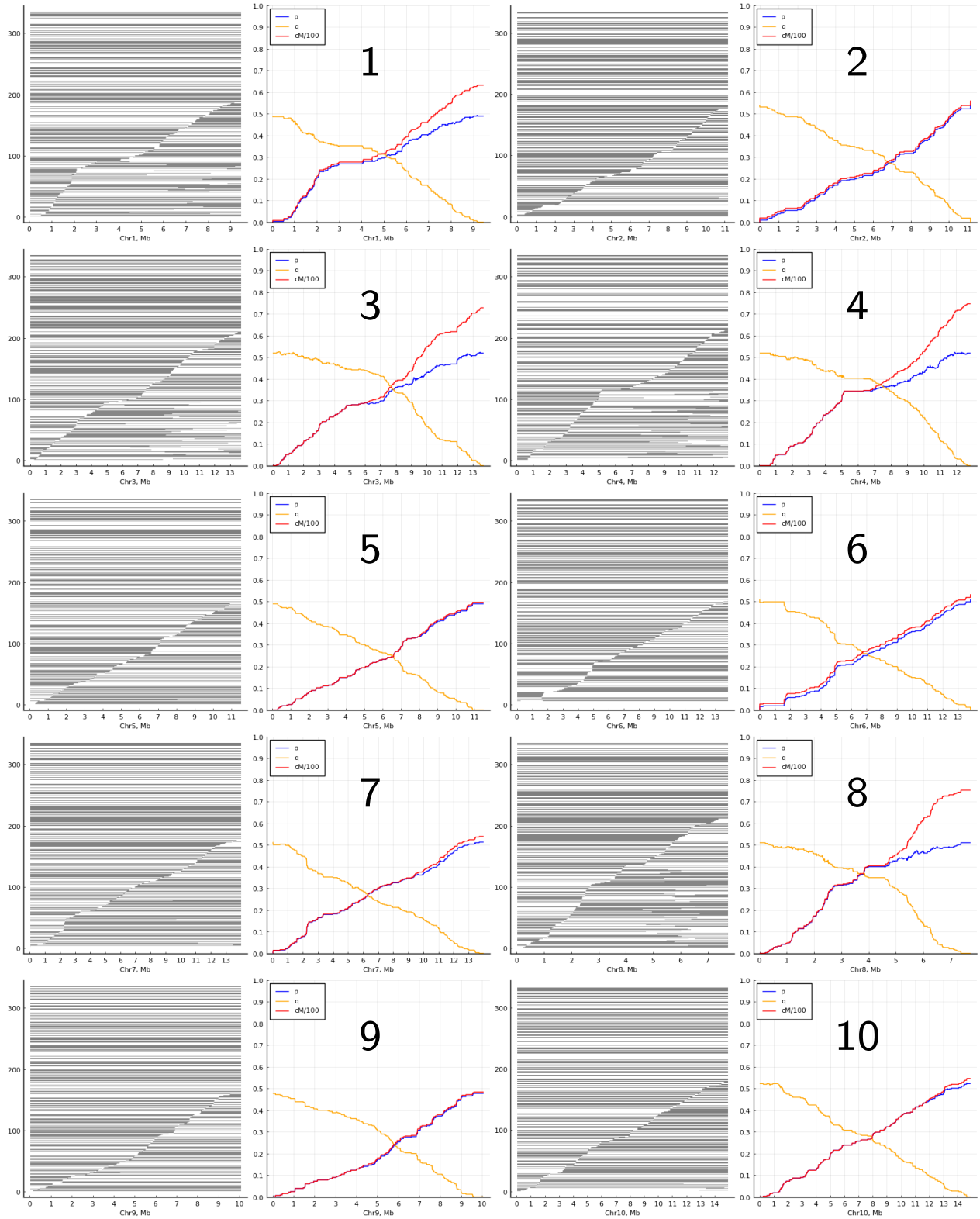


Figure S5: Inferred paternal haplotypes among all backcross individuals on chromosomes 1-10 in *Papilio*. Blue curves show the recombination probability of each marker to the left end of each chromosome. Yellow curves show the recombination probability of each marker to the right end of each chromosome. Red curves are linkage maps measured in cM.

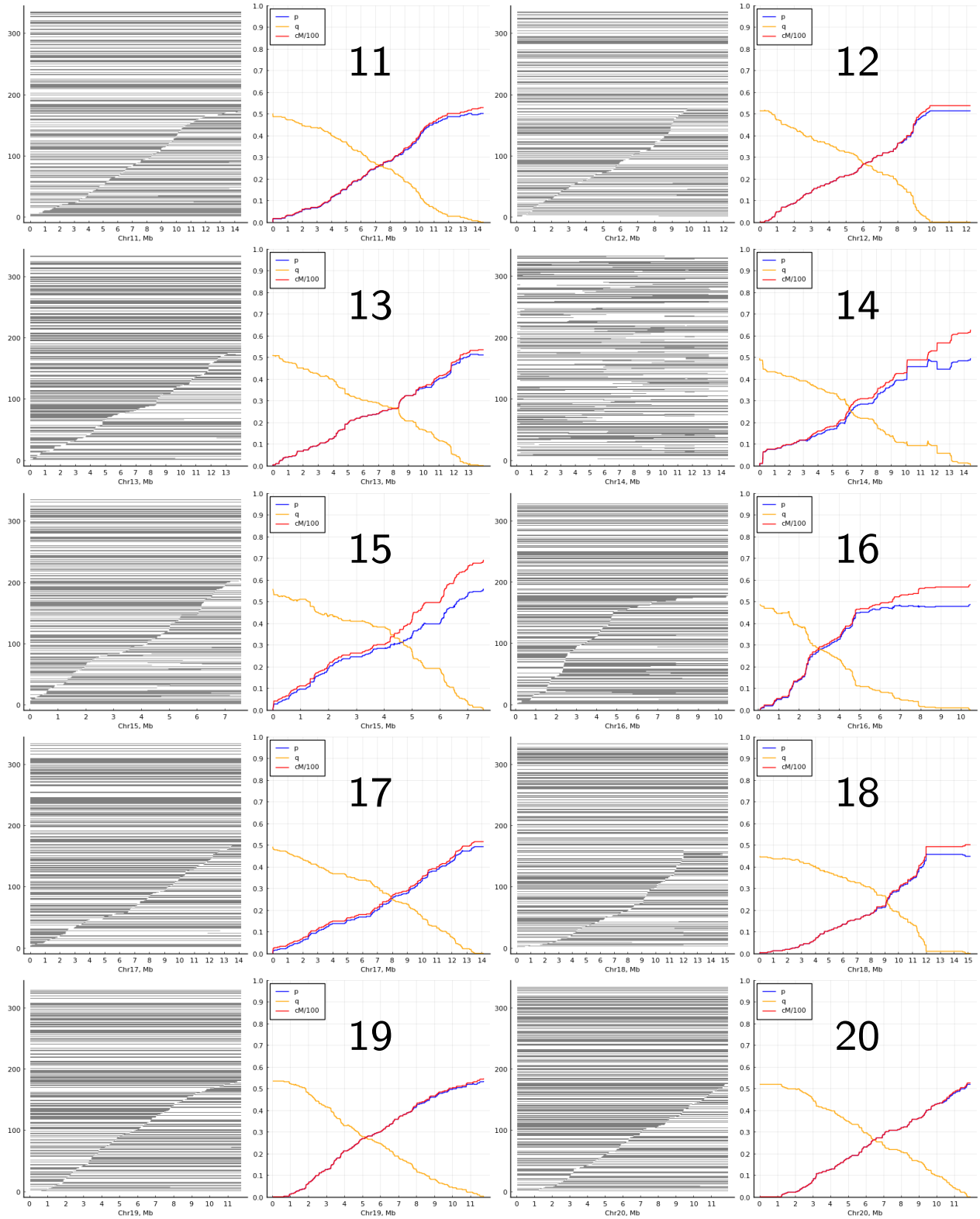


Figure S6: Inferred paternal haplotypes among all backcross individuals on chromosomes 11-20 in *Papilio*. Blue curves show the recombination probability of each marker to the left end of each chromosome. Yellow curves show the recombination probability of each marker to the right end of each chromosome. Red curves are linkage maps measured in cM.

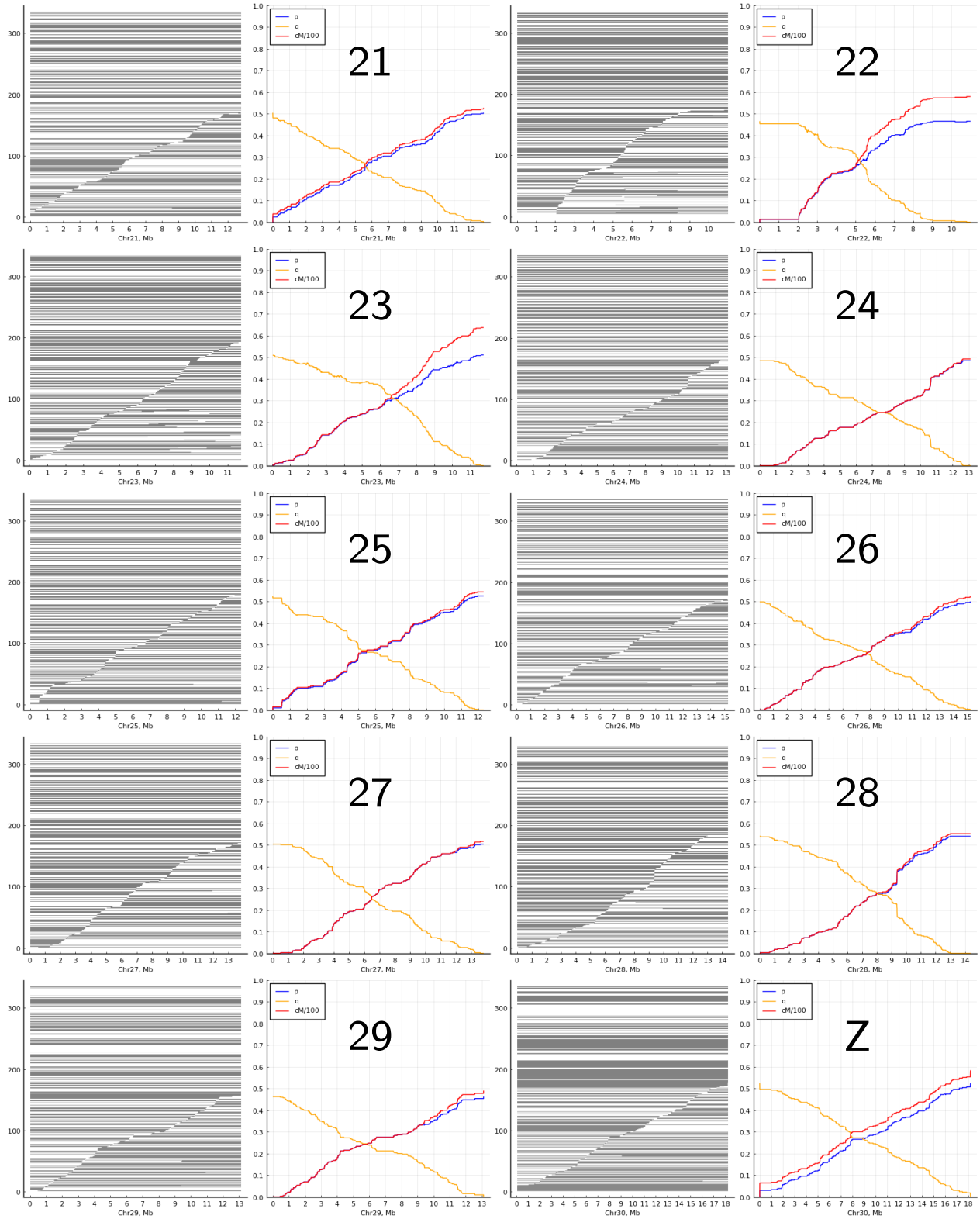
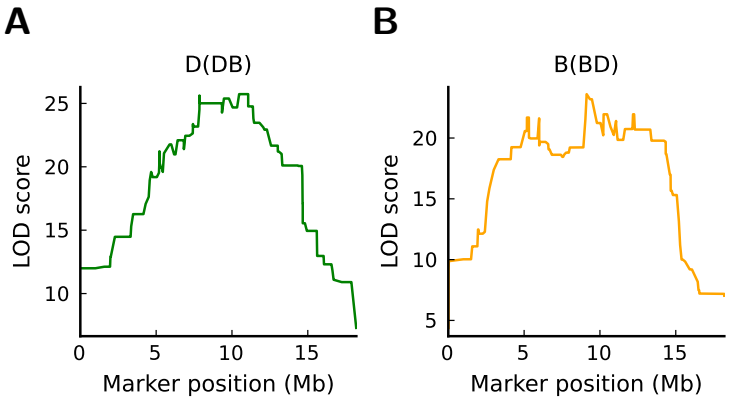


Figure S7: Inferred paternal haplotypes among all backcross individuals on chromosomes 21-Z in *Papilio*. Blue curves show the recombination probability of each marker to the left end of each chromosome. Yellow curves show the recombination probability of each marker to the right end of each chromosome. Red curves are linkage maps measured in cM.



C

Cross	Peak (Mb)	95% CI (Mb)
D(DB)	11.06879	[7.855832, 11.37611]
B(BD)	9.124007	[9.124007,12.254113]

Figure S8: One-marker scans of pupal weight in *Papilio* on the Z chromosome by r/qtl2. **(A,B)** LOD scores on the Z chromosome. **(C)** Peaks identified by r/qtl2 and their confidence intervals (CI) at the 95% level.

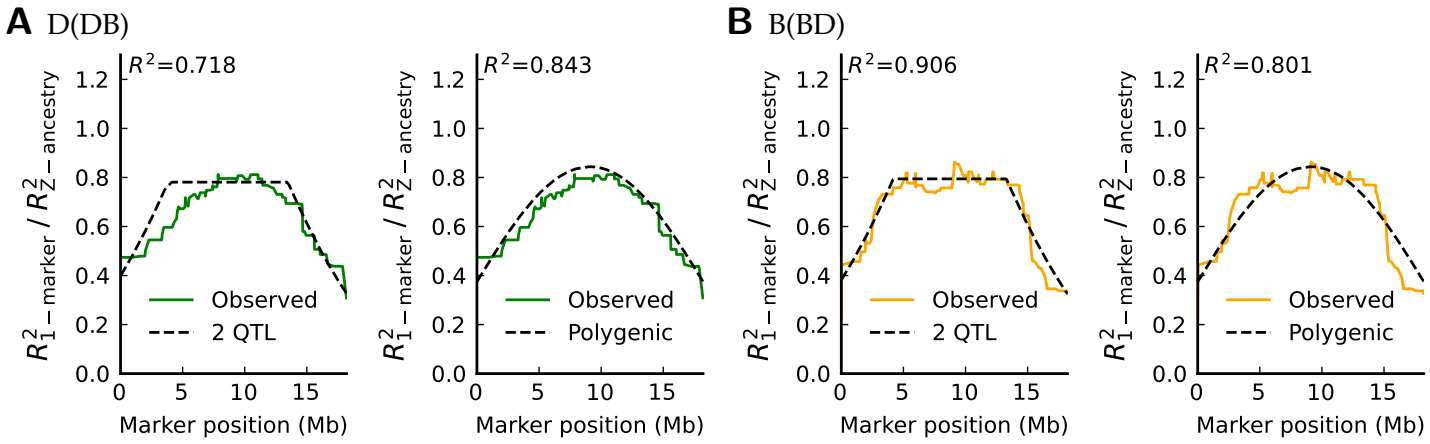


Figure S9: Expected results of 1-marker scans with the polygenic model versus 2-QTL models that best fit the observed curves in *Papilio* pupal weight. **(A)** In D(DB) females, the polygenic model can better fit 1-marker scans. For the best 2-QTL model, the relative locations of the two QTLs are 0.21 and 0.74. **(B)** In B(BD) females, the fully polygenic model is worse at fitting 1-marker scans than the best 2-QTL model. For the best 2-QTL model, the relative locations of the two QTLs are 0.23 and 0.73.

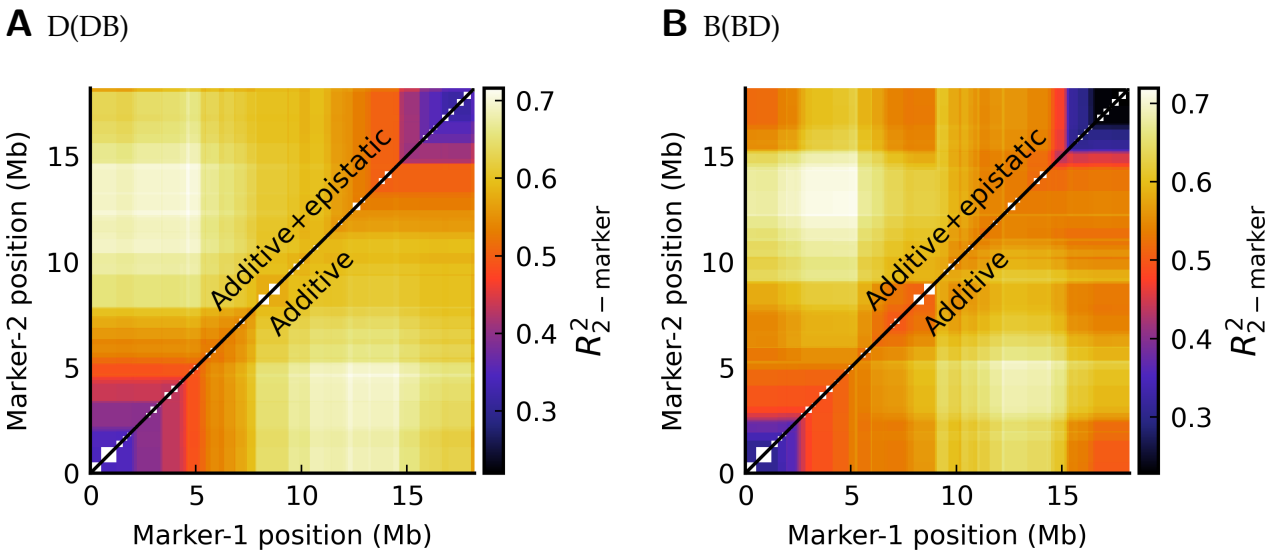


Figure S10: Results of 2-marker regression on pupal weight in *Papilio*. Model prediction powers are nearly identical between the additive model ($W \sim p_{l_1} + p_{l_2}$) and the full model with an extra epistasis term ($W \sim p_{l_1} + p_{l_2} + p_{l_1} p_{l_2}$). Thus, epistasis adds little information to predicting pupal weight in backcrosses.

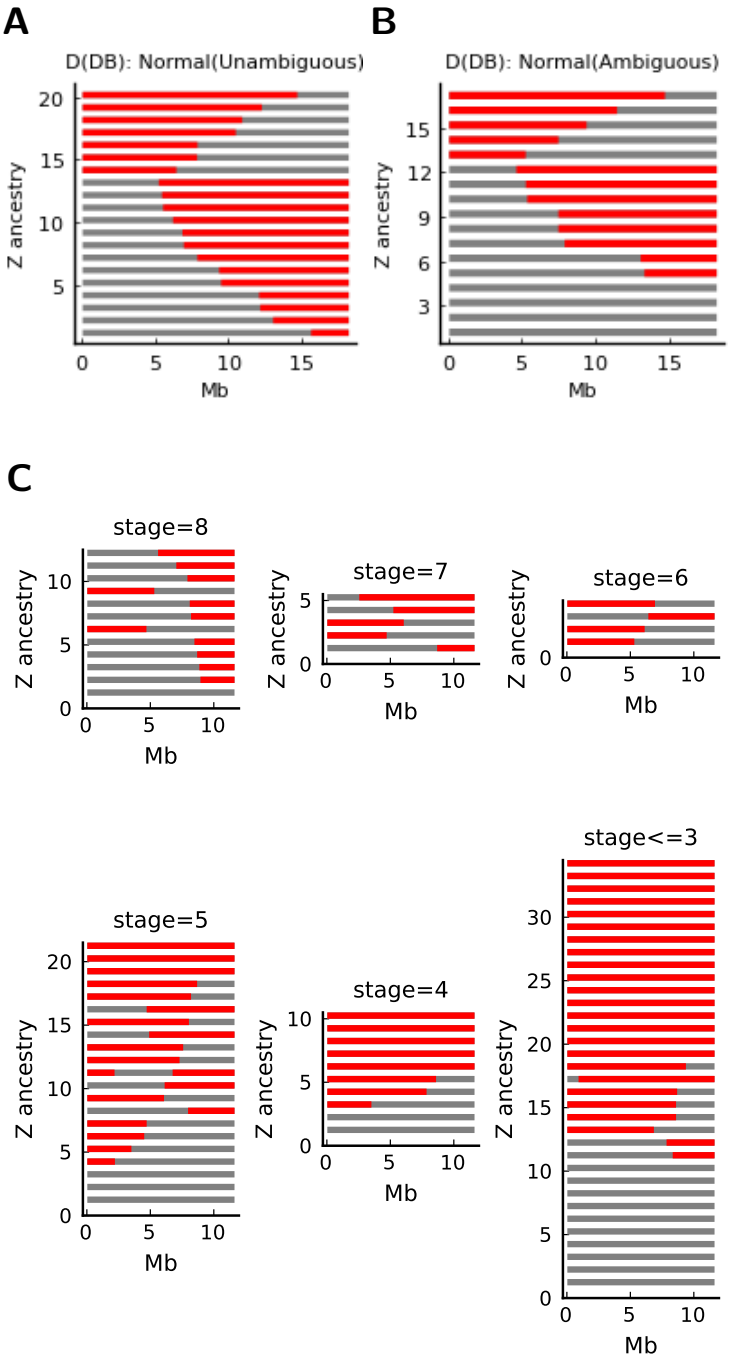


Figure S11: Z chromosome ancestry haplotypes in D(DB) females and *Heliconius* backcrosses. **(A,B)** Z chromosome haplotypes in D(DB) females associated with phenotype Normal. This phenotype is associated with Z chromosomes recombined in either direction. Gray: inherited from *P. dehaanii*; Red: inherited from *P. bianor*. **(C)** Z chromosome haplotypes in *Heliconius* backcross females grouped by ovary stages (larger=more normal). Gray: inherited from *H. pardalinus butleri*; Red: inherited from *H. p. sergestus*.

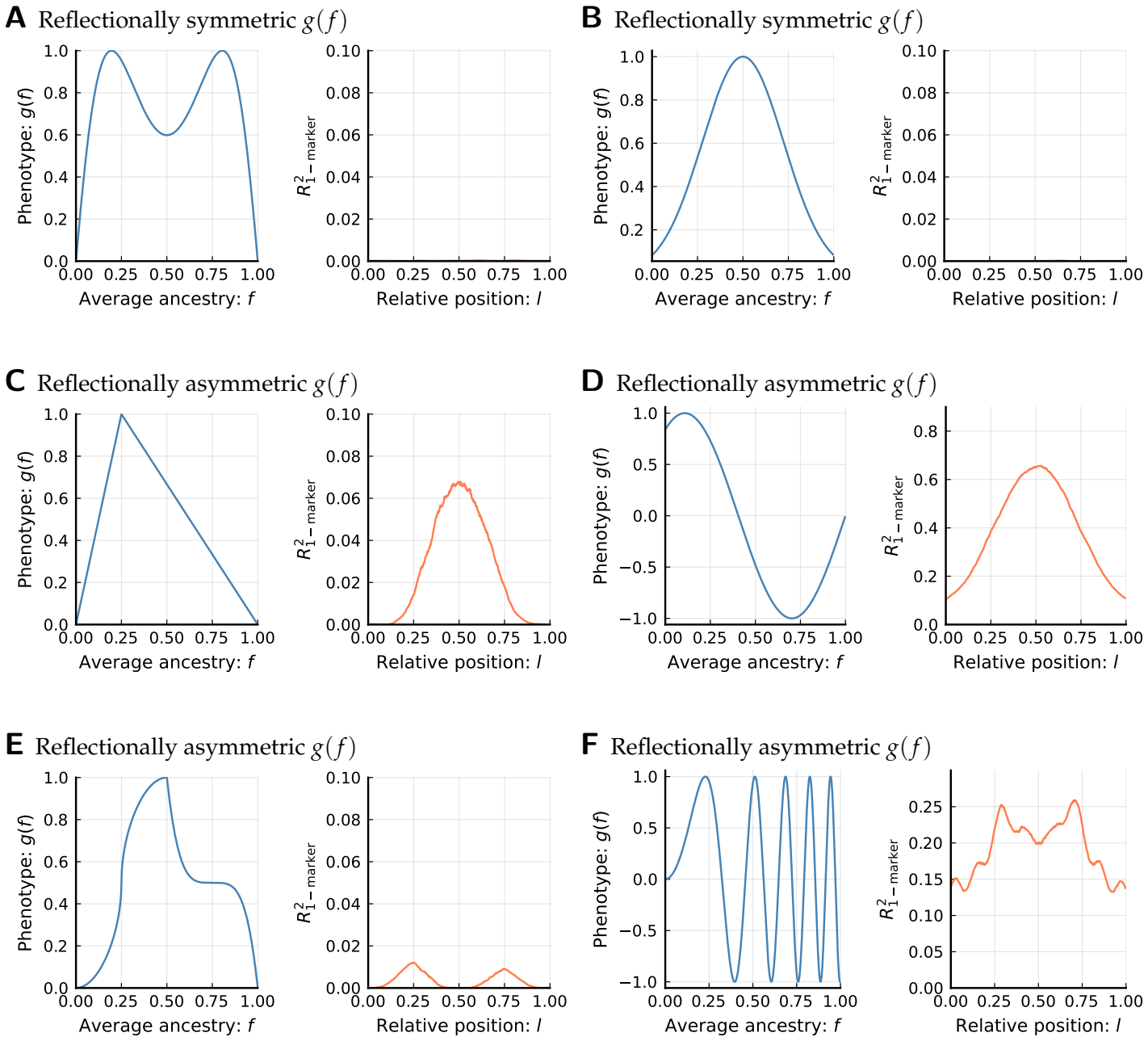


Figure S12: Different reflectional symmetry of $g(f)$ leads to different results of 1-marker scans. The uneven distribution of R^2 does not reflect an uneven distribution of phenotypic effects, because the model is fully polygenic. Simulated using 10^4 backcross individuals with uniform crossover positions. **(A,B)** A reflectionally symmetric $g(f)$ w.r.t. $f = 0.5$ produces no marker-phenotype association in 1-marker scans. **(C,D)** A reflectionally asymmetric $g(f)$ satisfying Theorem 8, i.e., no zeros in $h(f)$ when $0 < f < 1/2$, produces a unique peak at the chromosome center. **(E,F)** A reflectionally asymmetric $g(f)$ violating conditions in Theorem 8 can produce multiple peaks in 1-marker scans, but the shape of R^2 is still symmetric w.r.t. $l = 1/2$.

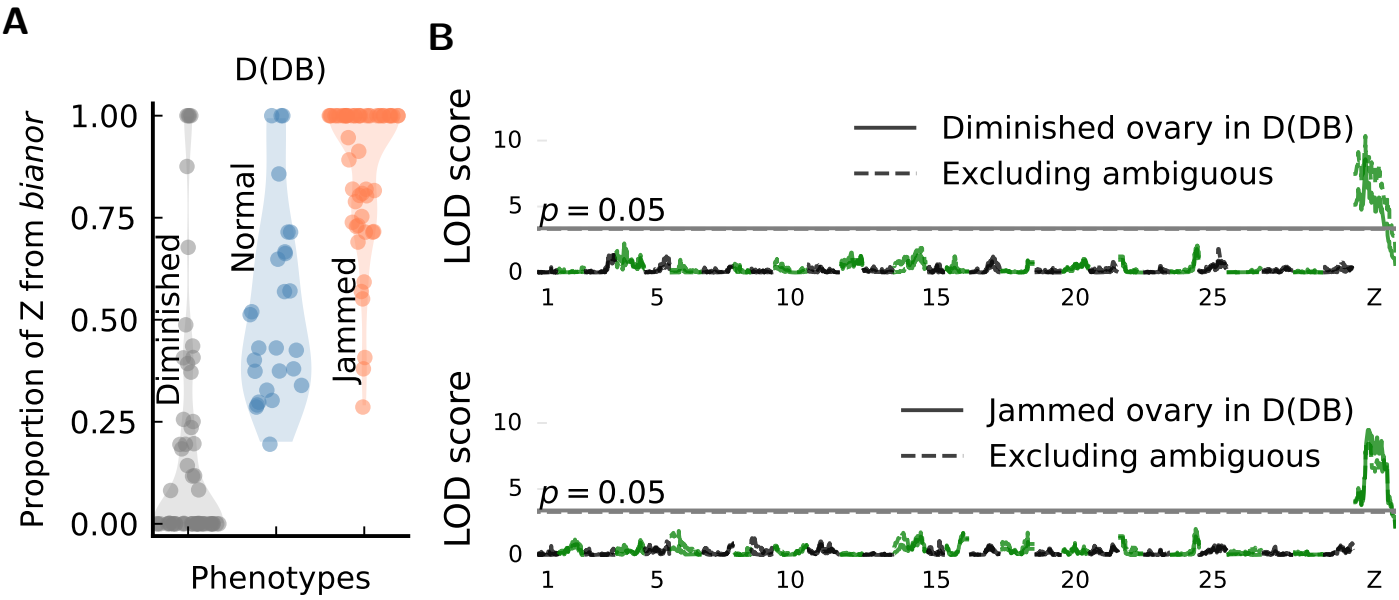


Figure S13: Ovary phenotypes in D(DB) females partitioned by the Z-chromosome ancestry fraction. **(A)** Ovary phenotypes in D(DB) females are well described by the Z-chromosome ancestry fraction. Sample dots represent one round of phenotype assignment for individuals with ambiguous phenotypes. **(B)** LOD scores of defective ovary phenotypes in D(DB) females using 1-marker scans. The Z chromosome is significantly associated with phenotypes Diminished and Jammed. This significant association is predicted by the polygenic model, because there is a strong reflectional asymmetry in $g(f)$ when the phenotype occurs only when the Z chromosome has little introgression (Diminished) or with nearly full introgression (Jammed).

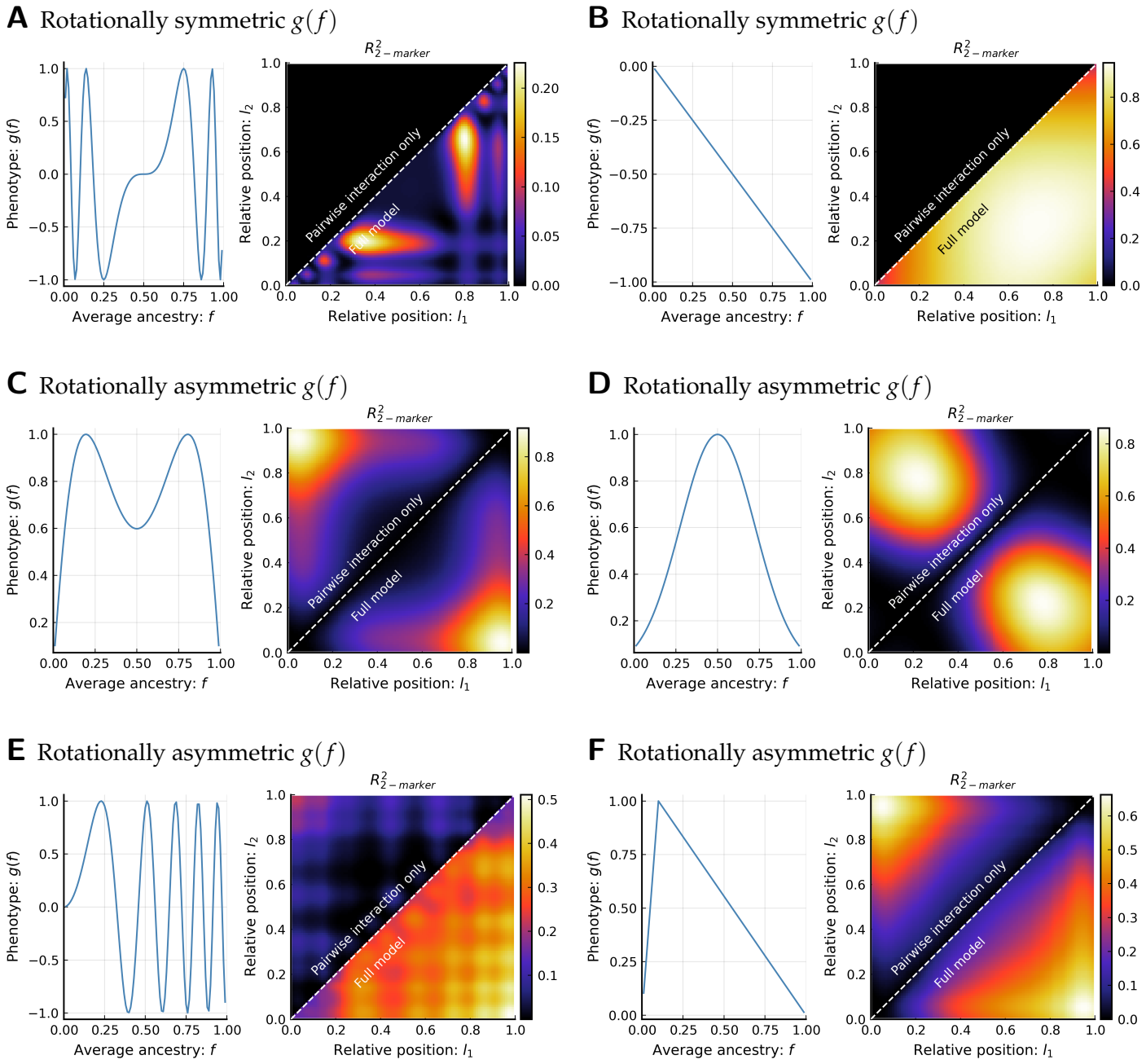


Figure S14: Different rotational symmetry of $g(f)$ leads to different results of 2-marker scans. The heatmap contains two genotype-phenotype models. The "Pairwise interaction only" model uses the regression $V \sim 1 + p_{l_1} p_{l_2}$, while the "Full model" includes additive terms: $V \sim 1 + p_{l_1} + p_{l_2} + p_{l_1} p_{l_2}$. The uneven distribution of R^2 does not reflect an uneven distribution of phenotypic effects, because the model is fully polygenic. Simulated using 10^4 backcross individuals with uniform crossover positions. **(A,B)** A rotationally symmetric $g(f)$ w.r.t. the function center produces no interaction between markers. **(C-F)** A rotationally asymmetric $g(f)$ w.r.t. the function center produces interaction between markers. If the function is also reflectionally symmetric (panels C and D), there will be no additive effects, so the interaction term dominates the full model.

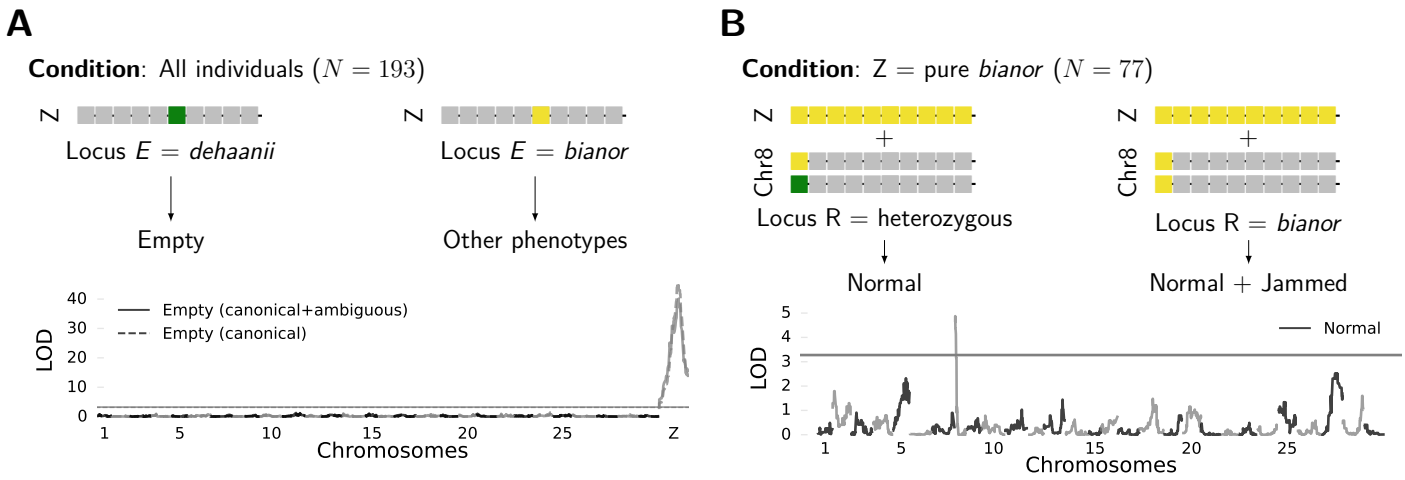


Figure S15: Two narrow regions of major effects control ovary dysgenesis in maternally *bianor* hybrids. **(A)** Phenotype Empty is dominantly controlled by Locus *E* on the Z chromosome. The LOD plot shows both the score for the canonical Empty phenotype as well as the score when we include a few ambiguous individuals that are classified as Empty. **(B)** If the Z chromosome is purely *bianor*, introgression on Locus *R* from *dehaanii* suppresses abnormal phenotypes.

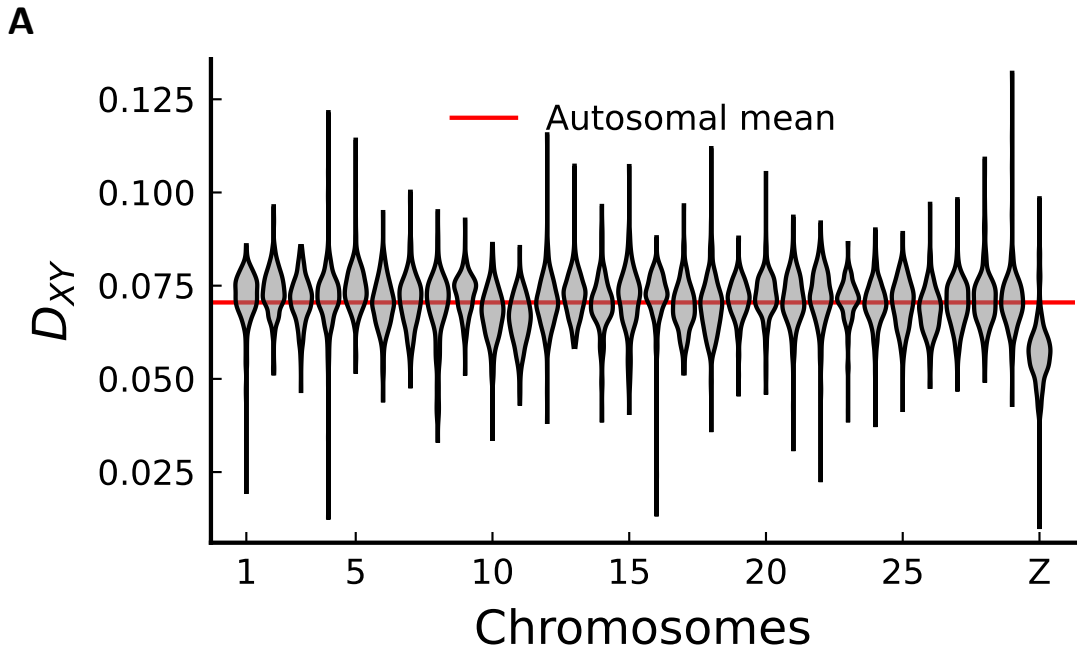


Figure S16: Average sequence divergence (D_{XY}) between parental *P. dehaanii* and *P. bianor* used in the experiment. Each data point is estimated for 50kb non-overlapping chromosomal windows.

Table S1: Summary of locus E and locus R in *Papilio*

Locus	Chromosome	Position (Mb)	LOD	95% Confidence interval (Mb)
E (canonical+ambiguous)	Z	12.18279	40.12502	[11.64904, 12.25411]
E (canonical)	Z	11.45892	45.56313	[11.37611 12.25411]
R	8	0.366353	4.875207	[0.005916 0.784124]

Table S2: The ratio of genetic variance between male and female pupal weight among backcross individuals in *Papilio*

Cross direction	D(DB)	B(BD)
$V_{g,\text{Male}} / V_{g,\text{Female}}$	0.37	0.23
95% Confidence interval of V_g ratio	(0.18, 0.62)	(0.05, 0.46)