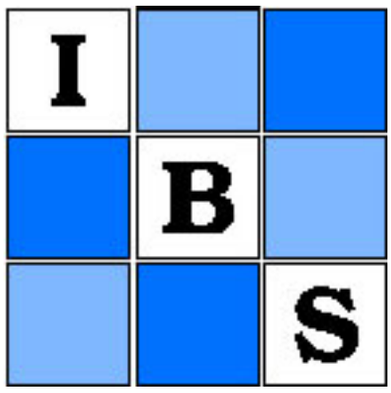

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Fisher's Contributions to Genetics and Heredity, with Special Emphasis on the Gregor Mendel Controversy

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SUMMARY

R. A. Fisher is widely respected for his contributions to both statistics and genetics. For instance, his 1930 text on *The Genetical Theory of Natural Selection* remains a watershed contribution in that area. Fisher's subsequent research led him to study the work of (Johann) Gregor Mendel, the 19th century monk who first developed the basic principles of heredity with experiments on garden peas. In examining Mendel's original 1865 article, Fisher noted that the conformity between Mendel's reported and proposed (theoretical) ratios of segregating individuals was unusually good, "too good" perhaps. The resulting controversy as to whether Mendel "cooked" his data for presentation has continued to the current day. This review highlights Fisher's most salient points as regards Mendel's "too good" fit, within the context of Fisher's extensive contributions to the development of genetical and evolutionary theory.

1. Introduction

Among Ronald A. Fisher's (1890–1962) many contributions to the furtherance of science, his advancements in the field of genetics and heredity are rivaled only by those in the field of biometry and statistics. The two efforts were often intricately related. Indeed, major advances in biometry are often catalyzed by subject-matter problems, and the study of genetics and heredity has provided great motivation for such advances in many quantitative fields (Stigler, 1986, p. 266). Some of history's greatest statisticians—among them Galton, Pearson, (Wilhelm) Weinberg, Weldon, Yule, and of course Fisher—were all deeply involved in genetic application areas (Olkin et al., 1990).

Fisher's interests in genetics, and more generally biological study, were stimulated while he was a student at Cambridge University, after reading Karl Pearson's work on mathematical aspects of evolutionary theory (Box, 1983; Yates and Mather, 1963). [A classic anecdote regarding Fisher's decision to study mathematics instead of biology relates his astonishment over the complex taxonomic classification of vertebrate bone structure. Apparently, during a visit to a museum, the young Fisher "happened on a cod's skull with all its bones separated and labelled . . ." (Yates and Mather, 1963, p. 92). The resulting impression of the intricacies and possible drudgery of biological nomenclature was strong enough to send him into the study of mathematics (Mather, 1963)!] Fisher went on to contribute over 150 articles, reviews, or books to the fields of genetics and heredity,

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evolution, and eugenics (Healy, 1963), many of which provided fundamental advancements to those specific fields and to scientific thought in general.

Fisher's early interest and research into problems of evolutionary theory coincided with the maturation of an embittered debate in the scientific community. This debate concerned the underlying mechanisms of hereditary effects and their implications for evolution. The mid-19th century publication of Charles Darwin's theory of continuous evolution based on natural selection began an exciting era of biological thought. The theory sparked great debates as to its accuracy and propriety, not the least of which occurred among biologists studying their implications. [Technically, one should discriminate between evolution as a theory of species' "descent with modification," and natural selection as a sort of differential fertility based on advantageous characteristics arising within or among species. Indeed, as a reviewer has noted, the current consensus among historians of science argues that around the turn of the century there was little support for Darwinism, when the latter was interpreted as a theory of evolution *by means of natural selection* in contrast to a theory of "descent with modification;" see, e.g., Hull (1985), Mayr (1985), and Provine (1985).]

Scientific study and discourse regarding Darwin's theories in the 1900s included that of a so-called "biometric" school of researchers. These luminaries attempted to corroborate Darwinian theories via quantitative investigations of the apparently continuous variability of traits in biological populations. They included Pearson and Weldon, and, as might be expected, early issues of *Biometrika* often provided important contributions to this corroborative research. This activity was contrasted, however, by the "rediscovery" in 1900 of (Johann) Gregor Mendel's (1865) classic treatise on simple inheritance in the garden pea. [Also see an English translation in Mendel (1966); Mendel's experiments will be discussed in greater detail in §3, below.] Mendel's data suggested that inheritance was, at the genetic level, a combination of discrete events leading to discretely varying traits (e.g., round vs wrinkled peas); this appeared to conflict with the observed data corroborating "Darwin's" continuous variability for genetic events. The ensuing controversy over the nature of heredity and evolution often reached bitter extremes, with the two camps ("biometricians" vs "Mendelians") yielding very little to each other's arguments (Provine, 1971, §3). Into this fray entered Fisher, whose contributions made major strides forward and, in fact, reconciled the two opposing theories. These and some other of Fisher's contributions are reviewed briefly in Section 2.

Part of Fisher's effort in the reconciliation of Mendelism and (biometric) Darwinism was the recognition that only superficial scientific scrutiny had been directed at Mendel's article by both its proponents and opponents (Box, 1978, p. 297). By the early 1930s, Fisher had undertaken to critically review Mendel's original article, going further—in a sense—than those earlier researchers. His conclusions included an astonishing calculation: Mendel's data appeared to fit his own expectations and proposed theories *far better* than expected by chance alone. The quality of the data (but *not* the theory itself), and even Mendel's historical reputation were soon called into question by the scientific community. Ironically, after reconciling one scientific controversy on the effects of heredity in evolution, Fisher's research helped spark another! In Sections 3 and 4, some of Fisher's calculations and the concerns he raised on the issue of Mendel's data are given particular emphasis, within the context of Fisher's many contributions to genetics and heredity.

2. Some Contributions to Genetics

As introduced above, Fisher was actively researching problems in genetical and evolutionary theory early in his career. By 1911, as a student at Cambridge, he was active in the University's Eugenics Society, and even spoke at its second meeting (Box, 1978, pp. 1–3,

27). His theme was "Mendelism and Biometry," wherein he outlined his perceptions that the two competing lines of thought on hereditary effects were, in fact, wholly compatible with one another, and with a Mendelian underpinning to a theory of evolution based on natural selection. To wit, the observation that continuous variability was seen throughout nature (and was quantified by Pearson and others) could be easily explained by accepting Mendel's observations that inheritance at its basic levels was discrete and discontinuous. One need simply recognize the apparently continuous variability as the cumulative effect(s) of many Mendelian, discretely inherited factors acting in an additive manner!

By 1916, Fisher had collected together his thoughts into a manuscript (Fisher, 1918) detailing how these calculations could be applied to correlations among relatives in human populations. Writing to Pearson in June 1916, Fisher noted that he had ". . . recently completed an article on Mendelism and Biometry which will probably be of interest . . . . I find on analysis that the human data is as far as it goes, not inconsistent with Mendelism" (Pearson, 1968, p. 454). [Pearson was unimpressed. His son reports a return correspondence, apparently dated 21 October 1918, in which the elder Pearson admitted, "I am afraid I am not a believer in cumulative Mendelian factors as being the solution to the hereditary puzzle" (Pearson, 1968, p. 456).] Fisher's manuscript was submitted to the Royal Society, where it met with an unfavorable reception. The reviewers were Pearson, the biometrician, and R. C. Punnett, a renowned geneticist whose Balfour chair at Cambridge Fisher would, ironically, later hold. Although Pearson and Punnett were major proponents of opposing theories of heredity, and ". . . may have agreed on nothing else in life, . . . they were united in rejecting Fisher's paper; each [discarding] it as a [minimal] contribution to his own subject" (Box, 1978, p. 59). The manuscript languished for two years until, with the help and urging of Leonard Darwin (Box, 1978, p. 60; Norton, 1976), Fisher was finally able to publish in the *Transactions of the Royal Society of Edinburgh* (Fisher, 1918).

Along with the important reconciliation the 1918 article provided between biometric and Mendelian theories of heredity, it effectively introduced the concept and study of "biometrical genetics" (Mather, 1963; Rao, 1964), a fusion of the two previously disparate fields (Box, 1978, p. 53). It gave also the first appearance of the technical use of *variance* as squared standard deviation to describe observed variability, and of the decomposition of variance into component parts. These were clearly important landmarks in the development of statistical and quantitative methods, and the 1918 article has since been recognized as one of Fisher's greatest scientific contributions (Box, 1983; Crow and Dove, 1990; Mather, 1963; Owen, 1962; Rao, 1964).

By 1918, Fisher's interests in genetics and heredity were developing rapidly. In particular, he directed great attention to the impact of those fields on the development of evolutionary theory. Besides reconciling the jump from Mendelian to continuous hereditary factors, Fisher's 1918 article also laid the foundations for further development of Darwin's ideas on evolution and natural selection. Indeed, part of the Mendelian–biometric controversy encompassed the pre-Fisher concern that Mendel's discrete hereditary factors did not conform with continuously varying *evolution* as proposed by Darwin. It was supposed that small variations and gradual selective pressure could not wholly account for the vast and varied differences observed among species. Some additional mechanism leading to externally expressed ("phenotypic") change was thought to be necessary—or at least concomitant—in the evolutionary process. To help explain this, the botanist Hugo de Vries introduced a theory of discontinuous evolution via alterations of old species' characteristics into new ones. He called these alterations "mutations" (de Vries, 1901), and employed them to describe the discrete, blatant changes observed within various botanical species. De Vries was, in fact, one of the rediscoverers of Mendel's original work, publishing his own investigation into segregation of hybrid plant varieties in 1900 (de Vries, 1900, 1966).

Along with other experimental results achieved by many of Mendel's proponents—including the biologist William Bateson—these showed that Mendel's findings on discrete inheritance were easily replicated. This provided strong evidence for discrete variation as the early Mendelians—including Bateson, Punnett, and others—had proposed (Bateson, 1913). These researchers "... felt their findings to be incompatible with the principle of evolution by natural selection and especially with the significance Darwin attached to small variations in adaptation and evolution" (Mather, 1963, p. 167). Fisher's entrance into the evolutionary debate via his 1918 article inspired further thought among biologists and biometricians on discontinuous-vs-continuous variations, and scientists began to recognize that many earlier concerns on the issue were perhaps overstated. Characteristically, Fisher led the way, and his subsequent researches culminated in publication of *The Genetical Theory of Natural Selection* (1930a). The book completed Fisher's reconciliation of Mendelian hereditary theory with Darwinian concepts of natural selection by emphasizing that natural selection's operation on biological systems led naturally to their evolution (Yates and Mather, 1963). In addition, the book contained Fisher's thoughts on the evolution of sexual selection, and began development on his theories of the evolution of dominance. Most importantly,

> ... just as Fisher had brought together Mendelism and biometry in the [1918 article] on correlations between relatives, and had displayed how these different instruments for the scientific representation of nature complemented each other, so in *The Genetical Theory of Natural Selection* he brought genetics together with evolution by natural selection, rewriting Darwin's score in the symbols of [the emerging field of] population genetics. (Box, 1978, p. 186)

In particular,

> ... it followed from Fisher's work that Darwin's hypothesis of environmental [causation] of variability was unnecessary .... The essentials remained [however]—natural selection operated on the abundant supplies of variance kept in being by the Mendelian mechanism [of inheritance]. (Owen, 1962, p. 452)

Fisher's 1930 text remains a watershed contribution to the development of evolutionary biology and genetics, much the way his classic text on *Statistical Methods for Research Workers* (1925) advanced the use and acceptance of the basic ideals of experimental statistics.

There were, of course, many more contributions made by Fisher to genetical study, and the next section provides some detail on his in-depth reconstruction of Mendel's original data (Fisher, 1936) and his concerns about those data. Before moving to that issue, though, it is worth mentioning briefly some of Fisher's other interesting and important forays into genetical thought. These include his famous breeding experiments with mice [see Box (1978, pp. 172–174) and, e.g., Fisher (1930b), Fisher and Mather (1936a; 1936b)]; studies of mutation and dominance in poultry (Fisher, 1934a, 1934b, 1935); of gene survival and spread (Fisher, 1937; Fisher and Ford, 1947), and of gene linkage in humans. The latter research led to advances in serology and furthered the development of human blood group classifications (Race, 1964), in which Fisher and a team of colleagues at University College's Galton Laboratory advanced a three-locus theory for inheritance of the Rhesus factor (Fisher, 1947a; Fisher, Race, and Taylor, 1944). The theory actually anticipated (correctly) the existence of two unobserved antisera and one unobserved (rare) allele (Yates and Mather, 1963).

Statisticians may find interesting Fisher's application of his theory of maximum likelihood to gene frequency and recombination frequency estimation. Estimation for the multinomial models employed in such studies is hindered by many factors, and many of

the special forms of statistical estimation these models require were contributed by Fisher; see Finney (1964). Further, his concepts of statistical orthogonality and factorial design played important roles in his (1947b) development of the theory of polysomic inheritance (Finney, 1964).

Interested readers are referred to the excellent reviews of Fisher's contributions to genetics, including those by Owen (1962), Mather (1963), and Race (1964), to Joan Box's informative biography of her father (Box, 1978), and also to the 1964 Fisher memorial issue of *Biometrics* (Vol. 20, No. 2). Lastly, of particular charm is a 1967 reminiscence by Cochran, including an amusing anecdote on Fisher as an "applied" geneticist:

> We were standing at the corner of Euston Rd. and Gower St. in London, waiting to cross .... Traffic was almost continuous and I was worried, because Fisher could scarcely see and I would have to [lead] him .... Finally there was a gap, but clearly not large enough to get us across. Before I could stop him he stepped into the stream, crying over his left shoulder "Oh, come on, Cochran. A spot of natural selection won't hurt us." (Cochran, 1967, p. 1462)

3. Fisher's Concerns with Mendel's Data

The confusion and misinterpretations of Mendel's theories that seemed to pervade early 20th century biological thought clearly struck Fisher as hindrances to the advancement of his own theories, and to scientific thought in general. Just as clearly was he struck by the contrasting clarity of thought and presentation in Mendel's original (1865) article, and this anomalous contrast led Fisher to examine "Mendel's genius in its own terms, and not as it had been perceived" (Box, 1978, p. 295). Taking advantage of an invitation to submit a work to the newly formed *Annals of Science*, Fisher prepared in late 1935 a quantitative reexamination and reconstruction of Mendel's data. The article appeared in 1936.

Mendel (1822–1884) was an Augustinian monk who served in the Monastery of St. Thomas in Alt-Brünn (in what is now Brno, Czechoslovakia), eventually becoming Abbot of the Monastery. A comprehensive, if perhaps idealized, biography of Mendel was given by Iltis (1932); more modern perspectives are available in Gustafsson (1968) and Olby (1985).

As part of his duties in the 1850s and 1860s, Mendel conducted breeding experiments in the monastery garden to examine and improve edibility, disease/pest-resistance, and yield in the vegetables grown for monastery consumption. During this time, he became interested in the pattern of inheritance of certain traits in various garden plants, and he conducted a series of experiments to study these patterns further. His experiments with the garden pea, *Pisum sativum* (and other associated species), examined seven simple traits: seed shape (*R*), seed color (*I*), flower color (*A*), pod shape (*P*), pod color (*Gp*), flower position (*Fa*), and plant height (*Le*). (The letters in parentheses are the modern symbols used in denoting the genes for these traits.) Each trait maintained one of two characteristic forms, either dominant or recessive. The modern notation indicates this by assigning an uppercase leading letter for a dominant gene—e.g., *A* or *Gp*—and all lowercase letters for a recessive—e.g., *le*. When an individual plant's internal genetic ("genotypic") character is mixed ("heterozygous") for a specific trait, such as *Aa* for flower color, it is known as a hybrid form. Mendel's work with the pea consisted of mono-, di-, and tri-hybrid (i.e., one-, two-, and three-trait hybrid) crosses. From them, he proposed laws of heredity that have become the cornerstone of our modern hereditary theory. His results from these experiments were reported to the Brünn Society for the Study of Natural Science in early 1865.

For example, Mendel's dihybrid experiment assessed hereditary activity of the phenotypic traits for seed shape and seed color. In *Pisum*, seed shape characters are either (dominant) round or (recessive) wrinkled (with the modern designation for the corresponding genotypes

given by $R-$ and rr); seed color characters are either (dominant) yellow ($I-$) or (recessive) green (ii). Under Mendel's theory, simple inheritance is based on the presence of copies of dominant or recessive genes: presence of at least one dominant copy assures expression of the dominant phenotype for an individual trait. The recessive phenotype is expressed only if both copies of the recessive gene are present. [One must be careful not to ascribe to Mendel the genotypic interpretation of inheritance denoted so simply and elegantly by our modern notation. It is not clear that in 1865 Mendel had actually conceptualized the outline of what we have come to call "Mendelian genetics" (Olby, 1979), and use of the modern notation herein is not intended to place Mendel in a 20th century frame that was not his own.]

To examine how these effects were expressed with two traits, Mendel prepared two pure-breeding, homozygous lines of round/yellow (RR/II) and wrinkled/green (rr/ii) parents, and then crossed these pure-breeding lines to achieve a first filial (F_1) generation of true hybrids: all offspring were round/yellow hybrids (Rr/Ii). He then crossed the hybrid F_1's, and observed in the F_2's a *segregation* of the original traits into four categories:

	Observed
$R-/I-$ (Round/Yellow)	315
$rr/I-$ (wrinkled/Yellow)	101
$R-/ii$ (Round/green)	108
rr/ii (wrinkled/green)	32
	556

Under our modern interpretation of Mendel's proposed theories, we would expect the F_2 offspring to exhibit a 9:3:3:1 phenotypic ratio among these four outcome categories; for $N = 556$ observations, this is 312.75:104.25:104.25:34.75.

Fisher's goal in examining these data was to reconstruct the observations in a chronological fashion. This then would allow him to gain a better understanding of Mendel's experimental and inferential techniques. As Box reports (1978, p. 295):

> [By] 1932 [Fisher had come to attribute] the misunderstanding of Mendel's work by its rediscoverers to their lack of appreciation of its logical implications. In contrast, to him the logical and mathematical aspects of Mendel's work were most impressive. He felt that despite the popularization of ideas attributed to Mendel as the father of modern genetical knowledge, very little attention had been paid to Mendel's paper itself. [Fisher's] was a missionary spirit, wishing to exhibit Mendel's genius in its own terms . . .

As part of his reconstruction, however, Fisher made a startling discovery: "The bias [in favor of expectation] seems to pervade the whole of the data . . ." (Fisher, 1936, p. 131). That is, Mendel's data appear to fit his expectations excessively well. For example, with the dihybrid data discussed above, the expected ratio under Mendel's theory is 9:3:3:1. The observations show a ratio of 9.05:2.93:3.11:0.91. Testing for goodness of fit gives a statistic of $\chi^2 = .47$ on 3 degrees of freedom. Since the upper-tail area, or P-value, corresponding to this test is $\Pr[\chi^2_3 \geq .47] = .925$, no significant deviation is evidenced from the proposed model. The very large P-value also suggests, however, that the data appear to fit the model very well. If this pattern were to be seen throughout, some question could be raised regarding the actual experimental validity of the data.

Fisher's attention was sparked by the following recognition: in testing for monohybrid ratios, Mendel crossed hybrid F_1's

$$Aa \times Aa$$

to achieve in the next (F_2) generation

AA	*Aa*	*aa*
Dominant		recessive

with expected phenotypic ratio (dominant:recessive) of

3 : 1.

It was then of interest to test the genotypic ratios of the dominant F_2 offspring (i.e., examine under what ratios the *AA* and *Aa* individuals were segregating). To do so, Mendel employed the following technique: first he self-crossed 100 Dominant F_2's. Then, he (randomly) examined *ten* F_3 offspring per cross. If any F_3 offspring exhibited the recessive trait (*aa*), the parent was classified as a hybrid, i.e., *Aa*.

Fisher reasoned that this design technique would generate a *misclassification probability* equal to $(\frac{3}{4})^{10}$, or 5.63%, of incorrectly identifying a hybrid F_2 as homozygous dominant. The corresponding expected genotypic ratio (homozygous dominant:hybrid) for the dominant F_2's is therefore not 1:2 (as Mendel hypothesized), rather 1.1126:1.8874.

Application to the six such experiments Mendel performed shows (Fisher, 1936, Table III):

Plants tested	*Pure dominant plants*	*Expected (uncorrec.)*	*Expected (correc.)*
600	201	200	222.5

From this, Fisher suggested that the data appeared to agree with Mendel's expectations far better than they agreed with expected values corrected for misclassification.

The expectation-vs-misclassification anomaly identified in the tests for genotypic ratios led Fisher to examine the entire data set in greater detail. With his reconstructed data set, Fisher calculated χ^2 to test goodness of fit on a series of pertinent subdivisions of Mendel's data. Throughout, there was exceptionally good agreement with (Mendel's) expectations. [For further details on the statistical issues, and some additional perspectives of Fisher's approach, see the reviews by Piegorsch (1983; 1986), Root-Bernstein (1983), and Edwards (1986).]

Overall, Fisher's analysis gave $\chi^2 = 41.6056$ on 84 degrees of freedom. [A corrected analysis by Edwards (1986) suggests $\chi^2 = 41.9509$.] Thus, e.g., $\Pr[\chi^2_{84} \geq 41.9059] = .99997$; similarly striking *P*-values were calculated for various subdivisions of the data. The implication was that the data appeared to agree with Mendel's expectations very well; i.e., as a whole, only 3 such experiments out of 100,000 would be expected to agree as well or better with Mendel's theory as he envisioned it.

Among Fisher's conclusions, therefore, one finds the concern that the data may have been "... falsified so as to agree closely with Mendel's expectations" (Fisher, 1936, p. 132). This perception has been taken by many to suggest, or at least to question whether Mendel actually performed the falsification (Broad and Wade, 1982; Mahoney, 1979; Zirkle, 1964). Based on Mendel's exemplary personal, community, scientific, and religious reputation (Weiling, 1984), this possibility seems unlikely. Even Fisher relented: "... it remains a possibility ... that Mendel was deceived by some assistant who knew too well what was expected" (Fisher, 1936, p. 132), and in a January 1936 letter to E. B. Ford, Fisher emphasized "I cannot conceive that Mendel himself had any hand in [any falsification of the data]" (Edwards, 1986, p. 295). Nonetheless, Mendel's hybridization data do exhibit unusually close agreement with (Mendel's) theory, even when mitigated by more modern attempts to understand the nature of χ^2 when applied to data of this form (Robertson, 1978; van der Wärden, 1968; Weiling, 1966, 1986, 1989). The controversy over the data's nature and origin remains unresolved.

4. Fisher's Conclusions Regarding Mendel's Work

The sensationalism Fisher's quantitative discovery engendered had a perhaps unintended, deleterious effect: it overshadowed Fisher's other conclusions regarding Mendel's experiments. In spite of the concern over the "cooked" data, Fisher argued that Mendel's experimental methodology was an advance well ahead of its time. Instead of the then-common practice of crossing different species to examine the hereditary effects of hybridization, Mendel made a conceptual advance by examining hybrids of closely related varieties of one species [although it is unclear whether Mendel realized the full value of his experimental technique; see Olby (1979)]. Thus, instead of encountering large numbers of differing traits or factors—i.e., unnecessarily large variability—in his hybrid progeny, as was often seen by his experimental contemporaries, Mendel reported on data with very specific endpoints (Fisher, 1936, p. 142). By simplifying the experimental question to one of dichotomous differences among (progeny of) pure-breeding lines, Mendel afforded himself the opportunity to describe the underlying, discrete nature of the pea's hereditary system.

These important methodological advances appeared to be ignored, or at least misinterpreted, well into the 20th century. Fisher's explanation as to why was, perhaps, his most important (and prophetic—see below) observation (Fisher, 1936, p. 137, emphasis added):

> Mendel's contemporaries may be blamed for failing to recognize his discovery, perhaps through resting too great a confidence on comprehensive compilations. It is equally clear, however, that since 1900, in spite of the immense publicity it has received, his work has not often been examined with sufficient care to prevent its many extraordinary features being overlooked, and the opinions of its author being misrepresented. *Each generation, perhaps, found in Mendel's paper only what it expected to find*; in the first period a repetition of the hybridization results commonly reported, in the second a discovery in inheritance supposedly difficult to reconcile with continuous evolution. Each generation, therefore, ignored what did not confirm its own expectations.

Thus Bateson and his colleagues read and understood Mendel's methodology only as it related to (and substantiated) their own experimental data. Similarly, Pearson, Weldon, and the opposing camp of "biometricians" may have recognized the "wonderfully consistent way in which Mendel's results agree with his theory" (Weldon, 1902, p. 232), but they also readily dismissed these results as inconsequential or inappropriate to the "greater" questions of human heredity and evolution.

Even Fisher's work has fallen prey to some modern writers' inabilities to read past their own (pre)conceptions. Fisher is often identified among popular writers as Mendel's intellectual assassin and scourge for his discoveries regarding the "too good" fit of Mendel's data. As noted above, however, Fisher *never* argued that the (possible) falsification of the data was Mendel's doing, and valiantly suggested another alternative: the "overzealous" assistant. Although this particular solution to the data falsification problem remains in doubt (Dobzhansky, 1967; Gustafsson, 1968), the fact remains that one of Fisher's basic conclusions espoused support and admiration for Mendel's scientific work. It is inappropriate and misleading to paint Fisher in any other light. Indeed, one cannot help but argue that both Mendel *and* Fisher provided substantive, groundbreaking advancements to the theory and practice of genetics. Clearly, they are both deserving of praise, support, and admiration for their varied and important contributions.

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anonymous reviewer for some very useful suggestions. Also, one must take Fisher's concern regarding careful examination of original works (see §4) to heart: I can only hope that I have examined all the sources quoted herein with the scrutiny and concern that Fisher himself would have deemed appropriate.

Résumé

R. A. Fisher est largement respecté pour ses contributions à la fois à la statistique et à la génétique. Par exemple son texte de 1930 sur *The Genetical Theory of Natural Selection* reste une contribution de référence dans le domaine. Les recherches correspondantes de Fisher le conduisire à étudier le travail de (Johann) Gregor Mendel, le moine du 19ème siècle qui le premier développa les principes de base de l'hérédité à partir d'expérimentations sur le pois. En examinant l'article original de Mendel de 1865, Fisher remarqua que l'accord entre les ratios, cités et proposés (théoriques) par Mendel, de ségrégation d'individus était anormalement bon, "trop bon" peut-être. La controverse qui s'en suivit porta sur une éventuelle falsification par Mendel de ses données sous une forme encore présentée actuellement. Cet article met en lumière les arguments les plus caractéristiques de Fisher vis à vis d'un ajustement "trop bon" par Mendel, dans le cadre des contributions de Fisher au développement de la théorie génétique et de l'évolution.

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