Plant breeding - principles and methods - practical exercises

Inheritance of qualitative traits – Analyze segregation results and predict the genetic basis of a trait

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Literature


Overview

Molecular basis of allele variations

Single gene inheritance: dominant and recessive
incomplete dominance
codominance, overdominance
recessive lethal alleles

Two genes inheritance: epistasis
additive genes

Chi-square Test ($\chi^2$-Test) to test for segregation ratios
Analyze segregation results and predict the genetic basis of a trait
Qualitative versus quantitative traits

**Qualitative traits** – controlled by one (few) major gene(s), phenotypic variation can be separated into distinct classes; generally, the environment has little influence.

**Quantitative trait** – controlled by several to many genes having small, cumulative effects, can be measured in quantitative units that are continuous, and is often considerably influenced by environment.

Some characters difficult to categorized. Major gene(s) modified by minor genes plus environmental effects -> the phenotype of such characters may show continuous variation.

-> The range of variation for a particular trait indicates the mode of inheritance of that trait.

To determine the mode of inheritance: mate plants having contrasting phenotypes and evaluate the performance of their offspring. The proportion of progeny exhibiting different phenotypes provides information about the proportion of progeny possessing different genotypes.
Molecular basis of allele variations

Alleles: alternative forms (of the same gene) at one genetic locus
Caused by mutations of the DNA codes (Substitution/Insertion/Deletion)
Many alleles at one locus possible: the ‘normal functional’ – wild-type allele and mutant alleles.

Point mutation: SNP (single nucleotide polymorphism) in gene Cbd of cotton

INDEL (Insertions-Deletions polymorphism): in gene Cbd of cotton

Molecular basis of allele variations

The expression of the products of wild-type alleles produces wild-type phenotype.

Null alleles produce no functional product. Homozygous ‘nulls’ have mutant phenotype - no gene product. Heterozygous produce less functional gene product than homozygous wild-types and may have mutant phenotype.

Mutant phenotype visible in heterozygous state?
Haplosufficiency of wild type allele (= dominant)  –  mutant allele not visible
Haploinsufficiency of wild type allele (= recessive)  –  mutant allele visible

Sanders modified
Molecular basis of allele variations

**Gain of function: Hypermorph mutation**

Excessive expression of the gene product leads to excessive gene action. The mutant phenotype may be more severe or lethal in the homozygous than in the heterozygous.

**Gain of function: Neomorph mutation**

The mutant allele has novel function that produces a mutant phenotype in homozygous and heterozygous organisms, and may be more severe in homozygous organisms.
Single gene inheritance: dominant and recessive

-> the purple inheritance factor is **dominant** and the factor governing “white” is **recessive**.

-> Complete dominance: homozygous dominant can not distinguished from heterozygous, $A/A = A/a$.

<table>
<thead>
<tr>
<th>Genotype:</th>
<th>1 $A/A$ : 2 $A/a$ : 1 $a/a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenotype:</td>
<td>3 $A$ : 1 $a$</td>
</tr>
</tbody>
</table>

$A/A$ → active enzyme → purple pigment

$A/a$ → active enzyme → purple pigment

$a/a$ → no active enzyme → white

Gametes

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>a</th>
</tr>
</thead>
<tbody>
<tr>
<td>F2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>AA</td>
<td>Aa</td>
</tr>
<tr>
<td>a</td>
<td>aA</td>
<td>aa</td>
</tr>
</tbody>
</table>
Incomplete dominance, partial dominance

The occurrence of intermediate phenotypes:
1:2:1 segregation in the F2
Classical example for flower colors:
In the heterzygous the amount of anthocyan is half – pink, dosage effect

Codominance

Codominance is defined as the expression in a heterozygote of both the phenotypes normally shown by the two alleles.
Overdominance

Phenotype of heterozygous outperforms phenotype of homozygous parental lines.

Overdominance is a genetic model for heterosis; it posits that increased heterosis is the result of positive interactions between two functional alleles that leads to a phenotypic value beyond the range of both homozygous parents.

Heterosis, hybrid vigor

Heterosis is a phenomenon whereby the phenotype of F1 hybrids is superior to that of their parents.

Other hypothesis: dominance, epistasis

Underdominance is the opposite of overdominance
**Recessive lethal alleles**

Mutant alleles of essential genes capable of causing death are called lethal alleles.

Mostly recessive, dominant lethal alleles are rarely maintained in populations.

Expected monohybride segregation ratio from 1:2:1 would just be found in zygotes. But zygotes with the homozygous lethal alleles do not survive and can not be counted -> 2:1 ratio.

Recessive sublethal allele: Letalität varies from 0 to 100 %, depending from the gene, rest of the genome and environment.
### Summary: alleles of one gene

<table>
<thead>
<tr>
<th>Type of dominance</th>
<th>( A^1/A^1 )</th>
<th>( A^2/A^2 )</th>
<th>( A^1/A^2 ) hybrids</th>
<th>( F_2 )</th>
<th>( F_\infty )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete</td>
<td><img src="image" alt="White square" /></td>
<td><img src="image" alt="Blue square" /></td>
<td><img src="image" alt="White square" /></td>
<td>( A^1 ) is dominant to ( A^2 ) ( A^2 ) is recessive to ( A^1 )</td>
<td>3:1</td>
</tr>
<tr>
<td>Complete</td>
<td><img src="image" alt="White square" /></td>
<td><img src="image" alt="Blue square" /></td>
<td><img src="image" alt="Blue square" /></td>
<td>( A^2 ) is dominant to ( A^1 ) ( A^1 ) is recessive to ( A^2 )</td>
<td>3:1</td>
</tr>
<tr>
<td>Incomplete</td>
<td><img src="image" alt="White square" /></td>
<td><img src="image" alt="Blue square" /></td>
<td><img src="image" alt="Light blue square" /></td>
<td>( A^1 ) and ( A^2 ) are incompletely dominant relative to each other</td>
<td>1:2:1</td>
</tr>
<tr>
<td>Codominant</td>
<td><img src="image" alt="White square" /></td>
<td><img src="image" alt="Blue square" /></td>
<td><img src="image" alt="Striped square" /></td>
<td>( A^1 ) and ( A^2 ) are codominant relative to each other</td>
<td>1:2:1</td>
</tr>
<tr>
<td>Overdominance</td>
<td><img src="image" alt="Light blue square" /></td>
<td><img src="image" alt="Blue square" /></td>
<td><img src="image" alt="Dark blue square" /></td>
<td>( A^1 ) and ( A^2 ) are together overdominant</td>
<td>1:2:1</td>
</tr>
<tr>
<td>Lethal allele</td>
<td><img src="image" alt="White square" /></td>
<td><img src="image" alt="Skull and crossbones" /></td>
<td><img src="image" alt="White square" /></td>
<td>( A^1 ) is dominant to ( A^2 )</td>
<td>1:2</td>
</tr>
</tbody>
</table>
Summary:
Interactions of alleles

The leaves of clover plants show several variations on the dominance theme. The different chevron forms (and the absence of chevrons) are determined by a series of alleles of one gene. The figure shows the many different types of interactions that are possible, even for one allele.

A gene can have several different states or forms — called multiple alleles. The alleles are said to constitute an allelic series, and the members of a series can show various degrees of dominance to one another.

Figure 6-12 Multiple alleles determine the chevron pattern on the leaves of white clover. The genotype of each plant is shown below it. (After photograph by W. Ellis Davies.)
Two genes inheritance

The F2 genotypes of 2 independently assorting genes with complete dominance result in a 9 : 3 : 3 : 1 ratio of phenotypes, provided there is no interaction between the genes.

If there is interaction that renders two or more of the phenotypes indistinguishable, then the F2 ratio is modified. Epistasis is the interaction of alleles at different loci. The value of an allele or genotype at one locus depends on the genotype at other epistematically interacting loci, complicating the picture of gene action.

- **no interaction**: unmodified ratio 2 dominant genes
  - 9:3:3:1
  - 1:1:1:1
- Dominant epistasis
  - 12:3:1
  - 2:1:1
- Dominant-inhibitory epistasis
  - 13:3
  - 1:3
- Duplicate dominant epistasis
  - 15:1
  - 3:1
- Duplicate recessive epistasis
  - 9:7
  - 1:3
- Recessive epistasis
  - 9:3:4
  - 1:1:2
- Polymeric gene interaction
  - 9:6:1
  - 1:2:1
- **no interaction**: Additive genes
  - 1:4:6:4:1
  - 1:2:1
Dominant epistasis

Also known as **masking action** and simple epistasis. A dominant allele at one locus masks the expression of both alleles (dominant and recessive) at another locus.

Example: color of the hull in oat seeds
Black-hull phenotype -> dominant allele A
Gray-hull phenotype -> another dominant allele B
this effect is apparent only in aa genotypes.

F1 genotype Aa Bb (black)
F2
9/16 A B  black hull
3/16 A  bb  black hull
3/16 aa B  gray hull
1/16 aa bb  white hull
Dominant-inhibitory epistasis

Also known as inhibitory gene interaction, or dominant-recessive interaction. A dominant allele (A) either homo- or heterozygous of one gene and a homozygous recessive allele (bb) of another gene produce the same phenotype.

The green colour of plants is governed by the gene A which is dominant over purple colour. The purple colour -> dominant B.
Duplicate dominant epistasis

Also known as **duplicate action**. Two genes control a trait and only one dominant is necessary to express the trait. Just the double recessive shows the mutation.

Example: bean flower color

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Phenotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>PpRr</td>
<td>Purple</td>
</tr>
<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
<td>PpRr</td>
<td>Purple</td>
</tr>
<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
<td>PpRr</td>
<td>Purple</td>
</tr>
<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
<td>PpRr</td>
<td>Purple</td>
</tr>
<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
<td>PpRr</td>
<td>Purple</td>
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<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
<td>PpRr</td>
<td>Purple</td>
</tr>
<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
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<td>Purple</td>
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<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
<td>PpRr</td>
<td>Purple</td>
</tr>
<tr>
<td>Pprr</td>
<td>White</td>
</tr>
<tr>
<td>PpRr</td>
<td>Purple</td>
</tr>
<tr>
<td>Pprr</td>
<td>White</td>
</tr>
</tbody>
</table>

Quelle: Sanders
Duplicate recessive epistasis

Also known as **complementary epistasis**. When recessive alleles at either of the two loci can mask the expression of dominant alleles at the two loci, it is called duplicate recessive epistasis.

At least one dominant allele from each of the two genes needed for the phenotype.

Example: flower color in sweet pea
The purple color is governed by two dominant genes A and B. When these genes are in separate individuals (AAbb or aaBB) or recessive (aabb) they produce white flower.
**Two genes inheritance**

### Recessive epistasis

Also known as **modifying action**. Wild-type alleles of two genes (\(w^+\) and \(m^+\)) encode enzymes catalyzing successive steps in the synthesis of a blue petal pigment. Homozygous \(m/m\) plants produce magenta flowers and homozygous \(w/w\) plants produce white flowers. The double mutant \(w/w ; m/m\) also produces white flowers.
Polymeric gene interaction

Two dominant alleles have similar effect when they are separate, but produce enhanced effect when they come together. The joint effect of two alleles appears to be additive or cumulative, but each of the two genes show complete dominance, hence they cannot be considered as additive genes. In case of additive effect, genes show lack of dominance.

Example: squash fruit shape

- **9:6:1**
- **9/16**: Precursor → Protein A → Disk
- **3/16**: Precursor → Protein A → Sphere
- **3/16**: Precursor → No protein A → Sphere
- **1/16**: Precursor → No protein A → Long

Quelle: Sanders
2 genes contribute to a trait in a quantitative fashion, each 'positive' allele increases the amount of gene product: sum of positive alleles determine level of the trait.

=> this is the transition to **Quantitative Genetics**

Discontinuous binomial distribution becomes a continuous normal distribution.

**Additive gene effects**

Example seed color in wheat ([Nilsson-Ehle, 1909](#))
Incomplete penetrance and variable expressivity are due to effects of other genes or environmental factors. Make genetic analysis difficult!

Penetrance and Expressivity

Penetrance: percentage of individuals having a particular genotype that express the expected phenotype

Expressivity is a related concept that describes the degree to which a character is expressed

In Figure 6-25, Pigment intensity as an example of penetrance and expressivity. Assume that all the individuals shown have the same pigment allele (P) and possess the same potential to produce pigment. Effects from the rest of the genome and the environment may suppress or modify pigment production in any one individual. The color reflects the level of expression.
Calculation of Chi-square ($\chi^2$) test for deviation from Mendelian ratios

“goodness-of-fit” test

 Breeders wonder if data support or fit a particular hypothesis and therefore help to explain the results.

A null hypothesis is formed that states there is no real difference between the observed and expected data. If differences are due to chance, then the hypothesis can be accepted, but if not, the null hypothesis is rejected and the breeder can modify the hypothesis in favor of a better one.

The equation used to calculate the ($\chi^2$) statistics is as follows:

$$\chi^2 = \sum \frac{(O - E)^2}{E}$$

for all classes

in which

- $E =$ expected number in a class
- $O =$ observed number in a class
- $\sum$ means “sum of.”
Example 1: CHI-SQUARE (2) TEST to test for Mendelian segregation ratios

Mendel’s results when phenotyping traits in his pea experiments

<table>
<thead>
<tr>
<th>Parental phenotype</th>
<th>F1</th>
<th>F2</th>
<th>F2 ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Round × wrinkled seeds</td>
<td>All round</td>
<td>5474 round; 1850 wrinkled</td>
<td>2.96:1</td>
</tr>
<tr>
<td>2. Yellow × green seeds</td>
<td>All yellow</td>
<td>6022 yellow; 2001 green</td>
<td>3.01:1</td>
</tr>
<tr>
<td>3. Purple × white petals</td>
<td>All purple</td>
<td>705 purple; 224 white</td>
<td>3.13:1</td>
</tr>
<tr>
<td>4. Inflated × pinched pods</td>
<td>All inflated</td>
<td>882 inflated; 299 pinched</td>
<td>2.95:1</td>
</tr>
<tr>
<td>5. Green × yellow pods</td>
<td>All green</td>
<td>428 green; 152 yellow</td>
<td>2.82:1</td>
</tr>
<tr>
<td>6. Axial × terminal flowers</td>
<td>All axial</td>
<td>651 axial; 207 terminal</td>
<td>3.14:1</td>
</tr>
<tr>
<td>7. Long × short stems</td>
<td>All long</td>
<td>787 long; 277 short</td>
<td>2.84:1</td>
</tr>
</tbody>
</table>

F2 ratio close to 3:1 ratio. BUT do these traits really segregate in the predicted ratio?
Example 1: CHI-SQUARE (2) TEST
to test for Mendelian segregation ratios

<table>
<thead>
<tr>
<th>Classes</th>
<th>Expected ratio</th>
<th>observed</th>
<th>expected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Round seed</td>
<td>3</td>
<td>5475</td>
<td>5494</td>
</tr>
<tr>
<td>Wrinkled seed</td>
<td>1</td>
<td>1850</td>
<td>1831</td>
</tr>
<tr>
<td>Σ</td>
<td></td>
<td>7325</td>
<td></td>
</tr>
</tbody>
</table>

1) Determine the expected frequencies
2) Calculate the test statistics
3) Test for significance

\[ \chi^2 = \sum \frac{(O - E)^2}{E} \]

\[ x^2 = \frac{(5475-5494)^2}{5494} + \frac{(1850-1831)^2}{1831} \]

\[ x^2 = 0.25597 \]
Critical Chi-square values for different degrees of freedom and $p$-values

Table 2-2

<table>
<thead>
<tr>
<th>df</th>
<th>0.995</th>
<th>0.975</th>
<th>0.9</th>
<th>0.5</th>
<th>0.1</th>
<th>0.05</th>
<th>0.025</th>
<th>0.01</th>
<th>0.005</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.000</td>
<td>0.000</td>
<td>0.016</td>
<td>0.455</td>
<td>2.706</td>
<td>3.841</td>
<td>5.024</td>
<td>6.635</td>
<td>7.879</td>
</tr>
<tr>
<td>2</td>
<td>0.010</td>
<td>0.051</td>
<td>0.211</td>
<td>1.386</td>
<td>4.605</td>
<td>5.991</td>
<td>7.378</td>
<td>9.210</td>
<td>10.597</td>
</tr>
<tr>
<td>3</td>
<td>0.072</td>
<td>0.216</td>
<td>0.584</td>
<td>2.366</td>
<td>6.251</td>
<td>7.815</td>
<td>9.348</td>
<td>11.345</td>
<td>12.838</td>
</tr>
<tr>
<td>4</td>
<td>0.207</td>
<td>0.484</td>
<td>1.064</td>
<td>3.357</td>
<td>7.779</td>
<td>9.488</td>
<td>11.143</td>
<td>13.277</td>
<td>14.860</td>
</tr>
<tr>
<td>5</td>
<td>0.412</td>
<td>0.831</td>
<td>1.610</td>
<td>4.351</td>
<td>9.236</td>
<td>11.070</td>
<td>12.832</td>
<td>15.086</td>
<td>16.750</td>
</tr>
<tr>
<td>6</td>
<td>0.676</td>
<td>1.237</td>
<td>2.204</td>
<td>5.348</td>
<td>10.645</td>
<td>12.592</td>
<td>14.449</td>
<td>16.812</td>
<td>18.548</td>
</tr>
<tr>
<td>7</td>
<td>0.989</td>
<td>1.690</td>
<td>2.833</td>
<td>6.346</td>
<td>12.017</td>
<td>14.067</td>
<td>16.013</td>
<td>18.475</td>
<td>20.278</td>
</tr>
</tbody>
</table>

df ... degree of freedom
= phenotypic classes -1

Example 1: CHI-SQUARE (2) TEST to test for Mendelian segregation ratios

Critical values of the $\chi^2$ distribution determine the probability ($P$) of the hypothesis being true for the observed distribution. Usually probabilities smaller than 5% ($p<0.05$) are chosen to define a cut-off when to reject a hypothesis.
Example 2: Analyze segregation results and predict the genetic basis of a trait

What hypothesis can we invent to explain the results?

%  24  57  19

-> use Excel file example 2
Example 2: Analyze segregation results and predict the genetic basis of a trait

**Incomplete dominance**

![Diagram of incomplete dominance]

- **F1 Generation**: Pink $Rr$
- **Gametes**: $\frac{1}{2}R, \frac{1}{2}r$
- **Eggs**: $\frac{1}{2}R, \frac{1}{2}r$
- **F2 Generation**: Pink $RR$, Pink $Rr$, White $rr$

**Counts**

- Incomplete dominance: 77, 182, 61
- recessive epistasis: 1, 2, 1

**Recessive Epistasis**

![Diagram of recessive epistasis]

- Dihybrid $w^+w^+/m^+m$
- Selled
- $\frac{9}{16} w^+m^+$: Both enzymes active
- $\frac{3}{16} w^+m^-$: Blocked at second enzyme
- $\frac{3}{16} w^-m^+$: Blocked at first enzyme
- $\frac{1}{16} w^-m^-$: No substrate
Example 2: Analyze segregation results and predict the genetic basis of a trait

Test both hypothesis with the $\chi^2$ test

1) Determine the expected frequencies
2) Calculate the test statistics
3) Test for significance

Incomplete dominance (1:2:1)

<table>
<thead>
<tr>
<th></th>
<th>$\chi^2$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>77</td>
<td>182</td>
<td>61</td>
</tr>
<tr>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>9</td>
<td>3</td>
</tr>
</tbody>
</table>

Recessive Epistasis (9:3:4)

<table>
<thead>
<tr>
<th></th>
<th>$\chi^2$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.650</td>
<td>0.022</td>
<td></td>
</tr>
<tr>
<td>0.151</td>
<td>0.927</td>
<td></td>
</tr>
</tbody>
</table>

We conclude that the results uphold the hypothesis of recessive epistasis.
Examples 3 and 4: Analyze segregation of breeding populations and predict the genetic basis of traits

Data from current research projects conducted at IFA-Tulln:
Resistance breeding for Fusarium head blight (FHB, *Fusarium* spp.) and bunt (*Tilletia* spp.)

**Example 3:**
111 F7 durum wheat lines of a cross DBC480 * Karur
Evaluated for FHB resistance, plant height, date of anthesis

**Example 4:**
126 F7 hexaploid wheat lines of a cross PI119333 * Rainer
Evaluated for bunt resistance and plant height

Illustrate segregation for the traits by histograms and correlations of the traits by scatter plots (e.g. using excel). Speculate about number of involved genes and possible dependencies of the traits.

-> use Excel file example 3 and 4
Analyze segregation of breeding population and predict the genetic basis of traits

Fusarium head blight severity

plant height (cm)

date of anthesis

DBC480

Karur

DBC480

Karur
Analyze segregation of breeding population and predict the genetic basis of traits.

Scatter plot for FHB severity and date of anthesis:
- % FHB severity vs. date of anthesis
  - Correlation coefficient: $r = -0.11$

Scatter plot for FHB severity and plant height:
- % FHB severity vs. plant height (cm)
  - Correlation coefficient: $r = -0.82$
Improvement of FHB Resistance in Durum Wheat

**DBC-480 x Karur**

Results: molecular-genetic analysis for FHB resistance

<table>
<thead>
<tr>
<th>Chr</th>
<th>Marker</th>
<th>Multi environment analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Add</td>
</tr>
<tr>
<td>3B</td>
<td>Umn10 (Fhb1)</td>
<td>57.6</td>
</tr>
<tr>
<td>4B</td>
<td>RhtB1</td>
<td>139.1</td>
</tr>
<tr>
<td>6A</td>
<td></td>
<td>-25.5</td>
</tr>
</tbody>
</table>

- Three QTL on chr. 3B, 4B, 6A associated with Fusarium resistance
- Major FHB resistance QTL associated with plant height (RhtB1)
- Despite that: several short lines carrying *Fhb1* show enhanced resistance and are integrated in durum wheat program

Results: Plant height

- One major plant height QTL on chr. 4B (RhtB1)
Common bunt: populations – PI119333 * Rainer

- 1 major resistance gene on chr. 7D (Bt12)
- 1:1 segregation of resistant (65) : susceptible (58)
- Least significant difference (LSD) for cutoff between resistant/susceptible (8.8%)

Several minor QTL involved
- QTL mapping detected 1 QTL on chr. 7B above the threshold
2 dominant genes
unmodified ratio

2 dominant genes
unmodified ratio

duplicate dominant epistasis

recessive epistasis

polymeric gene interaction

dominant epistasis

dominant (inhibitory) epistasis

duplicate recessive epistasis

Additive genes

9:3:3:1

15:1

12:3:1

13:3

9:7

9:6:1

1:4:6:4:1
## Summary - Segregation ratios for single and 2 genes inheritance in the F2 and F∞

<table>
<thead>
<tr>
<th>generation</th>
<th>phenotypic class</th>
<th>F2</th>
<th>F∞</th>
</tr>
</thead>
<tbody>
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