Inheritance of Radial Fruit Cracking Resistance in Tomatoes
(Solanum lycopersicum L.)

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Abstract. The shift of tomato cultivation from highlands to lowlands caused damage to the fruit due to fruit cracking. The damage was caused by high temperatures. This study aimed to determine the inheritance pattern of radial fruit cracking resistance and the characters that are directly related to fruit cracking so that it can recommend the right selection method for assembling radial fruit cracking resistant tomato. This study used six sets of population namely P1 female parents who were resistant to radial fruit cracking (IPB T64), P2 male parents who were sensitive to radial fruit cracking (IPB T73), F1, F1R, BCP1, BCP2, and F2. Mendel's analysis was employed to analyze the qualitative characters, and joint scaling test was used to the estimation of the actin gene of quantitative character. The results of the analysis showed that the character of the number of locules, the thickness of the fruit flesh and percentage of fruit cracking per plant were controlled by polygenes and there was no maternal effect. The heritability in a broad and narrow sense for all characters was moderate to high with a proportion of additives variance to total high genetic variability. The gene action for the number of locules was dominant epistasis x dominates and duplicates, for thicknesses of fruit flesh and fruit additive x dominan duplicate epistasis, and the number of fruit cracking by dominant epistasis gene x dominance, which was complementary. Radial fruit cracking which was controlled by the action of dominant epistasis gene by both parents with resistant genotype was FC₃-FC₄, FC₃-FC₄, FC₃-FC₄ and genotype which was susceptible to radial fruit cracking was FC₃-FC₄, FC₃-FC₄, and FC₃-FC₄. The selection method which was appropriate for the development of the best type of tomatoes that were resistant to fruit cracking was the pedigree method.

1. Introduction
Tomatoes are one of the most important horticultural plants in the world including Indonesia. Tomatoes have many uses, both for household and industrial consumption. Tomatoes are the main source of lycopene which has a radical oxygen binding capacity so that they are very useful to prevent cancer, cardiovascular disease, and other chronic diseases. It was also reported that vitamin C was also highly contained in tomatoes after oranges [1].

Tomatoes mostly require low temperatures or cold climates to produce high production and good fruit quality. In the tropics, low temperatures are mostly found in areas with high altitude. The limited availability of land for tomato production in the highlands of several tropical countries has led to the
development of tomato planting in the middle to lowland areas. In Indonesia, 60% of tomatoes are cultivated in the highlands, and 40% are cultivated in the middle to lowland [2,3]. Reported that there was a 35% decrease in the production of adaptive highland to lowland tomatoes. An adaptable variety of tomatoes in the middle to high land which was grown in medium plains also showed a decreased product as many as 50-60%. Conversely, if the variety were planted in the highlands (800 mm.a.s.l), it would have higher production [4]. The ability of tomatoes to produce fruit depends greatly on the interaction between plant growth and its environmental conditions [5].

The low production in the lowland was caused by a limited number of superior varieties with high potential production [6,7]. The shift of tomato cultivation areas to the lowlands caused the risk of a decrease in fruit quality and fruit production. The high temperature at lowland not only affected the fruit ripe time but also the tomato growth rate [8]. High temperatures caused a rapid rate of fruit growth that triggers fruit cracking. Fruit cracking in tomatoes can allow the entry of pathogens, which could reduce the appearance leading to increasing substantial crop losses [9–14].

The assembly of superior adaptive variety in lowland is an effective and efficient method to increase tomato production in the lowland. To get superior varieties, it is necessary to select superior and breakable genotypes of fruit that can be used as donor parents and to determine selection criteria as well as the character inheritance pattern to facilitate the selection process.

Fruit cracking is a physiological disease which is a complex phenomenon caused by genetic and environmental factors [15]. Tomato cracking can be divided into burst cracking, concentric cracking, russetting, and radial cracking [16]. Radial cracking is often found in fully ripe fruit; concentric cracking often occurs in ripe green fruit. This concentric cracking does not reduce the fruit production in the field much more than the radial cracking does [17]. Environmental factors that affect fruit cracking are rainfall, temperature, light intensity, and air humidity [9,18–21]. The environmental influence is very difficult to control. Treatment of cultivation techniques can reduce losses caused by fruit cracking. However, it is only temporary and less effective because it must be done in every planting season and requires a high cost. The use of fruit-cracking resistant varieties is a more effective solution [20]. Genetic analysis of resistance to fruit cracking is a preliminary requirement for selection to produce resistant varieties [22].

The success of assembly plants is largely determined by appropriate selection methods and criteria [23-25]. Characters used as selection criteria must be chosen based on the value of heritability and the closeness of the relationship with the desired character. A study conducted [26] showed that the characters having a direct influence on the fruit breaking index are the number of locules and thickness of fruit flesh. Both characters have high heritability values. The information related to inheritance studies of resistance to fruit cracking has not been consistent [27], reported that the nature of fruit cracking in tomatoes was controlled by a simple single gene. According to [28] radial fruit cracking was controlled by two major pairs of the gene, namely cr cr and Ir Ir. Furthermore [29,30] concluded that fruit cracking in tomatoes was controlled by multiple genes which had a partial dominant nature. [26] concluded that concentric fruit cracking in tomatoes was controlled by two pairs of double recessive epistasis genes or complete dominance by both genes which were complementary. The fruit cracking in chili is dominant with some additive influences [31]. The research results of [32,9] proved that genes controlling resistance to fruit cracking were quantitative (polygon).

The information on the study of character control gene inheritance and characters that had direct influence was very effective to determine the selection method to be used. This information was very useful in the selection stage so that the selection was more effective and efficient. Therefore, inheritance research on the resistance properties of radial fruit cracking on tomatoes was important to do. This study aimed to determine the pattern of inheritance of the resistance of radial fruit cracking in tomatoes and the characters that were directly related to fruit cracking. Finally, the right selection method for assembling radial fruit resistant tomato varieties can be recommended.
2. **Material and Method**

The plant material used consisted of 20 female parent (P1) tomatoes which were resistant to radial fruit cracking (IPB T64), male parent (P2) tomatoes which were susceptible to radial fruit cracking (IPB T73) consisting of 20 plants; first derivative (F1) consisting of 20 plants; reciprocal parents (F1R) consisting of 20 plants; backcrossing on female parents (BCP1) consisting of 100 plants, backcrossing on male parents consisting of 100 plants, and second derivative population (F2) consisting of 200 plants.

2.1. *The Formation of Populations*

The populations formed were F1, F1R, BCP1, BCP2, and F2. Emasculation and pollination were carried out between 6:00 a.m. and 9:00 a.m. F2 was produced from the results of selfing-F1. BCP1 and BCP2 were produced from the results of F1 crossing between its two parents.

2.2. *Observations*

The characters observed for the selection of fruit cracking resistance were the number of locules (locules), the thickness of fruit flesh (mm), the percentage of fruit cracking per plant (%), and radial fruit cracking index (R-FCI). To calculate the fruit cracking index, the researchers used the method [22][26] as follows

\[
\text{Fruit Cracking Index} = 100 - \frac{\sum (n_i \times \text{Score})}{\sum n \times \text{Maximum Score}} \times 100 \%
\]

2.3. *Description*

\( n_i = \) amount of fruit in Score i (i = 0, 1, 2, 3, 4; maximum score = 4). The score was determined based on the 'Crack Resistance Score' method [22,26,31,33–35], which was modified i.e 0: no fruit cracking, 1: slightly radial fruit cracking (<25%), 2: medium radial fruit cracking (25% - 50%), 3: heavy radial fruit cracking (50% - 70%), 4: heavy radial fruit cracking (> 75%). The scoring illustration of fruit cracking can be seen in Figure 1. The FCI value was then used to group the genotype resistance level to the fruit, which has been modified with the criteria: Highly Resistant (HR) if FCI = 100%; Resistant (R) if 95% < FCI ≤100%; Moderately Resistant (MR) if 90% < FCI ≤95%; Slightly Susceptible (SS) if 80% < FCI ≤90%, Susceptible (S) if 60% < FCI ≤80%, and Highly Susceptible (HS) if FCI <40% [26,34,35].

![Figure 1 Scoring Illustration of Radial Fruit Cracking](image)

2.4. *Data analysis*

The inheritance study on the fruit cracking was analyzed by using 6 population methods (P1, P2, F1 / F1R, F2, BCP1, and BCP2) following the method performed by [22]. The qualitative characteristic was the fruit cracking index and the analysis was carried out using the Mendel analysis, while the analysis of quantitative characters (number of locules, thickness of the fruit flesh, and percentage of radial fruit cracking) followed the method used by [36],[26,37–39], namely estimating the influence of female parents, degree of dominance (Potential Ratio), feasibility of genetic models, and heritability in broad and narrow sense.
3. Results and discussion

3.1. Maternal Effect
The results of the test of the influence of maternal effect using comparing mean test (t-test) showed that there were no significant differences between F1 and F1R on the character of the number of locules, the thickness of fruit flesh, and percentage of the number of fruit cracking in tomatoes (Table 1). The results of the t-test showed that there was no influence of female parents and only genes in the nucleus controlled the inheritance of these characters.

| Population | Number of locules | The thickness of Fruit Flesh | Percentage of Radial Fruit Cracking |
|------------|------------------|-------------------------------|-------------------------------------|
| F1         | 3.58 ± 0.15      | 4.92 ± 0.18                   | 24.77 ± 2.01                        |
| F1R        | 3.57 ± 0.17      | 4.92 ± 0.17                   | 24.59 ± 2.19                        |

Prob > t hit:
F1: 0.60<sub>m</sub>, F1R: 0.98<sub>m</sub>, t hit: 0.97<sub>n</sub>, 0.95<sub>n</sub>

In: significant level of α 5%

3.2. Heritability in Narrow and Broad Sense
The heritability in broad sense (h²bs), character number of locules, and percent number of fruit cracking per plant were high and the heritability in the narrow sense (H²ns) was also high (Table 2). Meanwhile for thickness characters of fruit flesh which had a heritability both in narrow and broad senses were included in the medium category. This shows that the character of the number of locules and percent of the number of fruit cracking was more controlled by genetic factors than environmental factors. The proportion of various additives to total genetic diversity was still very high, so the effect of additives was greater than the dominant influence. Character number of locules and percent number of fruit cracking were controlled by genes that worked additively. The proportion of various additives to total genetic diversity in thickness of fruit flesh was still quite high, namely 57.77. This shows that the effect of additives was greater than the dominant influence, so the thickness of the fruit flesh was controlled by genes that worked additively in that combination.

| Components | Number of locules | The thickness of Fruit Flesh | Percentage of Radial Fruit Cracking |
|------------|------------------|-------------------------------|-------------------------------------|
| Heritability in broad sense (h²bs) | 0.86              | 0.45                          | 0.85                               |
| Heritability in narrow sense (h²ns) | 0.70              | 0.26                          | 0.88                               |
| (h²m / h²bs) x 100% | 81.39            | 57.77                         | 103.52                             |

P1: female parent; P2: male parent; F1: first derivative; BCP1: backcross to female parent; BCP2: backcross to male parent; F2: second derivative.

The high estimated heritability value indicated that the number of locules and the percentage of fruit cracking could be used as a selection criterion for selecting genotypes that were resistant to radial fruit cracking. According to [40,41]. The estimated heritability can be used to select characters that will be used as criteria for selection. Heritability estimation values with high criteria can be used directly as a selection character in the early generation [42,43]. The results of research by [44] also showed high heritability in the number of fruit locules. Heritability in high broad meaning means that the characters observed were more controlled by genetic factors than environmental factors. Genetic diversity was expressed in the phenotypic appearance of plants [25].
3.3. Genetic Model

The genetic model which was suitable for the character of the number of fruit locules was additive-dominant (m[d][h][l]) because this model had shown a difference where \( \chi^2 \) count was less than \( \chi^2 \) table. Suitable models for thickness of fruit flesh were additive-dominant with interaction effect between dominant x and dominant (m[d][h][i]), additive-dominant with interaction effect between dominant x and dominant (m[d][h][l]), additive-dominant with the interaction effect between additives x additives and x dominant (m[d][h][i][j]), additive-dominant with the interaction effect of additive and dominant x dominant (m[d][h][i][l]) (Table 3). The most suitable genetic model in the combination of IPBT64 x IPBT73 was m[d][h][i][j], because it only had two values of genetic components that were not significant. Whereas the model (m[d][h][i][l]) had the smallest chi-square value but the effect of interaction on all genetic components was not significant.

Table 3. Fit Test of Character Genetic Model

| Genetic Model   | Number of locules | The thickness of Fruit | Percentage of Fruit |
|-----------------|-------------------|-----------------------|---------------------|
|                 | Chi-square | Prob     | Chi-square | Prob     | Chi-square | Prob |
| m [d]           | 31.15*     | 0.000    | 76.83*     | 0.000    | 43.77*     | 0.000 |
| m [d] [h]       | 7.65m     | 0.954    | 25.85*     | 0.000    | 41.02*     | 0.000 |
| m [d] [h] [i]   | 6.46*     | 0.040    | 1.42m      | 0.492    | 18.44*     | 0.000 |
| m [d] [h] [j]   | 6.88*     | 0.032    | 25.67*     | 0.000    | 40.53*     | 0.000 |
| m [d] [h] [l]   | 7.64*     | 0.023    | 4.43m      | 0.109    | 35.79*     | 0.000 |
| m [d] [h] [i] [j] | 5.23*  | 0.022    | 0.85m      | 0.356    | 18.22*     | 0.000 |
| m [d] [h] [i] [l] | 0.001m | 0.971    | 0.56m      | 0.454    | 2.49m      | 0.114 |
| m [d] [h] [j] [l] | 6.83*   | 0.009    | 3.98*      | 0.046    | 35.70*     | 0.000 |

Prob: Probability test with the level of significance of \( \alpha \) 5%; * model is not suitable with the significant level of \( \alpha \) 5%; tn: model is suitable with the significant level of \( \alpha \) 5%

The genetic model that was suitable with the percentage of fruit cracking was additive-dominant with the effect of additive x dominant and dominant x dominance interaction (m[d][h][i][l]) because it shows the lowest chi-square value and the only model was not significant. The most suitable genetic model was m[d][h][i][l] because it shows the lowest and not significant chi-square values and all the genetic components were significant. According to [45], the most suitable model based on a combined scale test was a model that showed the smallest value of \( \chi^2 \) count and smaller than \( \chi^2 \) table (prob <0.05).

The genetic component for the character of the fruit locules was controlled by the action of the dominant additive gene with gene action value and significant dominance (Table 4). The value of the action of the dominant gene was higher than the action of the additive gene. This shows that the character of the number of fruit locules was controlled by the action of the dominant gene. The genetic component to the character of the fruit locules was controlled by the action of the dominant additive gene with the value of the action of the additive gene. The value of the action of the dominant gene was higher than the action of the additive gene. The action of the dominant gene was opposite to its interaction so that the action of the gene was duplicated. This shows that the action of genes that had more influence on the number of fruit locules was the dominant epistatic x dominant which was duplicate. According to [46], the interaction of additive x dominant and dominant x dominant genes was better than the additive x additive interaction as a result of inheritance on tomatoes. Dominant gene interaction allows the chance of the emergence of dominant genes. The higher the result of the dominant gene interaction is, the greater the next generation will be. For further information it will be more comprehensive if it includes genetic diversity and mating systems [47,48]. According to [45] if the dominant and additive values are opposite to each interaction, the type of interaction that has the most dominant role is duplicate interaction. Conversely, if the type of interaction is the same, the complementary interactions are the most dominant.
The genetic component in the thick character of fruit flesh has a significant additive component value and no significant dominance. This shows that the action of additive genes was more influential than the action of dominant genes was. The effect of gene interaction shows that only the action of the dominant x additive gene was significant. Additive (positive) gene action was in the opposite direction with the dominant x additive interaction which had a positive value. Thus, the action of the gene was duplicated. This shows that the action of genes that were more influential on thick fruit flesh was the action of the dominant x additive epistasis gene which was duplicate.

**Table 4. Estimation of Genetic Components**

| Cross Combination | Genetic Component |
|-------------------|-------------------|
|                   | m     | d     | H     | i     | j     | l     |
| Number of locules | 4.45* | -2.12* | -0.86* | - | - | - |
| Thickness of Fruit Flesh | 4.59* | 0.64* | 0.24* | 0.30* | -1.13* | - |
| Percentage of Fruit Cracking | -19.74* | -26.38* | 92.95* | 46.49* | - | 48.44* |

m: mean value; d: additive effect; h: dominant effect; i: interactive effect between additive and additive; j: interactive effect additive x dominant; l: interactive effect between dominant x dominant; in: no difference in the level of at 5%.

The genetic component in the character of the percentage of the number of fruit cracking has a significant additive and dominant component value. The value of the action of the dominant gene was significantly positive and higher than the action value of the additive gene. Thus, the action of the dominant gene was more influential than the action of the additive gene. The effect of gene interaction shows that the action of the dominant x gene was dominantly significant and higher (48.44) than the effect of additive x additive (46.49) so that the action of the dominant x interaction gene was more influential. The action of the dominant gene was in the direction of its positive interaction so that the action of the gene was complementary. This shows that the action of genes that had more influence on the percentage of the number of fruits cracking per plant was the action of dominant x dominant epistasis genes that were complementary.

**3.4. Inheritance of Qualitative Character (Fruit Cracking Index)**

The parents used for inheritance of radial fruit cracking resistance were fruit-resistant genotypes (IPBT64 as P1) and genotypes that were susceptible to fruit cracking (IPBT73 as P2). The population of F1 generation and BCP1 generation for radial fruit cracking tended to lead to resistant parents, namely P1. BCP2 generation tended to lead to susceptible parents namely P2. F2 generation tended to be resistant parents. This indicated that the resistance of fruit cracking had a greater dominant effect on susceptible genotypes. This was contrary to the result reported by [22] in melons, where the F1 population was more likely to lead to parents which were susceptible to fruit cracking.

**Table 5. \(\chi^2\) Count Value of the Resistance to Radial Fruit Cracking in P1, P2, BCP1, BCP2 and F2 at Tomatoes**

| Population | Phenotype | Ratio | Expectation | Observation | \(\chi^2\)hit | \(\chi^2\)tab |
|------------|-----------|-------|-------------|-------------|---------------|---------------|
| P1, F1, F1R, BCP1 | 100% resistant | | | | | |
| P2 | 100% susceptible | | | | | |
| BCP2 | resistant: susceptible | 1 : 3 | 19 : 57 | 15 : 61 | 0.56* | 3.84 |
| | resistant: susceptible | | | | | |
| F2 | 11 : 5 | 132 : 60 | 140 : 52 | 2.13* | 3.84 |
The results of Mendel's genetic analysis on F2 generation for radial fruit cracking (IPBT64 x IPBT73) were 11 resistant: 5 susceptible, and the phenotype ratio in the backcross population to male parents (BCP2) was 1 resistant: 3 cracking (Table 5). This suggests that radial fruit cracking was controlled by two sets of genes where the complete dominance was of both genes only if there was a dominant allele in both genes, and if there were no dominance, recessive phenotype would emerge [28,49,50] reported that the radial fruit cracking was controlled by two major genes namely $cr$cr and $lr$lr. Genotypes that were resistant to radial fruit cracking were $FC_r FC_r$, $FC_r FC_r fc fc$, $fc FC_r FC_r fc$, and genotypes which were susceptible to radial fruit cracking were $FC_r FC_r fc$, $fc FC_r FC_r fc$, $FC_r FC_r fc$ and $fc FC_r FC_r fc$. The right selection method for the development of superior tomato varieties which were resistant to radial fruit cracking was the pedigree method because it can be observed in the early stages namely F2 generation.

4. Conclusion
1. Characters on the number of locules, thickness of fruit flesh, and percentage of fruit cracking per plant were controlled by many genes, and there were no external effects.
2. Heritability in broad sense and narrow sense for all characters was moderate to high with various proportions of additives to totally high genetic diversities.
3. Gene action based on the genetic model for the number of locules was the dominant action of epistasis genes x dominance and duplicate, for thicknesses of fruit flesh in the form of additive epistasis gene dominance and duplicate, and the number of pieces of fragmentation by epistasis dominance x genes was complimentary.
4. Radial fruit cracking was controlled by the action of the complete dominant epistasis gene by both parents with resistant genotypes namely $FC_r FC_r$, $FC_r FC_r fc fc$, $fc FC_r FC_r fc$, $FC_r FC_r fc$, and genotypes which were susceptible to radial fruit cracking were $FC_r FC_r fc$, $fc FC_r FC_r fc$, $FC_r FC_r fc$and $fc FC_r FC_r fc$. The right selection method for developing the best type of tomatoes that were resistant to the fruit cracking was the pedigree method.

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[49] Klug, Cummings, Spencer and Palladino 2011 Concepts of genetics (California, San Francisco: Pearson Education) pp 66-70

[50] Sobir and Syukur 2013 Genetika Tanaman (Bogor: IPB Press) pp 540–3