WHATEVER HAPPENED TO THE GREGOR MENDEL CONTROVERSY?

by

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Abstract

Ever since R. A. Fisher published his 1936 article, "Has Mendel's Work Been Rediscovered?", the surprisingly high conformity between Gregor (Johann) Mendel's observed and expected ratios in his famous experiments with peas has fascinated both historians of biology and statistics alike. Fisher's calculated χ^2 statistic of the experiments, taken as a whole, suggested that results on a par or better than those Mendel reported could only be expected to occur about three times in every 100,000 attempts, and the ensuing controversy as to whether or not the good Father "sophisticated" his data continues, unanswered, to this very day. In recent years the controversy has focused upon the more technical question of why Mendel failed to encounter some evidence of linkage in his data, even though some of the traits he worked with were not on independent linkage groups.

I examine the controversy in an historical and comparative perspective, considering the changes it has gone through, and what statements can be made concerning its current status.

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Many students of biology are introduced to Gregor Mendel's laws of heredity as early as junior high school, and the 3:1 or 9:3:3:1 ratios are quickly recognized in genetics classrooms across the land. Yet it is a strange happenstance that the particulars of Mendel's discovery, and the controversies that arose from it, are correctly understood today by only a select few. First reported to the Brünn Society for the Study of Natural Science in early 1865, Mendel's results stirred little interest in the scientific community, and they remained relatively unknown – though not completely ignored [1] – until 1900, when Hugo DeVries, Carl Correns, and Erich von Tschermak simultaneously "rediscovered" the 1866 paper. What followed was a rush of both support and, at times, bitter opposition to this new "Mendelian" doctrine of heredity [2].

As the twentieth century progressed so did the interest in Mendel's experiments. One of the many researchers they attracted was a young scientist named Ronald Aylmer Fisher. Included among his many works on statistical and genetic applications is a 1936 paper published in the Annals of Science [3], "Has Mendel's Work Been Rediscovered?" In this paper, Fisher attempted a quantitative reconstruction of Mendel's experiment, then went on to examine Mendel's ratios statistically, employing goodness-of-fit testing. His results were surprising; quickly summarized, they suggest that

Mendel's data conformed so well to the expected ratios that, treated as a whole unit, only three such experiments in 100,000 attempts would show ratios as close or closer to agreement with theoretical ratios as Mendel's did, i.e., the probability of, by chance alone, observing an "event" as good as or better than Mendel's result is 0.99997 [4]. This unusually good result might have one of a few possible explanations; Fisher's suggestion was that the data "... [had] been falsified so as to agree closely with Mendel's expectation" [5].

The crux of this 1936 paper was the goodness-of-fit testing Fisher performed. He not only utilized the formal chi-square test and its additive properties [6], but also reported simple units of deviation from expected values. The chi-square is a well-known statistical tool for fit-testing, and Fisher used it not only to examine the data Mendel reported from his experiments, but to critically examine what he was able to reconstruct as Mendel's results from the later years of experimentation, 1863 in particular. With these calculated chi-square statistics, he was able to report the probability that an appropriate chi-square variable (in terms of degrees of freedom) would exceed these results, i.e., the probability of observing an event as likely or less likely than the particular one in question (the so-called "tail" of the distribution). In goodness-of-fit hypothesis testing, we reject the given hypothesis of acceptable fit in favor of the alternative (unacceptable fit) when this probability drops below some small value, commonly 0.05. When this probability (or P-value) is very high, say 0.90, the results suggest an unusually good fit, i.e., only one out of every ten experiments would come as close to the expected results. What Fisher found with his chi-square tests of Mendel's data was precisely this: the P-values of the experiment as a whole, and many of its subdivisions, were uncomfortably high. Fisher reports a chi-square statistic of 41.6056 over the 84 degrees of freedom associated with Mendel's experiments

(to achieve a P-value of 0.05, the chi-square statistic would have to swell to 106.3917). He also partitions the 84 degrees of freedom into pertinent subdivisions and reports their particular chi-square values and P-values (e.g., in the available data from 1863, he reports a chi-square statistic of 15.5464 with 41 degrees of freedom [5]. The associated P-value is 0.999894). The only subdivision whose P-value is less than 90% is that for the test of genotypic ratios in the monohybrid crosses: there it is 0.74. The conclusion to draw from these results seemed clear to Fisher: "... the bias [in favor of expectation] seems to pervade the data," especially in the later years of experimentation [5]. We might give Mendel the benefit of the doubt for deformities or discolorations in seeds or pods which caused a subconscious, favored misclassification, but Fisher dismissed this as a minor consideration, certainly inapplicable to tests based on classification of whole plants. Coupled with the especially high P-values of the later years of experimentation, one might conclude that some "sophistication" of the data had taken place.

Even the case of the tests of genotypic ratios of the monohybrid crosses (Mendel performed eight of these – six for the five plant characters under study), with its P-value of 74%, is marred by controversy. To find the genotypes of the dominant plant character F_2's, Mendel grew ten seeds from each individual F_2 plant, then selfed these plants to produce an F_3. If <u>any</u> of the ten F_3's exhibited a recessive trait he would have concluded that the parental F_2 was heterozygous for the specific gene in question; if not, he would have classified it as dominant homozygous. Fisher established that a ten-seed selfing from a given heterozygote could result in ten dominant F_3's, with probability $(0.75)^{10} = 0.0563$. This would cause Mendel to misclassify about 5 or 6% of the parents as dominant homozygous, so the expected ratios from these crosses should be 1.8874:1.1126 (heterozygous to homozygous), instead of the usual 2:1. Applying

this to the 600 plants which Mendel tested in this manner yields an expected segregation of 222.5 homozygous plants, instead of 200 [7]. Mendel reports a total of 201 segregating plants in the 600 offspring, a unit deviation of +1.0 from the uncorrected expectation. Yet the corrected expectation suggests a deviation of -21.5. Not taking the correction factor into account therefore helps to substantiate the proposed theory. Expressing this statistically in terms of the chi-square distribution, the uncorrected expectation yields a statistic of 4.575 (P-value = 0.602) with six degrees of freedom, while the corrected statistic swells to 7.6582 (P-value = 0.2681). Thus the data fit the uncorrected expectation far better than they fit the corrected result. Again, some "sophistication" in the data is implied.

Fisher's presentation, however, lacks consistency. In reporting the chi-square statistic for the experiment as a whole, he decomposes down the eight degrees of freedom for this test of genotypic ratios in the monohybrid crosses with a chi-square (sub)statistic of 5.1733. Decomposing this further into two degrees of freedom for the earlier-observed seed characters (shape and color) and six degrees of freedom for the later plant characters, he gives chi-square values of 0.5983 and 4.575, respectively. With the seed characters there is no discrepancy in terms of a corrected or uncorrected expectation since Mendel worked with over 500 seeds for each of the two separate seed characters (565 for shape and 519 for color). Thus, since $(0.75)^{500} \approx 10^{-38}$ is effectively zero, we would expect 0% misclassification in these trials (i.e., the odds of getting all – over 500 – homozygous dominant F_3 are effectively zero). However, we've seen that this is not the case with the plant characters, where the correct expected ratio is 1.8874:1.1126. What is so curious here is that Fisher, after making a great fuss about misclassified data, reports the chi-square value derived from the 2:1 expected ratio! Had he used his own corrected ratio to

get a chi-square value of 7.6582, the overall chi-square would become 44.6888; at 84 degrees of freedom this yields a (lower) P-value of 0.999867.

In any case, the question here is clear, and Conway Zirkle's 1964 statement puts it simply enough: "Could the good Father Mendel have fudged his results just a little?" [8]. Since Mendel had earned a healthy respect for both his community efforts and his scientific interests, an affirmative response to Zirkle's query seems improbable. Even Fisher, after chi-squaring Mendel's data to death, relents: "... it remains a possibility ... that Mendel was deceived by some assistant who knew too well what was expected" [5]. In a note on personal memories of Fisher [9], P. C. Mahalanobis expounds further:

> "Mendel had announced in his last scientific publication that he would publish in another paper his results on three-factor segregation, but did not do so. Fisher had an almost irresistible urge to find out why Mendel ceased publication. Searching through old records, Fisher traced the original observations which Mendel had intended to use for his unpublished paper, and found that there was perfect agreement between observed and expected results. Fisher surmised that such agreement had raised a suspicion in Mendel's mind that his assistant, who had been helping him in these experiments, had deliberately changed the records to make them agree with expectations; Mendel had refrained from publishing as he could not guarantee their accuracy ..."

This "over-zealous assistant" explanation was only one example from a list of possibilities, and by the late 1960's that list had grown quite long. Perhaps the most celebrated piece is a letter to Curt Stern from Sewall Wright [10], in which Wright responded to a letter from Stern asking his opinion of the matter. In general Wright is supportive of Mendel, rejecting the possibility of any deliberate fraud, proposing instead that Mendel had fallen prey to occasional subconscious bias. Even today, such errors are not uncommon; Wright cites an example in which "15 trained observers obtained extraordinary differences in sorting and counting the same 532 kernels of corn" [11]. One might question how well current-day genetic count data would stand up to such rigorous goodness-of-fit testing, especially in light that the chi-square test is

very sensitive when applied to samples of large size. In such cases the slight effects of unconscious bias would be made to seem unduly important, and as Wright suggests, it would hardly seem fair to "... accuse Mendel of fraud for not meeting standards of objectivity in 1865 that few experimenters meet today" [12]. It is in fact particularly ironic that these experimental procedures and results should fall under Fisher's scrutiny, when his own scientific activities were of a surprisingly chauvinistic nature. Though acknowledged as one of the greatest biometricians of our time, he had a pestiferous habit of misrepresenting – and even ignoring at times – prior results. An example occurs as close as our own back door, when Fisher, in failing to carefully consider Mendel's letters to the biologist Carl Nägeli when reconstructing his experiments, miscalculated by a year and began them in 1857 instead of 1856 (an error he, however, later corrected). As Wright has pointed out, we certainly need to take these inconsistencies in Fisher's professional approach into account "in evaluating his attack on Mendel's integrity" [12].

In his letter to Stern, Wright also considered the misclassification discrepancy. He suggested that Mendel's knowledge of Pisum was perhaps advanced enough for him to discern the difference between a segregating group and a non-segregating group in the tests for monohybrid genotypic ratios, thus partially invalidating Fisher's "corrected" ratios as being too extreme. He went further to suggest that [13]:

> "... from [Mendel's] description of the heterozygotes for grey and white seed coat there would seem little doubt that in this case at least the occurrence of segregation of AA and Aa would be obvious in a group of 10 in the absence of recessives. I suspect that he used seed coat with the two real seed traits in his three-factor cross for this reason."

Also, as Wright's letter pointed out, Mendel may have planted "... more than ten seeds in each case to be sure to have at least ten to examine for segregants," which would also bring the probability of misclassification down. In general

he portrayed Mendel's work in a positive light, and states with confidence that he felt there was " ··· no deliberate attempt at falsification" [13].

In the same year that Curt Stern published Wright's letter (1966), Franz Weiling published a paper on the controversy, at times picking apart parts of Fisher's analysis in the same spirit with which Fisher disassembled Mendel's work. For example, in the misclassification problem he proposes an argument similar to Wright's, realizing that ten seeds could not consistently yield ten offspring. In sowing ten seeds, losses due to poor germination, birds, etc., could bring the number available for misclassification down. Thus, citing reports from The Agricultural Research Station in Brno which suggest a laboratory germination rate of 80-100% and a field rate of 57.5-100%, he supposes an average eight seedlings available for germination. From this he concludes that the probability of agreement with expectation in all of Mendel's pea experiments is effectively similar to the probabilities calculated from experiments with peas by Correns, von Tschermak and others [14]. His result is a "corrected 'corrected' ratio" of 1.7998:1.2002. The resulting chi-square for the six degrees of freedom would be 14.813, bringing the 84 degree of freedom total up to 51.8431. The corresponding P-value dips down to 0.997755.

Weiling's general argument centers about Mendel's data sampling technique. He points out that count data is based on binomial sampling, whose basic model is an "urn model with replacement". When the "balls" occupying the "urn" are thoroughly mixed, the sampling occurs at random, and a statistic can be constructed which, at least approximately, converges to the chi-square distribution. Since genetic segregation ratios occur in plants on the basis of a union of specific pollen cells with specific egg cells, this "urn model" will apply only when this union occurs totally at random. When this biological relationship is not random, perhaps more "semi-random", Weiling suggests that the

calculated chi-square statistic will be an underestimate, which he claims is the case with Fisher's calculated chi-square for Mendel's data. He was unable to bound the value of the constant by which Fisher is in error any better than $1 \leq c \lesssim 1.7$ [15], but his results do raise interesting questions for statisticians and experimenters interested in genetic count data.

By 1968 the goodness-of-fit controversy had attracted a number of authors to the fray. Fisher's explanation of the over-zealous assistant was considered by Åke Gustafsson in his 1968 semi-biographical piece on Mendel, but the lack of acceptable candidates for this wily assistant (the intellects of both of Mendel's closest servants were said to be lacking) led him to dismiss this as a viable explanation [16]. Theodosius Dobzhansky agreed [17], suggesting then a simpler possibility:

> "Few experimenters are lucky enough to have no mistakes or accidents happen in any of their experiments, and it is only common sense to have such failure discarded. The evident danger is ascribing to mistakes and expunging from the record perfectly authentic experimental results which do not fit one's expectations ... Mendel may have, in perfect conscience, thrown out some crosses which he suspected to involve contaminations with foreign pollen or other accidents."

G. A. Marx concurs, calling upon his experience as one of the few geneticists working with Pisum today to point out that the classification of plants into discontinuous categories is not always easy [18]. In those cases where the discontinuities are slight and less obvious, there exists a tendency to add one's own personal bias (cf. Pearl's "personal equation" in note [11]). In Mendel's case, the discontinuities are relatively distinguishable for most of the characters he studied – indeed, Pisum was a wise choice for precisely this reason. As early as 1913 William Bateson pointed out that "varieties in cultivation are distinguished by striking characters recognizable without trouble" [19], plant height being perhaps the greatest exception. Yet, with the large numbers of

individuals examined, there seems room enough for some error (Wright's "unconscious bias") to creep in.

It is also possible that Mendel was in some sense aware of this personal bias, and he may have chosen not to report what he felt were questionably derived ratios (Dobzhansky's "mistakes or accidents," e.g., diseases, insects, recording errors, etc.) in his final work. If this were the case, then Fisher's chi-square statistic would have only tested the fit of the most consistent ratios, and show, as it did, an extraordinarily good fit. Analogously, R. C. Olby (1966) suggested that Mendel stopped scoring the results as he saw that he was approaching the necessary values. With this, there is no direct falsification in the data, they are merely, in a sense, incomplete [20]. At about the same time, Jaroslav Krizenecky and L. C. Dunn proposed similar explanations [21], making this "incomplete data" proposition surprisingly popular. However, Gustafsson again disagreed, pointing out that von Tschermak's 1900 pea results were also of an unusually good fit (as were Corren's 1900 and Darbishire's 1908-9 results [15]), and to have all these authors employ this scoring procedure (as Olby suggests) "seems a bit thick". To Gustafsson, the interpretations of Wright and Weiling seemed more plausible.

As the controversy entered the 1970's, discussion over the excessively good fit diminished somewhat as fewer plausible explanations were proposed. Still unanswered, the question found itself growing over time, maturing with the amassing store of knowledge about Pisum. The continuing construction of a genetic map of Pisum's seven chromosomes helped transform the controversy into one of a more technical nature, centering about the distribution and locations of the genes for the seven traits Mendel worked with, and questioning whether or not he should have observed linkage.

For Mendel to develop a law of independent assortment, he had to have had

independently assorting characters. It was, in fact, Fisher who pointed out in his 1936 paper that " ··· a factor such as linkage would have been a complication extraneous to [Mendel's] theory, as he conceived it, which he would only have taken seriously had the observations forced it under his notice" [22]. One obvious assumption one could have made in 1936 – and still shared by many today – is that each of the seven genes Mendel worked with was located on a different chromosome. Since Pisum has only seven chromosomes, this would have been a very fortuitous choice indeed (the only other possibility is that, if there were any linkage, the distance between any pair of genes would have to have been quite large. Then, as crossing over of adjacent chromatids occurred, the dependent relationship of the two genes would seem erased, making the evidence for recombination virtually undetectable). The one gene per chromosome case is untrue. As early as 1951, Erst Nilsson suggested a distribution of the seven genes Mendel worked with (Table I) of <2,0,0,2,1,1,1>, locating the genes on only five of the seven chromosomes [23]. Seventeen years later, in 1968, Herbert Lamprecht published a gene map [24], in which he listed the seven traits on only four different chromosomes (Illustration I); a distribution of <2,0,0,3,1,0,1>. Unfortunately, Nilsson wrote in Swedish, Lamprecht in German, and although I. C. Murfet had attempted to publish some indication of linkage of Mendelian characters in English as early as 1972 [18], it was not until 1975 that Blixt finally succeeded in bringing this to general attention [25].

In examining the two distributions we see that they agree in all but one case, the pod shape locus. Pod shape is controlled by either of the two genes p and v in Pisum, as illustrated in Table II [26]. The difference between the two single dominant forms is only discernable to the trained observer through close inspection of the inside of the pod, and even then the difference is not always easily detected [18]. Given that Mendel studied only one locus

TABLE I

The <2,0,0,2,1,1,1> Distribution of the Seven Mendelian Characteristics
(adapted from Novitski and Blixt, 1978)

Character	Expression/Modern Symbol Dominant	Recessive	Chromosome	Locus
Seed Shape	Round R	wrinkled r	7	60
Seed Color	Yellow I	green i	1	204
Flower Color (and seed coat)	Violet A (grey-brown)	white a	1	0
Pod Shape (inedible-edible)	Smooth P (inedible)	constricted p	6	10
Pod Color (unripe)	Green Gp	yellow gp	5	21
Flower Position	Axial Fa	terminal fa	4	78
Plant Height (internode length)	Tall Le (long)	dwarf le	4	199

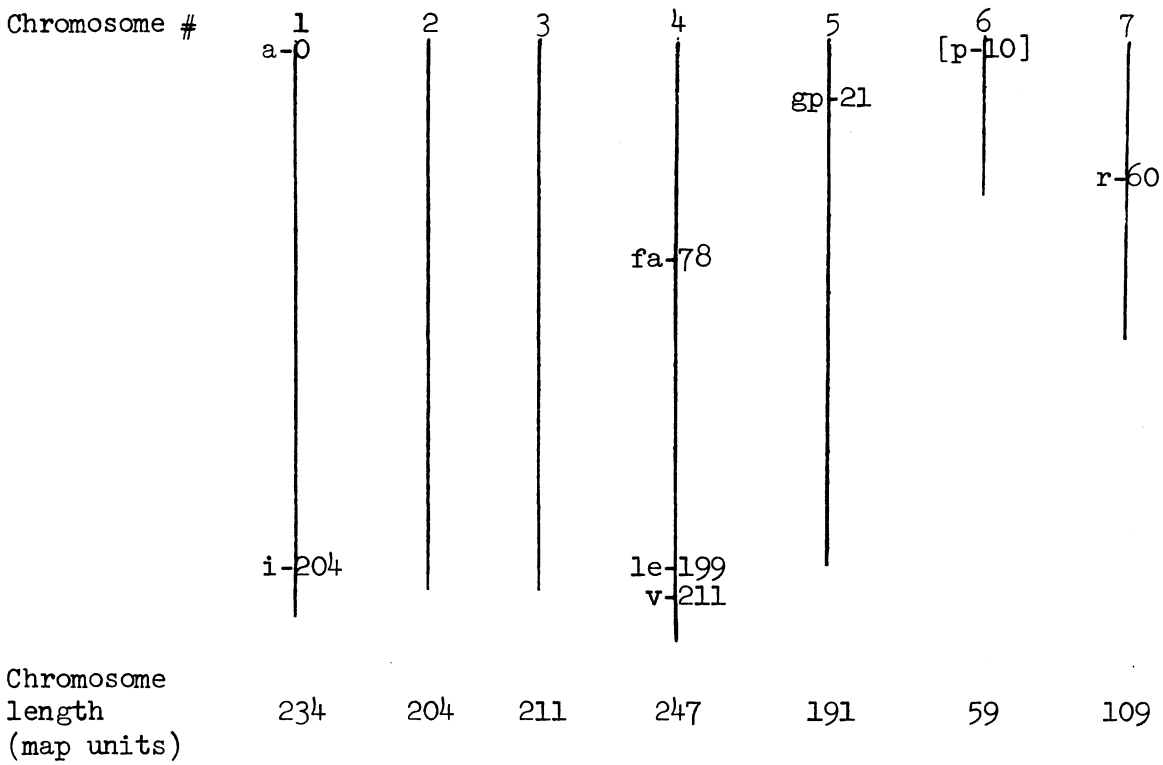
TABLE II

Pod Shape Expression by Genotype
(adapted from Blixt et al., 1978)

Genotype	Effect
P- V-	Strong membrane; normal parchmented pod
pp V-	Strip of sclerenchyma on inner pod edge
P- vv	Inner membrane reduced to patches of sclerenchyma
pp vv	No parchment; constricted pod (absence of sclerenchyma makes pods edible)

ILLUSTRATION I

The Alleles and Loca of the <2,0,0,3,1,0,1> Distribution;
Including the p-Locus (Brackets)
(adapted from Novitski and Blixt, 1978)



for edible pod – as his monohybrid data suggest – we question which of these two genes' effect it was. Nilsson suggested that it was the action of the p-locus on chromosome 6, while Lamprecht believed it was the v-locus of chromosome 4. If indeed Mendel had studied the action of the p-locus, then the only instances of linkage occur (a - i and fa-le) with such great chromosomal separation that crossing over would have rendered any evidence of recombination unnoticable. However, if Mendel had studied the effects of the v-locus, we would expect that he should have encountered some recombination in a dihybrid cross of plant height and pod shape (see Illustration I). Mendel indicated that he carried out all possible combina-

tions of crosses for the seven characters, though with smaller numbers than those of the reported multihybrid experiments [27]. Since he never mentioned anything as extraneous as recombination, stating instead that the outcomes were approximately similar to the main experiments, we can only speculate as to which locus it was, then consider the associated ramifications.

If Lamprecht's suggestion is correct, then one must question why Mendel didn't find (or at least didn't report) some evidence of linkage. Edward Novitski and Lee Douglas have proposed one possible explanation for this problem, considering Mendel's statement that "... the length of the stem varies greatly in individual varieties" [28]. As noted above, height, i.e., internode length, was probably the most difficult character for Mendel to easily classify, and though he stated that he was careful in his crossings with the trait, this is the only case where he expressed some ambiguity in classification. This might suggest that Mendel may not have examined those hybrid crosses with the le-locus in as great a detail as the others. Novitski and Douglas speculate further:

> "... the alternative that [Mendel] might even have had data suggesting le-v linkage raises a question: in view of his doubts about the constancy of le phenotypes, would he have recognised the linkage as a distinct biological phenomenon, or would he only have become more skeptical about classifying le/le? It seems reasonable to speculate that he would have been skeptical – especially since le-v is the only pair, of the $\binom{7}{2} = 21$ he could have constructed, giving 'abberant' [sic] assortment (i.e., linkage)." [23]

Also, the area of chromosome 4 about le seems to act in a particularly unstable manner; e.g., Novitski and Blixt point out a reverse mutation rate as high as 40% [29], while Lamprecht found crossover percentages between le and v ranging from $2.6 \pm 1.05\%$ to $38.5 \pm 4.32\%$ [30]. Had Mendel been working with a strain exhibiting values at the extreme ends of these spectra, the question of his not observing linkage would not be as pertinent.

If, on the other hand, Nilsson's <2,0,0,2,1,1,1> distribution is correct, then the question becomes truly academic, i.e., Mendel didn't observe linkage because no observable linkage existed. Novitski and Douglas have calculated the multinomial probabilities of randomly observing a given gene distribution in Pisum [31], and their results seem to give some weight to this possibility. The probability derived for Nilsson's proposal is Pr(<2,0,0,2,1,1,1>) = 0.2745, which is the most probable of any of the 15 possible distributions. However, Lamprecht's suggestion, favoring the v-locus, is the second most probable; Pr(<2,0,0,3,1,0,1>) = 0.2495. Further, as Blixt and Novitski point out [29],

> "... only one variety homozygous for p seems to have existed in Mendel's day, Sugarpea de Grace (Buchsbaum). This was genetically le-V-p, a weak variety, which was generally recommended only for greenhouse cultivation."

Unfortunately, Mendel never specified the varieties that he worked with. Thus, with the same spirit as the earlier question of goodness-of-fit, we might conclude that Mendel's abilities must have been sharp enough to not let anything like recombination escape his attention. Therefore unless he was working with some extreme strain (which is doubtful since he spent two years screening his stocks [32]), he must have been working with the p-locus. However, this is at best a speculative conclusion, and it is Blixt and Novitski who sum it up best:

> "[The answers] might be determined, given enough time and effort, by a search through the records of H. Lamprecht, who grew all the varieties Mendel is supposed to have used, or by a study of the 19th century German seed catalogues. However, the great extinction of old varieties in Europe during the 1940's and 1950's makes it almost certain that the original strains are not now in existence. We, therefore, doubt that absolute certainty can ever be reached ..." [29]

In the end, the only conclusion is that there are no conclusions, and one finds that there is very little to definitively conclude on the problem of linkage or on that of the "too good" fit. The storm of proposed explanations arising from the great abundance of intellectual excitement over the controversy

in and around the mid-1960's (not coincidentally about the time of the centennial of Mendel's 1865 presentation) has abated, leaving only a trickle of papers in the late-1970's as its wake. All that truly remains is the mystery.

Acknowledgements

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NOTES

[1] It is, by the way, incorrect to believe that Mendel's work was untouched for 34 years. In "The Life of Gregor Johann Mendel – Tragic or Not?", Heriditas, v. 62, p. 244, Åke Gustafsson (1969) points out that "the first outside quotation of Mendel's Pisum article was made by the professor of botany at Giessen, H. Hoffmann", in 1869. The interested reader is referred to a particularly extensive review by Alexander Weinstein (1977), "How Unknown Was Mendel's Paper?", in The Journal of the History of Biology, v. 10, pp. 341-364.

[2] A tremendous literature exists on this topic. For both a fascinating discussion and detailed citations, see William B. Provine's book, The Origins of Theoretical Population Genetics, Chicago, 1971.

[3] R. A. Fisher (1936), "Has Mendel's Work Been Rediscovered?", Annals of Science, v. 1, pp. 115-137.

[4] Ibid., p. 131. Fisher actually reported a value of 0.99993, an error which probably crept in due to a lack of precision in the algorithm or computing machine he was using at the time. The updated value of 0.99997 was calculated using Cornell University's IBM-370/168.

[5] Fisher (1936), pp. 130-132.

[6] The theoretical χ^2 variable with n degrees of freedom is a sum of squared Gaussian random variables. Thus if two χ^2 variables, with n and m degrees of freedom, are added together the resulting random variable has a χ^2 distribution with n + m degrees of freedom. If we were to test the goodness-of-fit of two or more independent events with qualitative data, we could use this fact to report the result in one statistic. Fisher (1936) used this additive property to report the goodness-of-fit of Mendel's work both as a whole and decomposed into pertinent subdivisions (p. 131).

[7] Fisher (1936), p. 125.

[8] C. Zirkle (1964), "Some Oddities in the Delayed Discovery of Mendelism", The Journal of Heredity, v. 55, p. 66.

[9] P. C. Mahalanobis (1964), "Some Personal Memories of R. A. Fisher", Biometrics, v. 20, p. 369.

[10] The letter was later published in The Origins of Genetics: A Mendel Source Book, C. Stern and E. Sherwood, eds., San Francisco, 1966, pp. 173-175.

[11] Ibid., p. 174. The example is due to Raymond Pearl, from his discussion on errors of personal equations in his book, Introduction to Medical Biometrics and Statistics (3rd ed., 1940), p. 85.

[12] Sewall Wright (1980), Laboratory of Genetics, Univ. of Wisconsin, Madison. Personal communication.

[13] Wright in Stern and Sherwood (1966), pp. 174-175.

[14] Franz Weiling, Has J. G. Mendel Been "Too Accurate" in His Experiments? The χ^2 Test and Its Significance to the Evaluation of Genetic Segregation, translated from the German version, in Der Züchter (1966) v. 36, by W. W. Piegorsch; Paper No. BU-718-M in the Cornell University Biometrics Unit Mimeo Series, 1980, p. 20.

[15] Ibid., pp. 13-14.

[16] Gustafsson (1969), p. 254.

[17] T. Dobzhansky (1967), "Looking Back at Mendel's Discovery", Science, v. 156, p. 1588.

[18] G. A. Marx (1980), editor, Pisum Newsletter, N.Y.S. Agricultural Experiment Station, Geneva, New York. Personal communication.

[19] W. Bateson, Mendel's Principles of Heredity, 3rd ed., New York, 1913, p. 8.

[20] Gustafsson (1969), pp. 256-257.

[21] V. Orel (1968), "Will the Story on 'Too Good' Results of Mendel's Data Continue?", BioScience, v. 18, p. 777.

[22] Fisher (1936), p. 133. Note that the linkage had not yet been discovered.

[23] L. Douglas and E. Novitski (1977), "What Chance Did Mendel's Experiments Give Him of Noticing Linkage?", Heredity, v. 38, p. 255.

[24] H. Lamprecht (1968), Die Nene Genenkarte von Pisum und warum Mendel in Seinen Erbsen-Kreutzungen keine Genenkoppelung gefunden hat. Arb. Steiner-märkischen Landesbibliothek Joanneum, Graz.

[25] S. Blixt (1975), "Why Didn't Gregor Mendel Find Linkage?", Nature, v. 256, p. 206.

[26] S. Blixt, G. A. Marx, and I. C. Murfet (1978), "Descriptive List of Genes for Pisum", Pisum Newsletter, v. 10, pp. 95, 100.

[27] G. Mendel in Stern and Sherwood (1966), p. 22.

[28] Ibid., p. 7.

[29] E. Novitski and S. Blixt (1978), "Mendel, Linkage, and Synteny", BioScience, v. 28, p. 34.

[30] Lamprecht (1968), p. 10.

[31] Douglas and Novitski (1977), pp. 255-256. Note that they have taken the lengths of the seven chromosomes into account, making the calculations far more precise than might be expected.

[32] G. Mendel in Stern and Sherwood (1966), p. 4.