washed cells in Penassay broth. The antibiotic in the concentration used (10 $\mu$g/ml) suppressed cell division. The number of colonies, $\times 10^6$ per 0.1 ml, was 16, 21, 6, and 10, respectively, after incubation of 0, 60, 180, and 300 minutes. Cells from the chloramphenicol broth became much larger than cells from plain broth, particularly by 300 minutes (compare Fig. 2, *d* and *e*, with Fig. 2f). These antibiotic-treated cells, which had been labeled by the direct method, showed no change at zero time (Fig. 2*a*). In the interval between 60 to 180 minutes, some irregularity of the fluorescent outline became increasingly apparent (Fig. 2, *b* and *c*). At 300 minutes of incubation, there was definite nonfluorescent, irregular interruption of the still-brilliant fluorescence on the cell wall (Fig. 2, *d* and *e*).

By the methods used, observation is restricted to behavior during replication of those cell wall components that are antigenic and accessible. If the described behavior of the labeled antigens is considered representative of the sequence of events occurring in the wall as a whole (including the mucopeptide), then it is apparent that the mode of cell wall replication in *Salmonella typhosa* is quite different from that in *Streptococcus pyogenes* (*1*). My observations indicate that for immunofluorescence to decrease with time of incubation, the bacteria so labeled must be living and dividing; the gradual decrease in fluorescence is attributed to wall replication by a process of continuous diffuse interaction of new materials into old wall. Chloramphenicol, though not preventing replication at the concentration used, slows or limits such a process, resulting in the appearance of unlabeled portions in multiple sites between regions of old and still-labeled wall of the enlarged but nondividing cells. This observation also appears to demonstrate visually that cell wall synthesis continues in *Salmonella typhosa* in the presence of chloramphenicol, although we have no concomitant evidence of the lack of protein synthesis such as that shown in prior biochemical studies of continued wall synthesis by *Staphylococcus aureus* grown with added chloramphenicol (*8*).

These findings are inconsistent with the "growing-point" hypothesis of wall replication of flagellated bacilli, as expressed by Bisset *et al.* (*9*), by which one "daughter" cell would appear with entirely new wall. Instead, the results support the ideas of Quadling, Stocker, and Kerridge (*10*), based on the study of the unilinear transmission of motility and of the sharing of parental flagella at division, that replication of the wall of the salmonellas must be by diffuse intercalation. Additional evidence for their view appeared during preparation of this report. By indirect immunofluorescence, May (*11*) has shown that the adsorbed antibody marker on the cell wall (of *Salmonella typhimurium*) became "uniformly dispersed" with time of incubation.

It therefore appears that the mode of cell wall replication may differ with the organism, although the result in all instances studied thus far appears to be (*11*) that each of the progeny after a cell division receives essentially half old wall and half new. It remains for similar methods to be applied to study of other microorganisms.

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## Multiple Authorship Trends in Scientific Papers

Abstract. *Since 1946 biomedical writers have shown no marked trend toward multiple authorship; the average number of authors per paper remains steady at about 2.3. This is in strong contrast to the conclusion of Price from a study of* Chemical Abstracts *that the chemists' trend toward four or more authors per paper has been during this period, and continues to be, steeply exponential.*

Price (*1*), apparently on the basis of a sampling of *Chemical Abstracts* for the period 1910–60, concludes that "a detailed examination of the incidence of collaborative work in science shows that this [the trend towards multiple authorship] is a phenomenon which has been increasing steadily and ever more rapidly since the beginning of the century."

I propose that, since this does not hold historically for biomedical papers, the generalization quoted, important as it is for students of changing patterns in scientific publication, is not valid for science as a whole. Authorship distributions in papers presented (*2*) at the annual meetings of the Federation of American Societies for Experimental Biology, from 1934 to 1963, were analyzed statistically for trend by using total counts or random samples. If a trend toward multiple authorship has existed since 1946, it is minute (Table 1 and Fig. 1).

In order to check the assumption that the papers in the data sources were randomly distributed with respect to different numbers of authors, random-sample counts (33 percent) as well as total counts were made of 2 years, 1942 and 1946. Random-sample percentage counts agreed in each authorship category within $\pm 2\sigma$ with the corresponding total count percentages; indeed, except in one instance, one-author papers for 1946, the agreement was within $\pm 1\sigma$.

Inspection of Fig. 1 leads immediately to a generalization: the factors in play during 1934–46 were different, in nature and quantitative effect or both, from those affecting authorship distributions from 1946 to date. Among the five curves after 1947 only that for one-author papers gives clear visual evidence of a trend. It was therefore decided to subject all the data to statistical tests for presence of trend.

Two tests were used: (i) The estimation of slope $b$ and its standard deviation $\sigma_b$ of the straight line fitted by least squares through the observed points (*3*); and (ii) the method of

mean square successive differences for detection of trend (*4, 5*).

This ratio of $b$ to $\sigma_b$ depends upon the slope and the error of the slope as estimated from the residuals of the data points from the fitted line. This error includes not only the sampling error but also any departure of the data from linearity. If the plotted points lie along a curve any straight line will be a poor fit and the value obtained for $\sigma_b$ will be larger than the sampling error. Thus there may be a trend in the data that goes undetected because $\sigma_b$ is large as a result of poor fit. The mean square successive differences technique does not postulate a linear trend in the data.

For this test a ratio is computed:

$$D^2/S^2 = \frac{\Sigma\delta^2_n}{\Sigma(y_i - \bar{y})^2},$$

where $\delta_n = (y_{n-1} - y_n)$. Bennett (*4*) computed values below which $D^2/S^2$ must fall, for the various numbers of observations $n$, before there is indication of a trend. By interpolation from Bennett's table, this value for $n = 17$ is 1.26 ($P = 0.95$). Table 2 summarizes the results of these tests.

All the curves, with the exception of that for two-author papers, exhibit a discontinuity at about 1946. No such discontinuity is evident in the Price (*1*) graphs for chemists.

The curves for the 1934–46 period parallel the Price graphs, except the curve for two authors. Incidentally, if the sampling error of a single point is 2 percent, the slope of a line based on 17 yearly points should be determined with $\sigma_b$ equal to 0.10. In this study the average value for $\sigma_b$ for one, two, three, and four or more authors is 0.114. This shows that the assumption of linearity is satisfactory over this range.

For the 1947–63 period, both test methods agree that there is no trend in the two- or three-author and the authors-per-paper curves. The evidence for one-author and four-or-more-author papers is inconclusive: the line fitting leads, by a small margin, to a negative slope for the one-author papers, while successive differences do not indicate a trend; and for four or more authors, line fitting gives no hint of a slope, while successive differences indicate a trend, again by a small margin.

Although the line-fitting method is inherently the more sensitive, it is probably conservative to hand down the Scottish verdict of "not proven," and to say that a slight trend *may* exist, or at least be in the making, for these data. One can only await the unrolling of several additional years, when new points on the curves will probably resolve the matter; for as $\Sigma x^2$ increases, the precision with which the slope may be estimated will become correspondingly greater.

However, this evidence touches only the periphery of a picture whose main features are quite clear. Since 1946 there has been no continuation of a marked trend toward multiple authorship among biomedical writers, and thus generalizing from a study of chemical literature to that of all science is not justified.

Table 1. Numbers of authors of biomedical papers.

| Year | Percentage of papers with 1, 2, 3, 4 or more authors, respectively | | | | Authors per paper | Sample size ($N$) |
|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 or more | | |
| 1934 | 34.2 | 42.7 | 18.3 | 4.7 | 1.95 | 459* |
| 1935 | 35.6 | 38.4 | 19.6 | 6.4 | 1.99 | 495* |
| 1936 | 34.7 | 42.2 | 17.4 | 5.7 | 1.96 | 562* |
| 1937 | 34.6 | 40.7 | 18.8 | 5.8 | 1.98 | 543* |
| 1938 | 34.8 | 42.2 | 17.8 | 5.1 | 1.94 | 669* |
| 1939 | 32.5 | 41.8 | 21.2 | 4.5 | 1.98 | 670* |
| 1940 | 35.2 | 38.4 | 20.5 | 5.8 | 1.97 | 667* |
| 1941 | 27.6 | 45.2 | 19.6 | 7.6 | 2.10 | 852* |
| 1942 | 31.9 | 39.8 | 21.4 | 6.9 | 2.06 | 681* |
| 1943 | 31.1 | 39.7 | 21.6 | 7.7 | 2.09 | † |
| 1944 | 30.5 | 39.5 | 21.8 | 8.5 | 2.12 | † |
| 1945 | 29.8 | 39.2 | 22.0 | 9.3 | 2.15 | † |
| 1946 | 28.9 | 38.7 | 22.2 | 10.2 | 2.19 | 855* |
| 1947 | 27.4 | 37.5 | 23.4 | 11.7 | 2.23 | 552 |
| 1948 | 25.5 | 40.5 | 22.9 | 11.1 | 2.27 | 494 |
| 1949 | 20.0 | 40.8 | 23.7 | 15.4 | 2.40 | 485 |
| 1950 | 24.2 | 36.2 | 25.3 | 14.3 | 2.35 | 467 |
| 1951 | 26.3 | 41.4 | 20.1 | 12.2 | 2.21 | 582 |
| 1952 | 28.0 | 39.4 | 21.1 | 11.5 | 2.20 | 497 |
| 1953 | 21.8 | 41.7 | 24.7 | 11.7 | 2.32 | 613 |
| 1954 | 23.8 | 39.5 | 26.1 | 10.6 | 2.28 | 564 |
| 1955 | 25.3 | 41.3 | 22.6 | 10.7 | 2.22 | 438 |
| 1956 | 20.7 | 42.5 | 21.8 | 14.9 | 2.36 | 522 |
| 1957 | 22.5 | 39.4 | 24.1 | 14.0 | 2.37 | 444 |
| 1958 | 24.0 | 39.8 | 22.9 | 13.3 | 2.30 | 437 |
| 1959 | 19.1 | 43.6 | 24.0 | 13.3 | 2.35 | 488 |
| 1960 | 22.1 | 42.1 | 24.4 | 11.4 | 2.30 | 503 |
| 1961 | 17.8 | 36.6 | 34.0 | 11.6 | 2.44 | 853 |
| 1962 | 23.3 | 43.9 | 21.8 | 11.0 | 2.24 | 570 |
| 1963 | 22.7 | 40.3 | 27.4 | 9.6 | 2.26 | 668 |

* For these years total counts were made. † During 1943, 1944, and 1945 no Federation meetings were held. Figures for these years were estimated from the graphs.

Table 2. Tests for trends in percentages of biomedical papers with different numbers of authors.

| Authors | Least squares test | | | | Successive differences test, 1947–63: $D^2/S^2$ |
|---|---|---|---|---|---|
| | 1934–46 | | 1947–63 | | |
| | $b$ | $2\sigma_b$ | $b$ | $2\sigma_b$ | |
| 1 | −0.543* | 0.250 | −0.292 | 0.244* | 1.65 |
| 2 | −0.193 | 0.294 | +0.144 | 0.212 | 2.58 |
| 3 | +0.364* | 0.138 | +0.238 | 0.294 | 2.18 |
| 4 or more | +0.391* | 0.144 | −0.088 | 0.162 | 1.17* |
| Authors per paper | +0.020* | 0.000 | +0.003 | 0.006 | 1.89 |

* A trend is indicated ($P = 0.95$).

Table 3. Comparative authorship distribution in 1963 *Chemical Abstracts* (C.A.), American Chemical Society (ACS) meeting, and Federation meeting.

| Data source | Percentage of papers with 1, 2, 3, 4, or more authors, respectively | | | | Authors per paper |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 or more | |
| C.A. (Price, *1*)* | 32.0 | 43.0 | 15.5 | 9.5 | |
| ACS meeting | 29.2 | 40.9 | 18.8 | 11.1 | 2.15 |
| Federation meeting | 22.7 | 40.3 | 27.4 | 9.6 | 2.26 |

* Extrapolated.

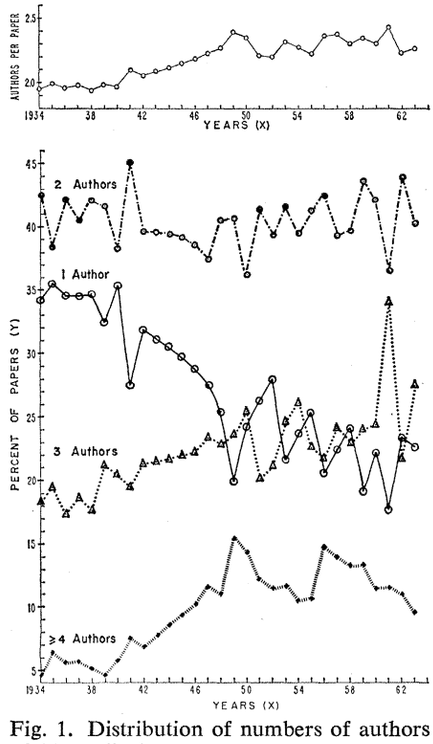


Fig. 1. Distribution of numbers of authors of biomedical papers.

As estimated from the Price curves (*1*) extrapolated (he publishes no data), papers cited in *Chemical Abstracts* by four or more authors increased from about 2.7 percent in 1946 to about 9.5 percent in 1963; even if our biomedical curve does show a slight trend, it is minuscule compared to this. Price writes (*1*): ". . . if the trend holds . . . by 1980 . . . we shall move steadily towards an infinity of authors per paper. It is one of the most violent transitions that can be measured in recent trends of scientific manpower and literature." At present this may be true of the chemists, but if the *lack* of trend reported here continues, planners of biomedical journals can expect that the average number of authors per paper will still be about 2.3 in the year 1980.

This striking contrast between chemists and biomedical scientists is puzzling. An explanation was sought in the fact that *Chemical Abstracts* is a heterogeneous universe compared with *Federation Proceedings,* since the former cites many papers in nonchemical disciplines. To test this idea, the program for the fall 1963 meeting of the American Chemical Society (*6*), which might be expected to represent chemists speaking to chemists, was analyzed for authorship distribution (a 50-percent sample). In each authorship category the correspondence with the Price curve extrapolated to 1963 (Table 3) was good, and thus there is no significant difference in authorship distribution between the "diluted" and the "pure" chemist universes.

It is interesting, also, that when these percentages for the chemists are compared with those for the 1963 biomedical papers (Tables 1 and 3), there is, in the four-or-more-author category, no significant difference. This, however, is apparently a coincidental crossing of two curves with different slopes; for the Price study shows that the distribution among chemical papers continues to change exponentially toward multiple authorship, whereas that of biomedical papers has reached an almost steady state.

As with all growth curves, this presently exponential curve for chemical papers must at some time pass through a point of inflection and eventually become asymptotic to some line parallel to the time axis. It appears that biomedical papers reached this inflection point almost two decades ago.

It is an attractive hypothesis that the multiple authorship trend among biomedical workers during the war years was caused by heavy pressure to get the research done, with forced emphasis on the team approach. But this trend did not hold for the chemists; and I have not been able to contrive an explanation.

Another speculation is that the difference between the authorship habits of the Federation members and those of the American Chemical Society may lie in the much higher qualifications for membership in the Federation. Perhaps the more mature and seasoned scientists who make up the Federation find less need for multiple research collaboration than do the chemical writers who are, *on the average,* less well established as independent investigators.

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7. I am indebted to W. J. Youden, consultant in applied statistics, National Bureau of Standards, for advice and assistance. Price wrote me, in a personal communication: ". . . sample counts taken for physics literature in general and for mathematics show just the same behavior as chemistry . . . so that one might have a good case for saying that the peculiarity you find is a significant particular property of social structures that occur perhaps in medical schools and not in university or industrial research establishments."

18 November 1963

## Antidromic Inhibition Accompanied by Ventral Root Positivities

Abstract. *Ventral-root positivities exhibiting the time course of antidromic inhibition were recorded in the cat. Hyperpolarization of the motoneuron occurs concomitant to antidromic inhibition without damage to the motoneuron. Under optimal conditions L7 and S1 ventral-root filaments show electrotonic potentials of positive sign with the time course of antidromic inhibition. Conditions predisposing to membrane depolarization are not responsible for this hyperpolarization. Antidromic inhibition was not found in roots caudal to S1.*

It is generally held that the inhibitory postsynaptic potentials (IPSP) of cat's motoneurons are responsible for much of the inhibition seen segmentally (*1*). Recent criticisms have been based on apparent temporal discrepancies between recordings made from the ventral roots and those made from the motoneuron pool (*2*). The critics hold that penetration of the motoneuron with a microelectrode produces depolarization, driving the resting potential away from the more slowly equilibrating inhibitory equilibrium potential. They have implied that such depolarization may even result from the poor condition of the animal.

Recently Araki *et al.* (*3*) showed the difference in timing between ventral-root and motoneuron recordings can be accounted for by conduction time and asynchrony in the ventral-root volley. They also recorded ventral-root positivities with the time course of inhibition in sacral segments using high gain amplification from electrodes placed at the point of exit of the root from the spinal cord. These ventral-root positivities were associated with the two-neuron so-called "direct" inhibition, and other forms of segmental inhibition were not described.

I have recorded ventral-root posi-