

Supplementary Note

1 The model reflecting the performance of homozygotes and heterozygote of one locus simulated according to Hill equation

The typical physiologihisical theory had pointed that most of the mutants of genes encoding enzymes are dominant^{1,2}, which has been preliminarily verified in yeast and human³. Recent study also noticed that enzyme unsaturation caused by insufficient substrate may be the direct cause of dominance⁴. However, it is still not clear whether insufficient substrate background is the general mechanism of dominant or even overdominance, and the relationship between the sufficiency of background and the occurrence of dominant or overdominance is still not clearly elucidated in literature. In this study, we systematically simulated occurrence of additive, dominant to over-dominant inheritance of target receptor genes under different level of ligand background supply.

It is generally recognized that a ligand X binds to a receptor Y and reacts to produce a product is a common mechanism in biology. Dynamically, the number of molecule product of Y produced per unit time is a function of the concentration of ligand X on its active form X*:

$$\text{Production rate of } Y = f(X^*) \quad (1-1)$$

Typically, the input function of $f(X)$ is a monotonic, S-shaped function. It is an increase function when X is an activator and a decrease one when X is a repressor⁵. The Hill input function for an activator is a curve that rises from zero and approaches a maximal saturated level:

$$f(X^*) = \frac{\beta X^{*n}}{K^n + X^{*n}} \quad \text{Hill function for activator (1-2)}$$

The Hill function has three parameters, K , β and n .

Parameter K is termed as the activation coefficient, and has units of concentration. It defines the concentration of active X needed to significantly activate production. From the equation, we can see that half-maximal production is reached when $X = K$. The value of K related to the chemical affinity between ligand X and its receptor, as well as additional factors.

Parameter β is the maximal production level of Y. Maximal production is reached at high activator concentration, $X \gg K$. Because at high concentration, X binds the receptor with high probability to generate more products per unit time.

Parameter n is known as Hill coefficient. It governs the steepness of the curve between two inflection points of the input function. Usually, it is moderately steep, with $n = 1 - 4$. The larger is n , the more step-like is the input function. Particularly, When $n = 1$, hill function is equal to Michaelis-Menten equation. As many functions in biology, the Hill function approaches a limiting value at high level of X , rather than increase indefinitely.

For a repressor, the Hill function is a decreasing S-shaped curve, whose shape depends on three similar parameters:

$$f(x) = \frac{\beta}{1 + \left(\frac{X^*}{K}\right)^n}$$

Hill input function for repressor (1-3)

The production of Y is balanced by two processes, degradation (destruction by specific proteins in the cell) and dilution (the reduction in concentration due to the increase of cell volume during growth). The degradation rate is a_{deg} , and the dilution rate is a_{dil} , giving a total degradation plus dilution rate (in units of 1/time) of

$$\alpha = \alpha_{\text{deg}} + \alpha_{\text{dil}} \quad (1-4)$$

The change in the concentration of Y due to the difference between its production and degradation plus dilution, as described by a dynamic equation:

$$\frac{dY}{dt} = f(X^*) - \alpha Y \quad (1-5)$$

At steady state, Y reaches a constant concentration Y_{st} . The steady-state concentration can be found by solving for $dY/dt = 0$. The steady-state concentration is:

$$Y_{st} = f(X^*)/\alpha \quad (1-6)$$

If reached its maximal level, we can also write as:

$$Y_{st} = \beta/\alpha \quad (1-7)$$

This makes sense: The higher is the production rate β , the higher will reach the steady-state concentration Y_{st} . The higher is the degradation/dilution rate α , the lower is Y_{st} .

Now let us consider one single locus with allele A and a, which are or code some kind of receptor and can be regulated by ligand X. The product of allele A is Y_1 at steady-state under concentration $[X_{11}^*]$ of active X (X_{11}^*), and that of a is Y_2 under concentration $[X_{22}^*]$. The production function of two alleles is expressed respectively as:

$$A: dY_1/dt = f([X_{11}^*]) - \alpha_1 Y_1 \quad (1-8a)$$

56 $a: dY_2/dt = f([X_{22}^*]) - \alpha_2 Y_2$ (1-8b)

57 Where $\alpha_1 > 0$ and $\alpha_2 > 0$ are the relative degradation rate.

58 Then, the product of two alleles at steady-state is respectively:

59 $A: Y_1 = f(X_1^*)/\alpha_1 = \frac{\beta_1}{\alpha_1} \frac{[X_{11}^*]^{n_1}}{K_{11}^{n_1} + [X_{11}^*]^{n_1}}$ (1-9a)

60 $a: Y_2 = f(X_2^*)/\alpha_2 = \frac{\beta_2}{\alpha_2} \frac{[X_{22}^*]^{n_2}}{K_{22}^{n_2} + [X_{22}^*]^{n_2}}$ (1-9b)

61 With $\mu_j = \frac{\beta_j}{\alpha_j}$, then Equations 1-9a and 1-9b are transformed into:

62 $A: Y_1 = \mu_1 \frac{[X_{11}^*]^{n_1}}{K_{11}^{n_1} + [X_{11}^*]^{n_1}}$ (1-10a)

63 $a: Y_2 = \mu_2 \frac{[X_{22}^*]^{n_2}}{K_{22}^{n_2} + [X_{22}^*]^{n_2}}$ (1-10b)

64 Regarding the relationship between products of homozygotes (AA and aa) and heterozygote (Aa) of the locus, we consider three scenarios. The general assumption for three scenarios is that: (1) the ligand background concentration keeps constant among two homozygotes (representing the parents) and heterozygote (representing the F₁ hybrid); so if two alleles share the same kind of ligand background, the ligand background concentration in homozygotes and heterozygote will be [X₁₁^{*}] or [X₂₂^{*}], with [X₁₁^{*}] = [X₂₂^{*}]; if two alleles have their respective ligand backgrounds, two homozygotes and the heterozygote will maintain the same concentration of both ligands, [X₁₁^{*}] and [X₂₂^{*}]; (2) the ligand background can be equally and randomly allocated to two alleles in the homozygote, and the reaction of two alleles in heterozygote is independent⁶ and the ligand background will be allocated to two different alleles under the rule as defined in different scenarios; (3) there is a basal product m in two homozygotes and heterozygote when there is no ligand.

75 Firstly, we consider the three scenarios under the situation that the ligand works as an activator.

76 Scenario 1: null allele vs one functional allele of one polymorphic site under one ligand background

77 (**Supplementary Fig 32 and Supplementary Fig 33a-b**). The ligand background concentration in two 78 homozygotes and heterozygote will be $2[X_{11}^*] = 2[X_{22}^*] = 2[X^*]$. The product of AA, aa and Aa at 79 steady state will be:

80 $AA: Y_{11} = m + \mu_1 \frac{[X^*]^{n_1}}{K^{n_1} + [X^*]^{n_1}} + \mu_2 \frac{[X^*]^{n_2}}{K^{n_2} + [X^*]^{n_2}}$ (1-11a)

81 aa: $Y_{22} = m + 0$ (1-11b)

82 Aa: $Y_{12} = m + \mu_1 \frac{(2[X^*])^{n_1}}{K_{11}^{n_1} + (2[X^*])^{n_1}}$ (1-11c)

83 Scenario 2: two alleles of one polymorphic site under two independent backgrounds, that is, two
 84 alleles of one polymorphic site of the receptor can be bound by two respective and independent ligands as
 85 the backgrounds of the receptor (**Supplementary Fig 34** and **Supplementary Fig 36**). The ligand
 86 background concentration in two homozygotes and heterozygote will be $2[X_{11}^*]$ and $2[X_{22}^*]$, but X_{11}^*
 87 can only be allocated to allele A and X_{22}^* to allele a. The product of AA, aa and Aa at steady state will be:

88 AA: $Y_{11} = m + \mu_1 \frac{[X_{11}^*]^{n_1}}{K_{11}^{n_1} + [X_{11}^*]^{n_1}} + \mu_1 \frac{[X_{11}^*]^{n_1}}{K_{11}^{n_1} + [X_{11}^*]^{n_1}}$ (1-12a)

89 aa: $Y_{22} = m + \mu_2 \frac{[X_{22}^*]^{n_2}}{K_{22}^{n_2} + [X_{22}^*]^{n_2}} + \mu_2 \frac{[X_{22}^*]^{n_2}}{K_{22}^{n_2} + [X_{22}^*]^{n_2}}$ (1-12b)

90 Aa: $Y_{12} = m + \mu_1 \frac{(2[X_{11}^*])^{n_1}}{K_{11}^{n_1} + (2[X_{11}^*])^{n_1}} + \mu_2 \frac{(2[X_{22}^*])^{n_2}}{K_{22}^{n_2} + (2[X_{22}^*])^{n_2}}$ (1-12c)

91 Scenario 3: two alleles of one polymorphic site with shared background, that is, two alleles of one
 92 polymorphic site of the receptor can be bound by the same ligand as the background of the receptor
 93 (**Supplementary Fig 38**). The ligand background concentration in two homozygotes and heterozygote
 94 will be $2[X_{11}^*] = 2[X_{22}^*] = 2[X^*]$. If the ligand background X^* was equally allocated to each of the two
 95 alleles in heterozygote as the simulation previously reported^{6,7}, the product of AA, aa and Aa at steady
 96 state will be:

97 AA: $Y_{11} = m + \mu_1 \frac{[X^*]^{n_1}}{K_{11}^{n_1} + [X^*]^{n_1}} + \mu_1 \frac{[X^*]^{n_1}}{K_{11}^{n_1} + [X^*]^{n_1}}$ (1-10a)

98 aa: $Y_{22} = m + \mu_2 \frac{[X^*]^{n_2}}{K_{22}^{n_2} + [X^*]^{n_2}} + \mu_2 \frac{[X^*]^{n_2}}{K_{22}^{n_2} + [X^*]^{n_2}}$ (1-10b)

99 Aa: $Y_{12} = m + \mu_1 \frac{[X^*]^{n_1}}{K_{11}^{n_1} + [X^*]^{n_1}} + \mu_2 \frac{[X^*]^{n_2}}{K_{22}^{n_2} + [X^*]^{n_2}}$ (Equal allocation) (1-10c)

100 As our simulation indicated, the locus will always appear to be additive under the situation of equal
 101 allocation ($X^* = (X_{11}^* + X_{22}^*)/2$). We need consider the situations that the background X^* was allocated
 102 to two alleles in an asymmetric way due to different affinities of two alleles with the ligand background.
 103 If the ligand background X^* was allocated to each of the two alleles in heterozygote in proportion to their
 104 respective affinities, the product of Aa at steady state will be:

105 Aa: $Y_{12} = m + \mu_1 \frac{(2[X^*]K_{22}/(K_{11}+K_{22}))^{n_1}}{K_{11}^{n_1} + (2[X^*]K_{22}/(K_{11}+K_{22}))^{n_1}} + \mu_2 \frac{(2[X^*]K_{11}/(K_{11}+K_{22}))^{n_2}}{K_{22}^{n_2} + (2[X^*]K_{11}/(K_{11}+K_{22}))^{n_2}}$ (Asymmetric allocation)

106 (1-10d)

107 We also proposed an optimal strategy to maximize the output of the heterozygote. Let $S_1+S_2 = 2[X^*]$,
 108 S_1 and S_2 represent the ligand concentration allocated to allele A and a in heterozygote, respectively, when
 109 the product of heterozygote Y_{12} is maximized at the ligand concentration $2[X^*]$ (**Supplementary Fig 39-40**). The product of Aa at steady state will be:

111 Aa: $Y_{12} = m + \max \left(\mu_1 \frac{S_1^{n_1}}{K_{11}^{n_1} + S_1^{n_1}} + \mu_2 \frac{S_2^{n_2}}{K_{22}^{n_2} + S_2^{n_2}} \right)$ (Maximized allocation) (1-10e)

112 Secondly, we consider the three scenarios under the situation that the ligand works as a repressor.

113 Regarding Scenario 1, null allele vs one functional allele of one polymorphic site under one ligand
 114 background (**Supplementary Fig 33c-d**). The product of AA, aa and Aa at steady state for negative
 115 regulation will be:

116 AA: $Y_{11} = m - \mu_1 \left(1 - \frac{K^{n_1}}{K^{n_1} + [X^*]^{n_1}} \right) - \mu_1 \left(1 - \frac{K^{n_1}}{K^{n_1} + [X^*]^{n_1}} \right)$ (1-11a)

117 aa: $Y_{22} = m - 0$ (1-11b)

118 Aa: $Y_{12} = m - \mu_1 \left(1 - \frac{K^{n_1}}{K^{n_1} + (2[X^*])^{n_1}} \right)$ (1-11c)

119 Regarding Scenario 2, two alleles of one polymorphic site under two independent backgrounds
 120 (**Supplementary Fig 37**). The product of AA, aa and Aa at steady state for negative regulation will be:

121 AA: $Y_{11} = m - \mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + [X_{11}^*]^{n_1}} \right) - \mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + [X_{11}^*]^{n_1}} \right)$ (1-12a)

122 aa: $Y_{22} = m - \mu_2 \left(1 - \frac{K_{22}^{n_2}}{K_{22}^{n_2} + [X_{22}^*]^{n_2}} \right) - \mu_2 \left(1 - \frac{K_{22}^{n_2}}{K_{22}^{n_2} + [X_{22}^*]^{n_2}} \right)$ (1-12b)

123 Aa: $Y_{12} = m - \mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + (2[X_{11}^*])^{n_1}} \right) - \mu_2 \left(1 - \frac{(K_{22})^{n_2}}{K_{22}^{n_2} + (2[X_{22}^*])^{n_2}} \right)$ (1-12c)

124 Regarding Scenario 3, two alleles of one polymorphic site with shared background
 125 (**Supplementary Fig 41-42**). The product of AA, aa and Aa at steady state for negative regulation will be:

126 AA: $Y_{11} = m - \mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + [X^*]^{n_1}} \right) - \mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + [X^*]^{n_1}} \right)$ (1-13a)

127 aa: $Y_{22} = m - \mu_2 \left(1 - \frac{K_{22}^{n_2}}{K_{22}^{n_2} + [X^*]^{n_2}} \right) - \mu_2 \left(1 - \frac{K_{22}^{n_2}}{K_{22}^{n_2} + [X^*]^{n_2}} \right)$ (1-13b)

128 $Aa: Y_{12} = m - \mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + [X^*]^{n_1}} \right) - \mu_2 \left(1 - \frac{K_{22}^{n_2}}{K_{22}^{n_2} + [X^*]^{n_2}} \right)$ (Equal allocation) (1-13c)

129 Or,

130 $Aa: Y_{12} = m - \mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + (2[X^*]K_{22}/(K_{11}+K_{22}))^{n_1}} \right) - \mu_2 \left(1 - \frac{K_{22}^{n_2}}{K_{22}^{n_2} + (2[X^*]K_{11}/(K_{11}+K_{22}))^{n_2}} \right)$ (Asymmetric allocation) (1-13d)

132 Or,

133 $Aa: Y_{12} = m - \max \left(\mu_1 \left(1 - \frac{K_{11}^{n_1}}{K_{11}^{n_1} + S_1^{n_1}} \right) + \mu_2 \left(1 - \frac{K_{22}^{n_2}}{K_{22}^{n_2} + S_2^{n_2}} \right) \right)$ (Maximized allocation) (1-13e)

134 Where $S_1 + S_2 = 2[X^*]$.

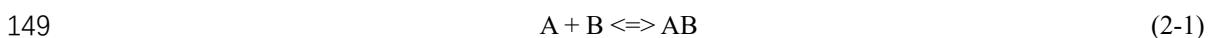
135 According the simulated values of Y_{11} , Y_{12} and Y_{22} , we calculated the degree of dominance (d/a) for
136 the locus as:

137 $d/a = (Y_{12} - (Y_{11} + Y_{22})/2) / |Y_{22} - Y_{11}|$ (1-14)

138 **2 The model reflecting the performance of homozygotes and heterozygote of one locus simulated
139 according to trimer ABA assembly**

140 The balance between genes involved in a biological complex is one important hypothesis about
141 heterosis. The typical example for gene balance was reported by Balazs and colleagues⁸. Their studies
142 indicated that mutation of the subunit in a trimer ABA complex can result in imbalance and thus is
143 harmful, which might impact gene imbalance on dominance. However, these studies did not consider the
144 effects from the counterpart background. Thus, we simulated the effects of complex background on
145 dominance of one single polymorphic locus that codes A or B.

146 In the system of trimer ABA complex, A and B are monomers, AB is the bridge dimer without active
147 function, the trimer ABA is the functional entity. The reaction among monomers, dimer and trimer could
148 be illustrated by the following chemical formula:



151 For simplicity, we consider a pseudo equilibrium state, that is: A and B were input once in an
152 enclosed environment and no degradation was considered; after a period of time, a chemical equilibrium
153 state will be achieved. Set S_A and S_B as the initial input concentration of A and B, k_{AB} as the association

rate from left to right in formula (2-1), m_{AB} as the dissociation rate from right to left of formula (2-1), k_{ABA} and m_{ABA} as the association rate from left to right and the dissociation rate from right to left in formula (2-2). And let $[A]$, $[B]$, $[AB]$ and $[ABA]$ represent the concentration of A, B, AB and ABA at equilibrium state. So we have:

$$S_A = [A] + [AB] + 2[ABA]$$

$$S_B = [B] + [AB] + [ABA]$$

$$k_{AB} \times [A][B] = m_{AB} \times [AB]$$

$$[AB] = k_{AB}/m_{AB} \times [A][B]$$

$$k_{ABA} \times [AB][A] = m_{ABA} \times [ABA]$$

$$[ABA] = k_{ABA}/m_{ABA} \times [AB][A]$$

$$[ABA] = k_{ABA}/m_{ABA} \times k_{AB}/m_{AB} [A][B][A]$$

We define association coefficient by the ratio of association to dissociation for two steps as:

$$K_1 = k_{AB}/m_{AB}$$

$$K_2 = k_{ABA}/m_{ABA}$$

then, we derived that

$$[AB] = K_1 \times [A][B]$$

$$[ABA] = K_1 \times K_2 \times [A][B][A]$$

According to the formula of stoichiometry balance, we can have:

$$S_A = [A] + K_1 \times [A][B] + 2K_1 \times K_2 \times [A][B][A]$$

$$S_B = [B] + K_1 \times [A][B] + K_1 \times K_2 \times [A][B][A]$$

Through the above equation, we get

$$[B] = S_B / (1 + K_1 \times [A] + K_1 \times K_2 \times [A]^2)$$

Then we introduced $[B]$ into S_A , we get,

$$S_A = [A] + S_B \times K_1 \times [A] / (1 + K_1 \times [A] + K_1 \times K_2 \times [A]^2) +$$

$$2 S_B \times K_1 \times K_2 \times [A]^2 / (1 + K_1 \times [A] + K_1 \times K_2 \times [A]^2)$$

We set a target function $f([A])$,

$$f([A]) = (S_A - ([A] + S_B \times K_1 \times [A] / (1 + K_1 \times [A] + K_1 \times K_2 \times [A]^2) +$$

$$2 S_B \times K_1 \times K_2 \times [A]^2 / (1 + K_1 \times [A] + K_1 \times K_2 \times [A]^2))^2$$

Among of the function of $f([A])$, only $[A]$ is the unknown parameters, S_A , S_B , K_1 and K_2 were all the predefined data, thus the value of $[A]$ that minimizes $f([A])$ is the solution of the concentration of A at

184 equilibrium state. Once the concentration of A at equilibrium state is obtained, the concentration of B, AB,
185 and ABA at equilibrium state could be easily calculated according to the above equations.

186 For given S_A , S_B , K_1 and K_2 , we solve the equation by using the optimize function in R and get the
187 concentration of A, B, AB and ABA at the equilibrium state, the solutions of parent and F_1 was follow the
188 same equations described above (**Supplementary Fig 63**).

189 We simulated two scenarios as following:

190 Scenario1, keep the input concentration of B fixed and constant among two homozygotes and the
191 heterozygote of A, and A was coded by one polymorphic locus (**Supplementary Fig 63b-c**): S_A ranges
192 from 0 to 20 nmol/L, with $S_B = 2.5$ nmol/L, $K_1 = 1$, $K_2 = 100$. The simulated data for the genotype of AA,
193 aa and Aa as follow:

194 AA: $S_{A(AA)} = 0 - 20$ nmol/L, $S_{B(AA)} = 2.5$ nmol/L

195 aa: $S_{A(aa)} = 0 - 20$ nmol/L, $S_{B(aa)} = 2.5$ nmol/L

196 Aa: $S_{A(Aa)} = (S_{A(AA)} + S_{A(aa)})/2$, $S_{B(Aa)} = 2.5$ nmol/L

197 Scenario2, keep the input concentration of A fixed and constant among two homozygotes and the
198 heterozygote of B, and B was coded by one polymorphic locus (**Supplementary Fig 63d-e**): S_B ranges
199 from 0 to 20 nmol/L, with $S_A = 5$ nmol/L, $K_1 = 1$, $K_2 = 100$. The simulated data for the genotype of AA, aa
200 and Aa as follow:

201 BB: $S_{A(BB)} = 5$ nmol/L, $S_{B(BB)} = 0 - 20$ nmol/L

202 bb: $S_{A(bb)} = 5$ nmol/L, $S_{B(bb)} = 0 - 20$ nmol/L

203 Bb: $S_{A(Bb)} = 5$ nmol/L, $S_{B(Bb)} = (S_{A(BB)} + S_{A(bb)})/2$

204 Same as the above simulation according to Hill function, according the simulated values of Y_{11} , Y_{12}
205 and Y_{22} , we calculated the degree of dominance (d/a) for the locus as:

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$$d/a = (Y_{12} - (Y_{11} + Y_{22})/2) / |Y_{22} - Y_{11}|$$

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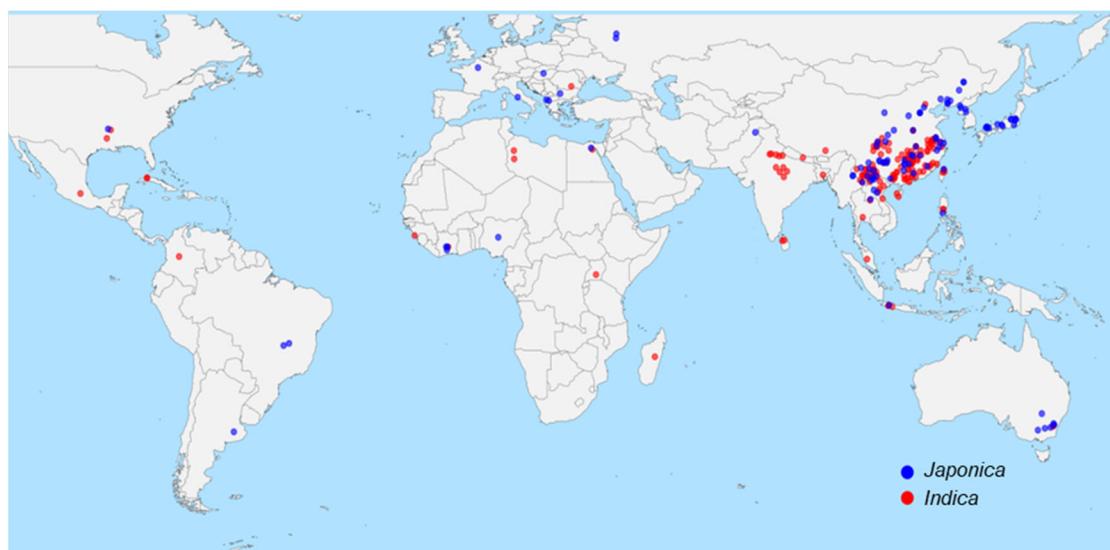
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244 **Supplementary Figures**

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247 **Supplementary Figure 1 Geographical distribution of 267 rice varieties.** The red dots represent *Indica*
248 varieties, the blue dots represent *Japonica* varieties.

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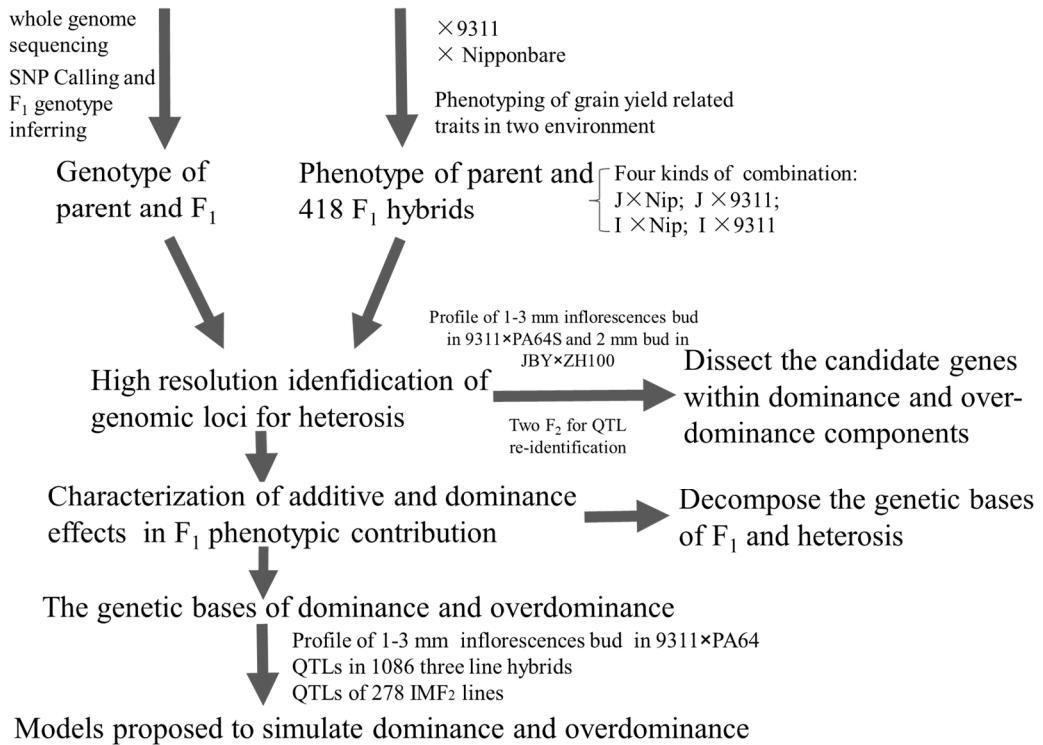
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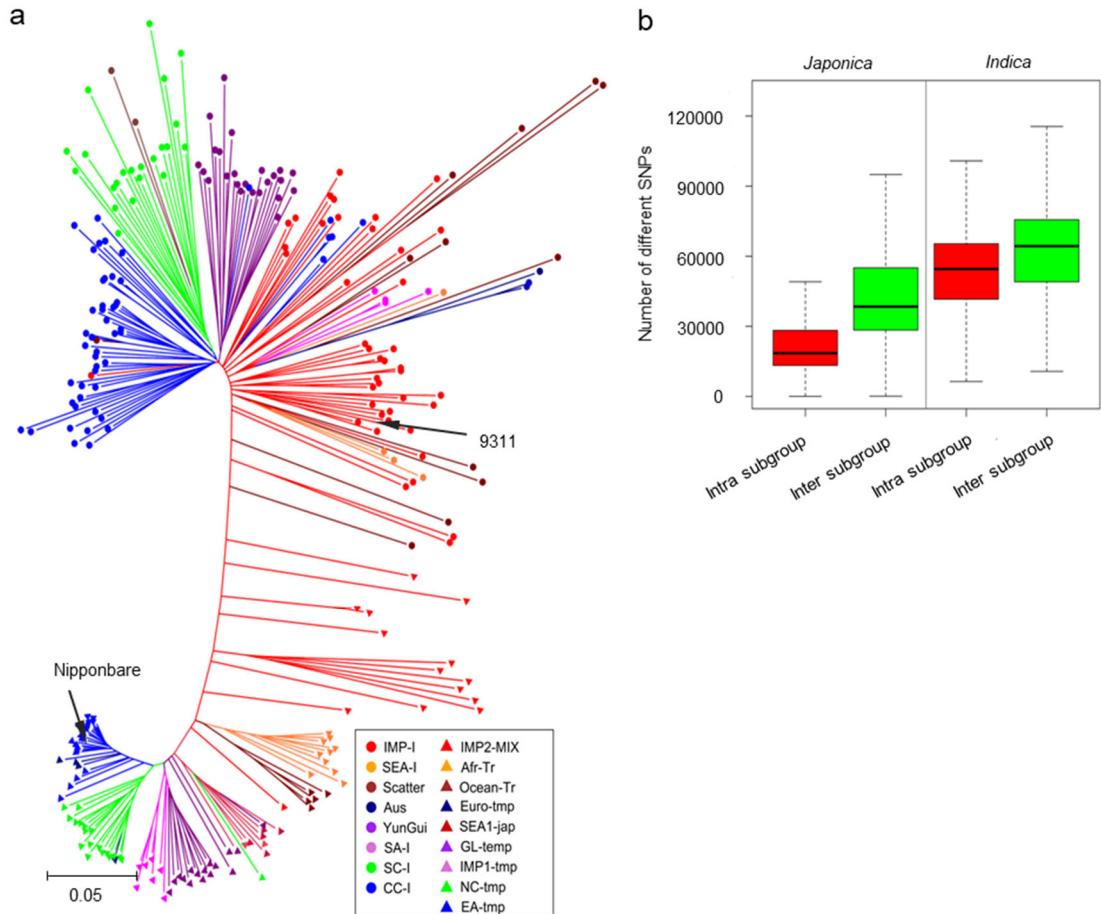
Selection of 267 MCC materials



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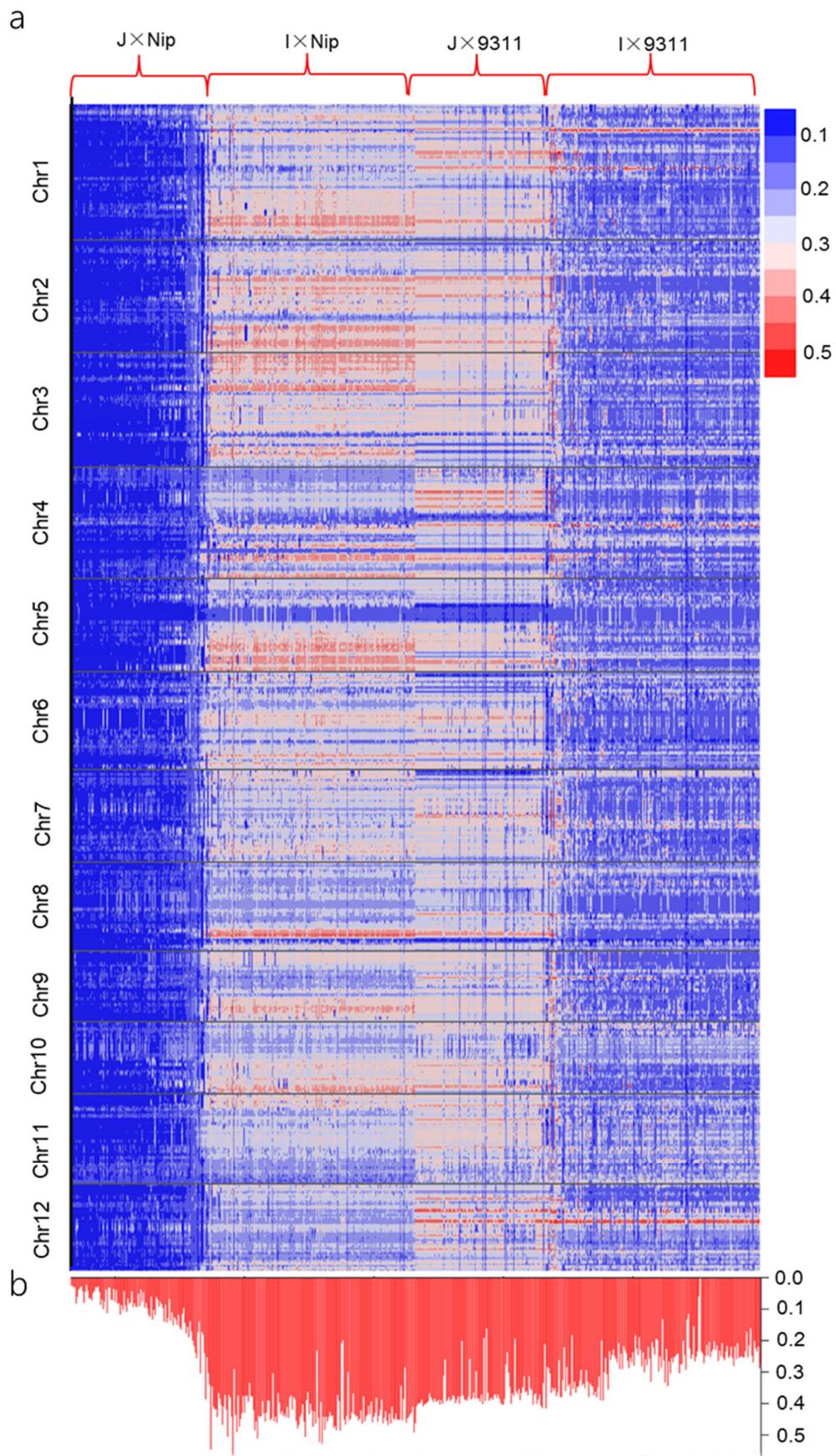
256 **Supplementary Figure 2 The experimental design and analysis procedure used in this study.**

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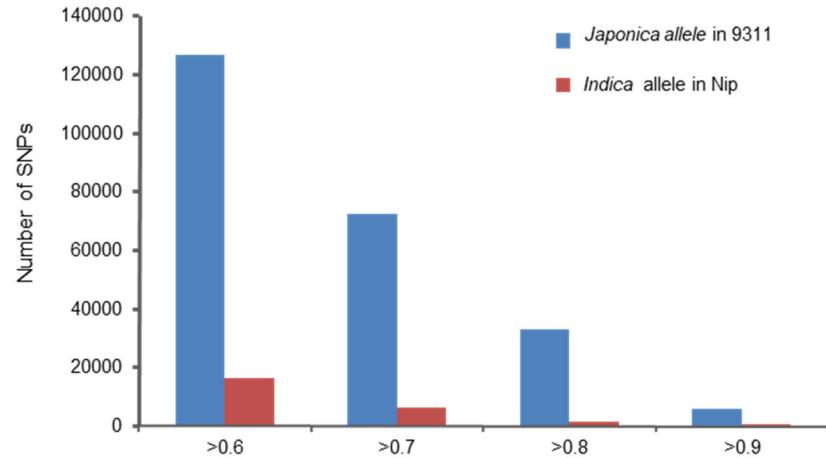
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259 **Supplementary Figure 3 Genetic structure of the 267 rice accessions.** (a) The neighbor-joining tree of
 260 267 rice accessions constructed from simple matching distance of 1.3 million SNPs. There were 8 and 9
 261 subpopulations were identified in *Indica* and *Japonica* subspecies respectively. *Indica* sub-population
 262 including: IMP-I, Improved *Indica*; SEA-I, South east Asian *Indica*; Scatter, landraces which contains the
 263 admixed fragment and several inter *Indica*-*Japonica* type materials; Aus, the aus sub population; YunGuI,
 264 Yunnan and Guizhou high altitude *Indica* from China; SA-I, South asian *Indica*; SC-I, South China
 265 *Indica*; CC-I, Center China *Indica*. *Japonica* sub-population including: IMP2-MIX, improved *Japonica*
 266 with admixed genomic fragment; Afr-Tr, Africa tropical *Japonica*. Ocean-Tr, Oceanica tropical *Japonica*.
 267 Euro-tmp, European temperate *Japonica*; SEA1-jap, Yunnan and Guizhou high altitude *Japonica* lines
 268 that subjected to Southeast Asian subtropical *Japonica*; GL-tmp, Yunnan and Guizhou high altitude
 269 Glutinous *Japonica*; IMP1-tmp, improved temperate *Japonica* lines; NC-tmp, north china temperate
 270 *Japonica*; EA-tmp from China, east asian temperate *Japonica*. (b) The average number of SNPs that
 271 different between pairwise individual of intra and inter sub-population in *Japonica* and *Indica*.

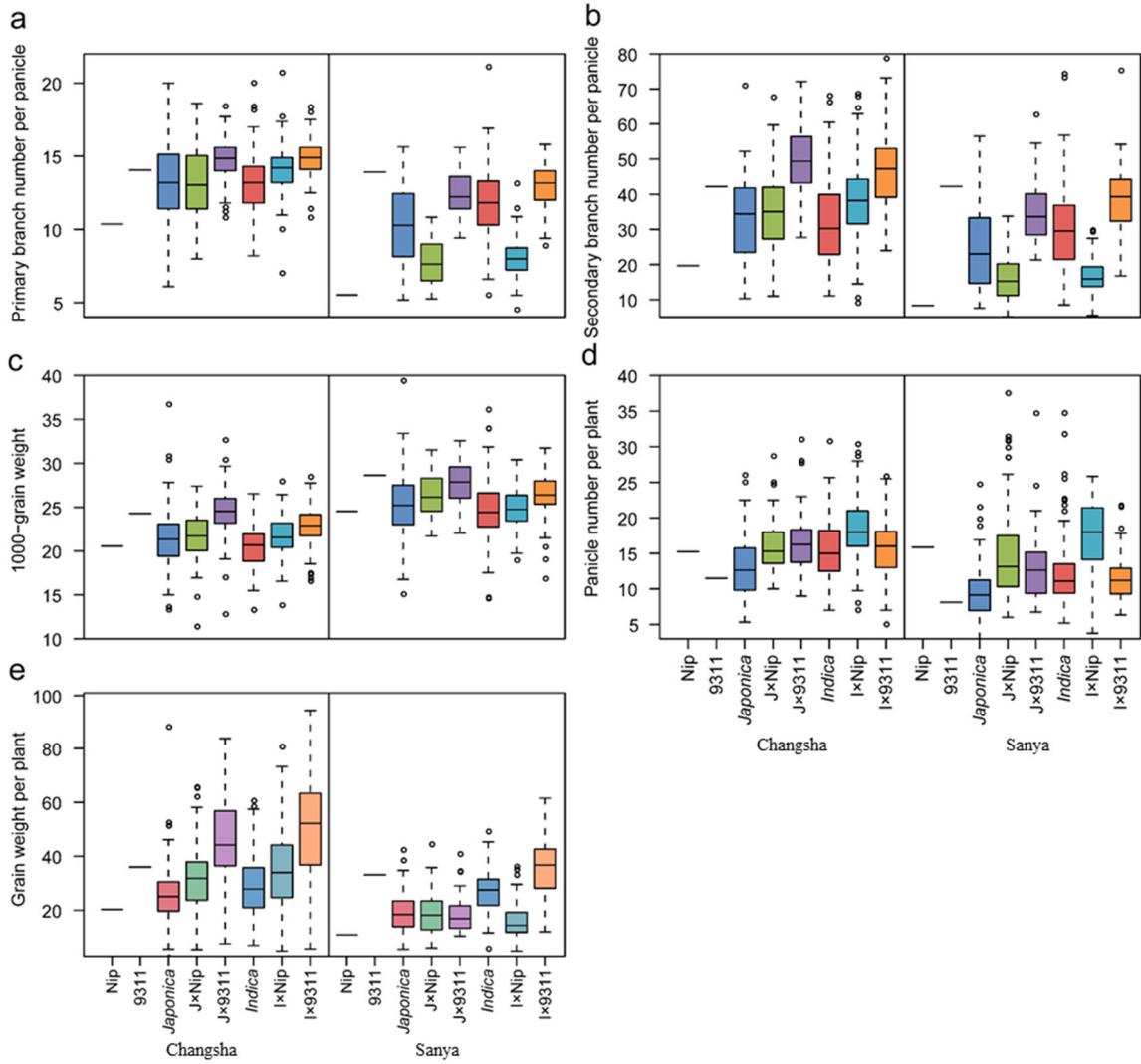


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273 **Supplementary Figure 4 Genome-wide heterozygosity for different kinds of combinations.** (a) The



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 281 **Supplementary Figure 5 Distribution of *Japonica* specific alleles in 9311 and *Indica* specific alleles**
 282 **in Nipponbare.** The total number of *Japonica* specific alleles that introgressed into 9311 (blue bar), and
 283 that of *Indica* specific alleles that introgressed into Nipponbare (red bar). >0.6, >0.7, >0.8 and >0.9 mean
 284 that the allele frequency in one subspecies are higher than 0.6, 0.7, 0.8 and 0.9 but equal and less than 0.4,
 285 0.3, 0.2 and 0.1 in the other subspecies.



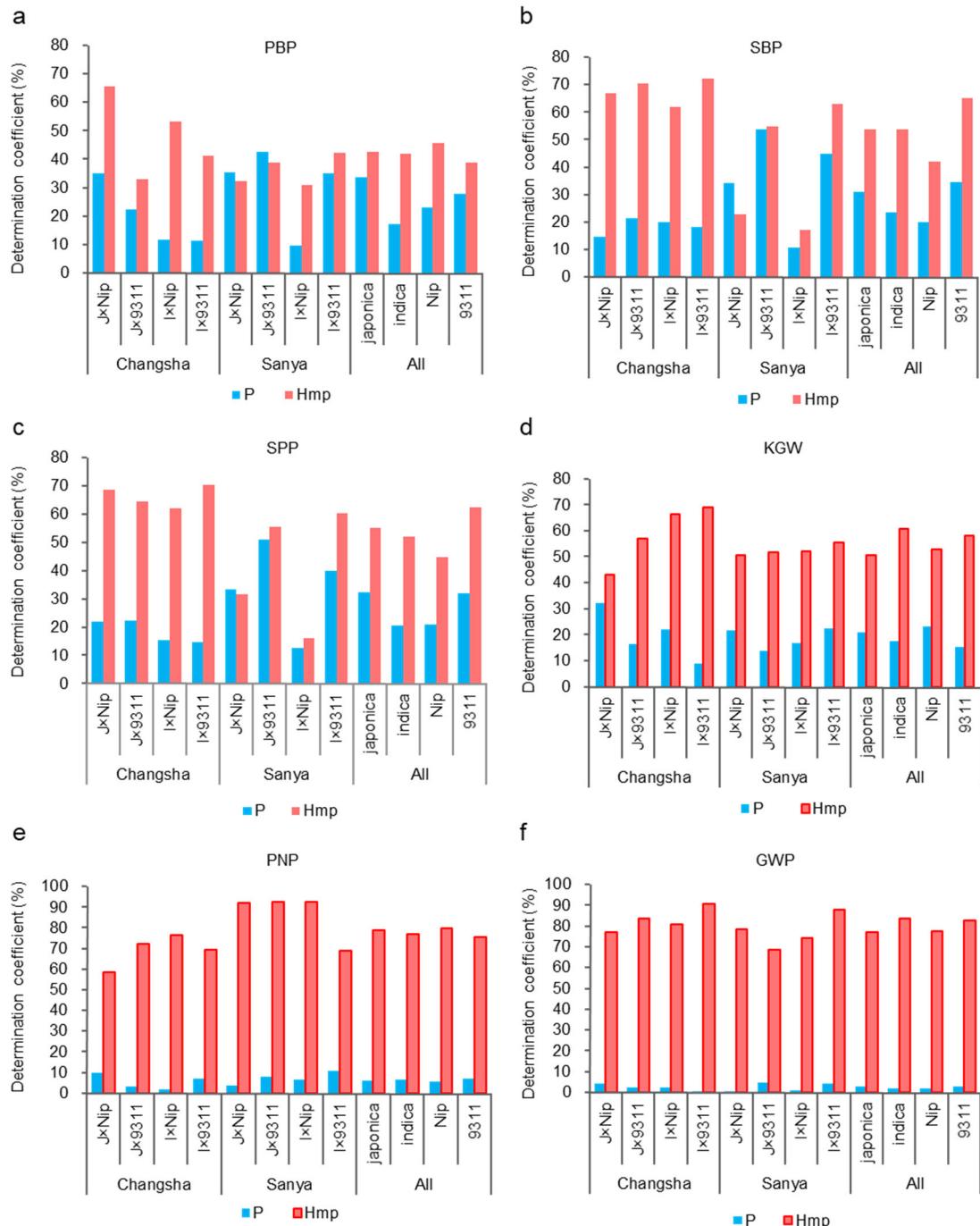
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Supplementary Figure 6 The phenotype distribution of parent and F₁ for different yield traits in Changsha and Sanya. Nip, Nipponbare; J×Nip, combination of Japonica and Nipponbare; I×Nip, combination of Indica and Nipponbare, and the others are similar.

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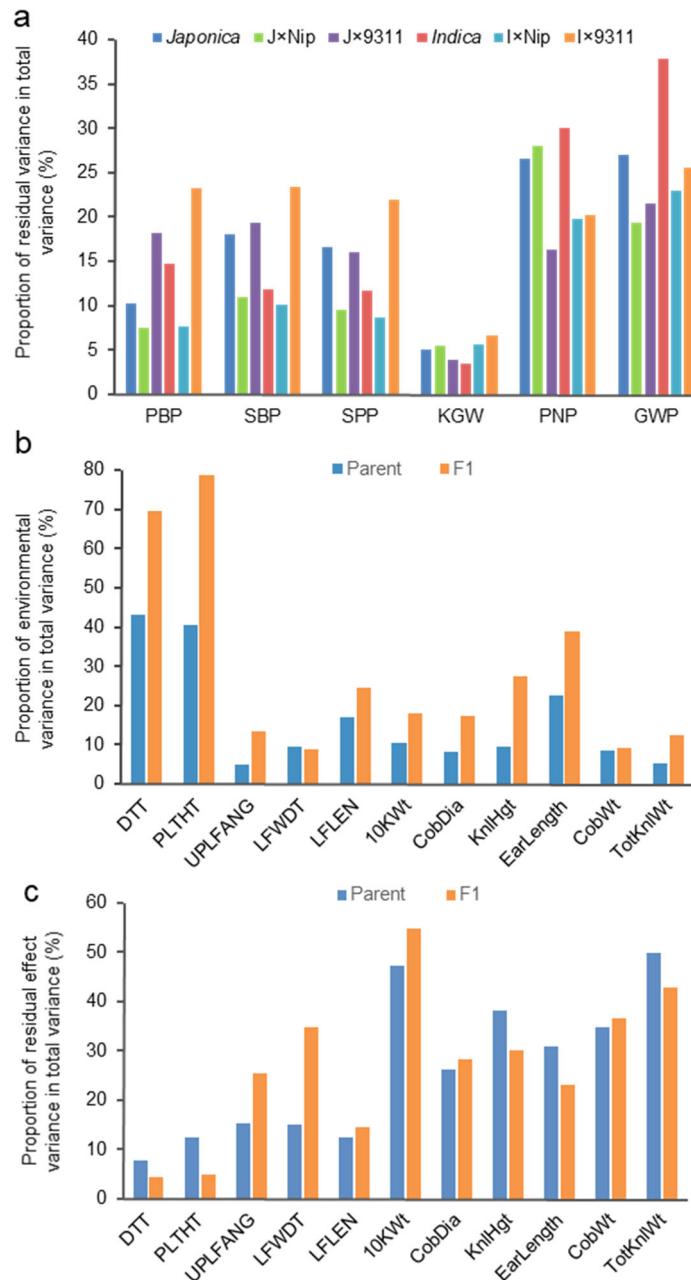
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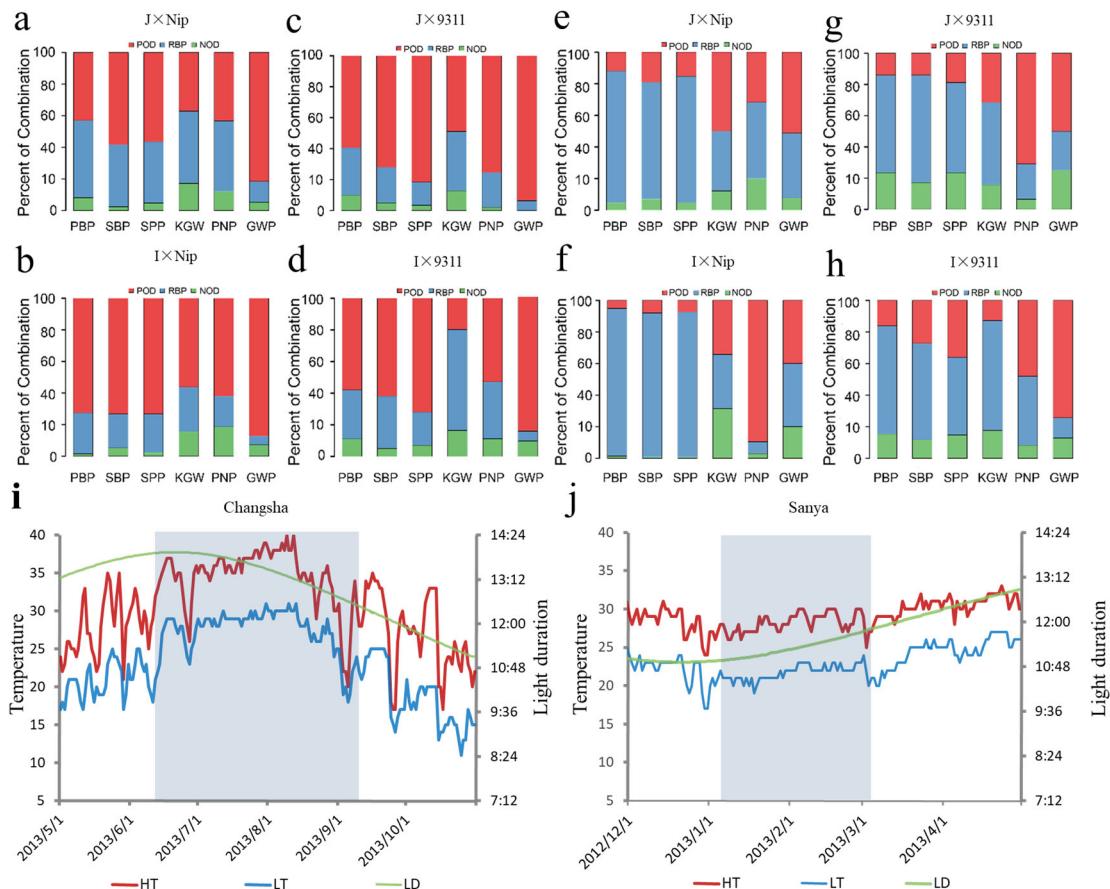
291 **Supplementary Figure 7 Phenotypic contribution of parental inbred genetic basis (P) and middle**
 292 **parent heterosis (Hmp) to hybrids.** PBP, primary branch number per panicle; SBP, secondary branch
 293 number per panicle; SPP, spikelet number per panicle; KGW, 1000-grain weight; PNP, panicle number
 294 per plant; GWP, grain weight per plant; all means all combinations for *Japonica*, *Indica*, *Nipponbare* and
 295 9311 in both environments.

296



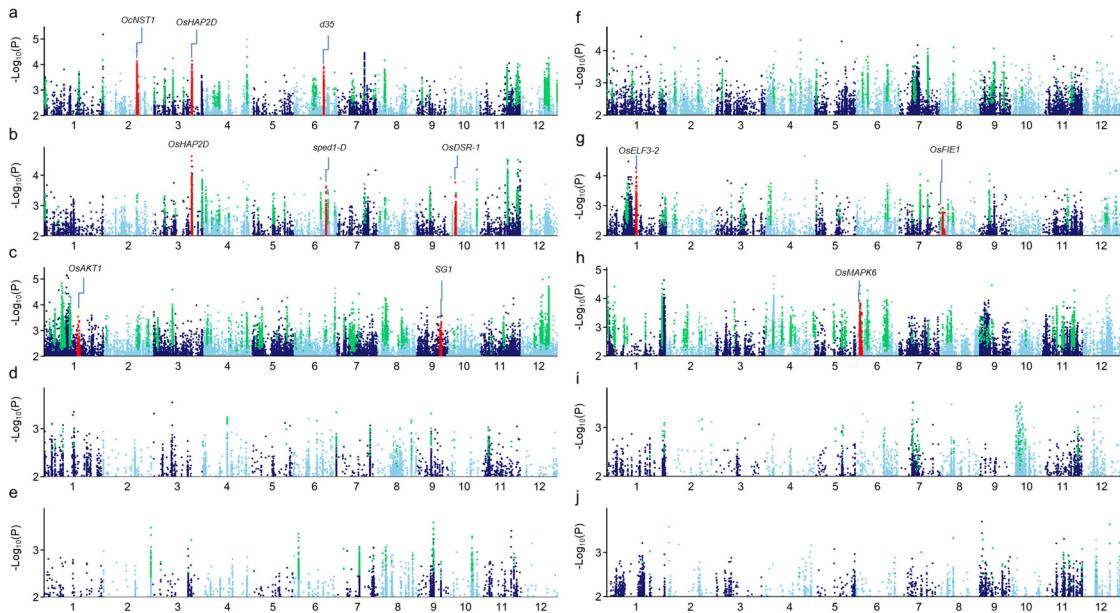
297

298 **Supplementary Figure 8 Supplementary Figure 8 Residual and environmental variance of yield**
 299 **traits in inbred parents and hybrids.** (a) The proportion of residual variance of each yield trait
 300 estimated in inbred parents and hybrid in rice. (b) Proportion of environmental variance in total variance
 301 for yield related traits in panels of inbred parents and hybrids in maize (data were collected from Flint-
 302 Garcia., Buckler. et al. 2009). (c) The proportion of residual variance of each yield trait estimated in
 303 parents and hybrid in maize. DTT, Days to anthesis; PLTHT, Plant height; UPLFANG, Upper leaf angle;
 304 LFWDT, Leaf width; LFLEN, leaf length; 10Kwt, 10 kernel weight; CobDia, cob diameter; KnHgt,
 305 kernel height; EarLength, Ear length; CobWt, Cob Weight; TolKnWt, Total Kernel Weight.



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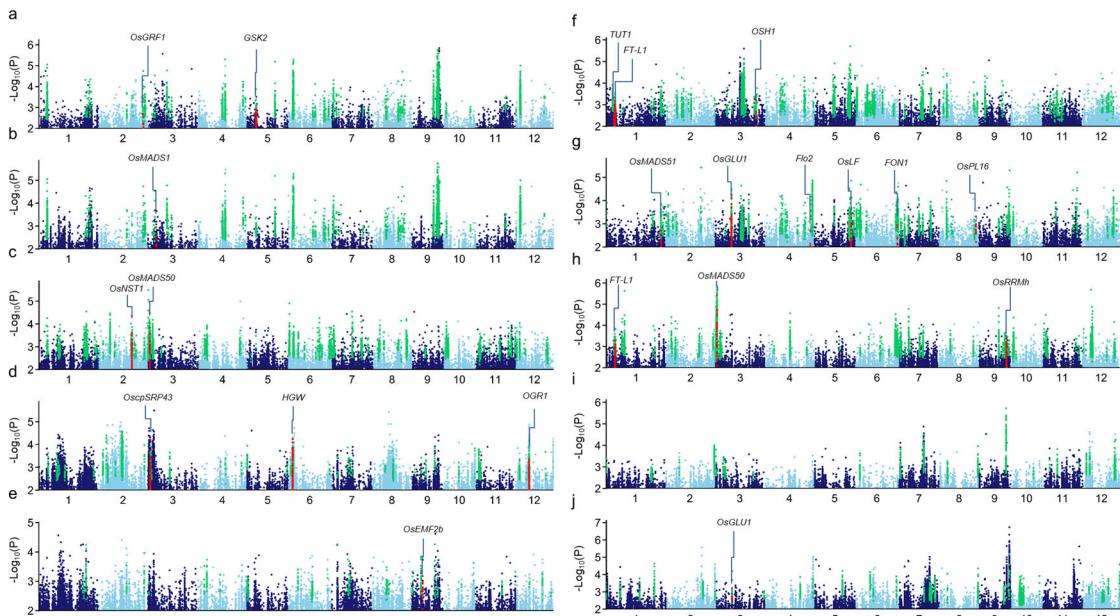
307 **Supplementary Figure 9 The distribution of combinations showing POD, RBP and NOD phenotype**
 308 **for different yield traits and different kinds of combinations under two environments.** (a-d) The
 309 distribution in Changsha. (e-f) The distribution in Sanya. (i) The temperature and light duration during the
 310 growth season in Changsha. (j) The temperature and light duration during the growth season in Sanya.
 311 POD, F_1 showing phenotype over the higher parent, referred as positive over-dominant (POD); RBP, F_1
 312 showing phenotype ranging between parents referred as RBP; NOD, F_1 showing phenotype below the
 313 lower parent, referred as negative over-dominant (NOD). The grey boxes indicated the stage from
 314 reproductive initiation to grain filling. HT, high temperature; LT, low temperature; LD, light duration.



315

316 **Supplementary Figure 10 Genome-wide association study of primary branch number per panicle**
 317 **(PBP) in Japonica parents and their combinations using compressed MLM.** (a) Manhattan plots
 318 for J×Nip F₁ phenotype in Changsha. (b) Manhattan plots for J×Nip mid-parent heterosis value
 319 in Changsha. (c) Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for
 320 J×9311 F₁ phenotype in Changsha. (e) Manhattan plots for J×9311 mid-parent heterosis value in
 321 Changsha. (f) Manhattan plots for J×Nip F₁ phenotype in Sanya. (g) Manhattan plots for J×Nip
 322 mid-parent heterosis value in Sanya. (h) Manhattan plots for phenotype of parents in Sanya. (i)
 323 Manhattan plots for J×9311 F₁ in Sanya. (j) Manhattan plots for J×9311 mid-parent heterosis in
 324 Sanya. Green dots represent the significant SNPs in identified QTLs, red dots represent the
 325 SNPs in cloned genes with 2 kb promoter.

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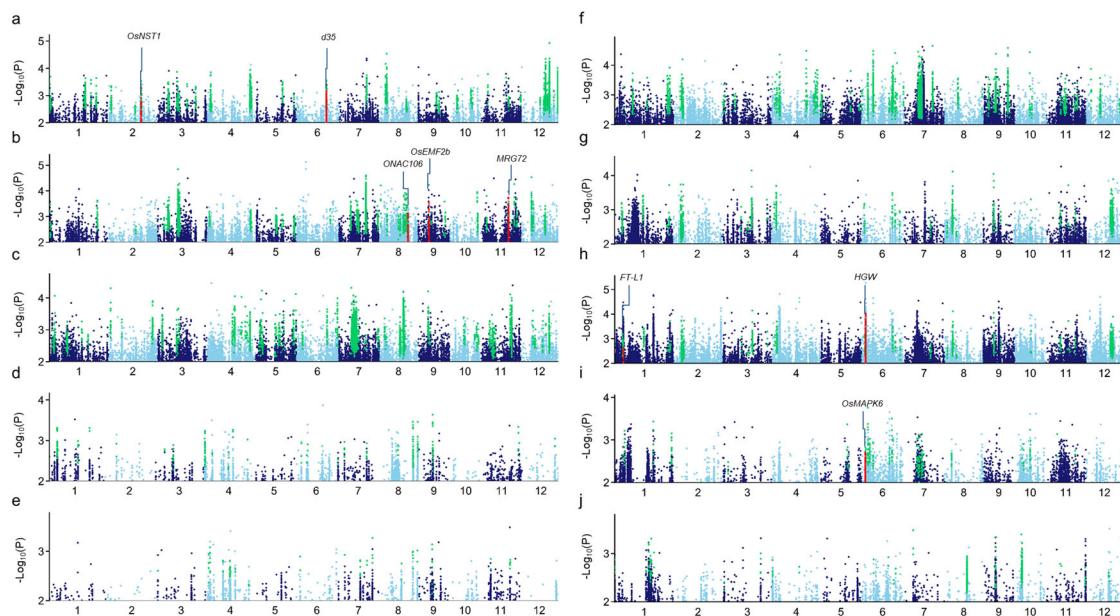


327

328 **Supplementary Figure 11 Genome-wide association study of primary branch number per panicle**

329 **(PBP) in Indica parents and their combinations using compressed MLM.** (a) Manhattan plots for
330 I×Nip F₁ phenotype in Changsha. (b) Manhattan plots for I×Nip mid-parent heterosis value in
331 Changsha. (c) Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for
332 I×9311 F₁ phenotype in Changsha. (e) Manhattan plots for I×9311 mid-parent heterosis value in
333 Changsha. (f) Manhattan plots for I×Nip F₁ phenotype in Sanya. (g) Manhattan plots for I×Nip
334 mid-parent heterosis value in Sanya. (h) Manhattan plots for phenotype of parents in Sanya. (i)
335 Manhattan plots for I×9311 F₁ in Sanya. (j) Manhattan plots for I×9311 mid-parent heterosis in
336 Sanya. Green dots represent the significant SNPs in identified QTLs, red dots represent the
337 SNPs in cloned genes with 2 kb promoter.

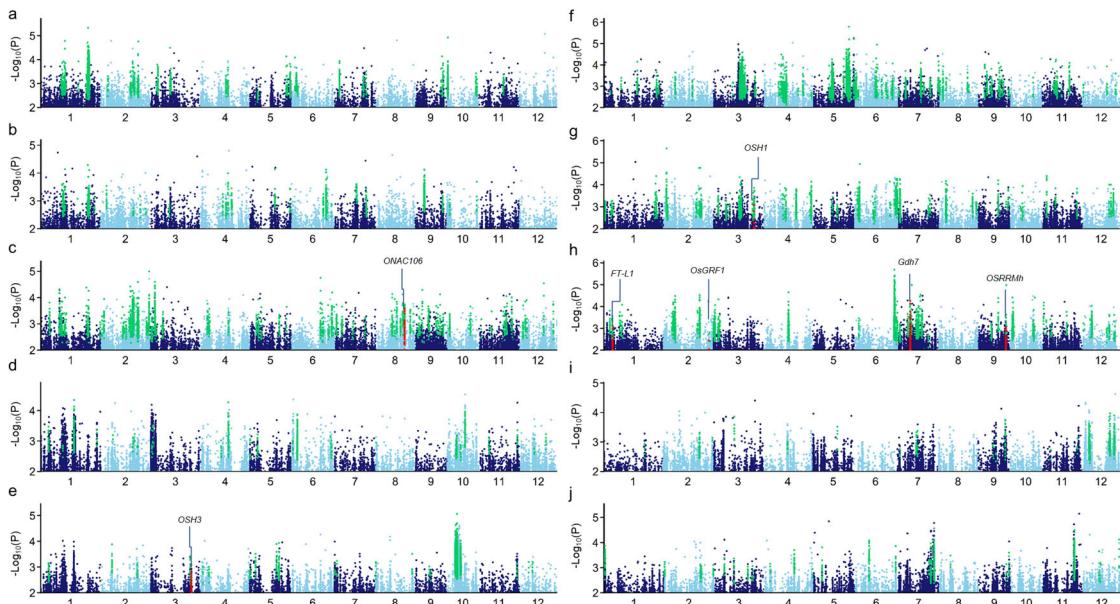
338



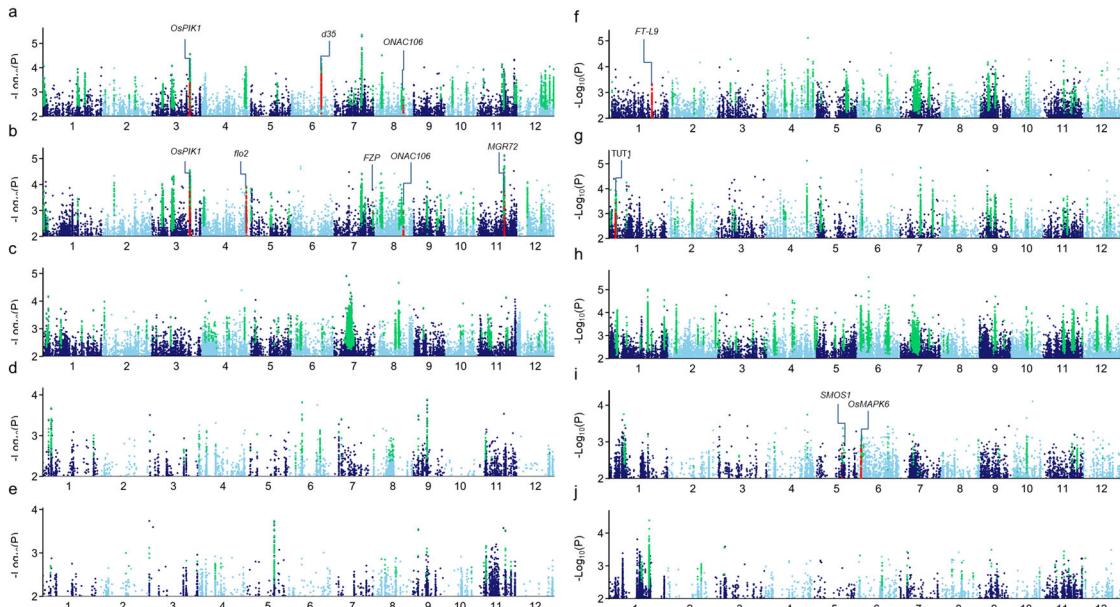
339

340 **Supplementary Figure 12 Genome-wide association study of secondary branch number per panicle**
341 **(SBP) in Japonica parents and their combinations using compressed MLM.** (a) Manhattan plots for
342 J×Nip F₁ phenotype in Changsha. (b) Manhattan plots for J×Nip mid-parent heterosis value in Changsha.
343 (c) Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for J×9311 F₁ phenotype
344 in Changsha. (e) Manhattan plots for J×9311 mid-parent heterosis value in Changsha. (f) Manhattan plots
345 for J×Nip F₁ phenotype in Sanya. (g) Manhattan plots for J×Nip mid-parent heterosis value in Sanya. (h)
346 Manhattan plots for phenotype of parents in Sanya. (i) Manhattan plots for J×9311 F₁ in Sanya. (j)
347 Manhattan plots for J×9311 mid-parent heterosis in Sanya. Green dots represent the significant SNPs in
348 identified QTLs, red dots represent the SNPs in cloned genes with 2 kb promoter.

349



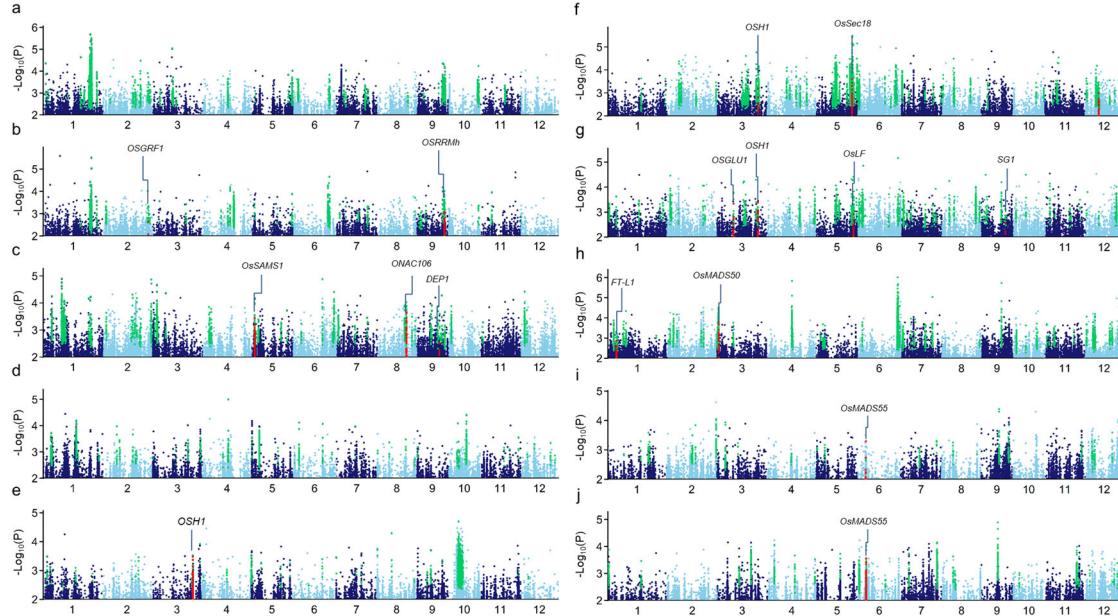
350
351 **Supplementary Figure 13 Genome-wide association study of secondary branch number per panicle**
352 **(SBP) in Indica parents and their combinations using compressed MLM.** (a) Manhattan plots for
353 I×Nip F₁ phenotype in Changsha. (b) Manhattan plots for I×Nip mid-parent heterosis value in
354 Changsha. (c) Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for
355 I×9311 F₁ phenotype in Changsha. (e) Manhattan plots for I×9311 mid-parent heterosis value in
356 Changsha. (f) Manhattan plots for I×Nip F₁ phenotype in Sanya. (g) Manhattan plots for I×Nip
357 mid-parent heterosis value in Sanya. (h) Manhattan plots for phenotype of parents in Sanya. (i)
358 Manhattan plots for I×9311 F₁ in Sanya. (j) Manhattan plots for I×9311 mid-parent heterosis in
359 Sanya. Green dots represent the significant SNPs in identified QTLs, red dots represent the
360 SNPs in cloned genes with 2 kb promoter.
361



362
363 **Supplementary Figure 14 Genome-wide association study of spikelet number per panicle (SPP) in**

364 **Japonica parents and their combinations using compressed MLM.** (a) Manhattan plots for J×Nip F₁
 365 phenotype in Changsha. (b) Manhattan plots for J×Nip mid-parent heterosis value in Changsha. (c)
 366 Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for J×9311 F₁ phenotype in
 367 Changsha. (e) Manhattan plots for J×9311 mid-parent heterosis value in Changsha. (f) Manhattan plots
 368 for J×Nip F₁ phenotype in Sanya. (g) Manhattan plots for J×Nip mid-parent heterosis value in Sanya. (h)
 369 Manhattan plots for phenotype of parents in Sanya. (i) Manhattan plots for J×9311 F₁ in Sanya. (j)
 370 Manhattan plots for J×9311 mid-parent heterosis in Sanya. Green dots represent the significant SNPs in
 371 identified QTLs, red dots represent the SNPs in cloned genes with 2 kb promoter.

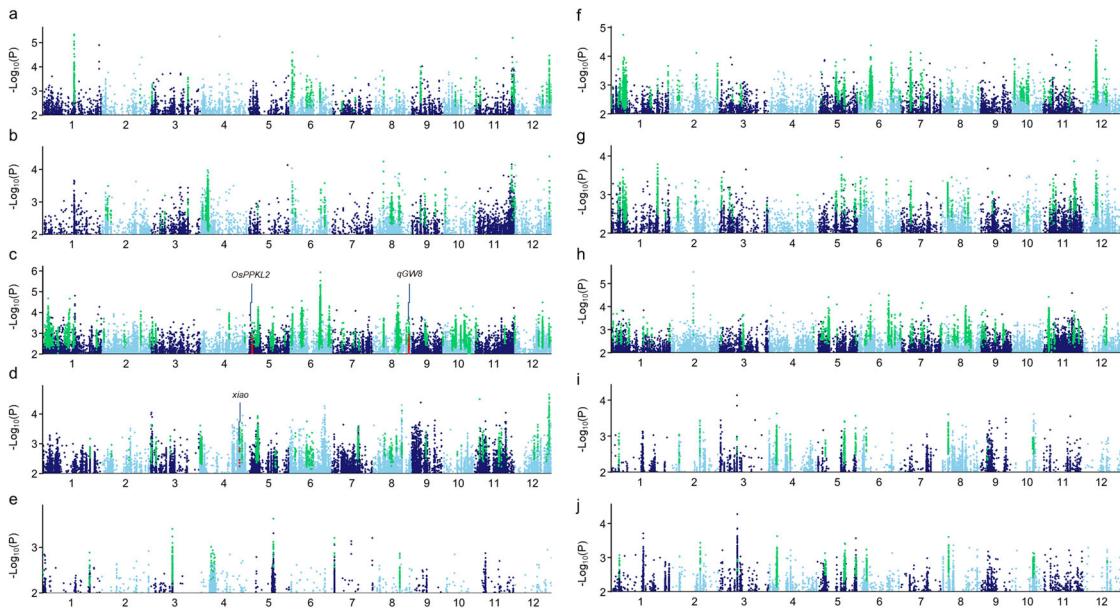
372



373

374 **Supplementary Figure 15 Genome-wide association studies of spikelet number per panicle (SPP) in**
 375 **Indica parents and their combinations using compressed MLM.** (a) Manhattan plots for I×Nip F₁
 376 phenotype in Changsha. (b) Manhattan plots for I×Nip mid-parent heterosis value in Changsha.
 377 (c) Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for I×9311 F₁
 378 phenotype in Changsha. (e) Manhattan plots for I×9311 mid-parent heterosis value in Changsha.
 379 (f) Manhattan plots for I×Nip F₁ phenotype in Sanya. (g) Manhattan plots for I×Nip mid-parent
 380 heterosis value in Sanya. (h) Manhattan plots for phenotype of parents in Sanya. (i) Manhattan
 381 plots for I×9311 F₁ in Sanya. (j) Manhattan plots for I×9311 mid-parent heterosis in Sanya.
 382 Green dots represent the significant SNPs in identified QTLs, red dots represent the SNPs in
 383 cloned genes with 2 kb promoter.

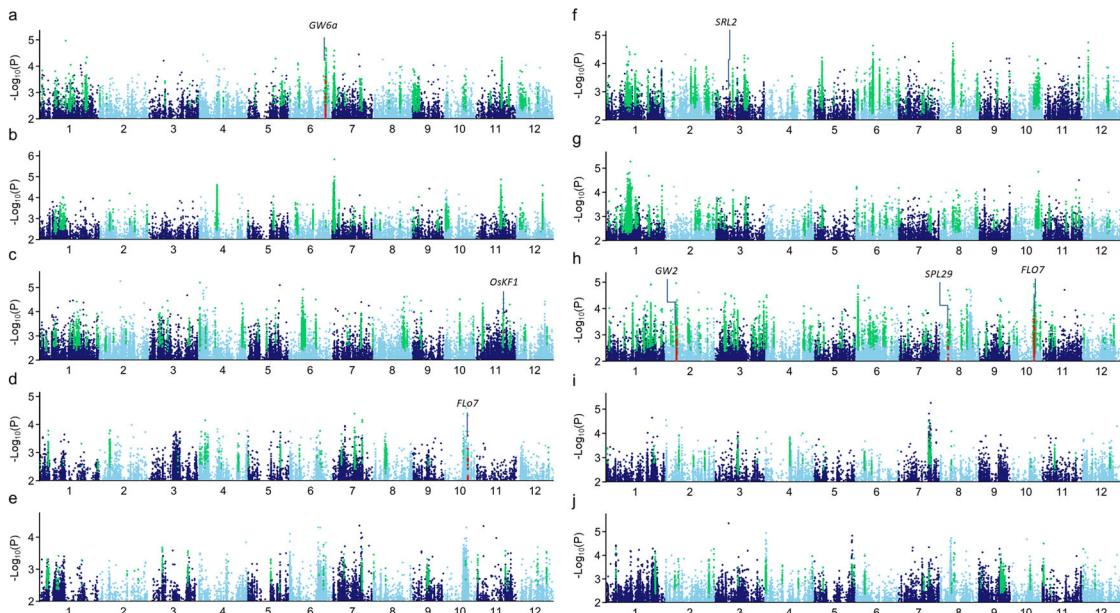
384



385

386 **Supplementary Figure 16 Genome-wide association study of 1000-grain weight (KGW) in *Japonica***
 387 **parents and their combinations using compressed MLM.** (a) Manhattan plots for J×Nip F1 phenotype
 388 in Changsha. (b) Manhattan plots for J×Nip mid-parent heterosis value in Changsha. (c) Manhattan plots
 389 for phenotype of parents in Changsha. (d) Manhattan plots for J×9311 F1 phenotype in Changsha. (e)
 390 Manhattan plots for J×9311 mid-parent heterosis value in Changsha. (f) Manhattan plots for J×Nip F1
 391 phenotype in Sanya. (g) Manhattan plots for J×Nip mid-parent heterosis value in Sanya. (h) Manhattan
 392 plots for phenotype of parents in Sanya. (i) Manhattan plots for J×9311 F1 in Sanya. (j) Manhattan plots
 393 for J×9311 mid-parent heterosis in Sanya. Green dots represent the significant SNPs in identified QTLs,
 394 red dots represent the SNPs in cloned genes with 2 kb promoter.

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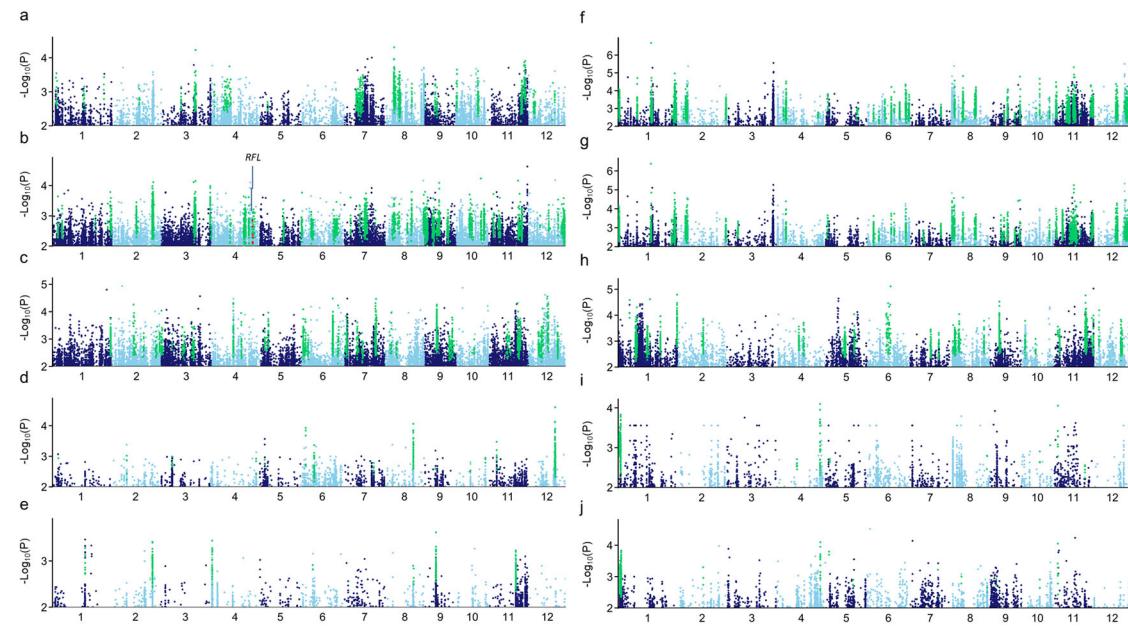


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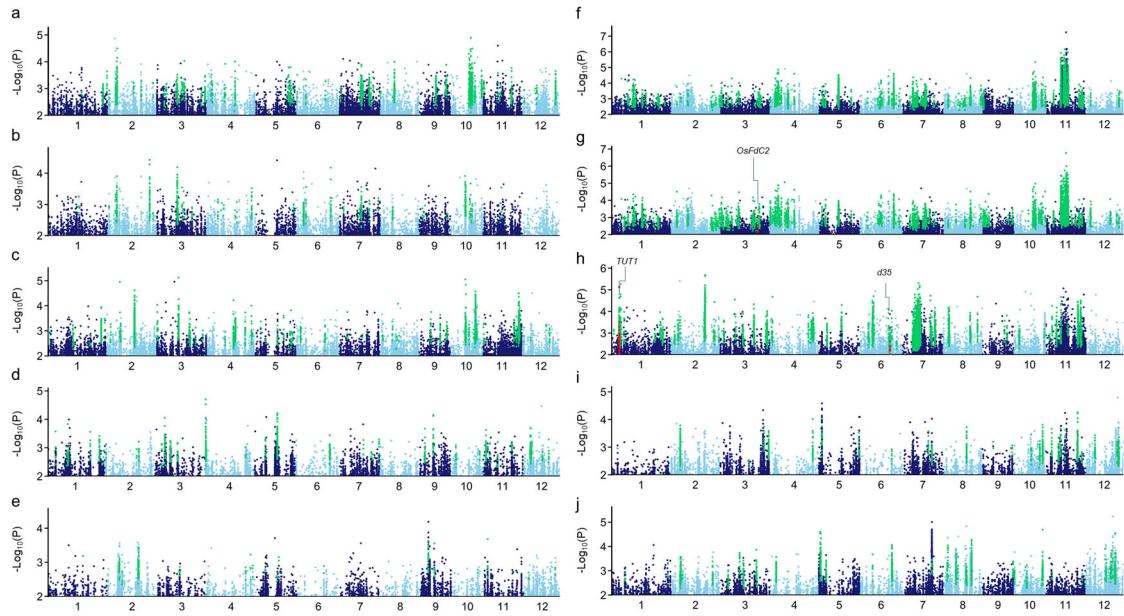
397 **Supplementary Figure 17 Genome-wide association study of 1000-grain weight (KGW) in *Indica***
 398 **parents and their combinations using compressed MLM.** (a) Manhattan plots for I×Nip F1 phenotype
 399 in Changsha. (b) Manhattan plots for I×Nip mid-parent heterosis value in Changsha.

400 (c) Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for I×9311 F₁
 401 phenotype in Changsha. (e) Manhattan plots for I×9311 mid-parent heterosis value in Changsha.
 402 (f) Manhattan plots for I×Nip F₁ phenotype in Sanya. (g) Manhattan plots for I×Nip mid-parent
 403 heterosis value in Sanya. (h) Manhattan plots for phenotype of parents in Sanya. (i) Manhattan
 404 plots for I×9311 F₁ in Sanya. (j) Manhattan plots for I×9311 mid-parent heterosis in Sanya.
 405 Green dots represent the significant SNPs in identified QTLs, red dots represent the SNPs in
 406 cloned genes with 2 kb promoter.

407



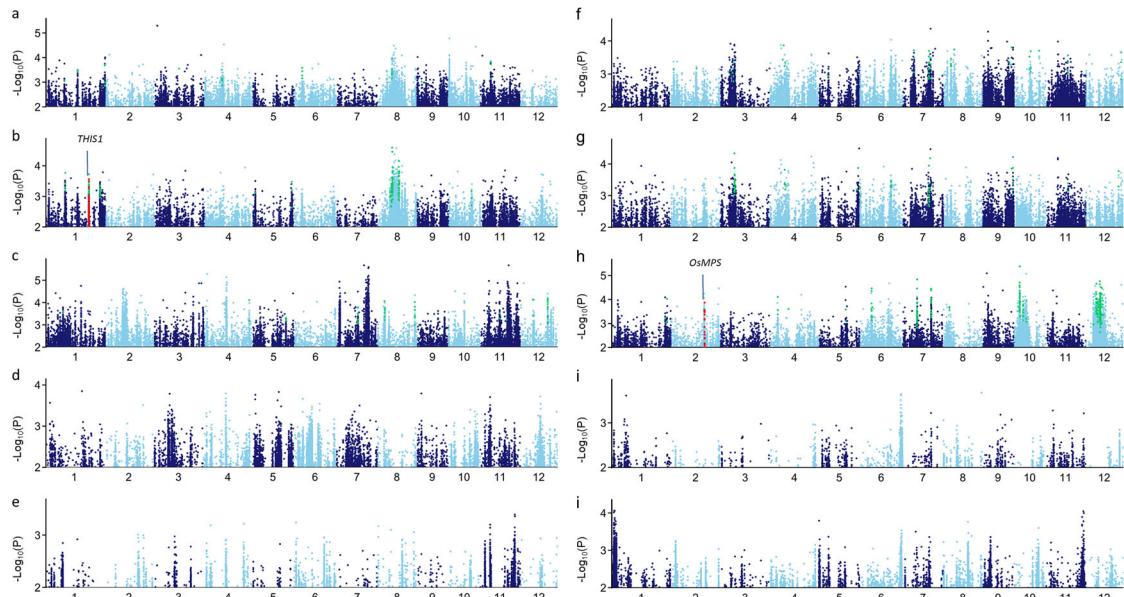
408
 409 **Supplementary Figure 18 Genome-wide association study of panicle number per plant (PNP) in**
 410 ***Japonica* parents and their combinations using compressed MLM.** (a) Manhattan plots for J×Nip F₁
 411 phenotype in Changsha. (b) Manhattan plots for J×Nip mid-parent heterosis value in Changsha. (c)
 412 Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for J×9311 F₁ phenotype in
 413 Changsha. (e) Manhattan plots for J×9311 mid-parent heterosis value in Changsha. (f) Manhattan plots
 414 for J×Nip F₁ phenotype in Sanya. (g) Manhattan plots for J×Nip mid-parent heterosis value in Sanya. (h)
 415 Manhattan plots for phenotype of parents in Sanya. (i) Manhattan plots for J×9311 F₁ in Sanya. (j)
 416 Manhattan plots for J×9311 mid-parent heterosis in Sanya. Green dots represent the significant SNPs in
 417 identified QTLs, red dots represent the SNPs in cloned genes with 2 kb promoter.



418

419 **Supplementary Figure 19 Genome-wide association study of panicle number per plant (PNP) in**
 420 ***Indica* parents and their combinations using compressed MLM.** (a) Manhattan plots for I×Nip F₁
 421 phenotype in Changsha. (b) Manhattan plots for I×Nip mid-parent heterosis value in Changsha. (c)
 422 Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for I×9311 F₁ phenotype in
 423 Changsha. (e) Manhattan plots for I×9311 mid-parent heterosis value in Changsha. (f) Manhattan plots
 424 for I×Nip F₁ phenotype in Sanya. (g) Manhattan plots for I×Nip mid-parent heterosis value in Sanya. (h)
 425 Manhattan plots for phenotype of parents in Sanya. (i) Manhattan plots for I×9311 F₁ in Sanya. (j)
 426 Manhattan plots for I×9311 mid-parent heterosis in Sanya. Green dots represent the significant SNPs in
 427 identified QTLs, red dots represent the SNPs in cloned genes with 2 kb promoter.

428

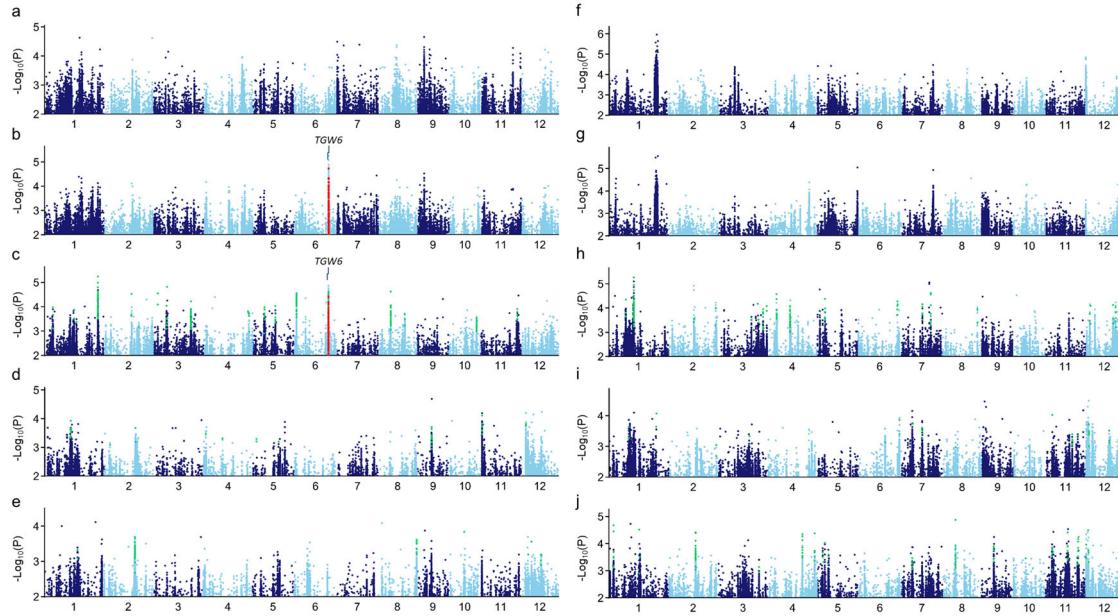


429

430 **Supplementary Figure 20 Genome-wide association study of grain weight per plant (GWP) in**
 431 ***Japonica* parents and their combinations using compressed MLM.** (a) Manhattan plots for J×Nip F₁
 432 phenotype in Changsha. (b) Manhattan plots for J×Nip mid-parent heterosis value in Changsha. (c)

433 Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for J×9311 F₁ phenotype in
434 Changsha. (e) Manhattan plots for J×9311 mid-parent heterosis value in Changsha. (f) Manhattan plots
435 for J×Nip F₁ phenotype in Sanya. (g) Manhattan plots for J×Nip mid-parent heterosis value in Sanya. (h)
436 Manhattan plots for phenotype of parents in Sanya. (i) Manhattan plots for J×9311 F₁ in Sanya. (j)
437 Manhattan plots for J×9311 mid-parent heterosis in Sanya. Green dots represent the significant SNPs in
438 identified QTLs, red dots represent the SNPs in cloned genes with 2 kb promoter.

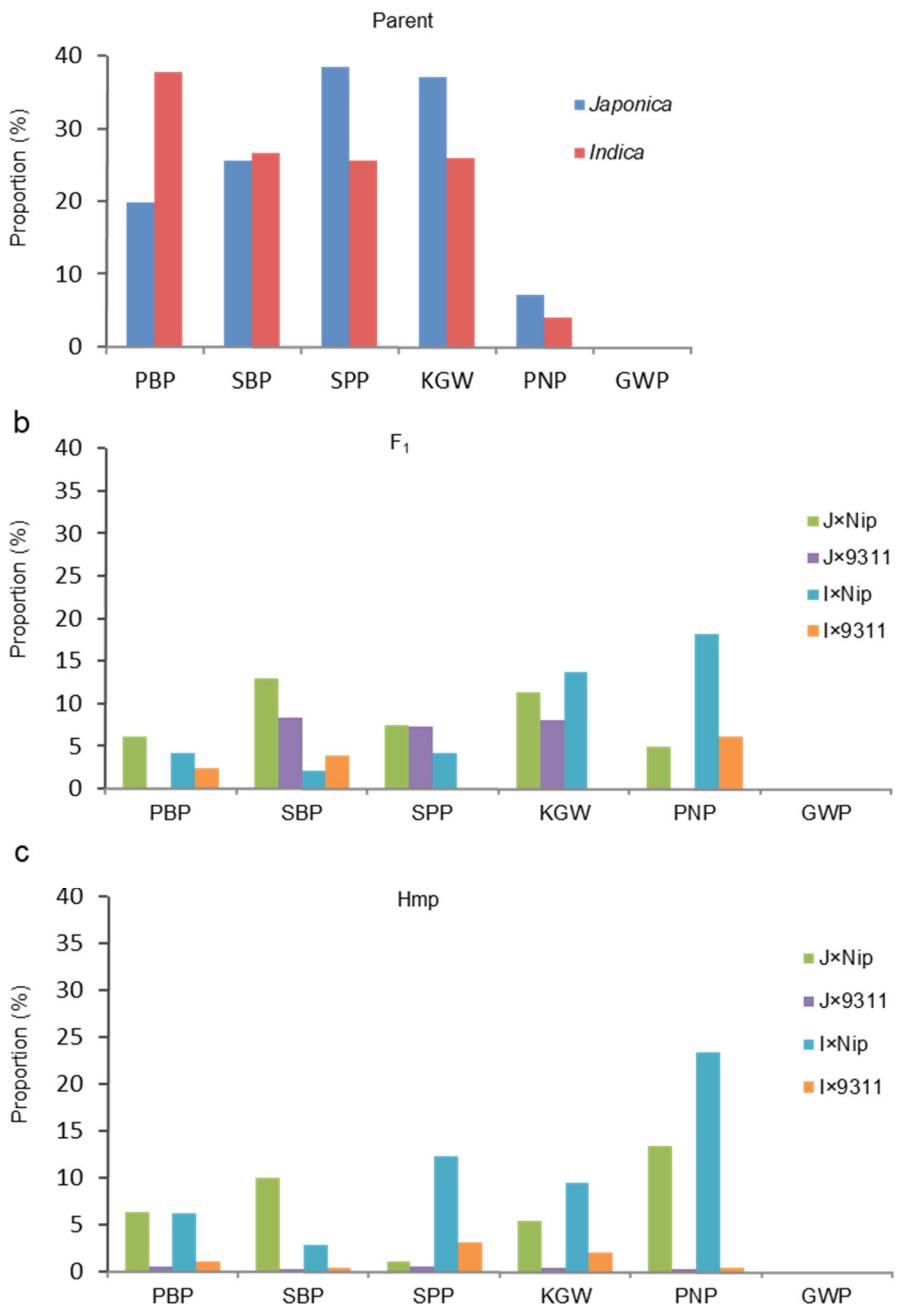
439



440

441 **Supplementary Figure 21 Genome-wide association study of grain weight per plant (GWP) in**
442 ***Indica* parents and their combinations using compressed MLM.** (a) Manhattan plots for I×Nip F₁
443 phenotype in Changsha. (b) Manhattan plots for I×Nip mid-parent heterosis value in Changsha. (c)
444 Manhattan plots for phenotype of parents in Changsha. (d) Manhattan plots for I×9311 F₁ phenotype in
445 Changsha. (e) Manhattan plots for I×9311 mid-parent heterosis value in Changsha. (f) Manhattan plots for
446 I×Nip F₁ phenotype in Sanya. (g) Manhattan plots for I×Nip mid-parent heterosis value in Sanya. (h)
447 Manhattan plots for phenotype of parents in Sanya. (i) Manhattan plots for I×9311 F₁ in Sanya. (j)
448 Manhattan plots for I×9311 mid-parent heterosis in Sanya. Green dots represent the significant SNPs in
449 identified QTLs, red dots represent the SNPs in cloned genes with 2 kb promoter.

450

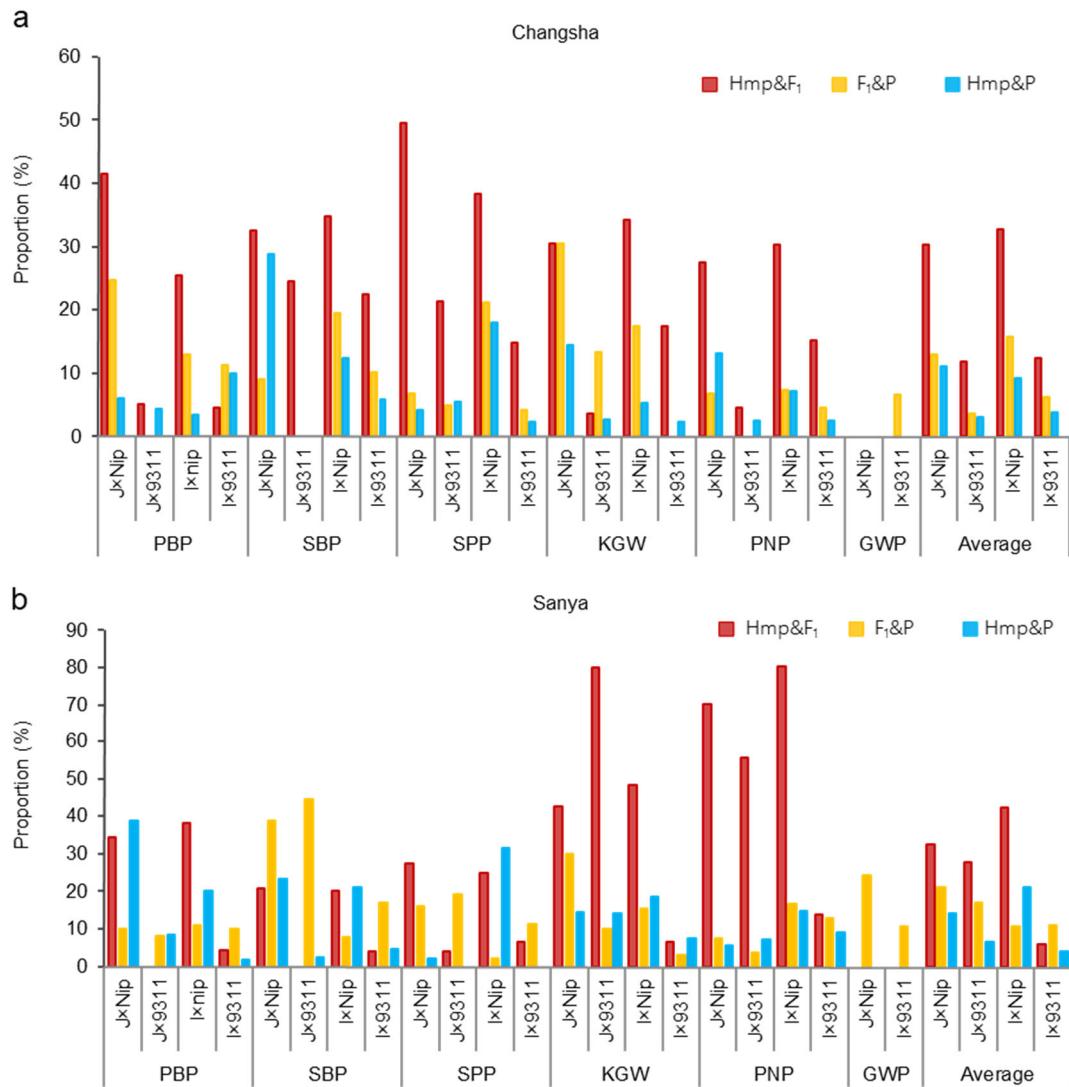


451

452 **Supplementary Figure 22 The colocalized QTL between two environments for yield and its sub-**
 453 **component traits.** (a) The proportion of colocalized QTL between Changsha and Sanya for P_QTL. (b)

454 The proportion of colocalized QTL between Changsha and Sanya for F₁_QTL. (c) The proportion of

455 colocalized QTL between Changsha and Sanya for Hmp_QTL.



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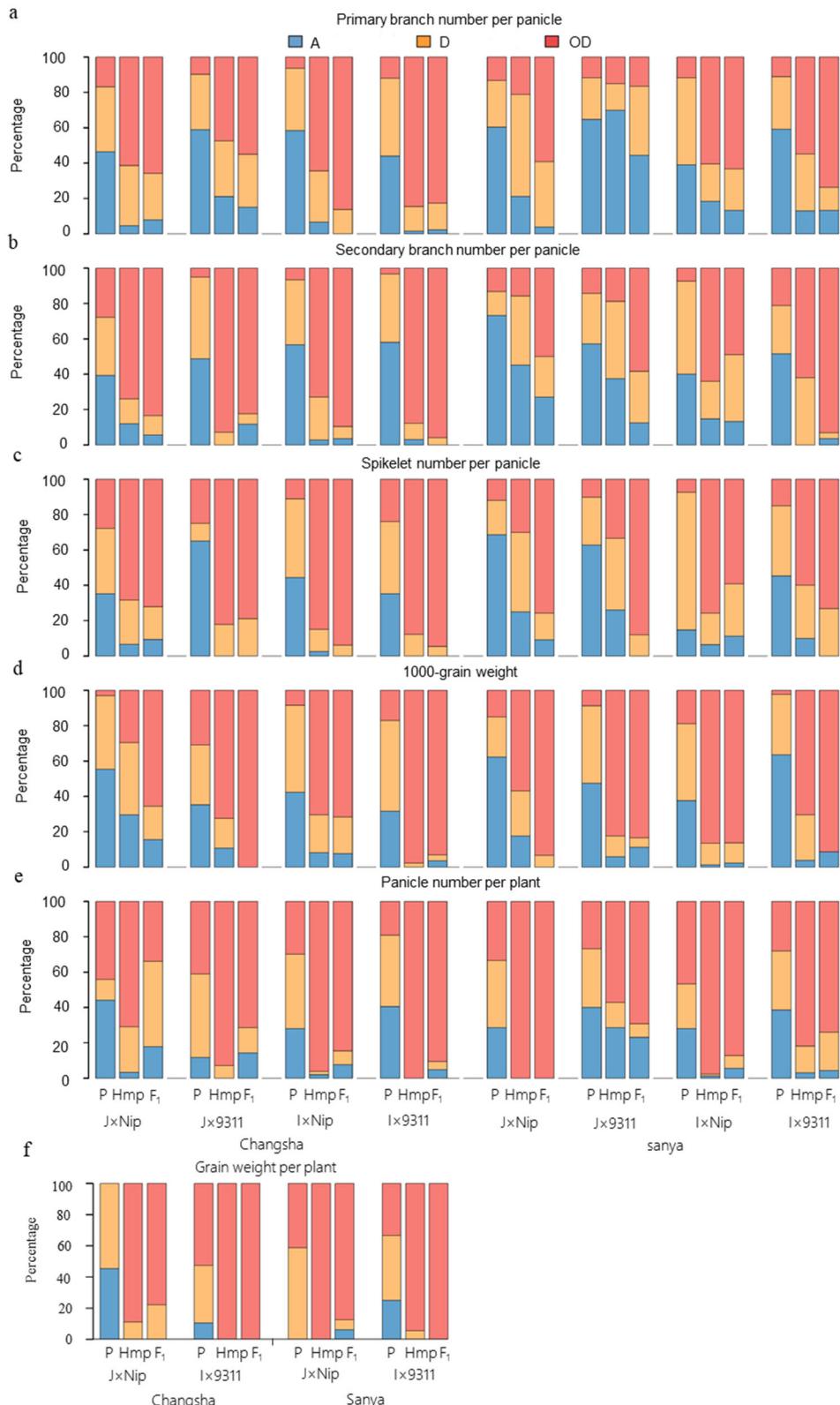
Supplementary Figure 23 The colocalized QTL between F₁_QTL, Hmp_QTL and _QTL for grain yield and its sub-component traits. (a) The proportion of colocalized QTL between F₁_QTL, Hmp_QTL and P_QTL for the trait of PBP, SBP, SPP, KGW, PNP and GWP in Changsha. (b) The proportion of colocalized QTLs between F₁_QTL, Hmp_QTL and P_QTL for the trait of PBP, SBP, SPP, KGW, PNP and GWP in Sanya.

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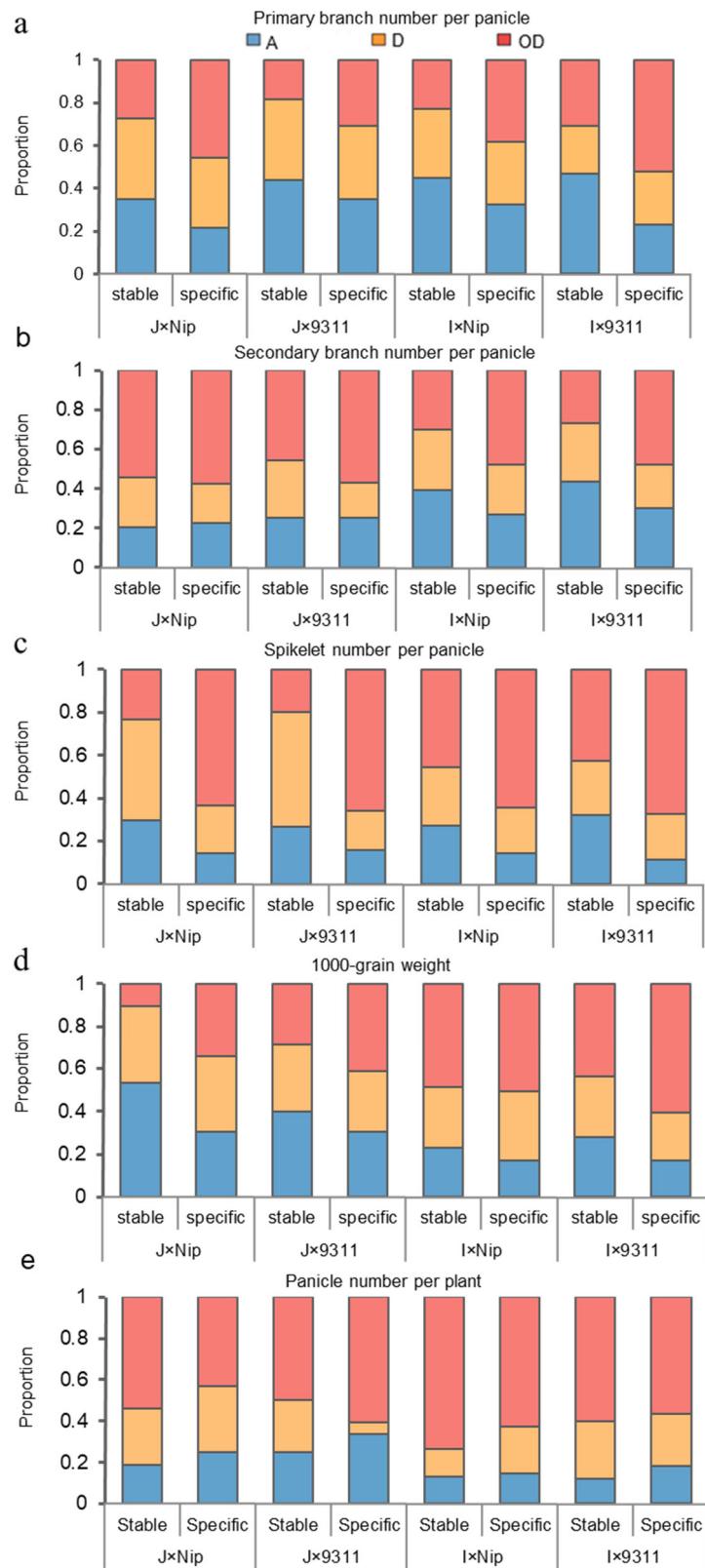
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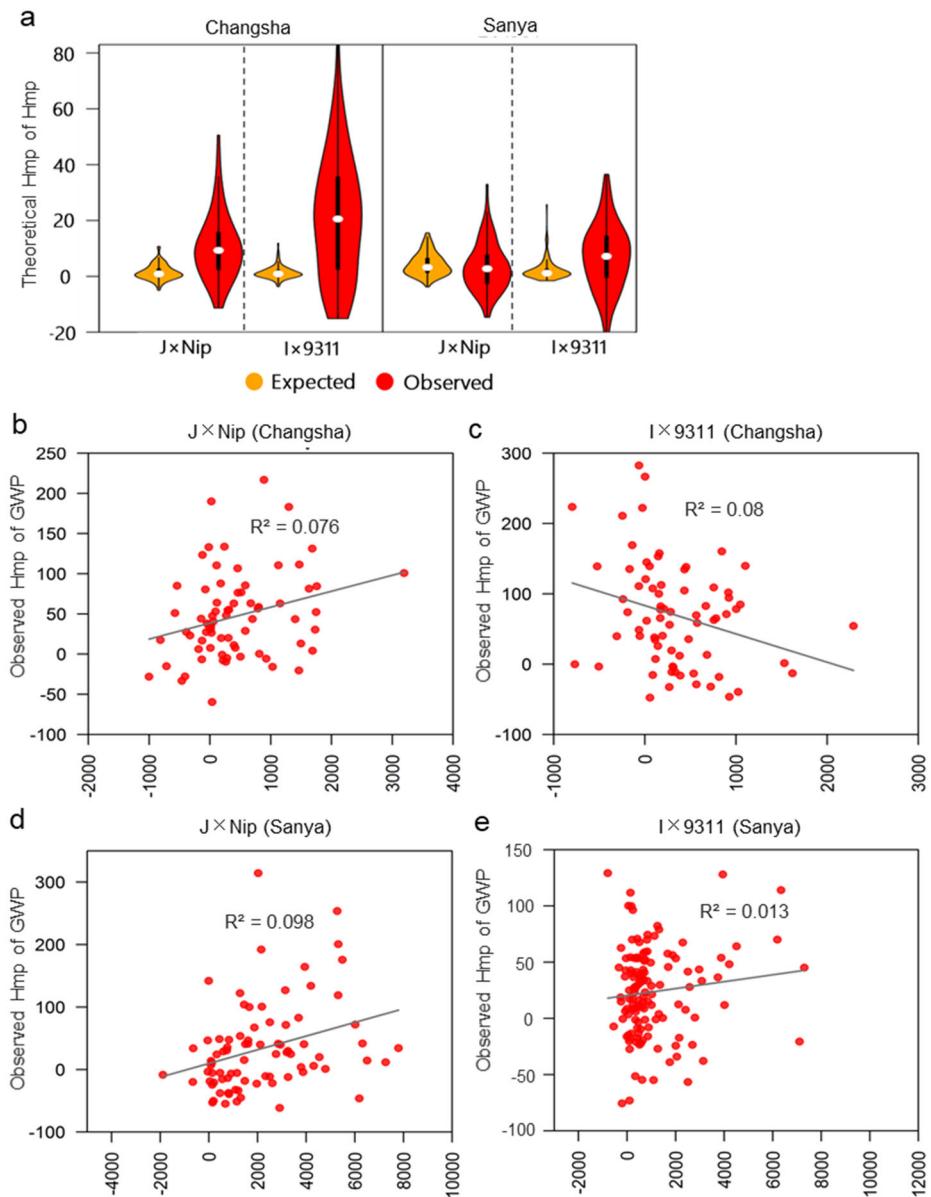
462

463 **Supplementary Figure 24** The percentage of detected QTLs with additive, dominant and over-
464 dominant effects for each yield trait using the phenotype of parents (P), the phenotype of F₁ (F₁)
465 and the middle parent heterosis value (Hmp) in different combinations under two environments
466 respectively. A, additive (in blue); D, dominant (in orange); OD, over-dominant (in red).



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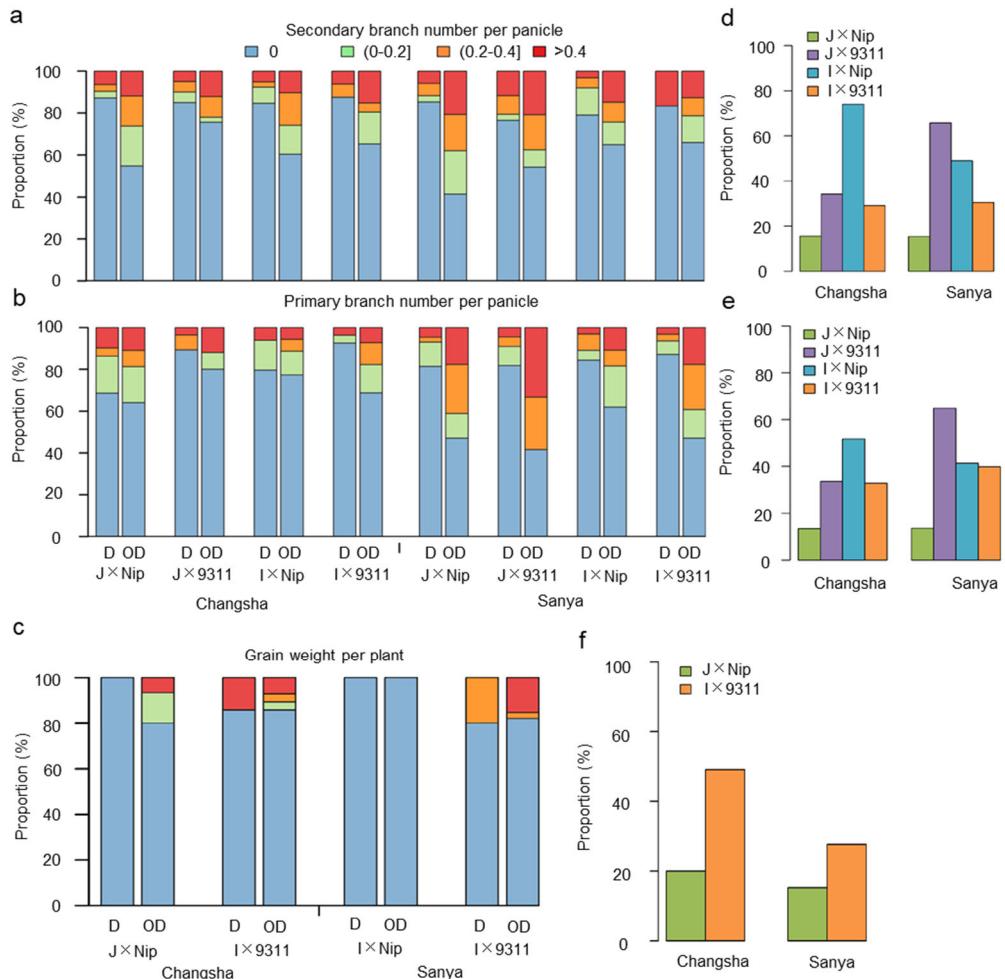
468 **Supplementary Figure 25 The proportion of colocalized QTLs between two environments with**
 469 **additive, dominant and over-dominant effects.** Stable, means the colocalized QTLs between two
 470 environments; Specific, means the QTLs can only be detected in one environment.



471

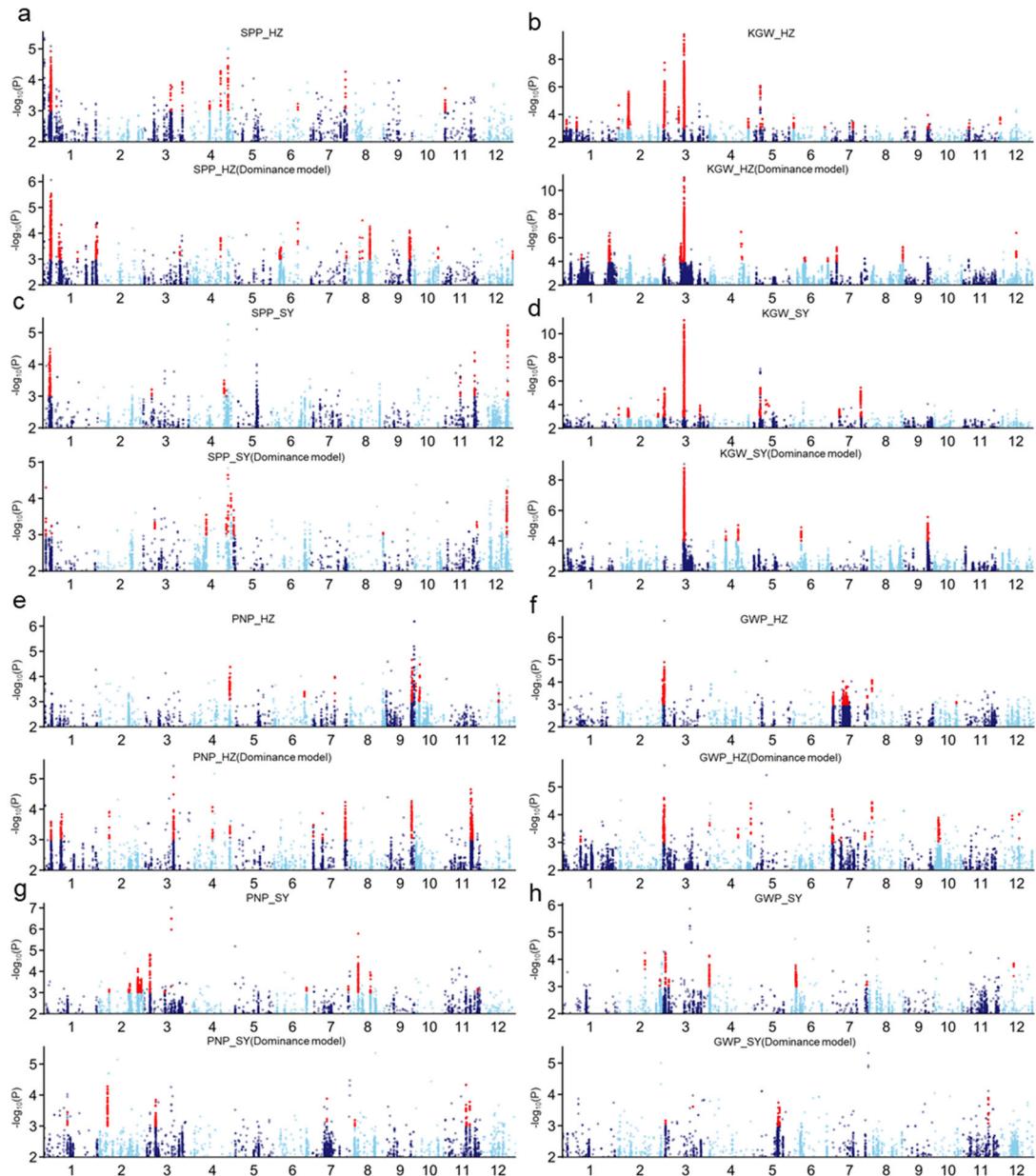
472 **Supplementary Figure 26 Comparison between the observed Hmp of GWP and the theoretical**
 473 **Hmp of GWP estimated according to the multiplicative from additive effect of three main yield**
 474 **components (SPP, KGW and PNP).** (a) The violin plot for the observed Hmp of GWP and the

475 **theoretical Hmp of GWP. (b-e) The scatter plot for the observed Hmp of GWP and the theoretical Hmp of**
 476 **GWP for different combinations in two environments.**



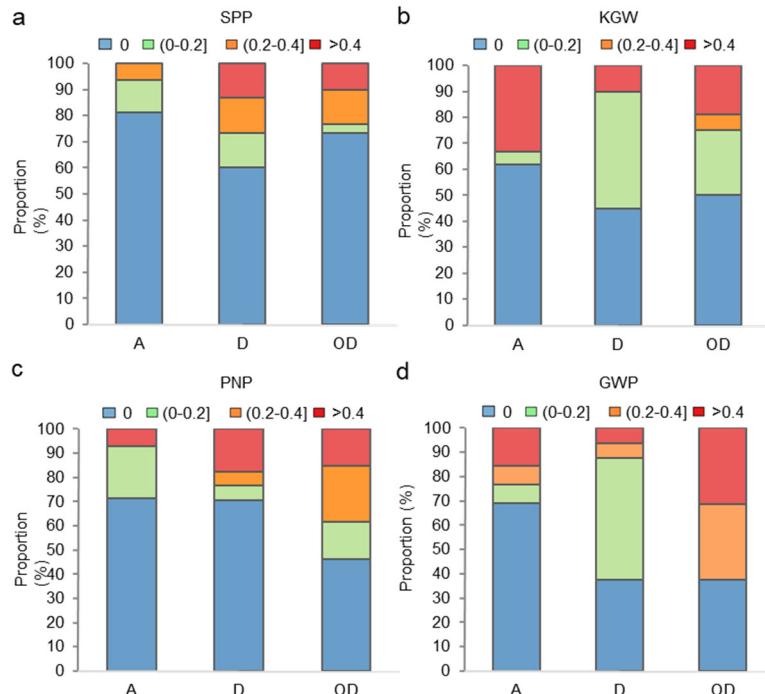
477

478 **Supplementary Figure 27 The repulsive degree in dominant and over-dominant QTLs.** (a-c) The
479 proportion of QTLs with different repulsive degrees; here, the sky blue represents that there is no
480 significant SNP with repulsive additive effects within the QTL, and the light green, orange and red
481 represent the repulsive degree within the range of (0-0.2], (0.2-0.4] and >0.4 within each QTL. (e-f) The
482 average proportion of combinations with repulsive effect alleles per QTL that contains repulsive effect
483 alleles.



484

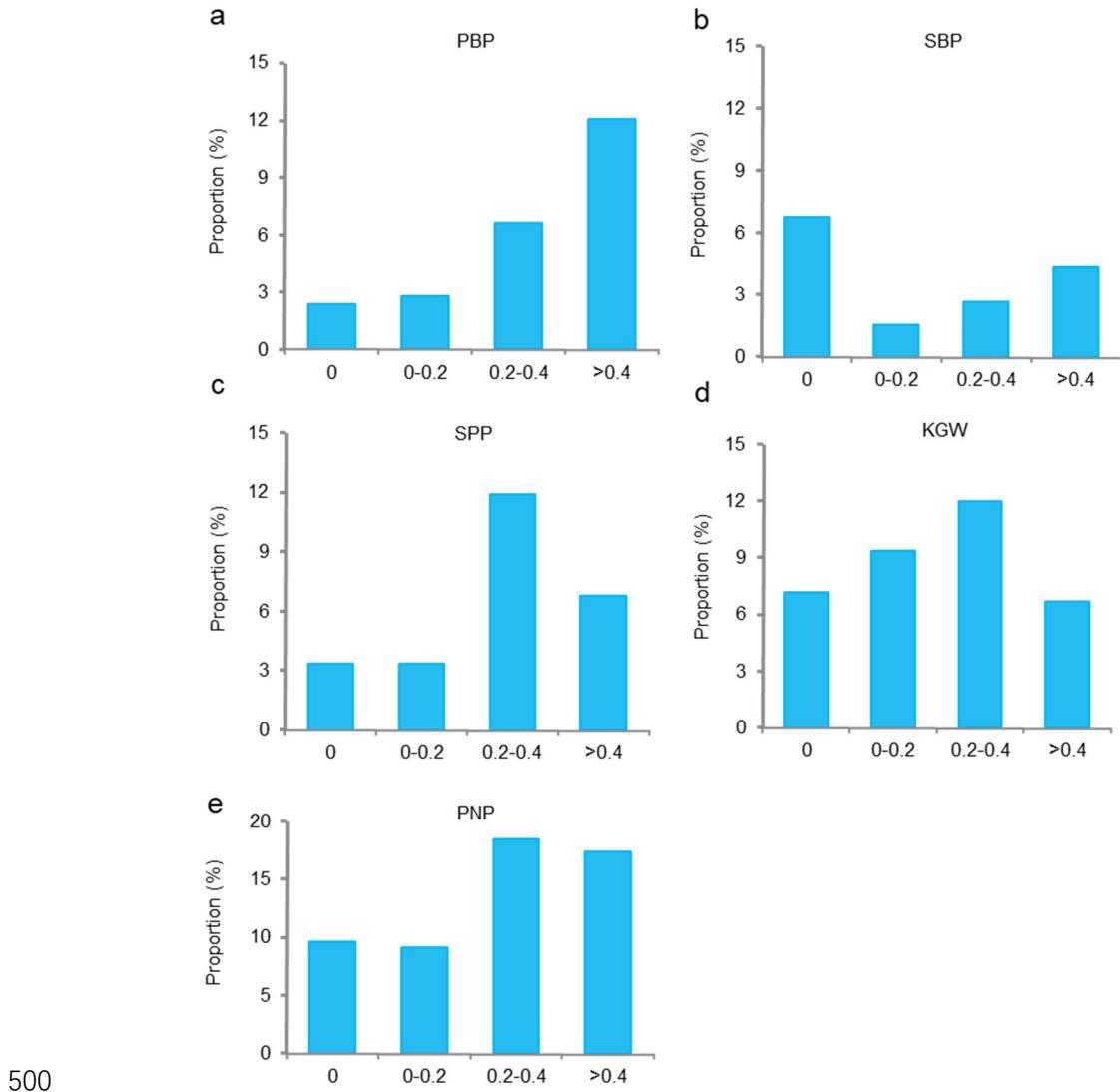
485 **Supplementary Figure 28 Genome-wide association study of yield traits in 1086 three-line system**
486 **hybrids using compressed MLM.** (a) Manhattan plot for SPP in Hangzhou (HZ). (b) Manhattan plot for
487 SPP in HZ by dominance coded genotype. (c) Manhattan plot for KGW in Sanya (SY). (d) Manhattan
488 plot for KGW in SY by dominance coded genotype. (e) Manhattan plot for PNP in HZ. (f) Manhattan plot
489 for PNP in HZ by dominance coded genotype. (g) Manhattan plot for GWP in SY. (h) Manhattan plot for
490 GWP in SY by dominance coded genotype. The red dots are the significant SNPs located within the
491 defined QTL. The data were collected from the published paper (Huang, X.H. et al. Genomic analysis of
492 hybrid rice varieties reveals numerous superior alleles that contribute to heterosis. *Nature*
493 Communications 6(2015)).



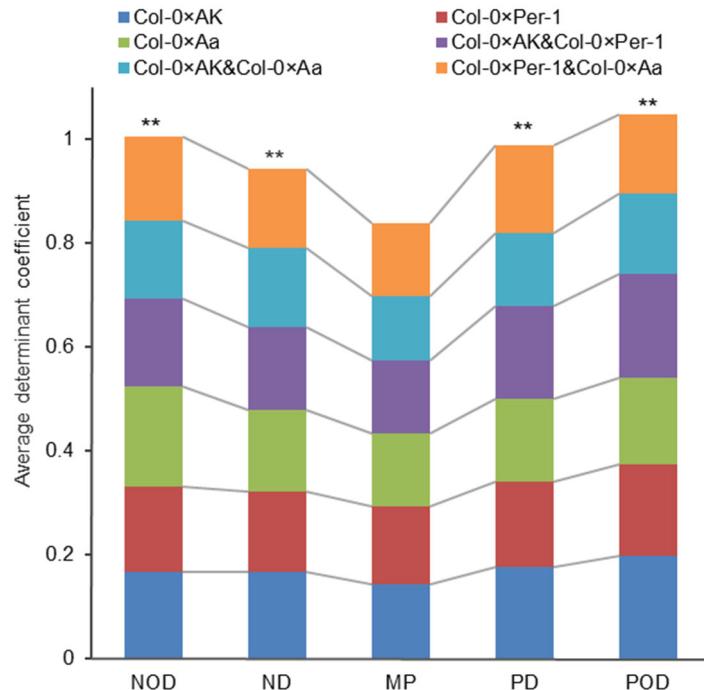
494

495 **Supplementary Figure 29 The repulsive degree in additive (A), dominant (D) and over-dominant**
496 **(OD) QTLs identified in 1086 three-line hybrids.** The sky blue represents that there is no significant
497 SNP with repulsive additive effects within the QTL, and the light green, orange and red represent the
498 repulsive degree within the range of (0-0.2], (0.2-0.4] and >0.4 within each QTL.

499

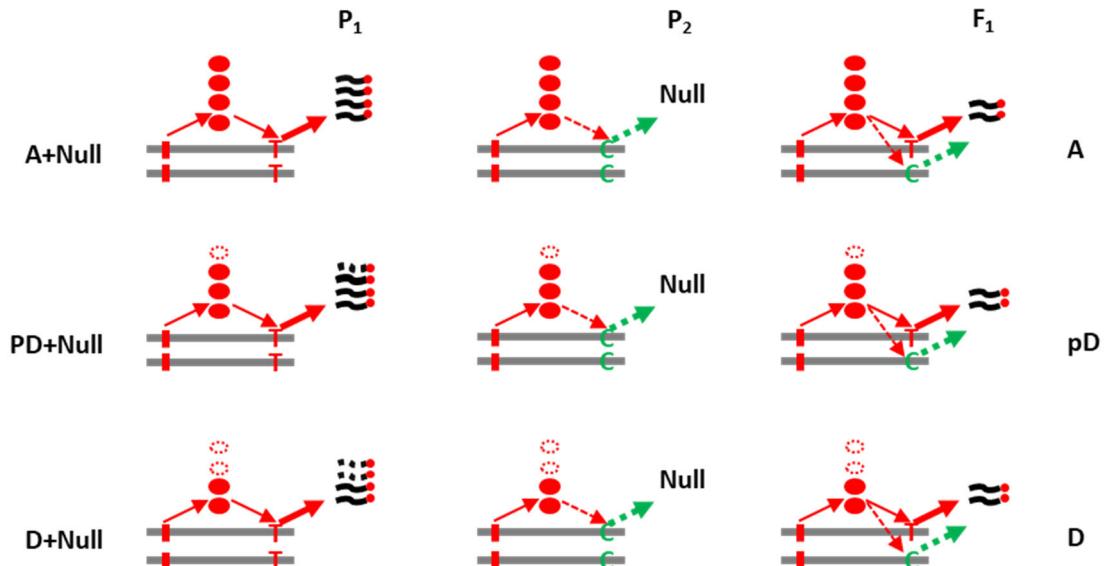


500
501 **Supplementary Figure 30 The colocalization proportion for over-dominant QTLs with different**
502 **repulsive degrees (0, 0-0.2, 0.2-0.4 and >0.4) for the trait of PBP (a), SBP (b), SPP (c), KGW (d)**
503 **and PNP (e).**



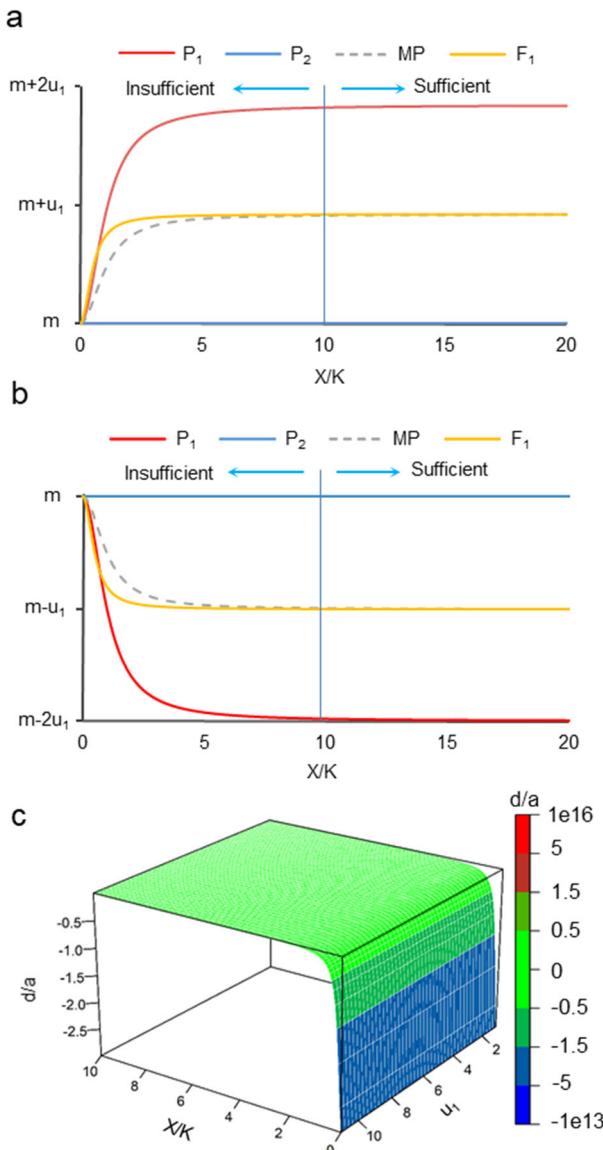
504

505 **Supplementary Figure 31 The average determinant coefficient between genes with different**
 506 **expression patterns and their transcription factors in three *A. thaliana* combinations.** The
 507 determination coefficient was estimated by six pairs of transcription levels in the first leaf between the
 508 gene and its transcription factor across three *A. thaliana* combinations (including Col-0×Per-1, Col-0×Aa,
 509 Col-0×Ak) and their parents. Here, Col-0×AK& Col-0×Per-1 refers to those genes that show the same
 510 expression pattern in both combinations Col-0×AK and Col-0×Per-1. And the others are similar. We
 511 estimated the significant difference of NOD, ND, PD and POD with MP by paired t-test; and “**”
 512 marked the significant level at 0.01. The raw data were collected from the published paper (Yang, M. et al.
 513 Genomic architecture of biomass heterosis. Proc Natl Acad Sci USA6 (2017)).



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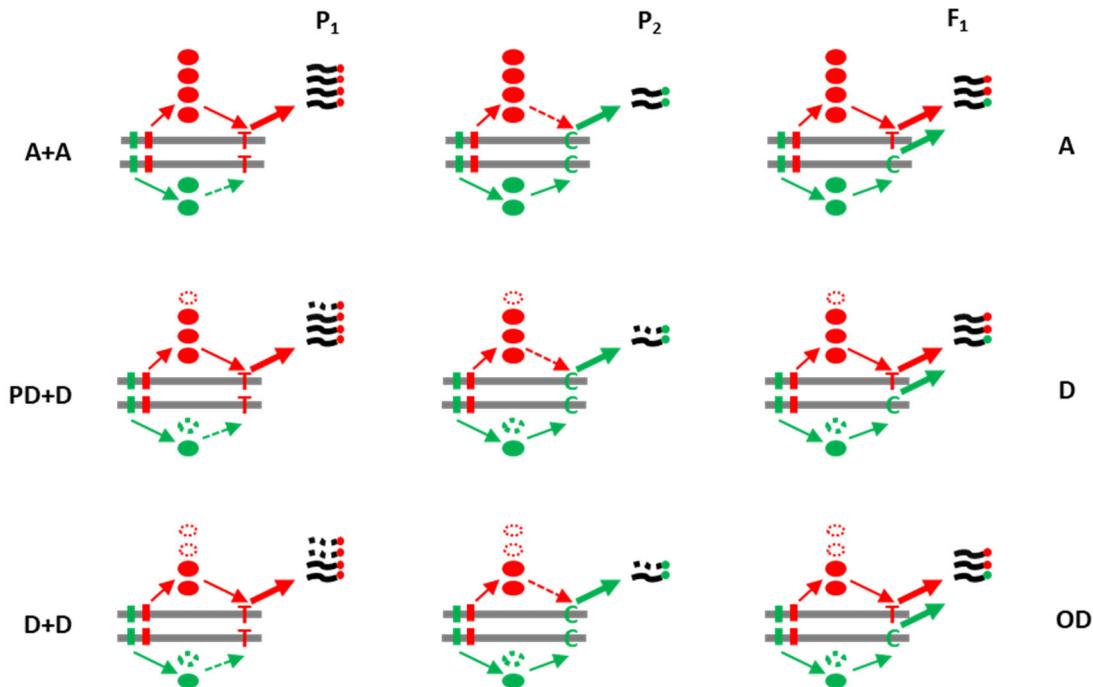
515 **Supplementary Figure 32 The schematic diagram of regulation model for molecular mechanism of**
 516 **additive and dominant effect produced by single site with null allele (C) and one functional allele (T)**
 517 **under one positive regulator background.** The grey thick lines show two chromosomes, the red bars on
 518 which are two homologous alleles of the regulator that are uniform among P_1 , P_2 and F_1 . The break and
 519 solid pies together represent the required regulator function that can maximize the function of T/T
 520 homozygote of the target site, and the solid pies represent different regulator functions and thus provide
 521 different backgrounds to the target site. The arrow represents the function process, and the break arrow
 522 represents the break function process. The break and solid curves together represent the maximum
 523 function of the T/T homozygote in parents or one T allele in F_1 , and the solid curves represent the real
 524 function. $A + Null$ shows the action mode for each allele and between two alleles (T vs C) that they are
 525 independent with T being additive and C being null; and $PD+Null$ and $D+Null$ are similar. A , pD and D
 526 mean additive, partial dominant, and dominant effect, respectively.



527

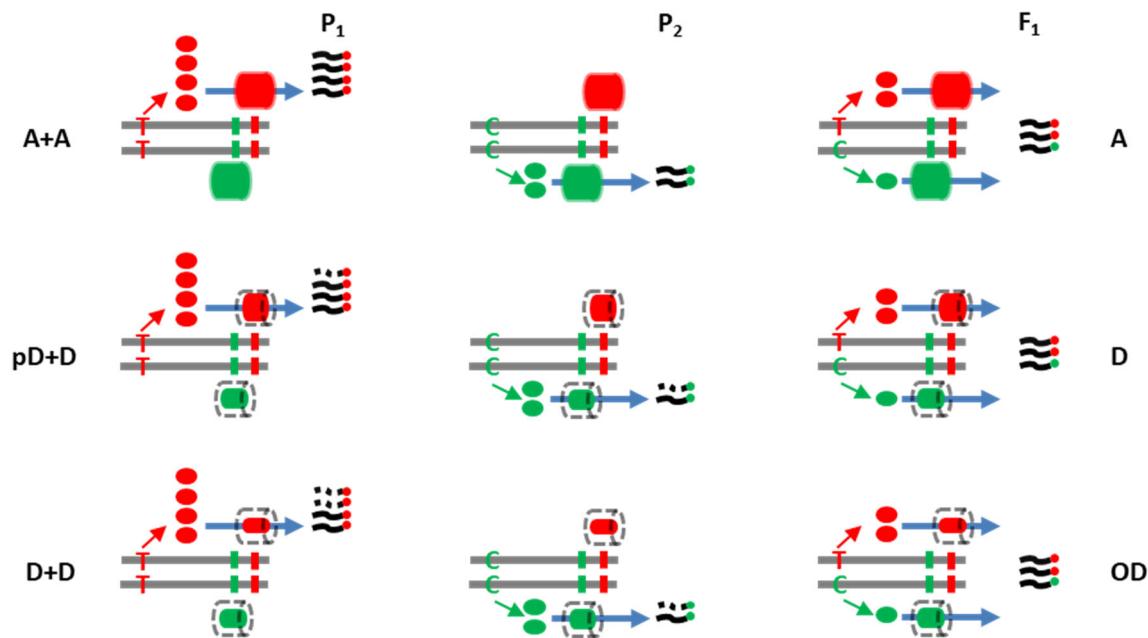
528 **Supplementary Figure 33 The simulated diagram of regulation model for molecular mechanism of**
 529 **additive and dominant effect produced by single site with null allele and one functional allele under**
 530 **one regulator background.** (a) The performance of the target site in two parents, F₁ and the middle
 531 parent (MP) under the activator background with different sufficiencies (X/K). It was simulated according
 532 to Hill function with $\mu_1 = 3$ and $n = 2$. μ_1 means the maximum function at steady state for one functional
 533 allele. n is the Hill coefficient. Left arrow represents a relatively insufficient activator background, and
 534 the right arrow represents the relatively sufficient activator background. (b) The performance of the target
 535 site in two parents, F₁ and the middle parent (MP) under the repressor background with different
 536 sufficiencies (X/K). It was simulated according to Hill function with $\mu_1 = 3$ and $n = 2$. μ_1 means the
 537 maximum function at steady state for one functional allele. n is the Hill coefficient. (c) The dominant

538 degree of the target site under the repressor background with different sufficiencies (X/K) and different μ_1 .



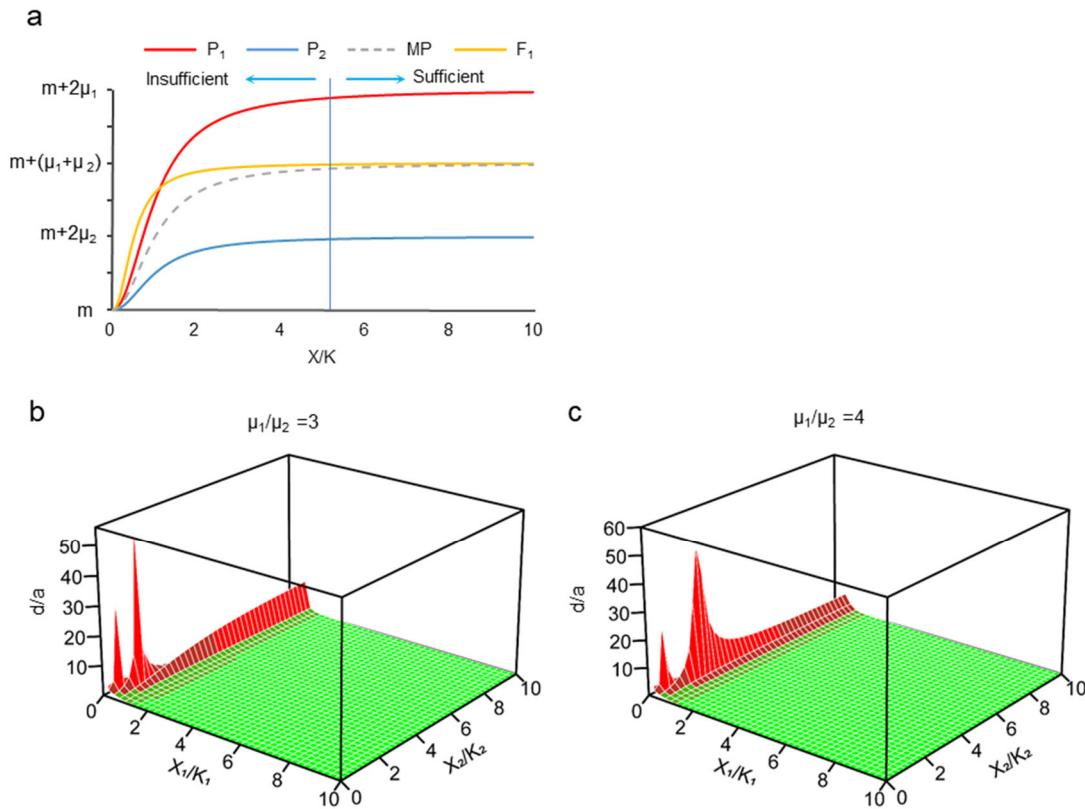
539

540 **Supplementary Figure 34 The schematic diagram of regulation model for molecular mechanism of**
541 **additive, dominant and over-dominant effect produced by the cumulated functions of two alleles of**
542 **one polymorphic site under two independent positive regulators as the upstream backgrounds.** The
543 grey thick lines show two chromosomes, the red and green bars on which represent the homologous
544 alleles of the regulators of T and C allele at target site, respectively; and the function of these regulators
545 keep constant and among P₁, P₂ and F₁. The break and solid pies together represent the required regulator
546 function that can maximize the function of the homozygote of the corresponding target allele, and the
547 solid pies represent different regulator functions and thus provide different backgrounds to the target
548 allele. The arrow represents the function process. The break and solid curves together represent the
549 maximum function of the homozygote in parents or one allele in F₁, and the solid curves represent the real
550 function; those curves with red or green dots represent the function of allele T or C respectively. A+A
551 shows the action mode for each allele and between two alleles (T vs C) that they are independent and
552 cumulative with T and C both being additive; and PD+D and D+D are similar. A, D and OD mean
553 additive, dominant, and over-dominant effect, respectively.



554

555 **Supplementary Figure 35 The schematic diagram of regulation model for molecular mechanism of**
 556 **additive, dominant and over-dominant effect produced by the cumulated functions of two alleles of**
 557 **one polymorphic site under two independent positive regulators or responsors as the downstream**
 558 **backgrounds.** The grey thick lines show two chromosomes, the red and green bars on which represent
 559 the homologous alleles of the regulators or responsors of T and C alleles at target site, respectively; and
 560 the function of these regulators or responsors keep constant among P_1 , P_2 and F_1 . Different numbers of red
 561 and green pies represent the maximum products of the homozygotes of allele T and C of target site,
 562 respectively. The dotted cylinder or the same size of solid cylinder represent the required regulator or
 563 responder function that can transform the full maximum function of the products of the homozygote of
 564 the corresponding target allele, with red corresponding to allele T and green to allele C; and the solid
 565 cylinders in dotted cylinder represent different regulator or response functions and thus provide different
 566 backgrounds to the target allele. The arrow represents the function process. The break and solid curves
 567 together represent the transformed maximum function of the homozygote in parents or one allele in F_1 ,
 568 and the solid curves represent the real transformed function; those curves with red or green dots represent
 569 the transformed function of allele T or C respectively. A+A shows the action mode for each allele and
 570 between two alleles (T vs C) that they are independent and cumulative with T and C both being additive;
 571 and PD+D and D+D are similar. A, D and OD mean additive, dominant, and over-dominant effect,
 572 respectively.



573

574 **Supplementary Figure 36 The simulated distribution of dominance to additive effect ratio (d/a)**

575 **with same backgrounds, but the two alleles in F₁ are regulated by different factors in the**

576 **background for positive regulation.** (a) The diagram of the performance of two parent, F₁ and middle

577 **parent (MP) under the condition of $\mu_1 = 3$, $\mu_2 = 1$ and $K_1 = K_2$ for positive regulation. left arrow means**

578 **background is relative insufficient and right arrow means background is relative sufficient. (b-c) The**

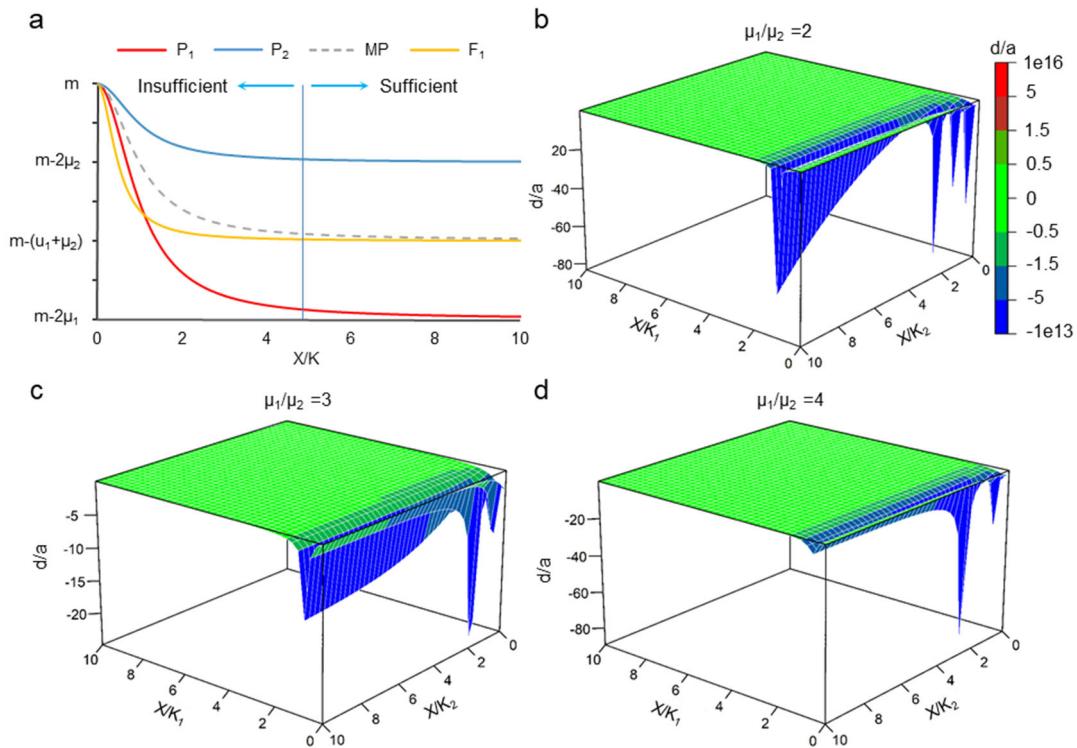
579 **simulated distribution of d/a with same homologous backgrounds, but the two alleles in F₁ are regulated**

580 **by different factors in the background. d/a means degree of dominance effect/additive effect, it can be in**

581 **both positive and negative direction. μ_1/μ_2 means the ratio of maximum function of P₁ genotype to**

582 **maximum function of P₂ genotype when their respective background afford to the complete expression of**

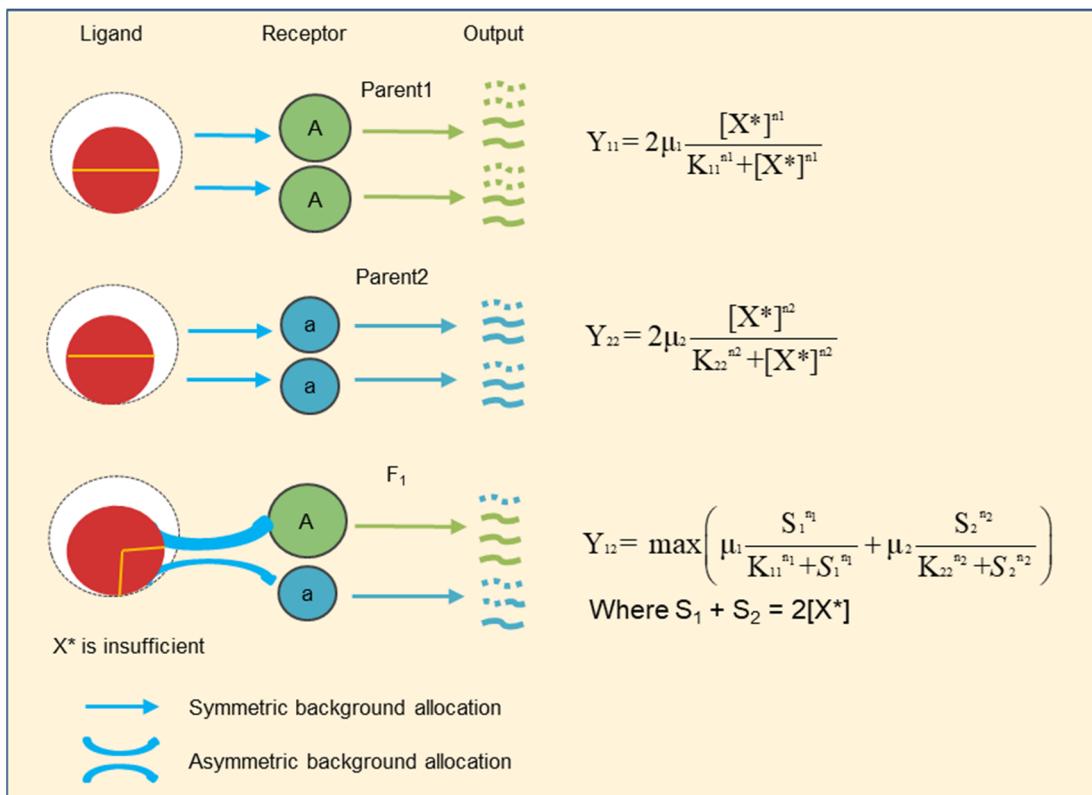
583 **the corresponding homologous genotype; d/a means the degree of dominance to additive effect ratio.**



584

585 **Supplementary Figure 37 The simulated diagram of regulation model for molecular mechanism of**
 586 **additive, dominant and over-dominant effect produced by the cumulated functions of two alleles of**
 587 **one polymorphic site under two independent negative regulators or responsors as the backgrounds.**

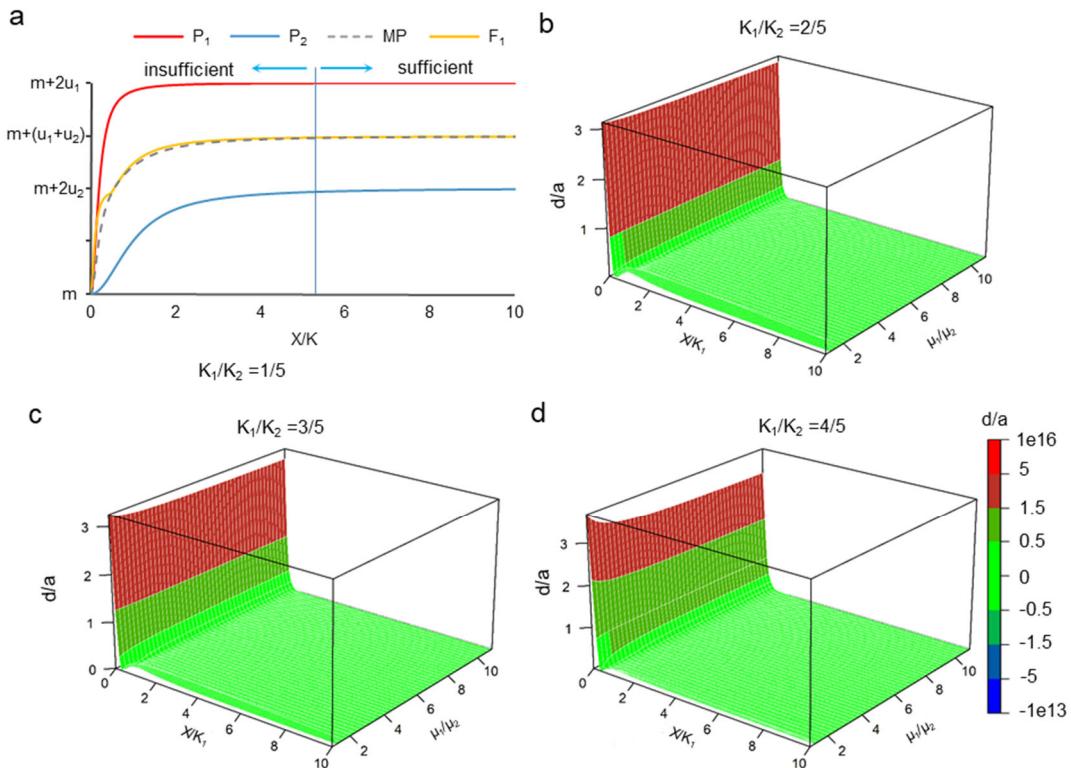
588 (a) The performance of the target site in two parents, F_1 and the middle parent (MP) under the repressor
 589 background with different sufficiencies (X/K). It was simulated according to Hill function with $\mu_1 = 3$, μ_2
 590 $= 1$, $K_1 = K_2$ and $n = 2$. μ_1 and μ_2 means the maximum function at steady state for allele with higher
 591 function and lower function, respectively. n is the Hill coefficient. Left arrow represents a relatively
 592 insufficient repressor background, and the right arrow represents the relatively sufficient repressor
 593 background. (b) The dominant degree of the target site under the repressor background with different
 594 sufficiencies (X/K) for two alleles of the target site with $\mu_1/\mu_2 = 2$. (c) The dominant degree of the target
 595 site under the repressor background with different sufficiencies (X/K) for two alleles of the target site
 596 with $\mu_1/\mu_2 = 3$. (d) The dominant degree of the target site under the repressor background with different
 597 sufficiencies (X/K) for two alleles of the target site with $\mu_1/\mu_2 = 4$.



598

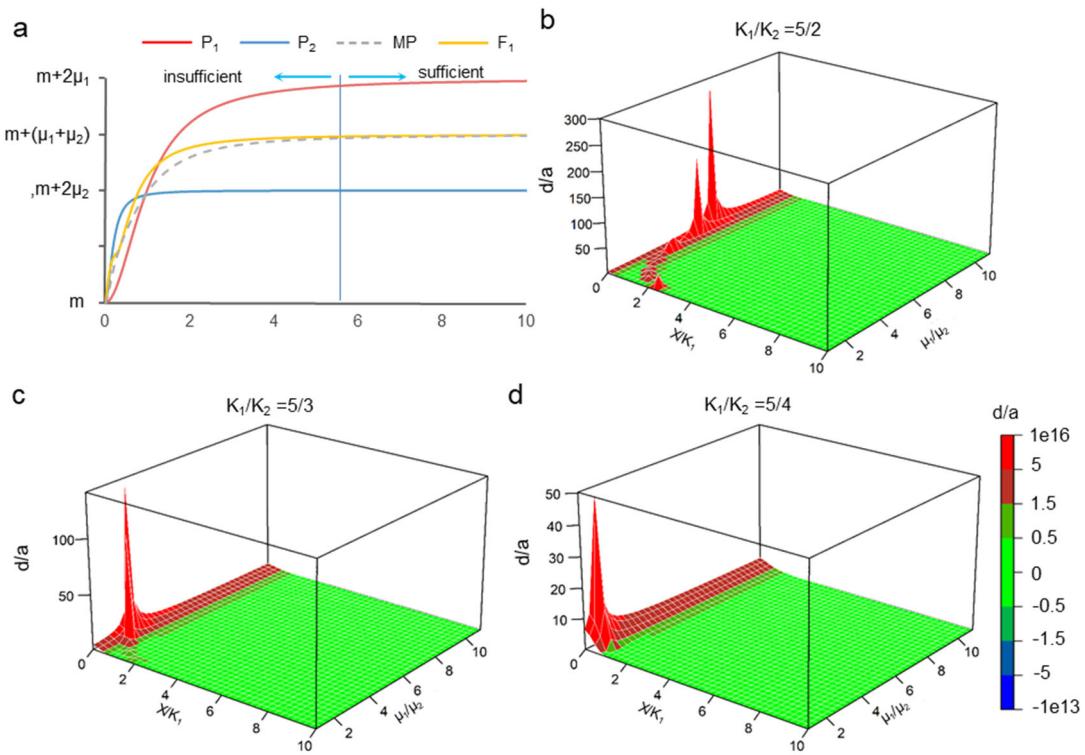
599 **Supplementary Figure 38 The regulation or response model for molecular mechanism of dominant**
 600 **and over-dominant effect produced by single locus with the same background.** Here, the hollow
 601 circle means the background level that satisfy the full potential of the target. The filled red circle means
 602 the background level that homozygous parent actually supplied. The orange line indicates the background
 603 allocation. When the background is insufficient and the affinity of the two genes is different, the two
 604 allele of A and a will be in a competitive use of limited background, the more competitive gene is likely
 605 to get relative more backgrounds in hybrid than that in the original homozygous parents. The parameter of
 606 μ , K and n are described in the **Supplementary Note**.

607



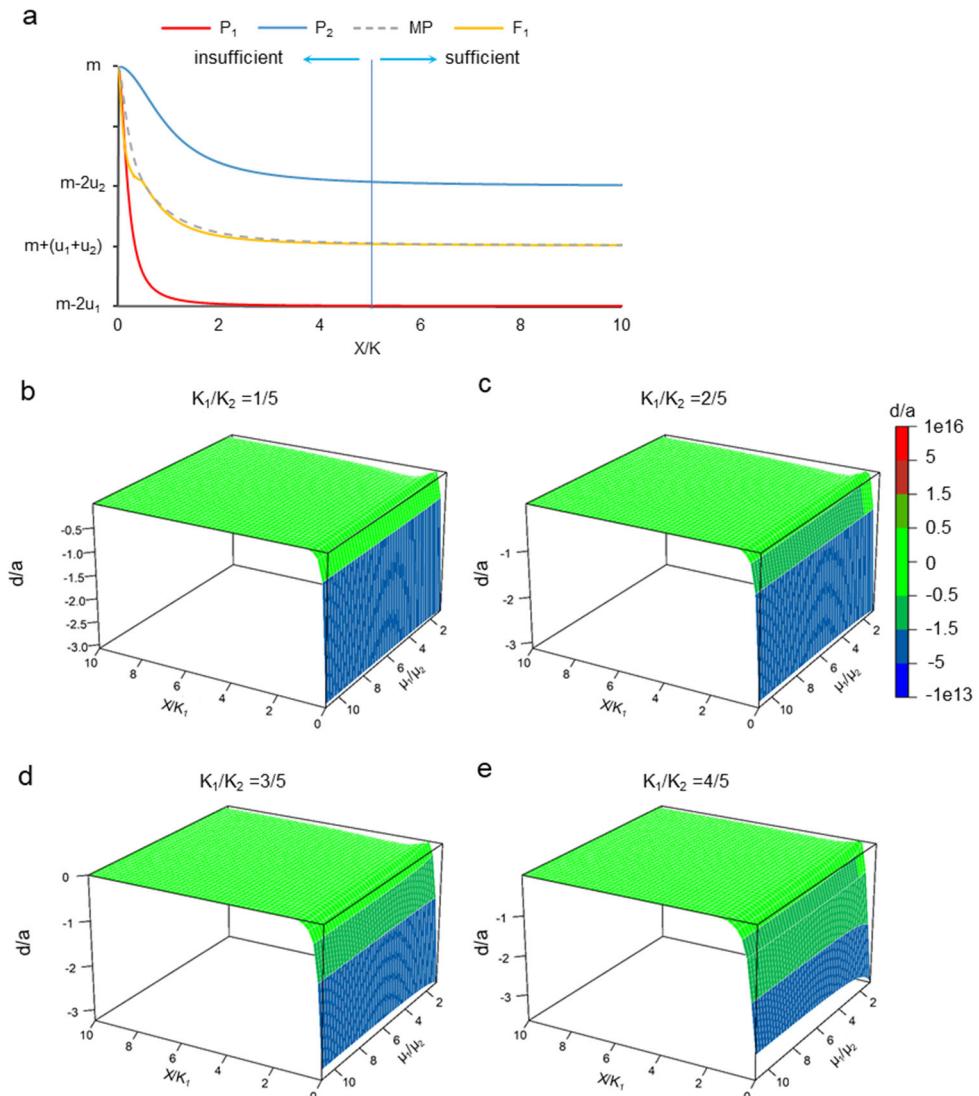
608

609 **Supplementary Figure 39 The simulated diagram of regulation model for molecular mechanism of**
 610 **additive, dominant and over-dominant effect produced by the cumulated functions of two alleles of**
 611 **one polymorphic site under the same positive regulators or responsors as the background when**
 612 **allele 1 showing higher maximum function and higher affinity ($\mu_1 > \mu_2$ and $K_1 < K_2$).** (a) The
 613 performance of the target site in two parents, F_1 and the middle parent (MP) under the activator
 614 background with different sufficiencies (X/K). It was simulated according to Hill function with $\mu_1 = 3$, μ_2
 615 = 1, $K_1 = 1$, $K_2 = 5$ and $n = 2$. μ_1 and μ_2 means the maximum function at steady state for allele with higher
 616 function and lower function, respectively. n is the Hill coefficient. Left arrow represents a relatively
 617 insufficient activator background, and the right arrow represents the relatively sufficient activator
 618 background. (b) The dominant degree of the target site under the activator background with different
 619 sufficiencies for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when K_1/K_2
 620 = 2/5. (c) The dominant degree of the target site under the activator background with different
 621 sufficiencies for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when K_1/K_2
 622 = 3/5. (d) The dominant degree of the target site under the activator background with different
 623 sufficiencies for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when K_1/K_2
 624 = 4/5.



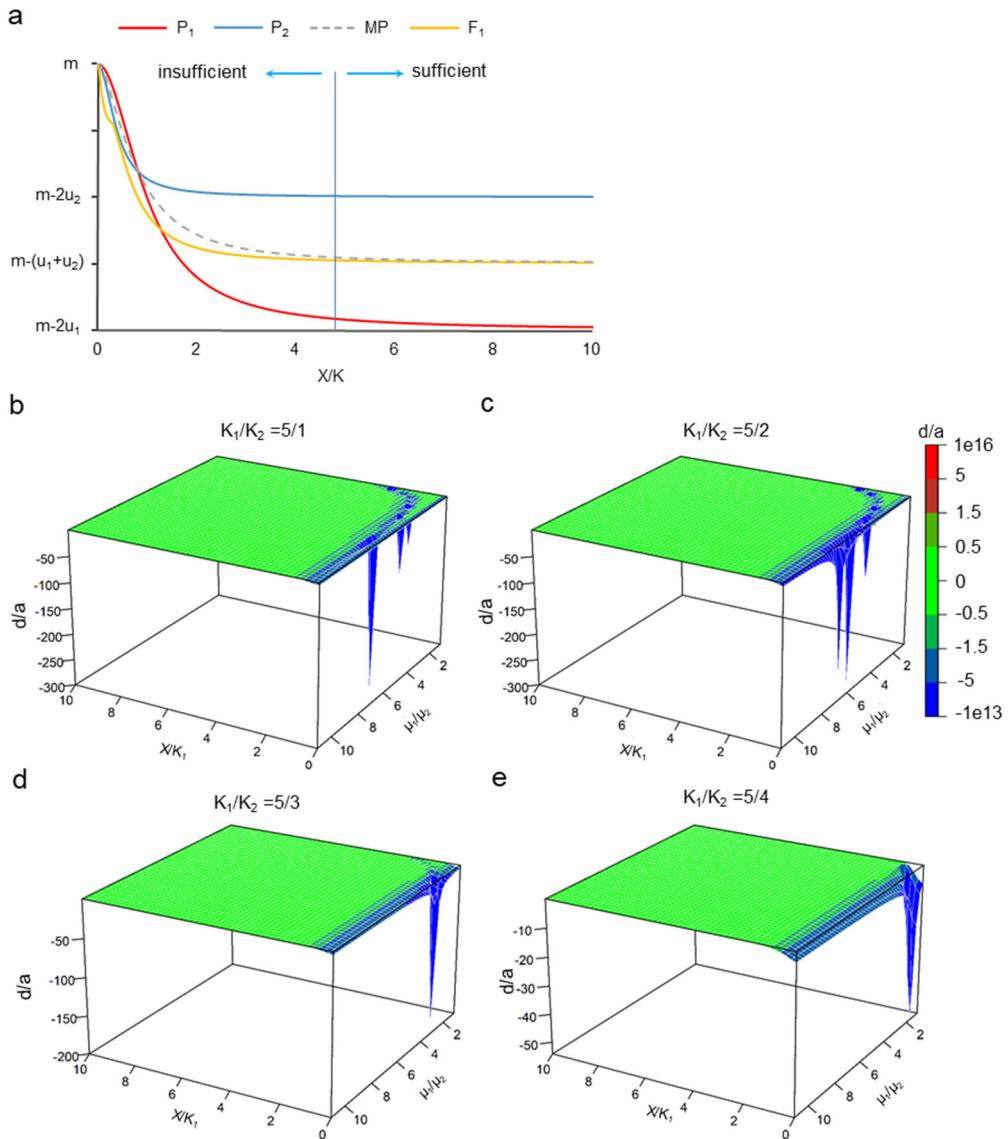
625

626 **Supplementary Figure 40 The simulated diagram of regulation model for molecular mechanism of**
 627 **additive, dominant and over-dominant effect produced by the cumulated functions of two alleles of**
 628 **one polymorphic site under the same positive regulators or responders as the background when**
 629 **allele 1 showing higher maximum function but lower affinity ($\mu_1 > \mu_2$ and $K_1 > K_2$).** (a) The
 630 performance of the target site in two parents, F_1 and the middle parent (MP) under the activator
 631 background with different sufficiencies (X/K). It was simulated according to Hill function with $\mu_1 = 3$, μ_2
 632 = 1, $K_1 = 5$, $K_2 = 1$ and $n = 2$. μ_1 and μ_2 means the maximum function at steady state for allele with higher
 633 function and lower function, respectively. n is the Hill coefficient. Left arrow represents a relatively
 634 insufficient activator background, and the right arrow represents the relatively sufficient activator
 635 background. (b) The dominant degree of the target site under the activator background with different sufficiencies
 636 for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 =$
 637 5/2. (c) The dominant degree of the target site under the activator background with different sufficiencies
 638 for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 5/3$. (d) The
 639 dominant degree of the target site under the activator background with different sufficiencies for allele
 640 with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 5/4$.



641

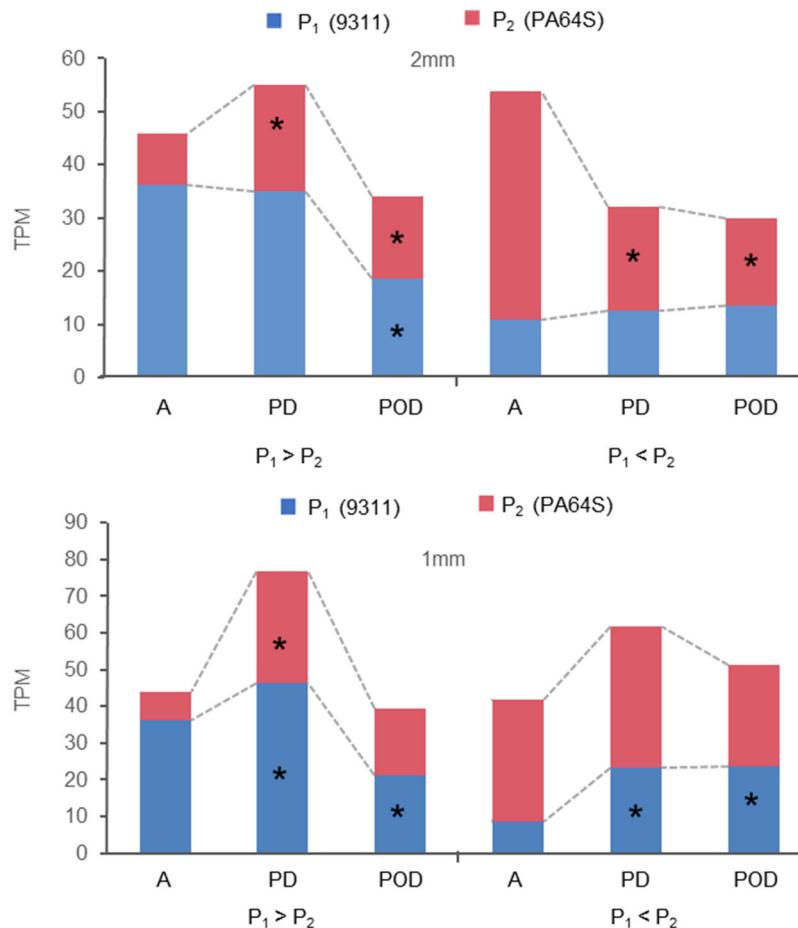
642 **Supplementary Figure 41 The simulated diagram of regulation model for molecular mechanism of**
 643 **additive, dominant and over-dominant effect produced by the cumulated functions of two alleles of**
 644 **one polymorphic site under the same negative regulators or responders as the background when**
 645 **allele 1 showing higher maximum function and higher affinity ($\mu_1 > \mu_2$ and $K_1 < K_2$).** (a) The
 646 **performance of the target site in two parents, F_1 and the middle parent (MP) under the repressor**
 647 **background with different sufficiencies (X/K). It was simulated according to Hill function with $\mu_1 = 2$, μ_2**
 648 **= 1, $K_1 = 1$, $K_2 = 5$ and $n = 2$. μ_1 and μ_2 means the maximum function at steady state for allele with higher**
 649 **function and lower function, respectively. n is the Hill coefficient. Left arrow represents a relatively**
 650 **insufficient repressor background, and the right arrow represents the relatively sufficient repressor**
 651 **background. (b) The dominant degree of the target site under the repressor background with different**
 652 **sufficiencies for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 =$**
 653 **1/5. (c) The dominant degree of the target site under the repressor background with different sufficiencies**
 654 **for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 2/5$. (d) The**
 655 **dominant degree of the target site under the repressor background with different sufficiencies for allele**
 656 **with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 3/5$. (e) The dominant**
 657 **degree of the target site under the repressor background with different sufficiencies for allele with higher**
 658 **function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 4/5$.**



660 **Supplementary Figure 42 The simulated diagram of regulation model for molecular mechanism of**
 661 **additive, dominant and over-dominant effect produced by the cumulated functions of two alleles of**
 662 **one polymorphic site under the same negative regulators or responsors as the background when**
 663 **allele 1 showing higher maximum function but lower affinity ($\mu_1 > \mu_2$ and $K_1 > K_2$).** (a) The
 664 performance of the target site in two parents, F_1 and the middle parent (MP) under the repressor
 665 background with different sufficiencies (X/K). It was simulated according to Hill function with $\mu_1 = 2$, μ_2
 666 = 1, $K_1 = 5$, $K_2 = 1$ and $n = 2$. μ_1 and μ_2 means the maximum function at steady state for allele with higher
 667 function and lower function, respectively. n is the Hill coefficient. Left arrow represents a relatively
 668 insufficient repressor background, and the right arrow represents the relatively sufficient repressor
 669 background. (b) The dominant degree of the target site under the repressor background with different
 670 sufficiencies for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 =$
 671 5/1. (c) The dominant degree of the target site under the repressor background with different sufficiencies
 672 for allele with higher function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 5/2$. (d) The
 673 dominant degree of the target site under the repressor background with different sufficiencies for allele with higher
 674 function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 5/3$. (e) The dominant
 675 degree of the target site under the repressor background with different sufficiencies for allele with higher

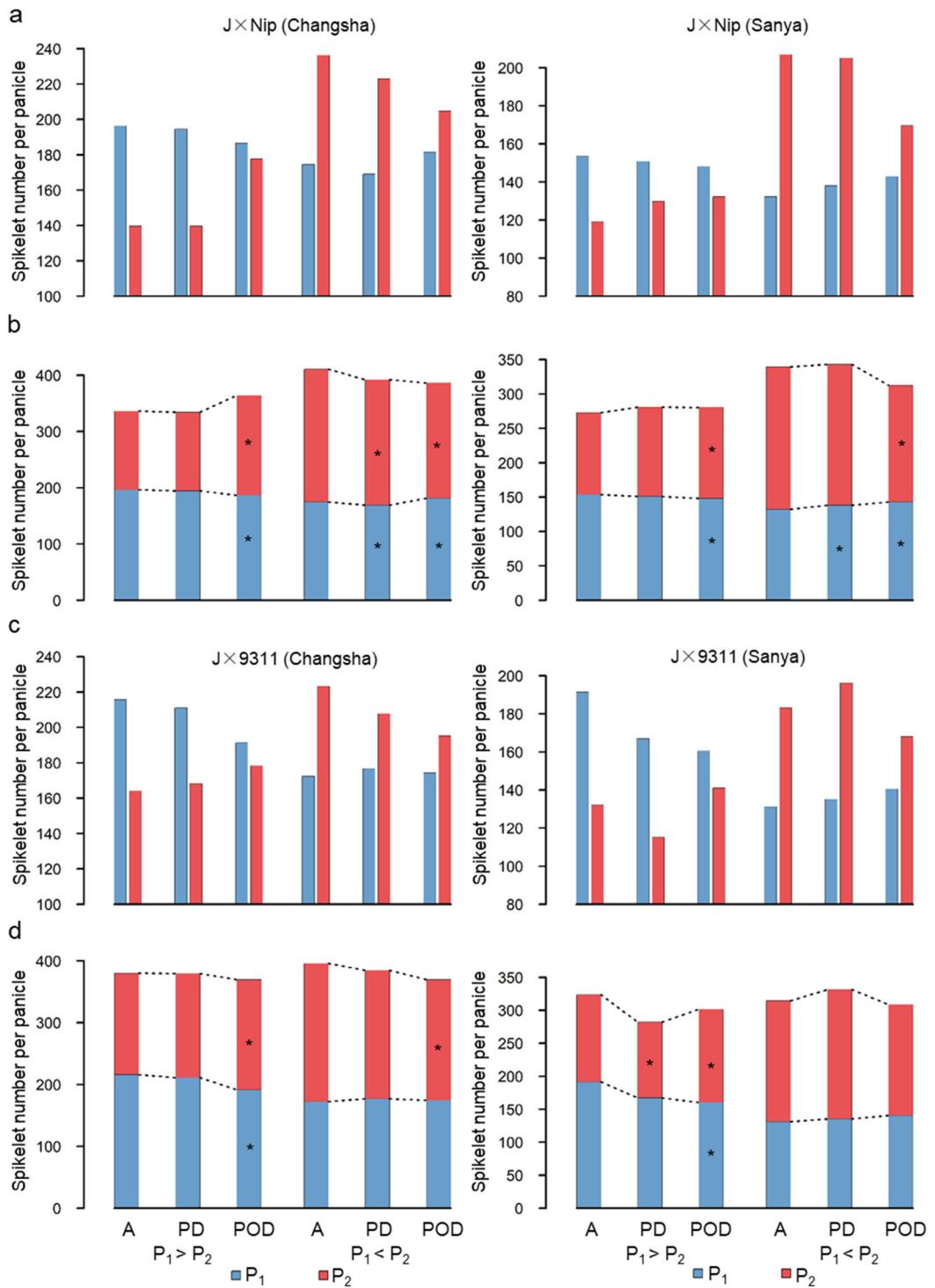
676 function (X/K_1) of the target site and with different μ_1/μ_2 when $K_1/K_2 = 5/4$.

677



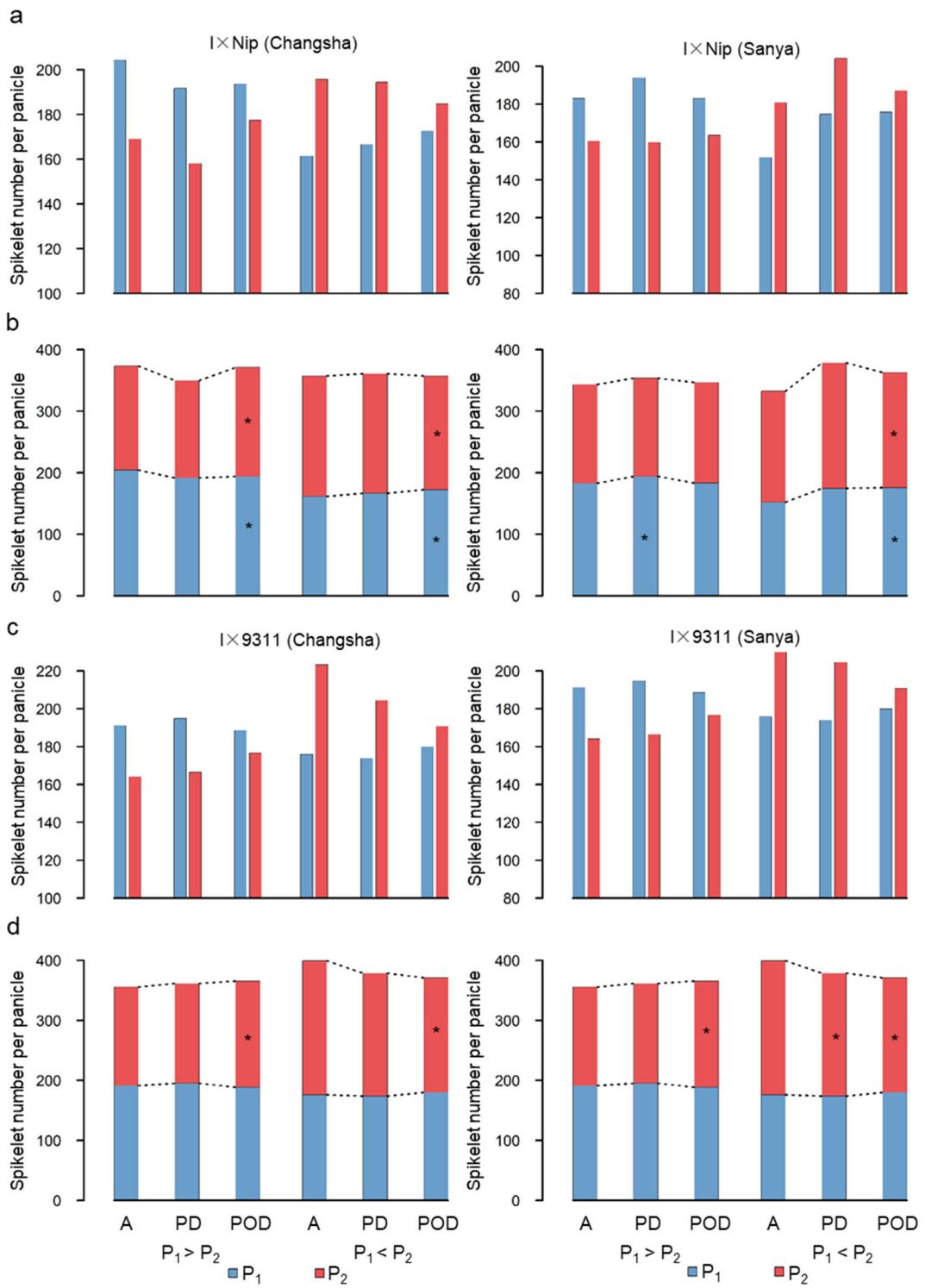
678

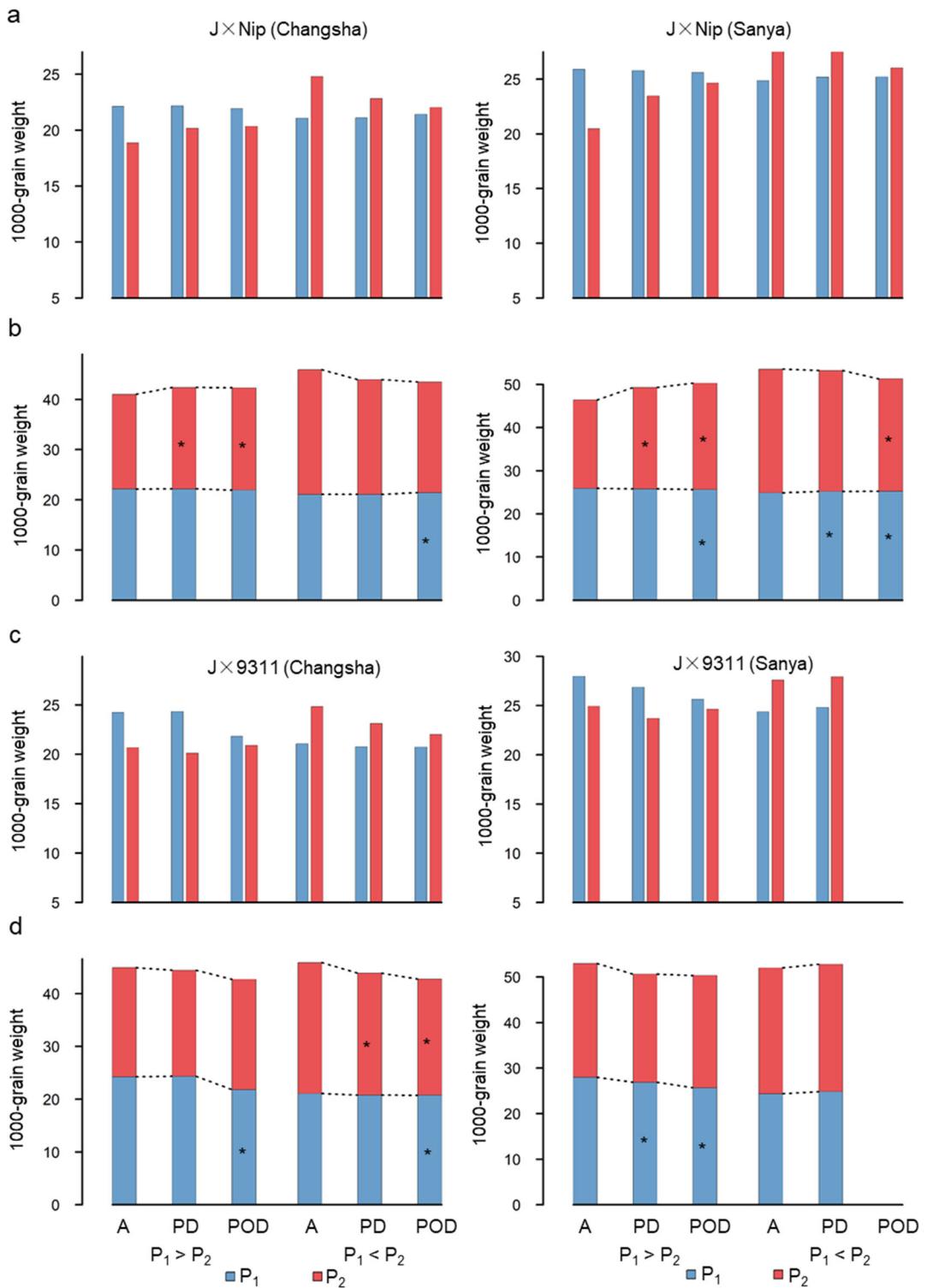
679 **Supplementary Figure 43 The expression level of genes with different expression patterns in 1 mm**
680 **and 2 mm young panicles of two parents.** Here, A, PD and POD mean the expression patterns
681 appearing additive, positive dominant and over-dominant, respectively; the star means significant
682 difference from the genes with additive expression pattern.



683

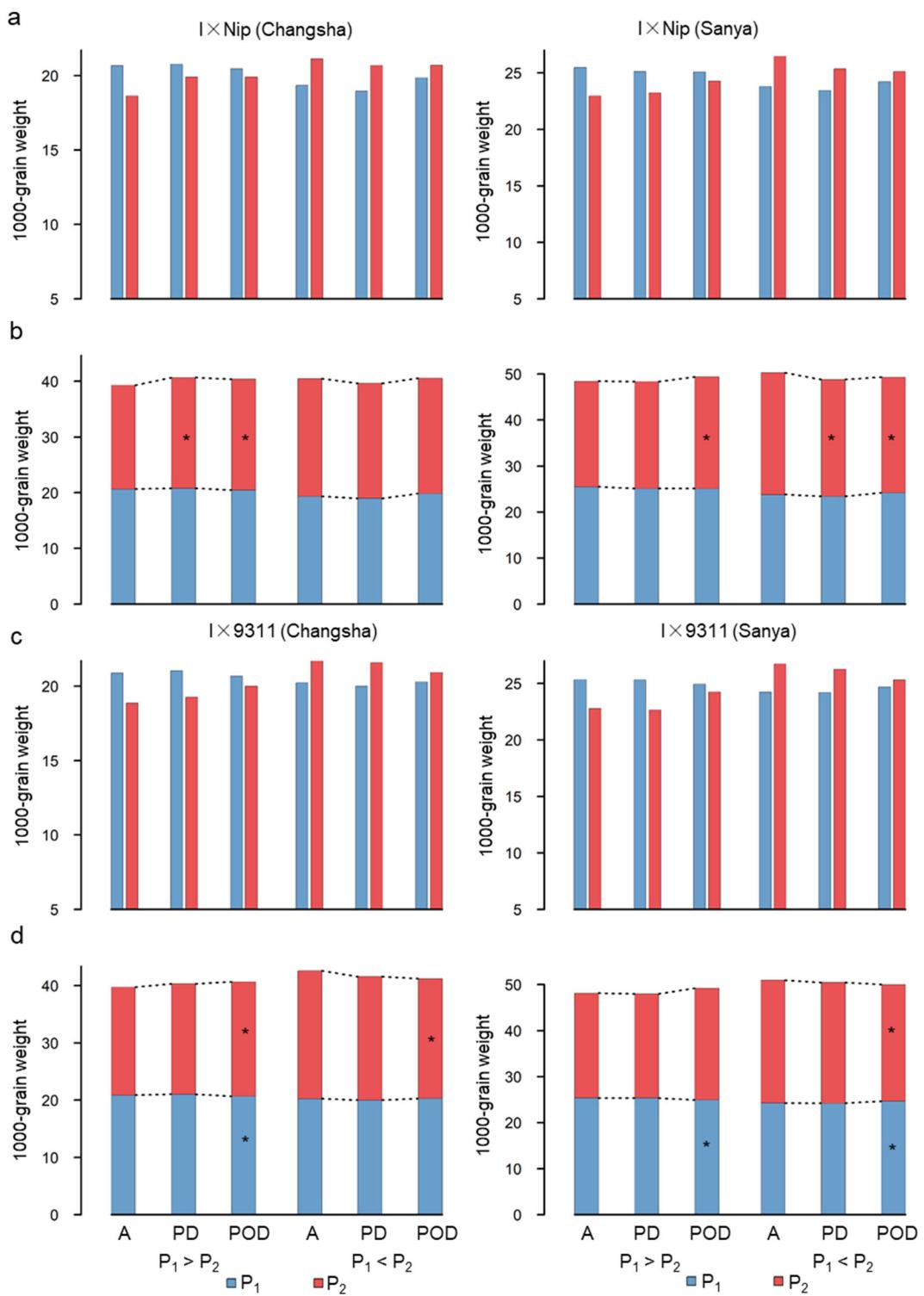
684 **Supplementary Figure 44 The spikelet number per plant (SPP) of parents with non-tester genotype**
 685 **(P₁) and parents with tester genotype (P₂) of the SPP QTLs showing different genetic effect types.**
 686 Here, A, PD and POD mean the additive, positive dominant and positive over-dominant QTLs,
 687 respectively. P₁ > P₂ means that P₁ contains the genotype with higher effect of the QTL, and on the
 688 contrary P₁ < P₂ means that P₂ contains the genotype with higher effect of the QTL. The star means
 689 significant difference from the additive QTLs.

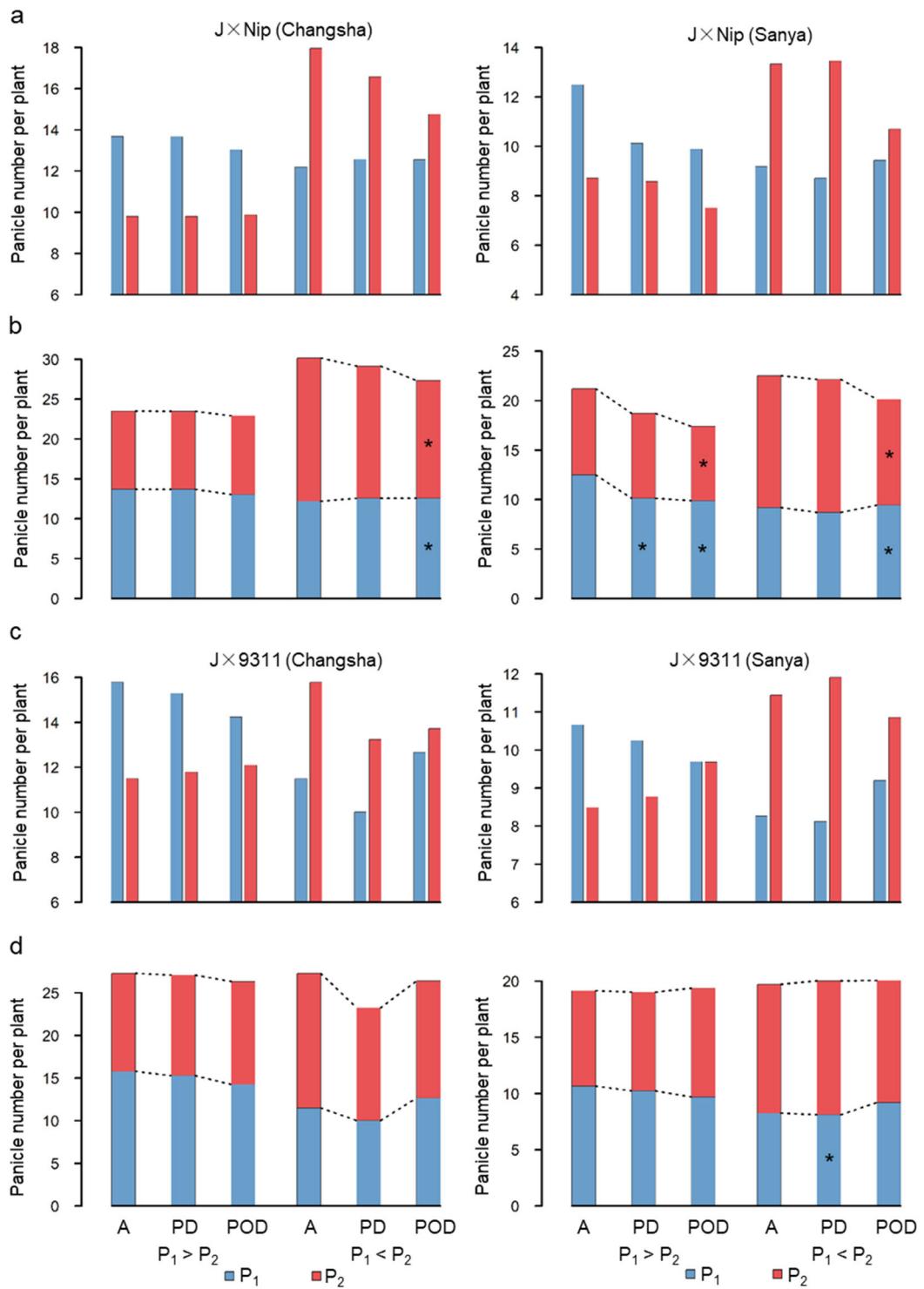




692

693 **Supplementary Figure 45** The 1000-grain weight (KGW) of parents with non-tester genotype (P_1)
694 and parents with tester genotype (P_2) of the KGW QTLs showing different genetic effect types. Here,
695 A, PD and POD mean the additive, positive dominant and positive over-dominant QTLs, respectively. $P_1 >$
696 P_2 means that P_1 contains the genotype with higher effect of the QTL, and on the contrary $P_1 < P_2$ means
697 that P_2 contains the genotype with higher effect of the QTL. The star means significant difference from
698 the additive QTLs.





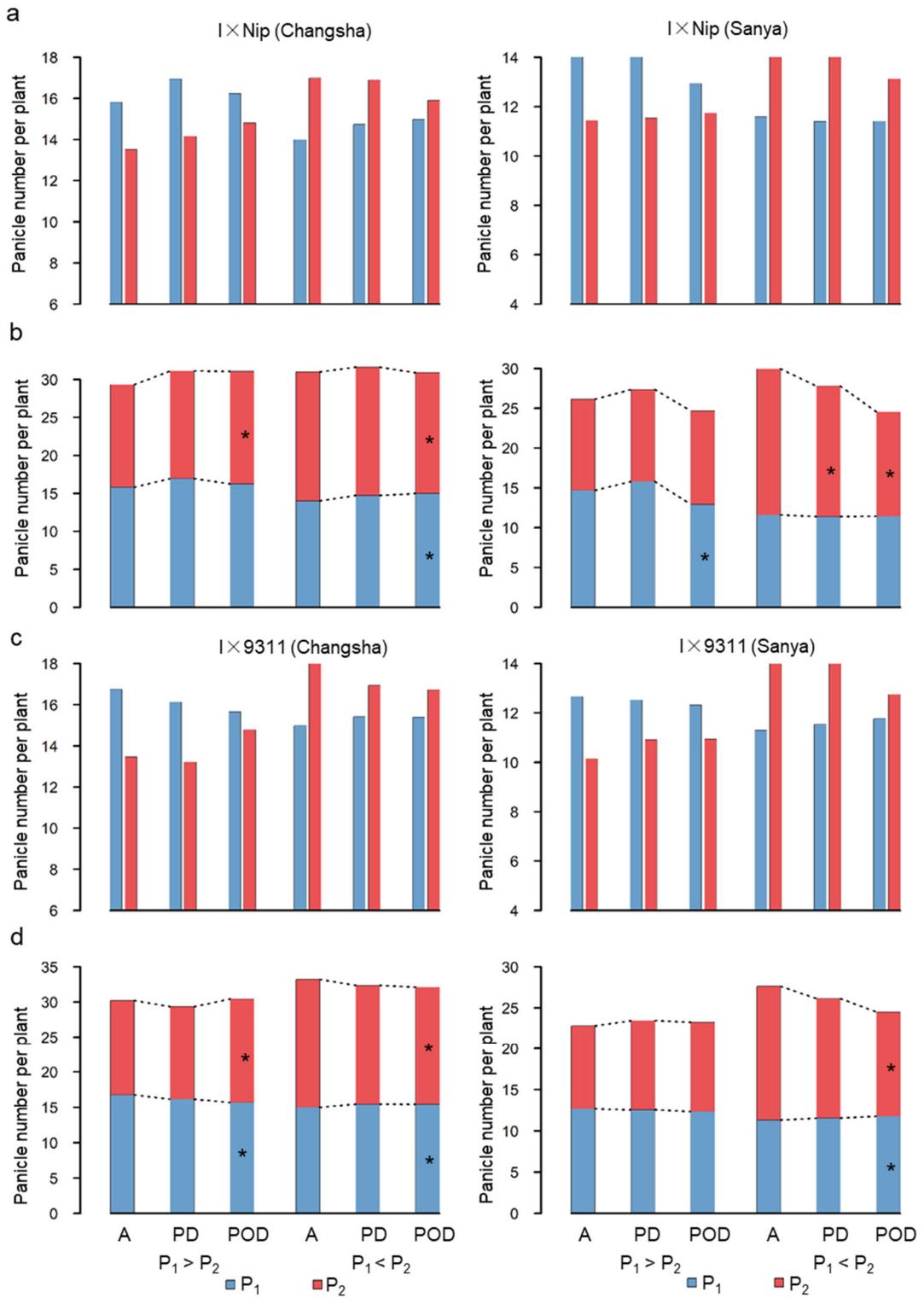
701

702 **Supplementary Figure 46** The panicle number per plant (PNP) of parents with non-tester genotype703 (P_1) and parents with tester genotype (P_2) of the PNP QTLs showing different genetic effect types.

704 Here, A, PD and POD mean the additive, positive dominant and positive over-dominant QTLs,

705 respectively. $P_1 > P_2$ means that P_1 contains the genotype with higher effect of the QTL, and on the706 contrary $P_1 < P_2$ means that P_2 contains the genotype with higher effect of the QTL. The star means

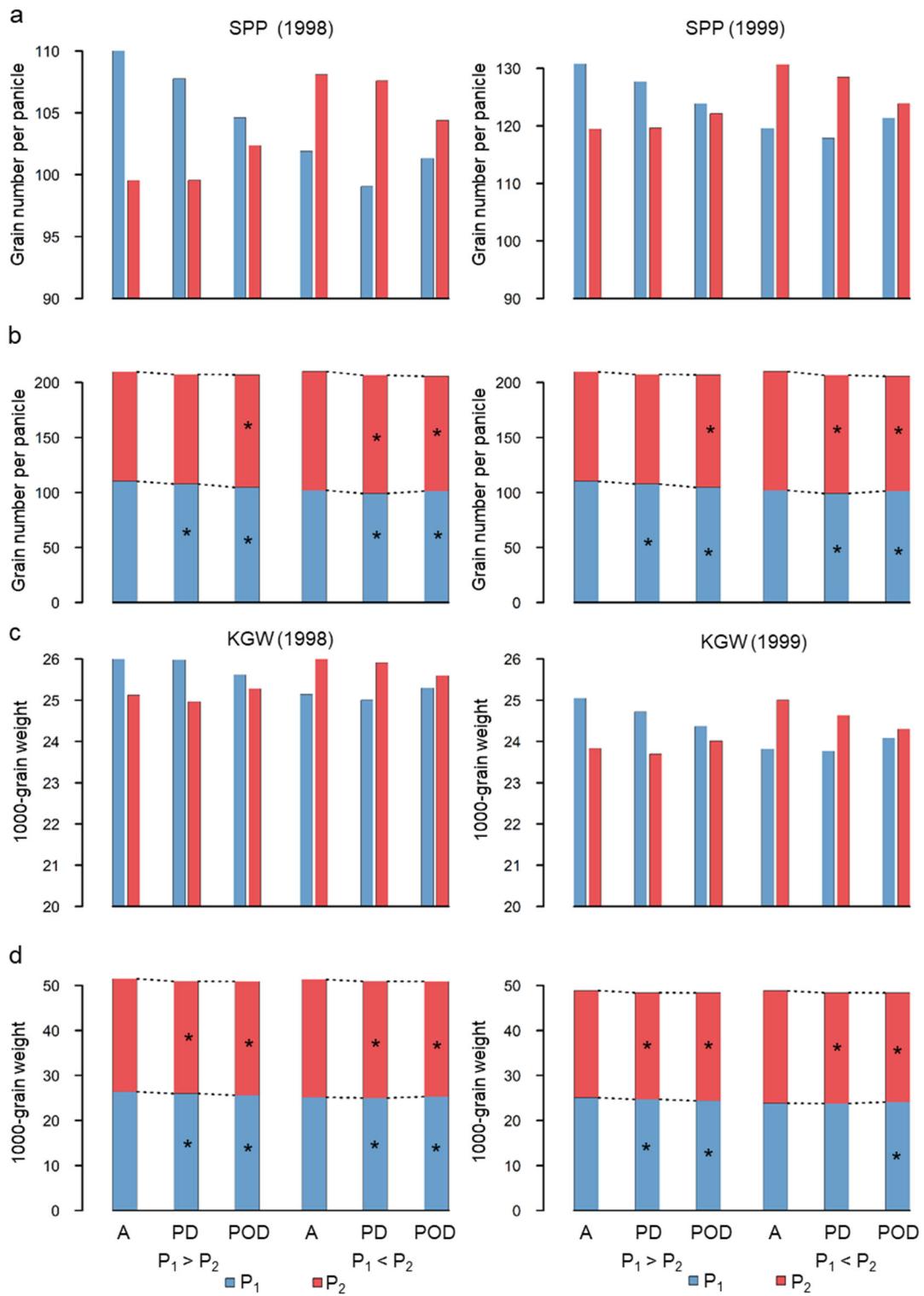
707 significant difference from the additive QTLs.



708

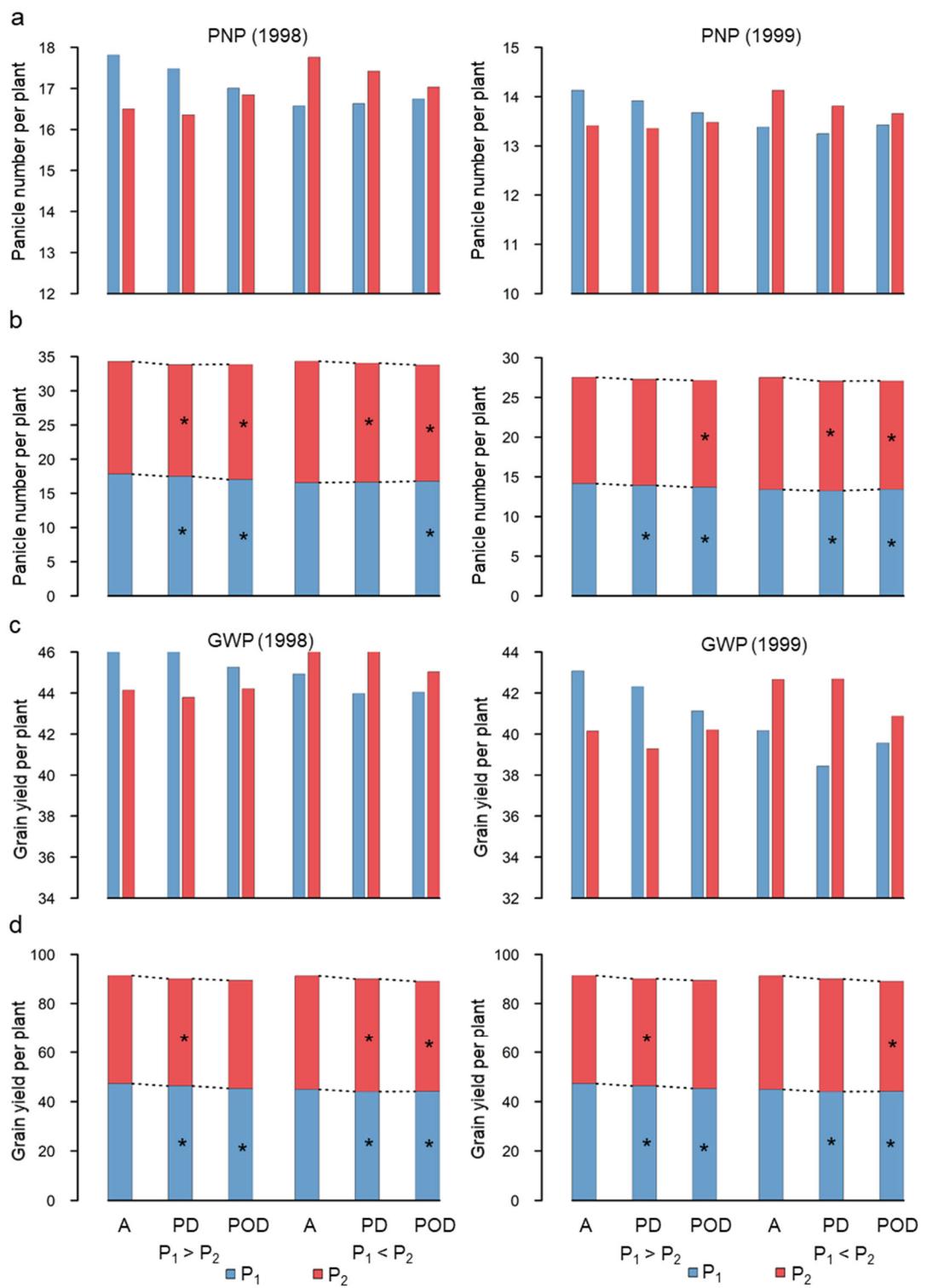
709 **Supplementary Figure 46 (continued)**

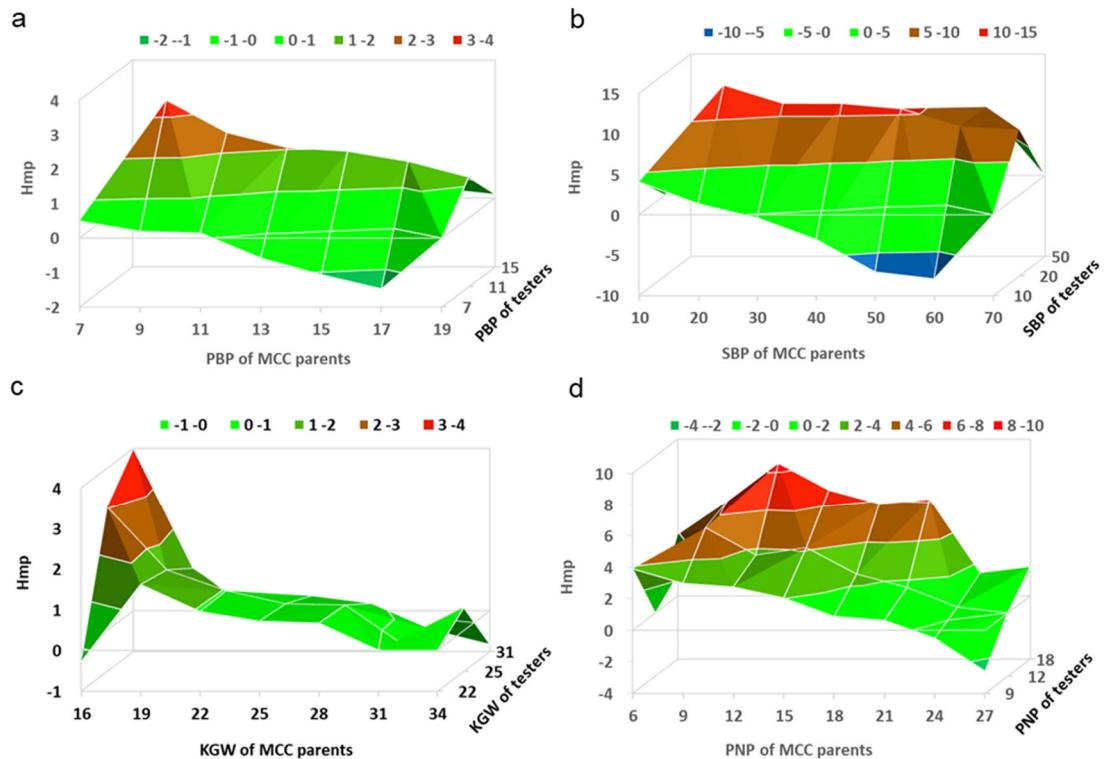
710



711

712 **Supplementary Figure 47** The yield traits of lines with Zhenshan97 (P_1) genotype and Minghui63
 713 (P_2) genotype of those QTLs with different genetic effect types in IMF₂ population in 1998 and 1999.
 714 Here, A, PD and POD mean the additive, positive dominant and positive over-dominant QTLs,
 715 respectively. $P_1 > P_2$ means that P_1 contains the genotype with higher effect of the QTL, and on the
 716 contrary $P_1 < P_2$ means that P_2 contains the genotype with higher effect of the QTL. The star means
 717 significant difference from the additive QTLs. The QTLs were identified according to the published data
 718 (Zhou, G. et al. Genetic composition of yield heterosis in an elite rice hybrid. Proceedings of the National

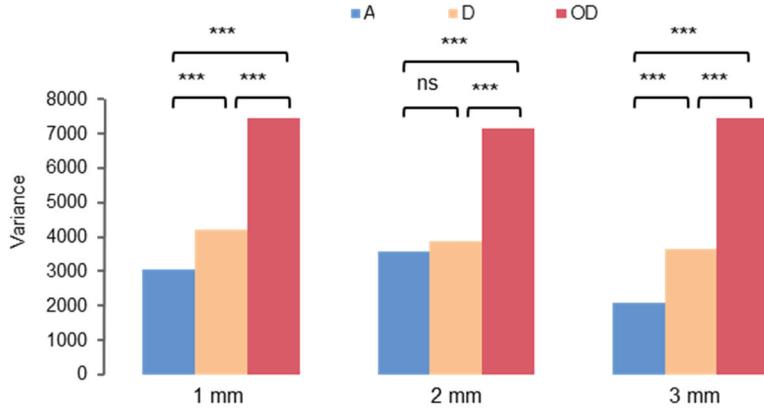




722

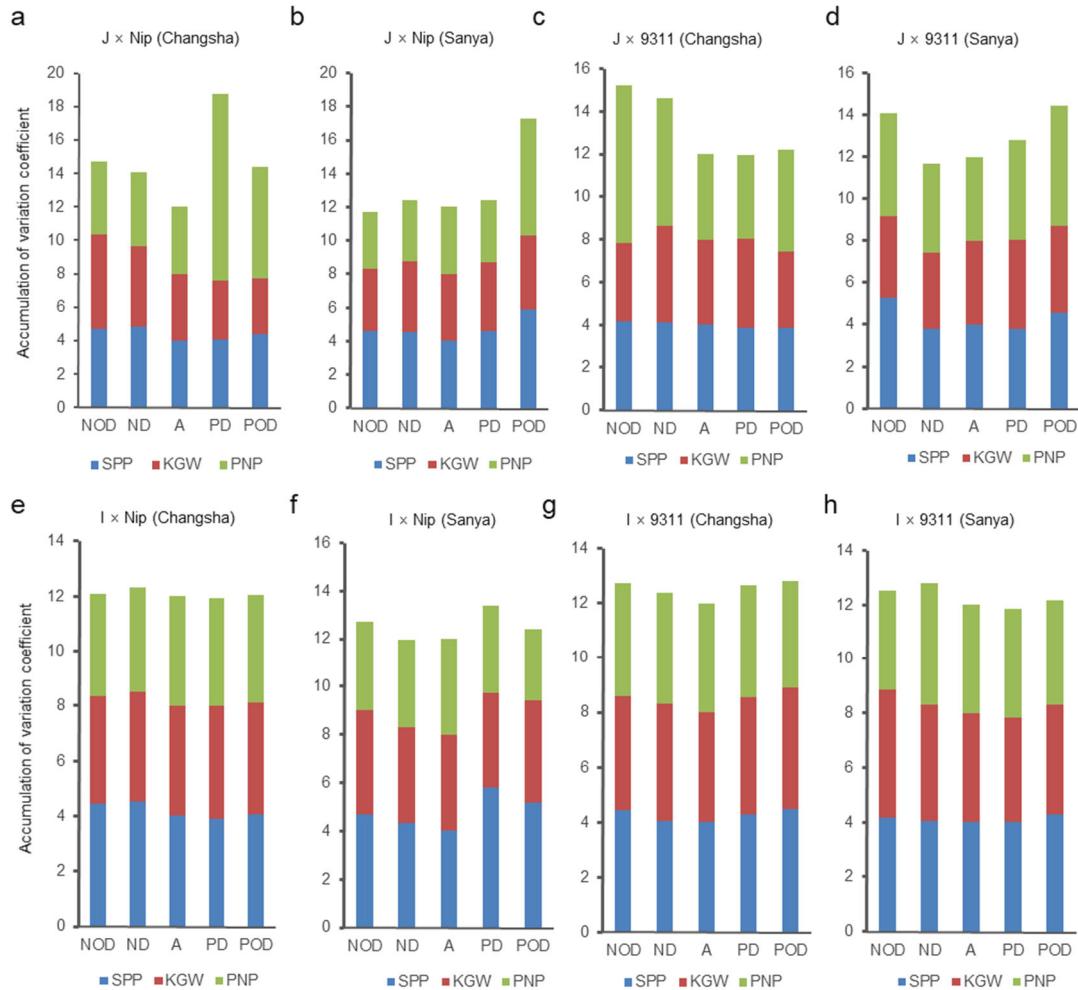
723 **Supplementary Figure 48** The relationship between the middle-parent heterosis (Hmp) and the
724 phenotypes of their parents for primary branch number per panicle (a), secondary branch number
725 per panicle (b), 1000-grain weight (c) and panicle number per plant (d). The results were calculated
726 according to the phenotype of 418 combinations of MCC in Changsha and Sanya.

727



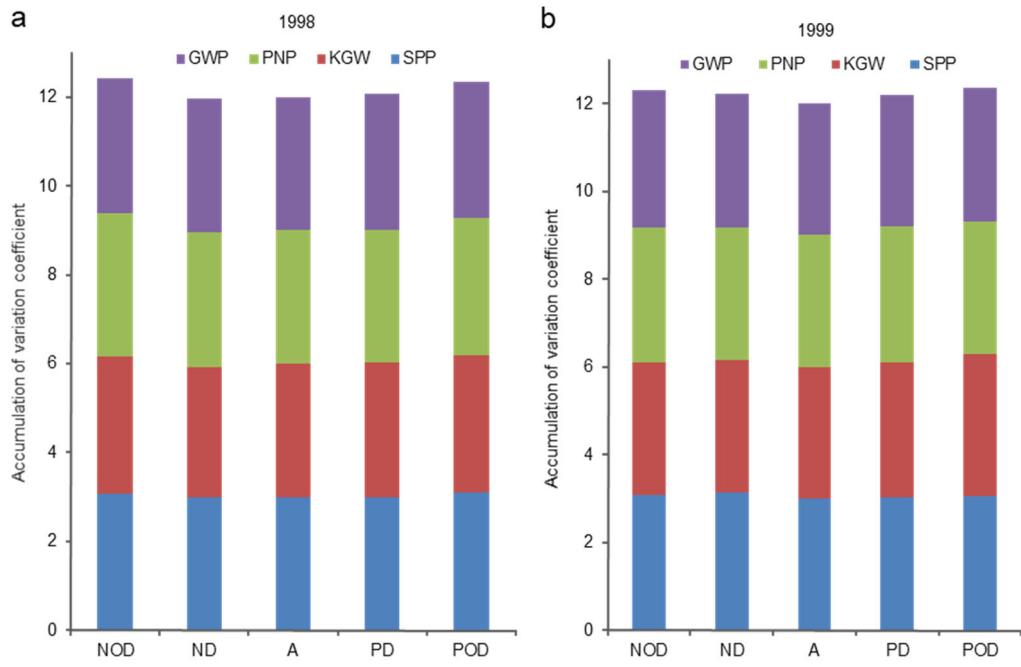
728

729 **Supplementary Figure 49** The variance of expression levels among different tissues (1 mm, 2 mm
730 and 3mm young panicles) for genes with additive, dominant and over-dominant expression patterns
731 in 1mm, 2mm or 3mm young panicles of hybrids. Triple-star means significant difference with p <
732 0.001; ns means no significance. The variance was estimated from 48 dataset collected from ricexpro
733 (<http://ricexpro.dna.affrc.go.jp/>)



734

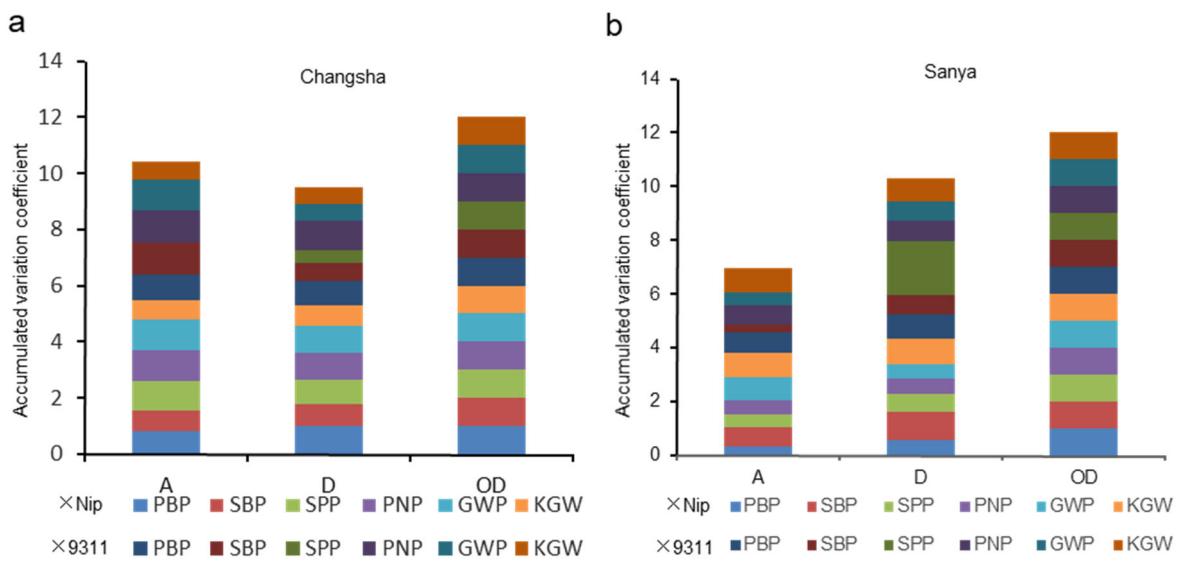
735 **Supplementary Figure 50 The accumulation of average variation coefficient estimated in each**
 736 **identified QTL across four kinds of combination for different type of genetic component.** Here NOD,
 737 ND, A, PD and POD represent the type of negative over-dominant, negative dominant, additive, positive
 738 dominant and positive over-dominant QTLs.



739

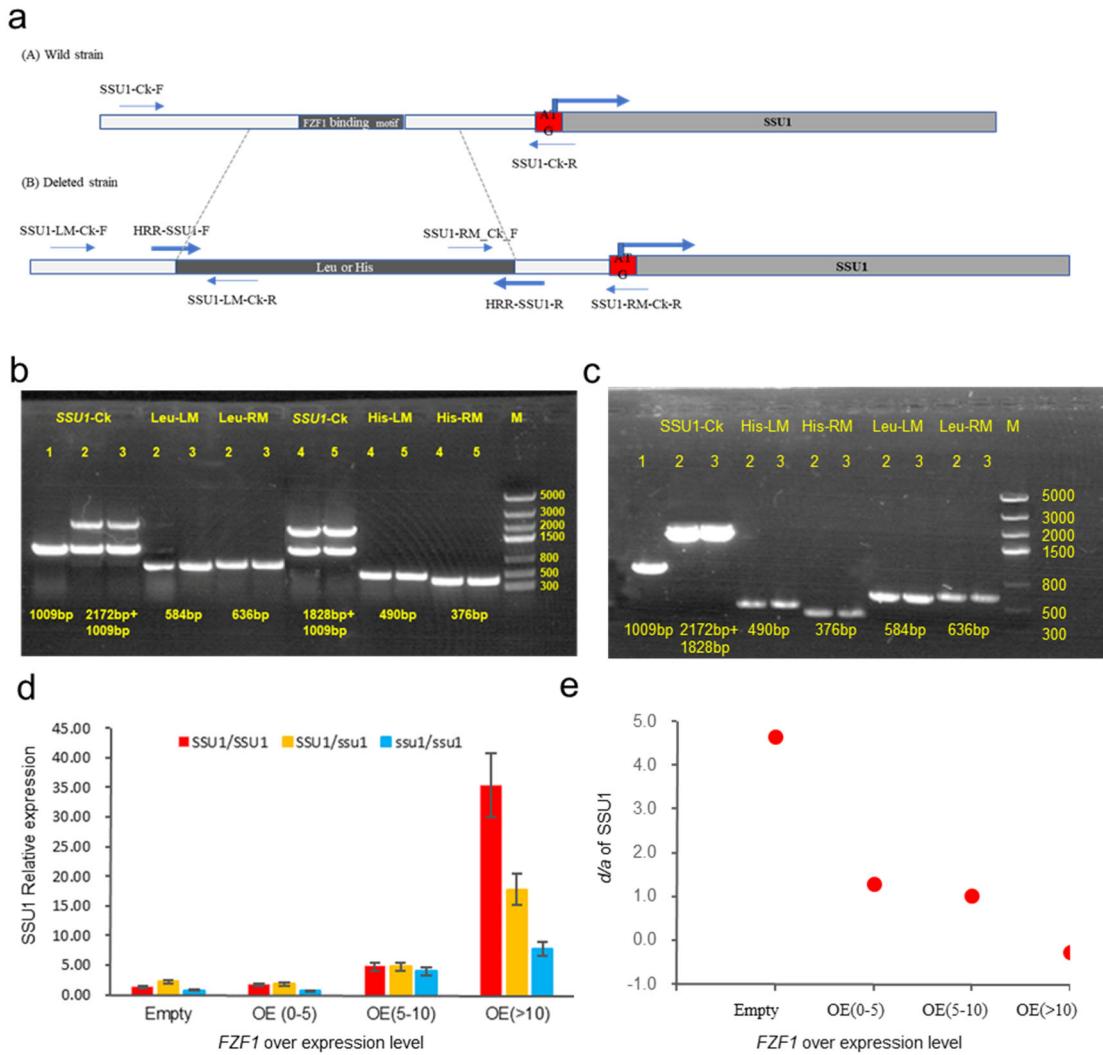
740 **Supplementary Figure 51 The accumulation of average variation coefficient estimated in each**
 741 **identified QTL for different type genetic component in IMF₂ population. (a) The comparisons of**
 742 **additive, dominance and overdominance for the accumulation of average variation coefficient estimated**
 743 **in each identified QTL at 1998. (b) The comparisons of additive, dominance and overdominance for the**
 744 **accumulation of average variation coefficient estimated in each identified QTL at 1999.**

745



746

747 **Supplementary Figure 52 The variance of different traits among environments for different degrees**
 748 **of dominant effects. (a) The accumulated variation coefficient of 6 yield related trait in Changsha. (b)**
 749 **The accumulated variation coefficient of 6 yield related trait in Sanya.**

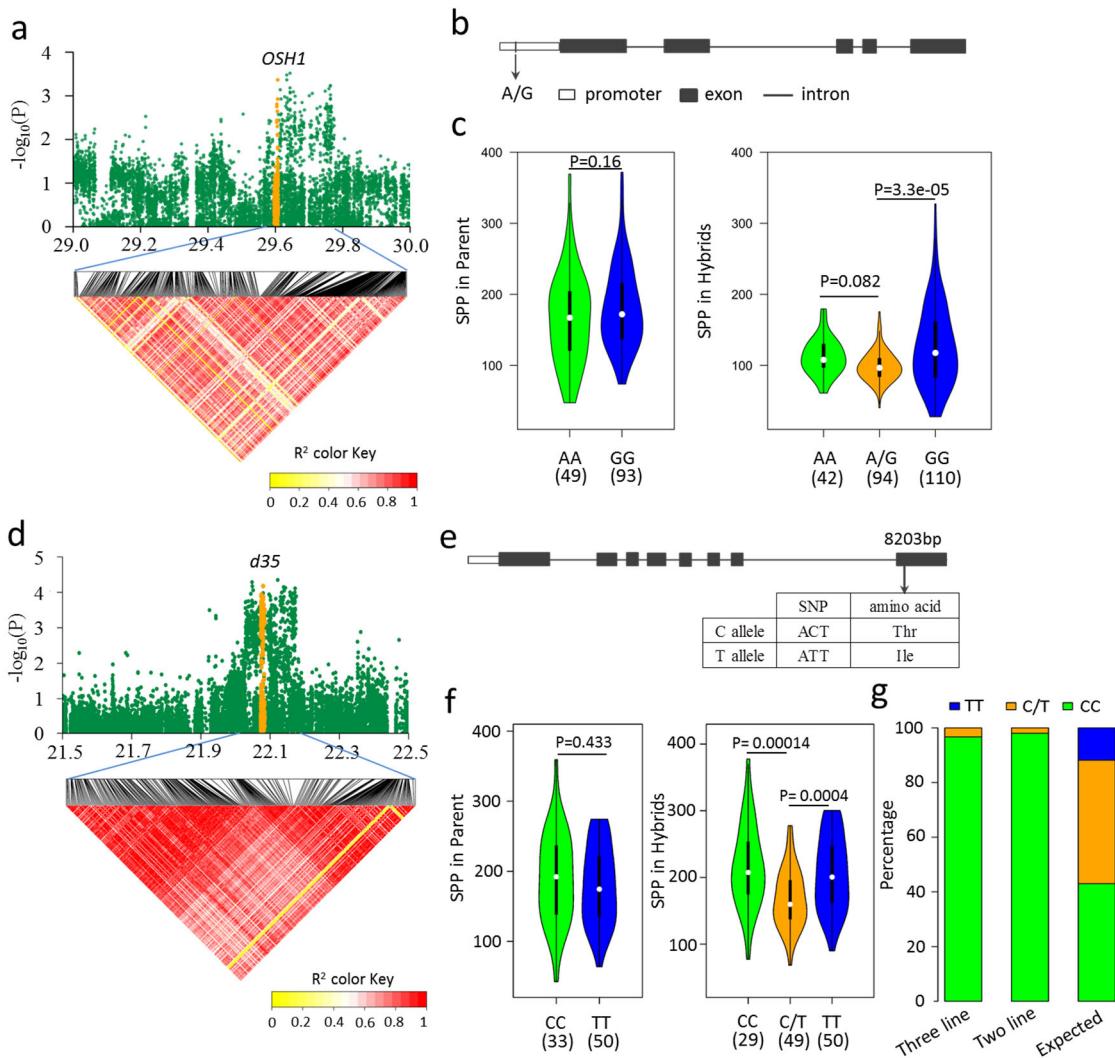


Supplementary Figure 53 Experimental validation of HoIIB model *Saccharomyces cerevisiae*. (a)

Primer design principles and requirements. SSU1_Ck-F and SSU1_Ck-R are located on both side of the transcription factor *FZF1* recognition motif in the *SSU1* gene promoter. SSU1-LM-Ck-R and SSU1-RM-Ck-F are located in inserted marker (marker can be Leu or His); HRR-SSU1-F and HRR-SSU1-R had 38bp homologous sequences on both sides of the *FZF1* recognition motif in *SSU1* promoter, and the outer 21bp sequence was the upstream and downstream primers for screening markers genes on the amplified plasmid (pfa6a-leu1mx or pFA6a-His3MX6). (b) 1 was genomic fragment containing the *FZF1* recognition motif in the wild type (BY4743), 2 and 3 were the genomic fragment (2.1kb and 1kb respectively) of heterozygous mutant that one copy of *FZF1* recognition motif was substituted by Leu and the other was remain unchanged; 4 and 5 were the genomic fragment (1.8kb and 1kb respectively) of heterozygous mutant that one copy of *FZF1* binding motif was substituted by His and the other was remain unchanged; Leu-LM was the primer used to amplify the left DNA fragment of Leu substitution genotype, Leu-RM was the primer used to amplify the right DNA fragment of Leu substitution genotype, His-LM was the primer used to amplify the left DNA fragment of His substitution genotype, Leu-RM was the primer used to amplify the right DNA fragment of Leu substitution genotype. (c) 1 was genomic fragment containing the *FZF1* recognition motif in the wild type (BY4743), 2 and 3 were the genomic fragment (2.1kb and 1kb respectively) of diploid mutant that one copy of *FZF1* recognition motif was substituted by Leu and the other was substituted by His; (d) The relative expression of gene *SSU1* in

769 different *SSU1* genotypes under different expression levels of its transcription factor (*FZF1*) in
770 *Saccharomyces cerevisiae* BY4743; here, AA, aa and Aa represent the homologous genotype of wild type,
771 the homologous genotype of mutant, and their heterozygous genotype, respectively; OE(0-5) means the
772 strain with upregulated *FZF1* by 0-5 folds, and similar for OE(5-10) and OE(>10), and Empty means the
773 strain with empty vector free of *FZF1*. (e) The dramatically decreased dominance degree of *SSU1* along
774 with the increase of upregulation levels of its transcription factor *FZF1* in *Saccharomyces cerevisiae*
775 BY4743.

776

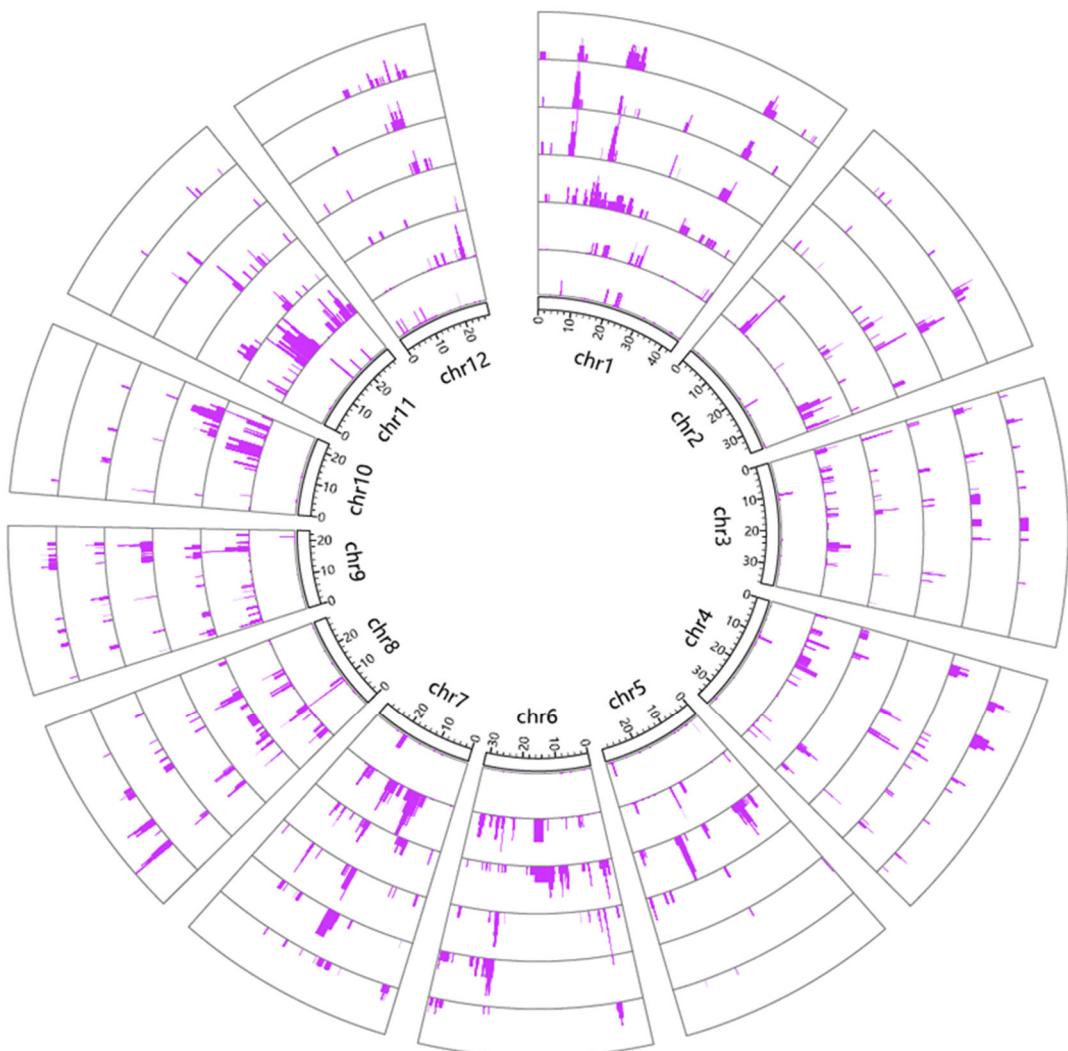


777

778 **Supplementary Figure 54 The association of previously identified genes related to rice spikelet**
779 **number per panicle (SPP).** (a) The associated QTL and LD heat map within the QTL for SPP gene
780 *OSH1*. The orange dots are the SNPs within gene *OSH1*. (b) The structure and the peak association signal
781 of gene *OSH1*. The SNP A/G on promoter significantly associates with the spikelet number per panicle in
782 F_1 of J×Nip. (c) Violin plots of *OSH1* genotypes for SPP in parents and hybrids. (d) The associated QTL
783 and LD heat map within the QTL for SPP gene *d35*. The orange dots are the SNPs within gene *d35*. (e)
784 The structure and the peak association signal of gene *d35*. The SNP C/T on the last exon significantly
785 associates with the spikelet number per panicle in F_1 of J×Nip. (f) Violion plots of *d35* genotypes for SPP
786 in parents and hybrids. (g) The genotype frequency of the SNP C/T in *d35* in the three-line and two-line

787 hybrids of *indica* subspecies, and the expected genotype frequency in combinations of *indica* varieties.

788



789

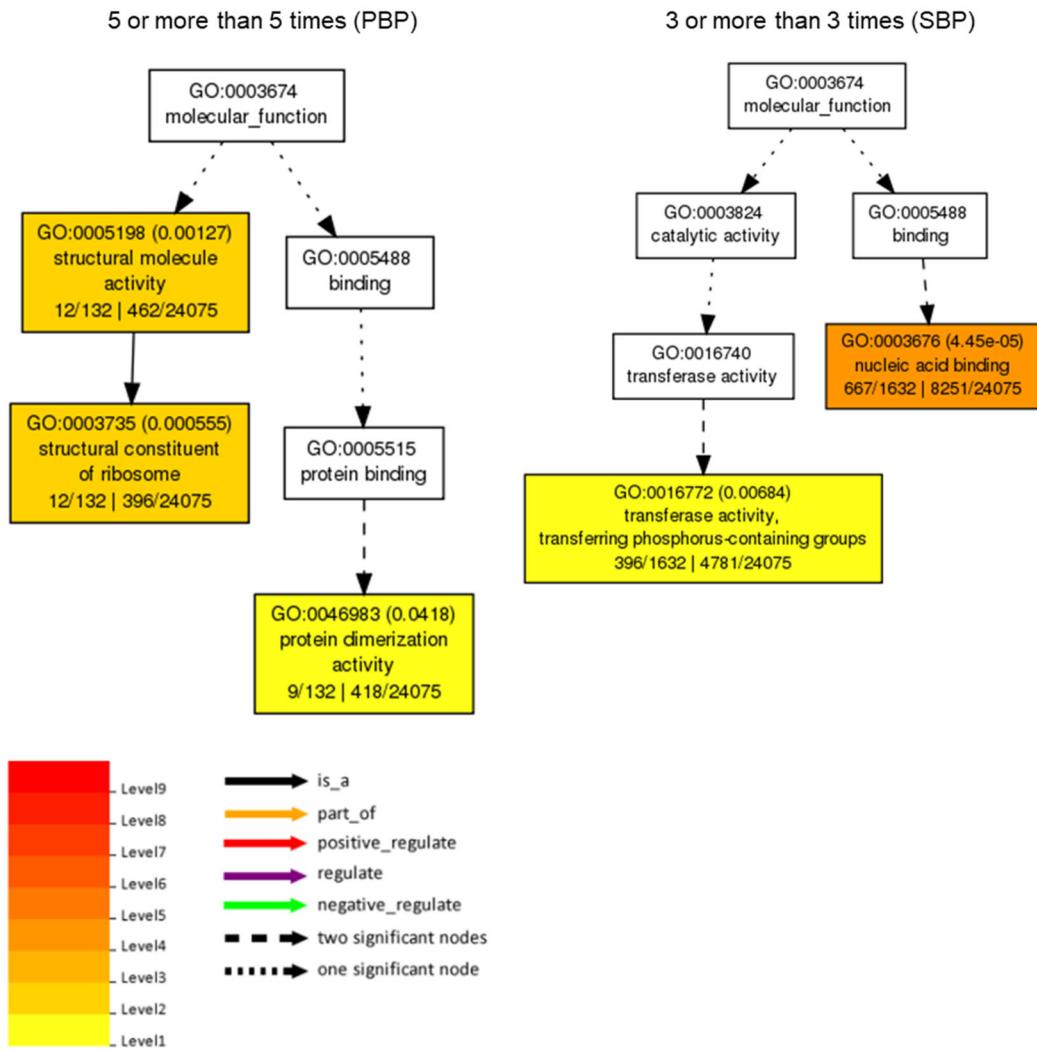
790 **Supplementary Figure 55 The times of associated genes that can be repeatedly identified in the**

791 **dominance and over-dominance QTLs across four kinds of combinations and two environments.**

792 Each line represents one associated gene. From the inner to outer layer, the height of lines represents 2-4

793 (for GWP), 3-6 (for PNP), 3-8 (for KGW), 3-6 (for SPP), 3-7 (for SBP) and 3-8 (for PBP) times,

794 respectively.



795

796 **Supplementary Figure 56 The GO enrichment of repeated identified non-additive genes in PBP,**
 797 **SBP, SPP, KGW, PNP and GWP across four kinds of combination and two environments for rice.**
 798 Only the term of molecular function was showed in figure, the other results were prepared in
 799 **Supplementary table 14.**

4 or more than 4 times (SPP)

GO:0003674
molecular_function

GO:0005488
binding

GO:0045735 (0.0184)
nutrient reservoir
activity
7/311 | 77/24075

GO:0043167
ion binding

GO:0043169
cation binding

GO:0046872
metal ion binding

GO:0046914
transition metal
ion binding

GO:0030145 (0.00697)
manganese ion
binding
7/311 | 58/24075

5 or more than 5 times (KGW)

GO:0003674
molecular_function

GO:0003824
catalytic activity

GO:0005488
binding

GO:0009055 (0.00584)
electron carrier
activity
25/381 | 599/24075

GO:0016787
hydrolase activity

GO:0016491
oxidoreductase activity

GO:0016788
hydrolase activity,
acting on ester bonds

GO:0004497 (0.00185)
monooxygenase activity
23/381 | 458/24075

GO:0042578
phosphoric ester
hydrolase activity

GO:0016791
phosphatase activity

GO:0003993 (0.00133)
acid phosphatase
activity
8/381 | 41/24075

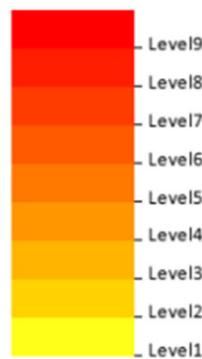
GO:0046906 (0.0033)
tetrapyrrole binding
26/381 | 608/24075

GO:0046872
metal ion binding

GO:0046914
transition metal
ion binding

GO:0005506 (0.0115)
iron ion binding
27/381 | 713/24075

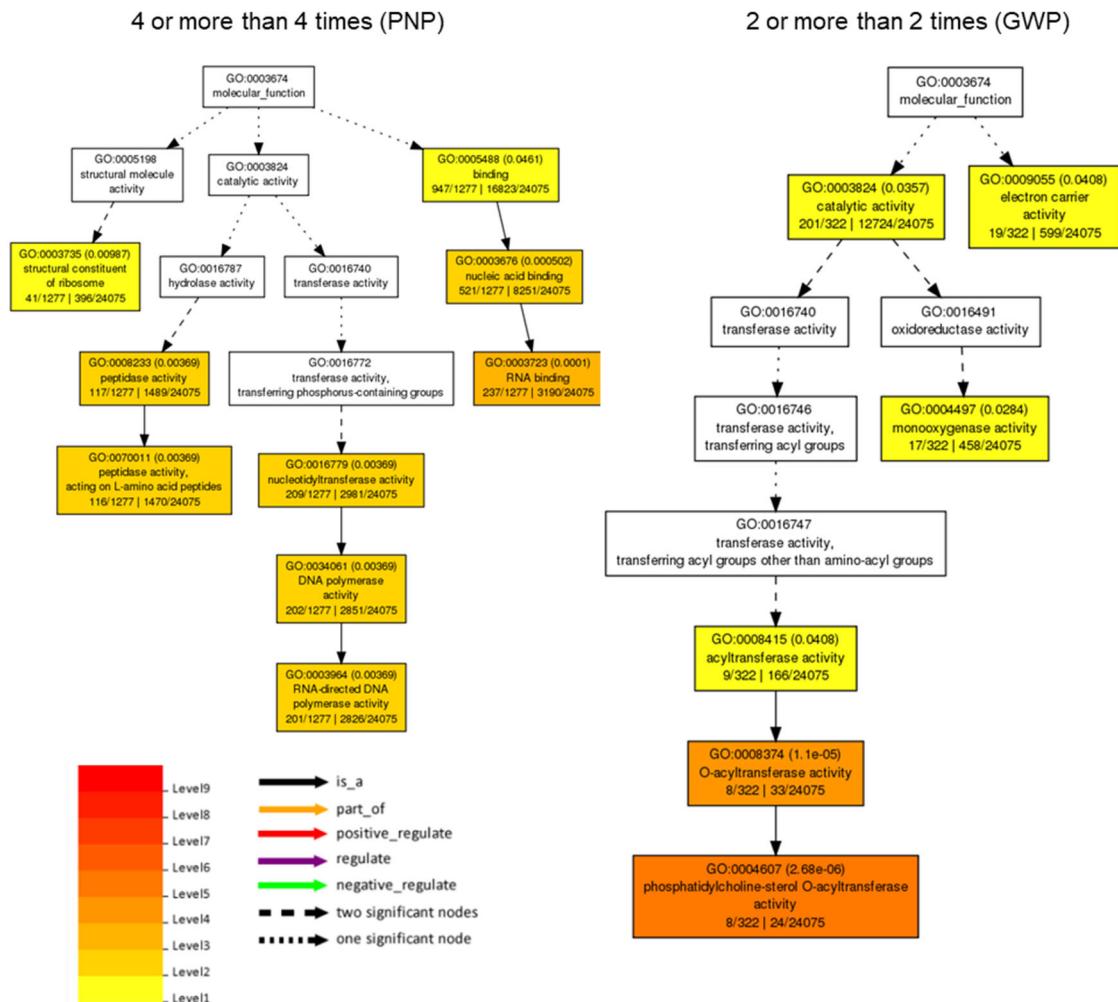
GO:0020037 (0.0033)
heme binding
26/381 | 600/24075



- is_a
- part_of
- positive_regulate
- regulate
- negative_regulate
- two significant nodes
- one significant node

800

801 **Supplementary Figure 56 continued**

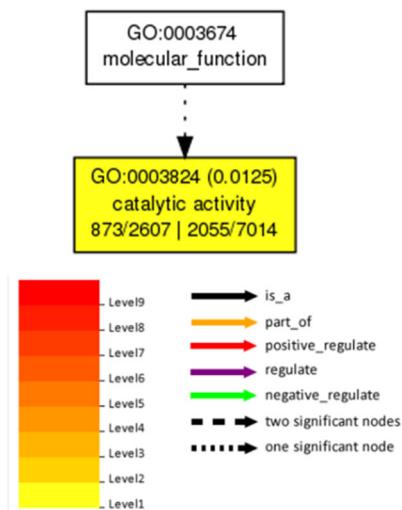


802

803 **Supplementary Figure 56 continued**

804

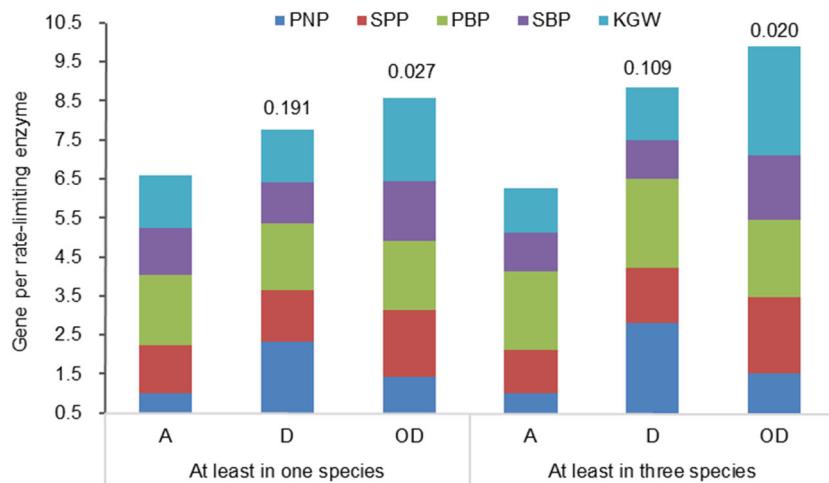
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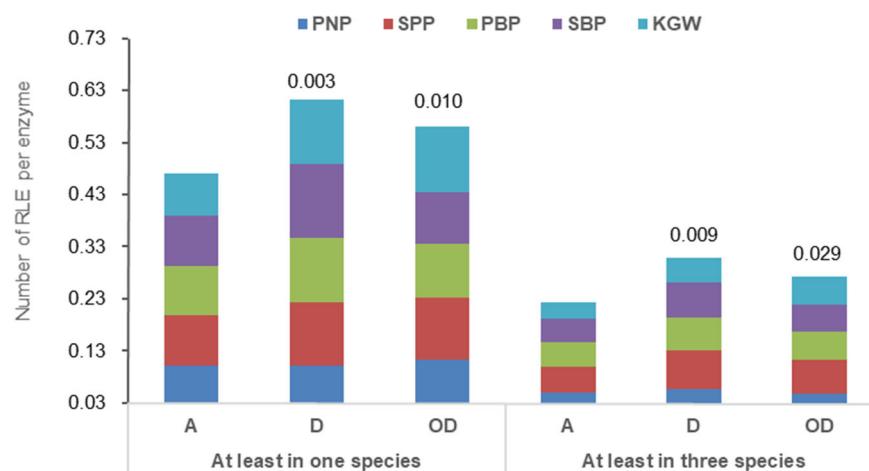
806

807 **Supplementary figure 57 The GO enrichment of repeated identified genes with non-additive**
 808 **performance in non-lethal deletion yeast strains grown in five media. Only the term of molecular**
 809 **function was showed in the figure.**

a

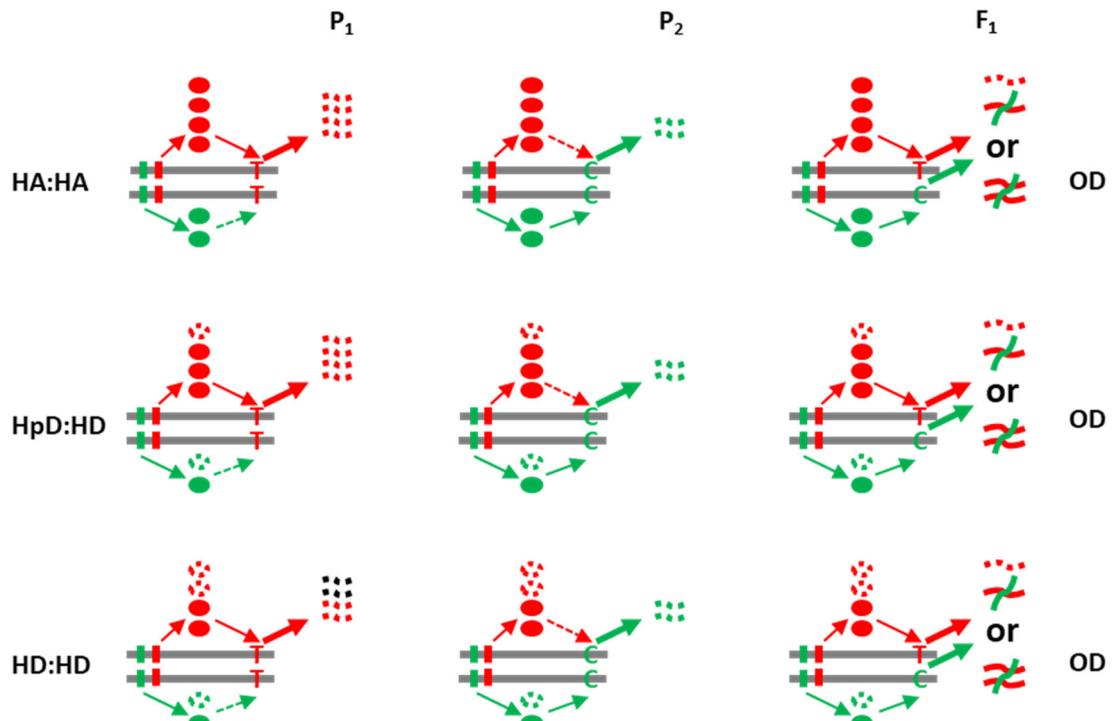


b



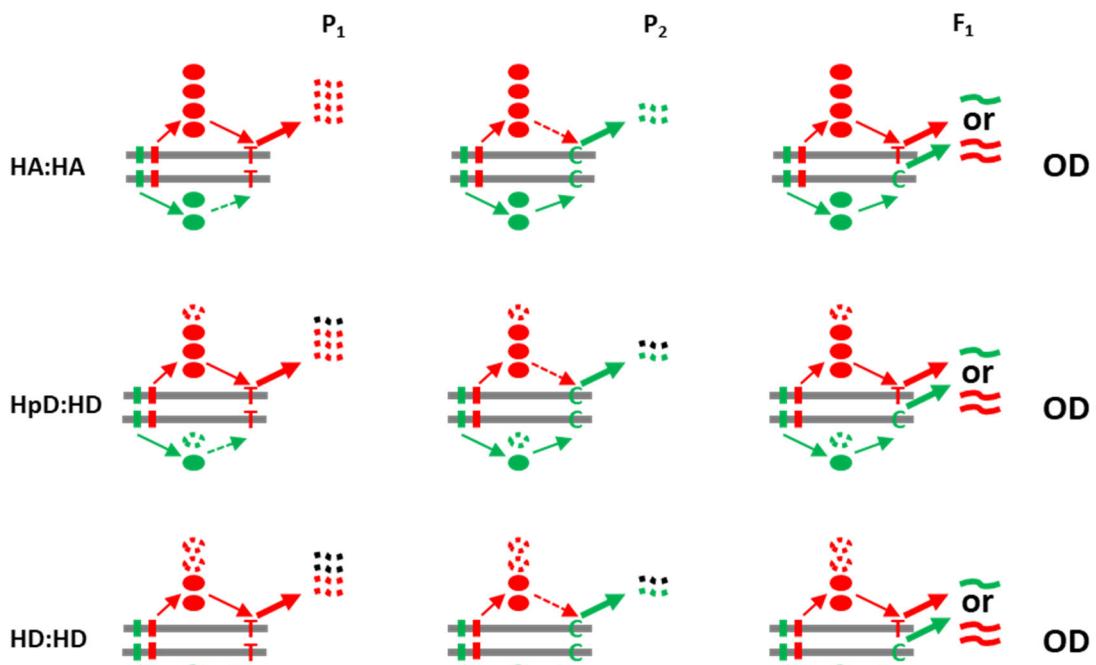
810

811 **Supplementary figure 58 The distribution of rate-limiting enzymes coded by candidate genes within**
812 **additive, dominant and over-dominant QTLs related to five yield component trait including PBP,**
813 **SBP, SPP, KGW and PNP.** The rate-limiting enzymes were identified in species yeast, mouse, and
814 Human. (a) the number of genes per enzyme for additive, dominance and over-dominance candidate
815 genes. (b) the number of genes per rate limiting enzyme for additive, dominance and over-dominance
816 candidate genes. The P value at the top of bar means significant difference from the additive.



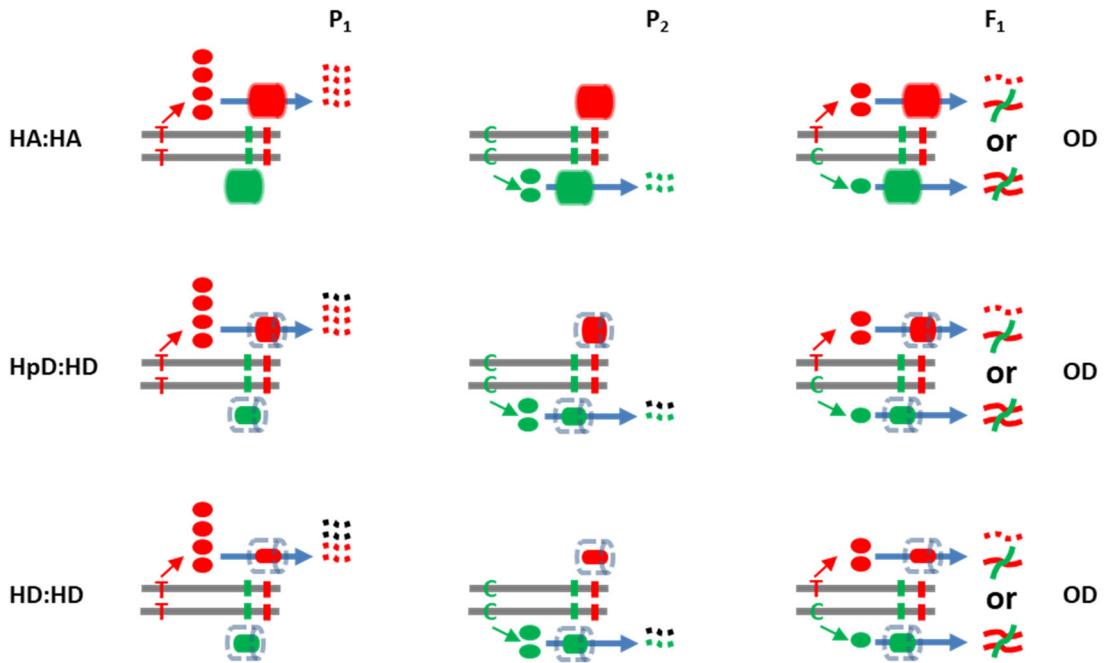
817

818 **Supplementary Figure 59 The schematic diagram of regulation model for molecular mechanism of**
 819 **additive, dominant and over-dominant effect produced by the parallel complementation of two**
 820 **alleles of one polymorphic site under two independent regulators as the upstream backgrounds.** The
 821 grey thick lines show two chromosomes, the red and green bars on which represent the homologous
 822 alleles of the regulators of T and C alleles at target site, respectively; and the function of these regulators
 823 keep constant among P₁, P₂ and F₁. The break and solid pies together represent the required regulator
 824 function that can maximize the output of the homozygote of the corresponding target allele, and the solid
 825 pies represent different regulator functions and thus provide different backgrounds to the target allele. The
 826 colored and black break curves together represent the maximum output of the homozygote in parents or
 827 one allele in F₁. The output of T without C or output of C without T does not take effect, and thus
 828 represented by the red or green break curves respectively for allele T or C; and the combined outputs of T
 829 and C can only take effect, as indicated by the cross solid curves. HA:HA means the complementation of
 830 the hidden additive effect of allele T and the hidden additive effect of allele C, in that the output of allele
 831 T is additive but non-functional itself and that of allele C does too; and HpD:HD and HD:HD are similar.
 832 HpD means hidden partial dominant.



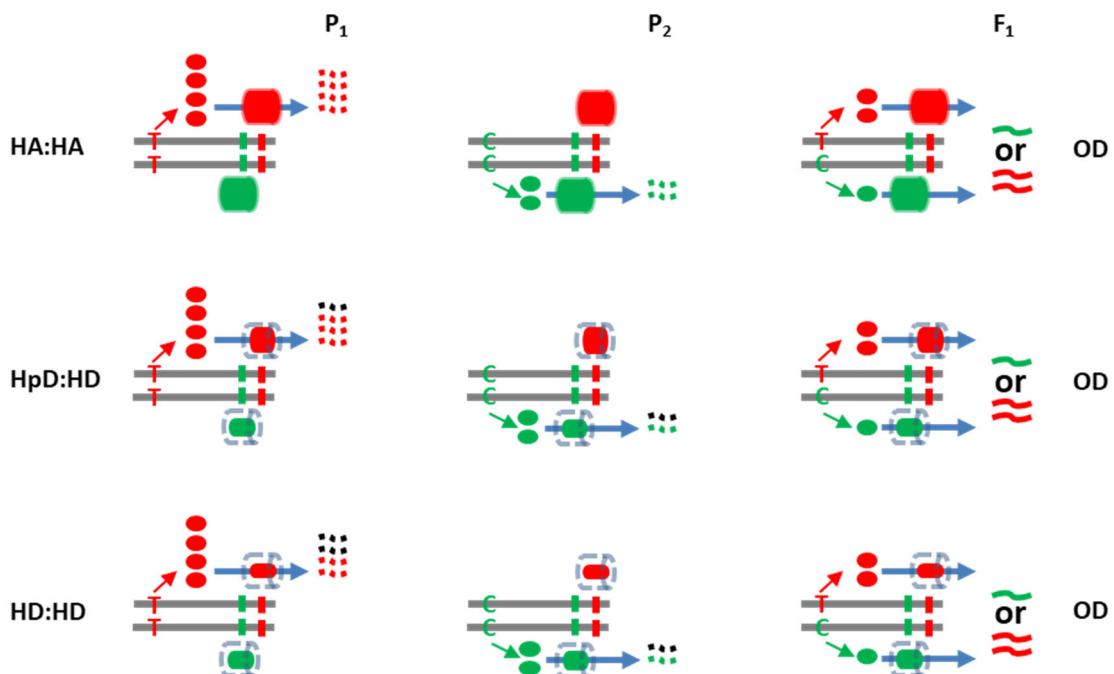
833

834 **Supplementary Figure 60** The schematic diagram of regulation model for molecular mechanism of
 835 additive, dominant and over-dominant effect produced by the sequential complementation of two
 836 alleles of one polymorphic site under two independent regulators as the upstream backgrounds. The
 837 grey thick lines show two chromosomes, the red and green bars on which represent the homologous
 838 alleles of the regulators of T and C alleles at target site, respectively; and the function of these regulators
 839 keep constant among P_1 , P_2 and F_1 . The break and solid pies together represent the required regulator
 840 function that can maximize the output of the homozygote of the corresponding target allele, and the solid
 841 pies represent different regulator functions and thus provide different backgrounds to the target allele. The
 842 colored and black break curves together represent the maximum output of the homozygote in parents or
 843 one allele in F_1 . The output of T without C or output of C without T does not take effect, and thus
 844 represented by the red or green break curves respectively for allele T or C; and the output of T following
 845 that of C or the output of C following that of T can only take effect, as indicated by the solid curves.
 846 HA:HA means the complementation of the hidden additive effect of allele T and the hidden additive
 847 effect of allele C, in that the output of allele T is additive but non-functional itself and that of allele C
 848 does too; and HpD:HD and HD:HD are similar. HpD means hidden partial dominant.



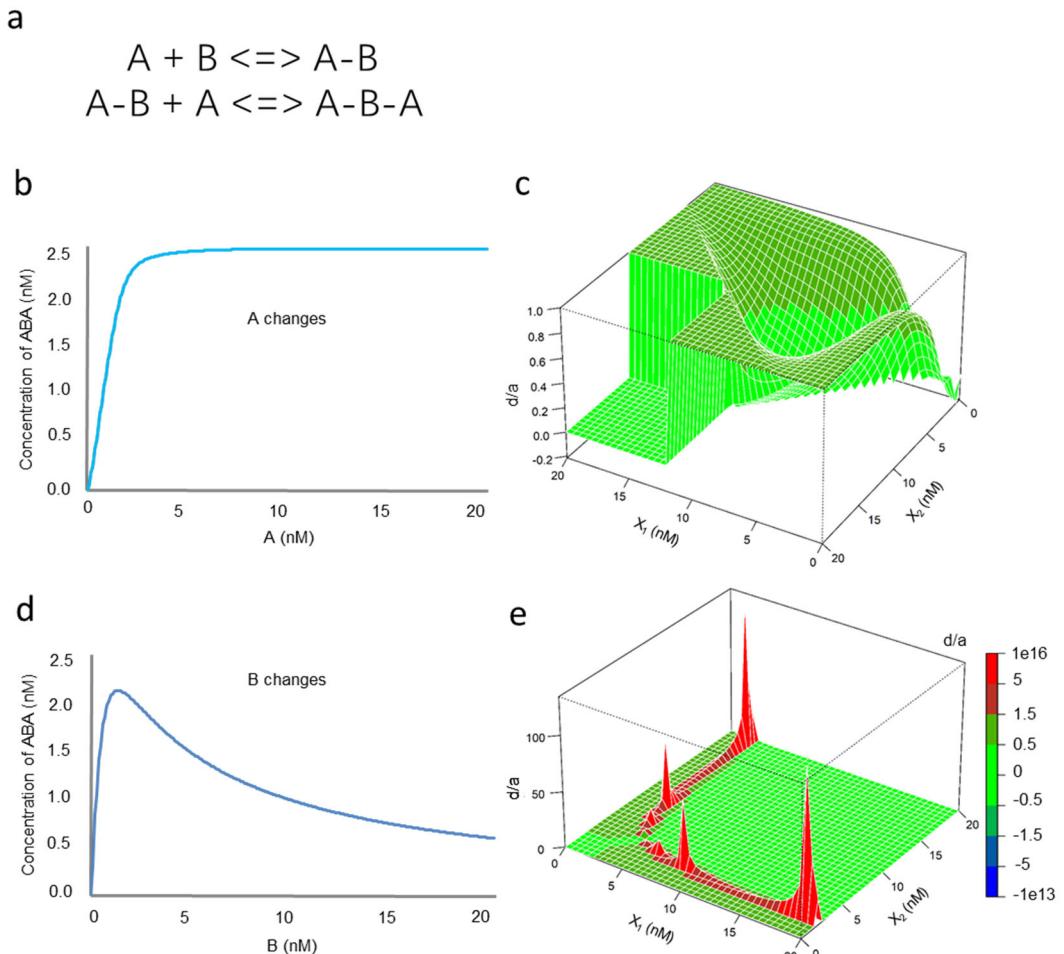
849

850 **Supplementary Figure 61 The schematic diagram of regulation model for molecular mechanism of**
 851 **additive, dominant and over-dominant effect produced by the parallel complementation of two**
 852 **alleles of one polymorphic site under two independent regulators or responders as the downstream**
 853 **backgrounds.** The grey thick lines show two chromosomes, the red and green bars on which represent
 854 the homologous alleles of the regulators or responders of T and C alleles at target site, respectively; and
 855 the function of these regulators or responders keep constant among P_1 , P_2 and F_1 . Different numbers of red
 856 and green pies represent the maximum outputs of the homozygotes of allele T and C of target site,
 857 respectively. The doted cylinder or the same size of solid cylinder represent the required regulator or
 858 responder function that can transform the full maximum output of the homozygote of the corresponding
 859 target allele, with red corresponding to allele T and green to allele C; and the solid cylinders in doted
 860 cylinder represent different regulator or response functions and thus provide different backgrounds to the
 861 target allele. The arrow represents the function process. The transformed output of T without that of C or
 862 the transformed output of C without that of T does not take effect, and thus represented by the red or
 863 green break curves respectively for allele T or C; and the combined transformed outputs of T and C can
 864 only take effect, as indicated by the cross solid curves. HA:HA means the complementation of the hidden
 865 additive effect of allele T and the hidden additive effect of allele C, in that the output of allele T is
 866 additive but non-functional itself and that of allele C does too; and HpD:HD and HD:HD are similar.
 867 HpD means hidden partial dominant.



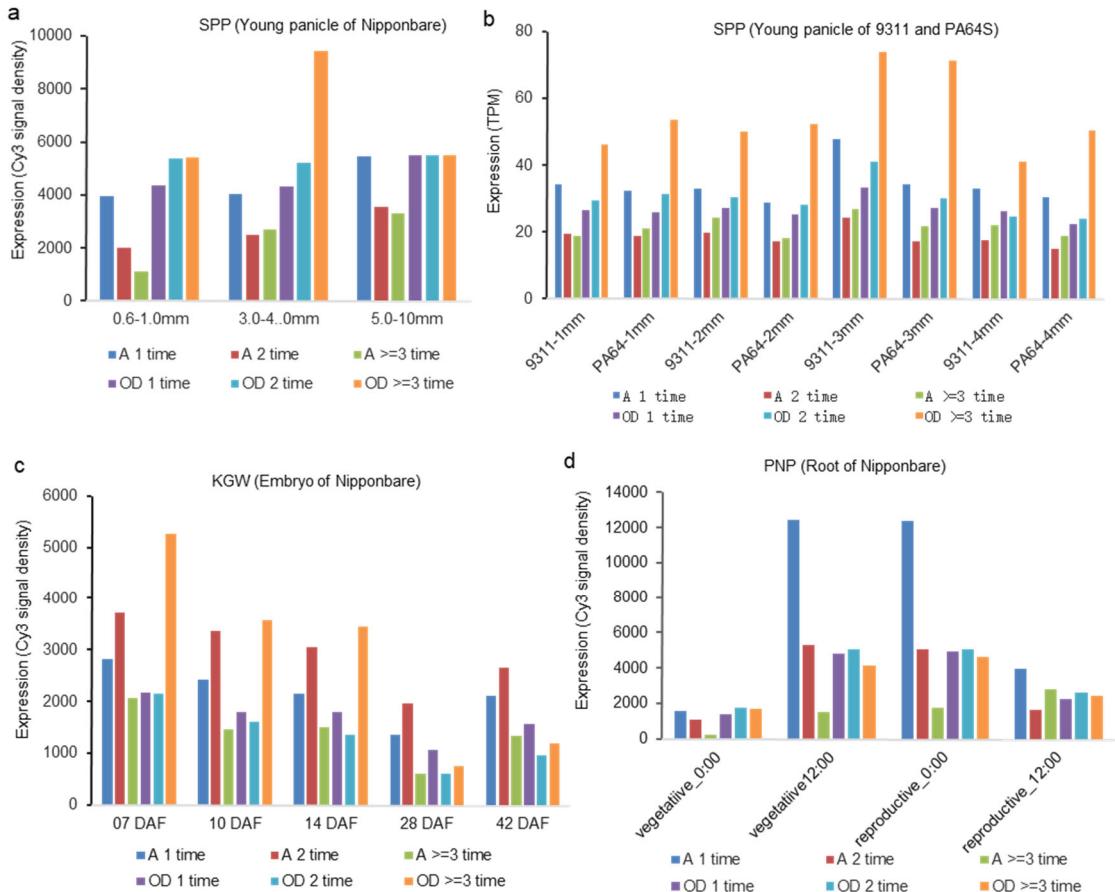
868

869 **Supplementary Figure 62** The schematic diagram of regulation model for molecular mechanism of
 870 additive, dominant and over-dominant effect produced by the sequential complementation of two
 871 alleles of one polymorphic site under two independent regulators or responsors as the downstream
 872 backgrounds. The grey thick lines show two chromosomes, the red and green bars on which represent
 873 the homologous alleles of the regulators or responsors of T and C alleles at target site, respectively; and
 874 the function of these regulators or responsors keep constant among P₁, P₂ and F₁. Different numbers of red
 875 and green pies represent the maximum outputs of the homozygotes of allele T and C of target site,
 876 respectively. The doted cylinder or the same size of solid cylinder represent the required regulator or
 877 responsor function that can transform the full maximum output of the homozygote of the corresponding
 878 target allele, with red corresponding to allele T and green to allele C; and the solid cylinders in doted
 879 cylinder represent different regulator or response functions and thus provide different backgrounds to the
 880 target allele. The arrow represents the function process. The transformed output of T without that of C or
 881 the transformed output of C without that of T does not take effect, and thus represented by the red or
 882 green break curves respectively for allele T or C; and the transformed outputs of T following the
 883 transformed output of C or the transformed outputs of C following the transformed out of T can only take
 884 effect, as indicated by the solid curves. HA:HA means the complementation of the hidden additive effect
 885 of allele T and the hidden additive effect of allele C, in that the transformed output of allele T is additive
 886 but non-functional itself and that of allele C does too; and HpD:HD and HD:HD are similar. HpD means
 887 hidden partial dominant.



888

889 **Supplementary Figure 63 The simulated diagram of regulation model for molecular mechanism of**
 890 **additive and dominant effect produced by single site in relation to the assembly of functional trimer**
 891 **ABA.** (a) The schematic diagram depicting the assembly of trimer ABA. In this model, the nonfunctional
 892 dimer AB can compete component B against the functional trimer ABA. (b) The simulated curve of ABA
 893 concentration in the equilibrium state with different concentrations of subcomponent A and keeping
 894 subcomponent B constant. The sigmoidal curve indicates that the sensitivity of ABA concentration to the
 895 concentration of A will decrease along with the decrease of background sufficiency as denoted by the
 896 increased ratio between A and B, and a nearly complete dominant effect will occur when the
 897 concentration of A reaches nearly 2X that of B. (c) The dominant degree of the timer ABA produced by
 898 different concentrations of subcomponent A in two parents (X_1 and X_2). (d) The simulated curve of ABA
 899 concentration in the equilibrium state with different concentrations of subcomponent B and keeping
 900 subcomponent A constant. The curve indicates that the sensitivity of ABA concentration to the
 901 concentration of B will decrease and nearly lost along with the decrease of background sufficiency as
 902 denoted by the increased ratio between B and A. (e) The dominant degree of the timer ABA produced by
 903 different concentrations of subcomponent B in two parents (X_1 and X_2). All the simulated results
 904 indicated that the non-additive effect usually generates under insufficient background. Detail of the model
 905 and the values of parameters can be found in **supplementary note**.



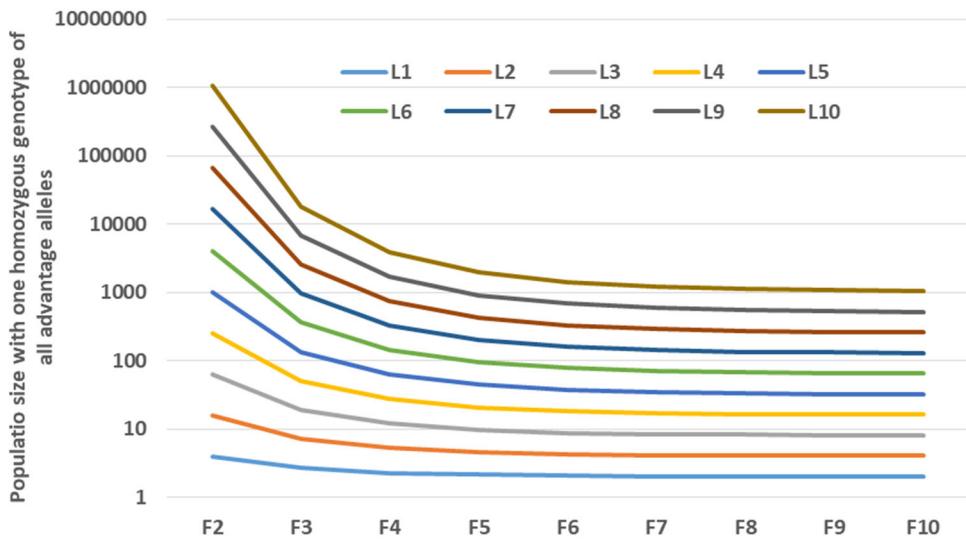
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907

Supplementary Figure 64 The expression level of associated candidate genes within additive (A) and over dominant (OD) QTLs. (a) The expression level of associated candidate genes of SPP QTLs in young panicles of Nipponbare. (b) The expression level of candidate genes of SPP QTLs in the young panicles of 9311 and PA64S. (c) The expression level of associated candidate genes of KWG QTLs in the embryo of Nipponbare. (d) The expression level of associated candidate genes of PNP QTLs in the root of Nipponbare. The raw data of gene expression in (a), (c) and (d) were obtained from the database of RiceXpro. 1 time, 2 time and ≥ 3 times means those candidate genes within the QTLs that can be detected in 1, 2, and 3 or more than 3 situations among four combinations under two environments.

914

915



916
917 **Supplementary Figure 65 The theoretical population size with at least one homozygous genotype of**
918 **all advantage alleles in different generations of two parents with different numbers of polymorphic**
919 **loci (from 1 to 10).** Here, we calculate the population under the hypothesis that there is no linkage
920 between loci and all loci are randomly combined; L1 - L10 mean the locus number from 1 to 10; F2 - F10
921 mean the self-crossing generations from 2 to 10.