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Prologue

THE INTRAFAMILIAL TRANSMISSION OF RHEUMATOID ARTHRITIS:
AN UNUSUAL STUDY

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THE SET of seven papers which follow constitutes a report which in its design complements the more common type of clinical investigation. The study sets out specifically to examine a pair of hypotheses: that rheumatoid arthritis is in part determined by a genetic mechanism and is in part determined by social effects within the family.

The study uses a carefully validated and thrice repeated interview to classify people with regard to rheumatoid arthritis. This interview technique is essential for it would be quite impossible to examine the widely distributed relatives of a national sample of arthritics.

The experimental design compares the frequency of rheumatoid arthritis in first degree relatives, who share one half of their genes with the key persons with arthritis, with the frequency in second degree relatives, who share one eighth of their genes, and with the frequency in unrelated persons, who share only a negligible percentage of their genes. The study also tackles the psychological side of rheumatoid arthritis with quantitative measuring instruments. The results of the studies support the findings in the clinical literature which are well summarized in a book not widely enough known [1].

Only those working at the boundary between medicine and social science will find what follows a fully familiar territory. Others may find much of the material is hard and slow reading. Also, measurement in social psychology has not yet reached the stage where crucial variables have been identified and adequate operationalizations agreed upon. Under such circumstances, it is desirable to take multiple bearings on a single variable and to try expecially hard to rule out the possibility that the relationships uncovered may be spurious.

The reader who persists will find:

(1) No evidence to support a genetic hypothesis, plus a discussion of the ways we may have been misled into believing that this was a familial disease.

(2) A pattern of associations in women which suggests that possibly the following sequence of events might contribute to rheumatoid disease: parental status stress (i.e. discrepancy between the several status indicators of mother and father), to mother's arbitrary treatment of daughter, to daughter's resentment as child and chronic anger as adult, to daughter's rheumatoid arthritis. The associations are not strong enough to suggest that this pattern is necessarily a major contributing factor for the disease; on the other hand, the time relationships in the above sequence are so suggestive that alternate interpretations—such as that the daughter's arthritis

affected the education and occupation status of the parents or that it caused the mother's arbitrary authority—appear rather implausible. To appreciate the exact state of the evidence, it is important to read all the details.

(3) A pattern of associations in men which, though somewhat more uncertain because of the small numbers involved, suggests a rather different sequence. In a variety of ways, it is demonstrated that the male rheumatoid is more controlled in the expression of his angry or aggressive feelings, and perhaps experiences them less frequently to start with. In addition, this male rheumatoid is less likely to have come from a status inconsistent family and is unlikely to report arbitrary treatment by his mother. These repeated differences between the male and female rheumatoids are interesting and may account for some of the discrepancies in the earlier literature.

(4) Evidence that marriages high on hostility are unduly likely to have a wife with rheumatoid arthritis and a husband with peptic ulcer. The psychosocial mechanisms involved are fully explored.

We conclude that a reasonably impressive body of evidence has been derived from diverse sources to suggest that people with rheumatoid arthritis have a good deal of conflict over their anger and its expression. There are presumably a variety of ways in which a person can come into conflict over the expression of his anger. The male and female patterns identified here may be specific to Western culture but are no more to be thought of as the only causes of this type of intrapsychic conflict than are the social and psychological factors to be thought of as the only factors contributing to the development of rheumatoid disease. As time goes on, the measurement of this conflict may improve. As it does, the associated physiologic changes may be identified. It would not surprise us if one of the associated physiologic changes turns out to increase susceptibility to rheumatoid disease. To this end we need a modern day Alexis St. Martin with a piece of synovial membrane exteriorized instead of a gastric fistula. In the meantime, it appears that we should all be reading the work of that distinguished orthopedic surgeon and former president of the American Rheumatism Association, LORING SWAIN [2, 3]. Perhaps some psychiatrist will be motivated to see if Swain's charismatic persuasion of his patients to "give their resentments to God" can be rationalized and operationalized to be useful to all of us in modifying the subsequent course of the disease.

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