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ABSTRACT

Homo sapiens is the only extant species for which there exists a significant post-reproductive period in the normal lifespan. Explanations for the evolution of this species-specific trait are possible through "non-deterministic" theories of aging positing "wear and tear" or the failure of nature to eliminate imperfection, or "deterministic" theories which explain longevity as the by-product of selection for some other features such as general constitutional "fitness" or optimal life-history strategy. All of these hypotheses, however, fail to propose a positive selective force which could have acted to favor longevity in evolving hominid populations. Consideration of the evolutionary advantages of long life suggests a model of how selection for longevity could have effected a positive feedback loop between inclusive fitness and social support networks, representing the central biological and sociocultural components of a biocultural theory. Results of preliminary testing of this theory, using data from New England family genealogies, suggest validity for this approach. (Author)

THE EVOLUTION OF HUMAN LONGEVITY: TOWARD A BIOCULTURAL THEORY

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ABSTRACT:

Homo sapiens is the only extant species for which we know there exists a significant post-reproductive period in the normal lifespan. Explanations for the evolution of this species-specific trait are briefly reviewed: "non-deterministic" theories of aging posit "wear and tear" or the failure of nature to eliminate imperfection (e.g., Haldane, Medawar, Comfort); "deterministic" theories, in contrast, explain longevity as the by-product of selection for some other features such as general constitutional "fitness" or optimal life-history strategy (e.g., Smith, Guthrie, Hamilton). All of these hypotheses, however, fail to propose a positive selective force which could have acted to favor longevity in evolving hominid populations. The theory developed here derives from a consideration of the evolutionary advantages of long life. It outlines a model of how selection for longevity could have effected a positive feedback loop between inclusive fitness and social support networks. These two concepts represent the central biological and sociocultural components of the theory. Results are presented of preliminary testing of this biocultural theory using data from New England family genealogies.

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THEORIES OF AGING

The many and diverse theories of aging can be viewed from an evolutionary perspective, within a framework which contributes heuristic value to both theory and research. This approach originates in response to the question: Has natural selection acted to determine the lifespan of members of a species? An answer in the affirmative yields "deterministic" theories of aging such as Pearl's (1928) "rate of living" concept or Guthrie's (1969) optimal life-history strategy. A negative response identifies "non-deterministic" theories of aging as exemplified by Weismann's (1882) early notion of wear and tear or any hypothesis which posits the failure of nature to eliminate imperfection (e.g., Schrodinger 1945).

The attempt to categorize theories or causes of aging is of course not new. Among others these previous schemes include: Medawar (1952) who distinguished between intrinsic and extrinsic factors; Strehler (1977) who divided age changes into determinate and ancillary or stochastic; and a recent review (Schofield and Davies 1978) in which programmed degeneration was contrasted with random damage. These dichotomies parallel each other in that they all can be reduced to a genetic vs. environmental distinction. In contrast the present scheme subsumes, under modern evolutionary theory, an implicit recognition of the fact that any complex phenotypic trait (such as longevity) is a multidetermined product of both genetic and environmental influences. A gene-environment interactionist approach thus supercedes the false nature-nurture dichotomy. By focusing on natural selection, furthermore, the deterministic vs. non-deterministic distinction explicitly brings to biological gerontology an extremely provocative and productive controversy from evolutionary biology:

I refer to the neutralist-selectionist debate.

A recent article on "The neutral theory of molecular evolution" (Kimura 1979) summarized the evidence which supports the notion that most[1] biochemical variability within a species is selectively neutral. As the mechanism of evolutionary change, proponents of this "non-Darwinian evolution" (King and Jukes 1969) posit random genetic drift of mutations or the chance substitution of one allele for another. If this genetic drift causes no change in fitness (when the effects of the replacing allele are compared to those of the allele being replaced) then the rate and nature of such substitutions will not be determined by natural selection. Thus a theory of aging which says that the deteriorative changes of senescence are due to "wear and tear" accumulating over time in different cells, tissues, organs, etc. is non-deterministic because, presumably, if one thing doesn't break down another will. In other words over evolutionary time, although some genes coding for more durable cells, tissues, organs, etc. may replace genes whose products are more vulnerable to deterioration, other mutations will merely result in substituting equally imperfect gene products which are still subject to senescent "wear and tear".

More sophisticated non-deterministic theories of aging include: Orgel's (1963) error-catastrophe model; the inactivation of enzymes proposed by Gershon and Gershon (1970); immunological (Burnet 1959, Comfort 1964) and autoimmunological (Walford 1969) processes; succession of somatic mutations (Maynard-Smith 1962); free radical (Harman 1956) and cross-linkage (Bjorksten 1974) hypotheses; and accumulation of waste products such as lipofuscin (Bourne 1973). It is safe to say, without discussing each one in detail, that these

1. Neutralists do not claim that positive natural selection has played no role in evolution — but they make a significant distinction between two different levels of evolution: "Even if Darwin's principle of natural selection prevails in determining evolution at the phenotypic level, down at the level of the internal structure of the genetic material a great deal of evolutionary change is propelled by random drift." (Kimura 1979: 126).

theories share a common theme: over time imperfections of "systems design" (cf. Strehler 1977) lead to decreased viability and increased vulnerability of the organism. The failure of natural selection to eliminate these causes of senescence reveals their status as neutral with respect to evolution.

Deterministic theories of aging reflect an opposite belief, namely, that evolution has influenced senescent processes in much the same way that it has affected developmental processes. Williams (1957) was perhaps the first theoretician to apply a neo-Darwinian understanding to the phenomena of longevity. His model of pleiotropic genes presents a trade-off between earlier-acting and later-acting effects. The trade-off point, at which time degenerative changes begin to predominate, is determined by natural selection. Refinement of this model by Hamilton (1966) concerns molding schedules of fertility and mortality (in Homo sapiens sapiens, among other species) by density-dependent adverse factors of the environment. His conclusion as to the inevitability of "senescence, or rather a tendancy to complete exhaustion by the reproductive effort, and consequent death" (ibid p. 26) is an important one. Explicitly evolutionary models such as Hamilton's, and those of Cutler and Sacher to follow, illustrate possible ways in which natural selection might act either to shorten or lengthen specific longevity within particular ecological contexts (Edney and Gill 1968). This type of theoretical approach has advantages for research which are briefly mentioned below.

Other deterministic theories of aging include: Strehler's (1977) codon restriction model; experimental evidence for the finite lifespan of normal cells in vitro (Hayflick 1965); depletion of genetic information as coded by non-repeated fractions of DNA (Medvedev 1972); limitations of ribosomal RNA (Gaubatz et al 1976); capacity of the

organism to repair damaged DNA (Hart and Setlow 1974); the extension of Maximum Lifespan Potential across species by a few simple regulatory gene changes which are "antibiosenescent" (Cutler 1978); and the evolution of "longevity assurance genes" which encode unspecified "enzymatic and physiological mechanisms" (Sacher 1978).

This brief review of some theories of aging (see Chart 1) by no means represents a complete listing of all such hypothetical models. A few of these have been discussed in more detail only to illustrate the reasoning behind the deterministic vs. non-deterministic categorization. This categorization was derived from a consideration of natural selection and longevity simply because evolutionary theory provides the prevailing paradigm (Kuhn 1970) of modern biology. However, in addition, there is great heuristic value in explicitly adopting this paradigm for gerontology.

An evolutionary perspective requires a comparative approach, one which recognizes the continuum of living forms as well as the uniqueness of species and individuals. Furthermore, within the present context, an evolutionary framework provides, at the biochemical levels of analysis, convergence from independent research programs and theoretical concerns. As evidence for and against the neutralist position accumulates, it adds data as well to the storehouse of biological gerontology. Finally, by recognizing that any particular theory of aging embodies evolutionary assumptions and implications, it becomes possible to compare types of research in order, for example, to assess areas of particular promise. I do not believe that this is the only way we ought to proceed; at least equally important is the likelihood for intervention and amelioration. But evolutionary theory does supply a universal organizing perspective and a vast amount of data at all levels of analysis, from the longevity of molecules to the lifespan of the biospher

CHART 1. SOME THEORIES OF AGING: AN EVOLUTIONARY PERSPECTIVE

Non-Deterministic Theories (Neutralist)

Wear and tear (Weismann 1882 and others)
Error-catastrophe (Orgel 1963)
Inactivation of enzymes (Gershon and Gershon 1970)
Immunological (Burnet 1959 and elsewhere)
Auto-immune (Walford 1969)
Somatic mutations (Maynard-Smith 1962 and others)
Free radicals (Harman 1956 and elsewhere)
Cross linkage (Bjorksten 1974)
Accumulation of wastes, e.g., lipofuscin (Bourne 1973 and others)

Deterministic Theories (Selectionist)

Pleiotropic genes (Williams 1957)
Evolutionary inevitability of senescence (Hamilton 1966)
Ecological "hazard factors" (Edney and Gill 1968)
Codon restriction (Strehler 1977)
Limited cell-doublings *in vitro* (Hayflick 1965 and elsewhere)
Depletion of genetic information (Medvedev 1972)
Ribosomal RNA dosage limits (Gaubatz et al 1976)
DNA repair capacity (Hart and Setlow 1974)
Antibiosenescent regulatory genes (Cutler 1978)
Longevity assurance genes (Sacher 1978)

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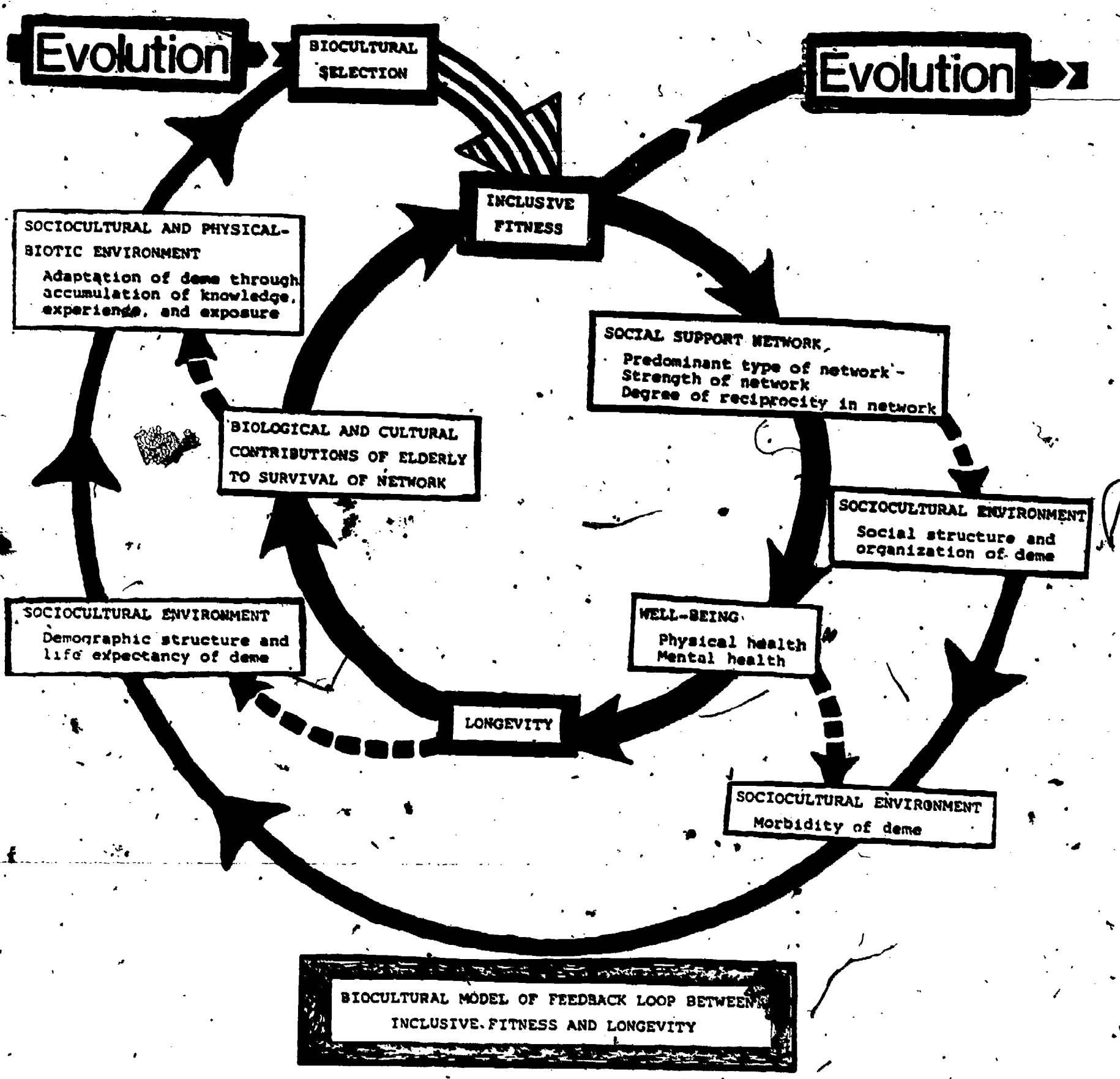

BIOCULTURAL MODEL OF FEEDBACK LOOP BETWEEN INCLUSIVE FITNESS AND LONGEVITY

EXPLANATION OF MODEL

EVOLUTION is a process which operates through selection acting on the individual; it is also a consequence of changes in gene frequencies in the population. Any evolutionary model, therefore, must account for the relationship between the level of the individual and the level of the population. The proposed bio-cultural model schematically presents relevant concepts at both levels as well as interactions between the levels. For the sake of simplicity and clarity, all possible interactions have not been indicated; rather, the model focuses specifically on the feedback relationship between inclusive fitness and longevity, between evolution and the individual.

The inner circle represents the mechanisms of the model at the level of the individual as a member of a social network. This social network includes non-kin as well as kin (consanguineal and affinal). The outer circle represents the effects of the individual and the social network on the deme and on the larger sociocultural and physical-biotic environment. The deme, or reproducing population, is comprised of any number of intersecting and interacting social networks. The sociocultural environment refers to human (hominid) behavior, sociology, ideology, and technology. The physical-biotic environment refers to all inorganic matter (e.g., stone, clay, salt), physical phenomena (e.g., weather, earthquake, water), flora and fauna with which the deme interacts. The concepts of the model can be briefly explained as follows.

BIOCULTURAL SELECTION refers to natural selection as it operates specifically on culture-bearing species (i.e., hominids). Natural selection acts on the phenotype through differential

fertility and mortality, thereby effecting changes in genotypes and, ultimately, gene frequencies. But phenotypes interact with the environment in which they live and reproduce, and thus they affect the conditions under which natural selection operates. This so-called Baldwin effect or genetic assimilation helps to explain the "rapid" evolution of the hominids by means of deviation amplifying consequences of extrasomatic adaptation, i.e., the positive feedback loop between culture and biology.

INCLUSIVE FITNESS is the representation of all of one's genetic material in the deme. Based on our increased understanding of the processes of genetic reproduction and modes of inheritance, it extends and modernizes the classic notion of evolutionary fitness as fertility. (See section titled "What is inclusive fitness").

SOCIAL SUPPORT NETWORK refers to a pattern of helping relationships which develops in response to many of the affective and instrumental needs of older persons. It comprises all of those individuals, both kin and non-kin, who contribute to the well-being of an elderly person. This conceptualization of social network emphasizes the centrality of elderly individuals as the focus of group support. It is therefore an egocentric network (with an elderly person as EGO) in which membership becomes operationally defined according to the level of participation of supportive others. The predominant type of network could be consanguineal kin, affinal kin, or individuals not at all related to EGO. In the latter two cases the genotypic and phenotypic consequences of reciprocal exchanges of support within the network must be ascertained in order for the model to be evolutionary.

The strength of the network can be defined in a number of

interrelated ways: number of individuals and their degree of relatedness to EGO, extent and type of support, geographic proximity of supporters to EGO, frequency and quality of interaction with EGO, etc. In general the value of the support to the elderly person can be determined from either an emic or an etic perspective, from subjective or objective evidence. The degree of reciprocity in a network refers at once to two different types of interactions. On the one hand it refers to the extent of mutuality in exchanges among members of the network. On the other hand it refers specifically to the ways in which an elderly person helps the network and hence is related to the biological and cultural contributions of elderly to survival of the deme. The arrangement, composition, and distribution of networks, and the interrelations among them, constitute, in part, the social organization and structure of the deme. (Also see discussion below of the contributions of elderly to survival of the deme.)

WELL-BEING of the elderly person (EGO) is a global concept which refers to the present state of the organism. It includes physical health and mental health and is the biological and psychological equivalent, at the level of the individual, to the demographic concept of morbidity. It is therefore the best predictor of the future state of the organism which, ultimately, becomes measured by longevity.

LONGEVITY of the elderly person (EGO) is measured by his or her age at death. The universal phenomenon of living past the actual (in the case of females) or virtual (in the case of males) end of the reproductive period is peculiar to human beings. The unique, longstanding, and widespread existence of significant

numbers of post-reproductive individuals qualifies the trait as species-specific. Whether the trait is of relatively recent origin or diagnostic of hominid status remains an empirical question. In either case the proposed model does not require that Plio-Pleistocene hominids lived past reproductive age in significant numbers. (Methodological questions aside, the scarcity of fossil hominid finds and the paucity of hominid data make such a determination unlikely.) Among the measurable effects of changes in longevity are the demographic structure and life expectancy of the deme.

BIOLOGICAL AND CULTURAL CONTRIBUTIONS OF ELDERLY TO SURVIVAL OF NETWORK include a wide variety of phenomena. Exposure to disease pathogens and subsequent acquisition of immunity is perhaps the most obvious and valuable example of a biological contribution to the social network. The choice and preparation of beneficial foods, and the avoidance of harmful substances, while not exclusively biological, nevertheless confer advantages of significant biological import. Knowledge of foodstuffs, like knowledge of many other scarce resources such as water holes, safe sleeping sites, and stone quarries, involves memory and experience. These capabilities, in combination with an enormous capacity for learning, are the hallmarks of advanced intelligence. Together with enhanced manual dexterity and heightened sensory-motor co-ordination, these traits characterize the biological potential of culturally-adapted creatures. They are further elaborated in human beings in extremely developed cognitive abilities including the consistent use of language.

Longer life naturally extends the possibilities of learning and increases opportunities for taking advantage of knowledge gained

through previous exposure and experience. Elderly individuals, therefore, represent a storehouse of "wisdom". They maintain continuity of traditions (e.g., tool-making, hunting strategies, child-rearing practices) and accumulate knowledge (e.g., cognitive maps, food preparation, familiarity with rare and occasional events), the benefits of which accrue to the social network. To the extent that consanguineal kin are members of the network, contributions to the survival of the network directly affect the inclusive fitness of the elderly contributor. To the extent that the network comprises affinal kin and non-kin, reciprocity determines, albeit indirectly, contributions to the inclusive fitness of the elderly contributor. Either way the value of elderly persons to the network becomes manifested in improved adaptation of the deme through the accumulation of knowledge, experience, and exposure.

The SOCIOCULTURAL ENVIRONMENT consists of human phenomena at all levels of integration, from the ideological to the technological. It represents the sum total of extrasomatic influences and elaborations of human origin within which culturally-dependent (i.e., human) organisms live. Because it is such a grand, overarching concept it must be highlighted in different aspects at different points of the model. The preceding discussions mention those aspects of the sociocultural environment which are most relevant to the particular concept being discussed. Since the deme is the evolutionary unit of analysis above the individual, aspects of the sociocultural environment which are observable in the deme are highlighted in the model. The PHYSICAL-BIOTIC ENVIRONMENT enlarges the concept of the sociocultural environment to include all known matter, phenomena and life in the biosphere. At this level of abstraction earth is the ecosystem within which all evolution occurs.

WHAT IS INCLUSIVE FITNESS?

INCLUSIVE FITNESS is the dispersion of an organism's genes in the deme. Conceptually it can be contrasted with fertility, the classic measure of evolutionary fitness. Fertility measures an organism's contribution of DNA to the gene pool (of the deme) solely through the production of offspring; inclusive fitness, in contrast, includes the reproductive behavior of all of one's blood relatives. Thus to use inclusive fitness is simply to recognize that consanguineal kin are descended from the same genetic stock and that, therefore, one can measure the likelihood that any two blood relatives have DNA identical by descent from a common ancestor.

Now since we know that each normal offspring of a diploid mating develops from the recombination of 50% of the DNA of each parent, it is possible to calculate probabilities of "shared genes" among blood relatives. In effect one traces through all of the parents that link the two individuals — each parental link dilutes the amount of "shared genes" by 50%. In this way two conceptually distinct probabilities can be calculated. As the originator of the concept inclusive fitness first discussed (Hamilton 1963), one can use knowledge of the degree of relationship between two organisms to measure the probability that they each possess a copy of the exact same gene. He later elaborated a slightly different way of understanding inclusive fitness: as "the measure of the proportion of replica genes in a relative" (Hamilton 1964:1; emphasis added). It is this second interpretation which complements current gerontological research on the value of social support networks and kinship to elderly individuals.

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1963 The evolution of altruistic behavior. The American Naturalist 97:354-356
1964 The genetical evolution of social behaviour. I. Journal of Theoretical Biology 7: 1-16

By adding up all of the proportions of replica genes among an elderly person's blood relatives, one can measure simultaneously an evolutionary and a sociocultural variable: the inclusive fitness for that person (EGO); and the size of the consanguineal kin network which is potentially capable of providing social support to that person. A conceptually appropriate unit of measurement for this summation is "'offspring equivalents', valuing each relative according to its relatedness compared to the young of the individual in question" (West Eberhard 1975:6; emphasis added). The relationship is to the young of the individual in question (and not to the individual him- or herself) in order to compare the quantity of inclusive fitness with that of classic fitness, or fertility, in which offspring is the unit of measure. The following chart (Table 8) contains calculations of degree of relatedness (cf. Wright 1922) between "the individual in question" (i.e., EGO) and a number of American kin terms. As can also be seen in the table, by multiplying each kin term by the frequency of people so related to EGO, and then summing across categories, one can calculate inclusive fitness, e.g., for the two EGOs (PETER and SANDY) in the hypothetical kinship diagram of Figure 5.

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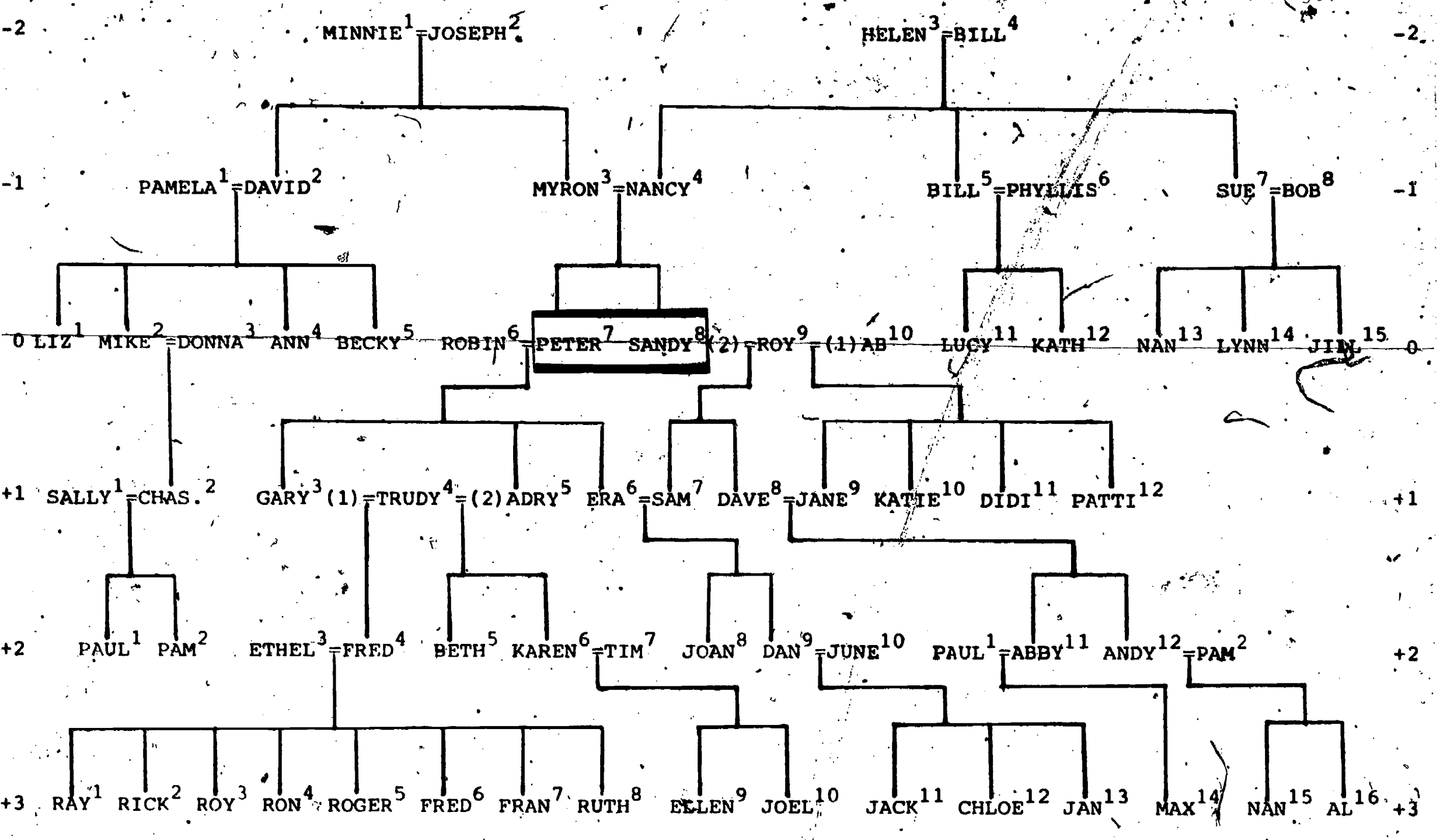
FIGURE 5. HYPOTHETICAL KINSHIP DIAGRAM



EXPLANATION OF HYPOTHETICAL KINSHIP DIAGRAM (FIGURE 5)

KEY

Diagram: DAVID[2] MYRON[3] Siblings (consanguineal kin)

HELEN[3]=BILL[4] Marriage (affinal kin).

NANCY[4] BILL[5] SUE[6] Offspring (consanguineal kin).

Numbers:

- Generations are counted relative to EGO. In Figure 5 EGO is each member of the sibship PETER[7] and SANDY[8].
- Within the same generation people are numbered consecutively by position from left to right.
- Any particular individual is designated by: generation number, position number.

Examples: Aunts of PETER (0,7) are: PAMELA (-1,1), PHYLLIS (-1,6) and SUE (-1,7).
Nephews of SANDY (0,8) are: GARY (+1,3) and ADRY (+1,5).
Great-grandchildren of NANCY (-1,4) are: FRED (+2,4), BETH (+2,5), KAREN (+2,6), JOAN (+2,8), DAN (+2,9), ABBY (+2,11) and ANDY (+2,12).

SOME RARE BUT REAL MARRIAGE ODDITIES

1. First cousin marriage, e.g., ERA (+1,6) married SAM (+1,7). Their children are simultaneously grandchildren and grand-niece/-nephew to PETER (0,7) and SANDY (0,8), thus $r_D = \frac{1}{4} + \frac{1}{8}$.
2. Half-sibling marriage, e.g., DAVE (+1,8) married JANE (+1,9). Their children are double grandchildren to ROY (0,9), thus $r_D = \frac{1}{4} + \frac{1}{4}$.
3. Siblings marrying siblings, e.g., siblings PAUL (+2,1) and PAM (+2,2) married siblings ABBY (+2,11) and ANDY (+2,12). Their children are double first cousins to each other, thus $r_D = \frac{1}{8} + \frac{1}{8}$.
4. Siblings marrying same spouse serially, e.g., TRUDY (+1,4) first married GARY (+1,3) and second married GARY's brother ADRY (+1,5). Their children are simultaneously first cousins and half-siblings, thus $r_D = \frac{1}{8} + \frac{1}{4}$.

TABLE 8. DEGREE OF RELATEDNESS, OFFSPRING EQUIVALENTS, AND INCLUSIVE FITNESS (see Fig. 5)

AMERICAN KIN TERMS	r_D DEGREE OF RELATEDNESS[a]	EXAMPLES (see Fig. 5)	OFFSPRING EQUIVALENTS[b]	PETER (0,7) Frequency	SANDY (0,8) Frequency
CHILDREN	$\frac{1}{2}$	RAY(+3,1) thru RUTH(+3,8) to ETHEL(+2,3)=FRED(+2,4)	1.0	3	2
GRANDCHILDREN	$\frac{1}{4}$	PAUL(+2,1) and PAM(+2,2) to MIKE(0,2)=DONNA(0,3)	0.5	5[c]	4[c]
GREAT-GRANDCHILDREN	$\frac{1}{8}$	ELLEN(+3,9) and JOEL(+3,10) to ROBIN(0,6)=PETER(0,7)	0.25	13[d]	6[d,e]
SIBLINGS	$\frac{1}{2}$	RAY(+3,1) thru RUTH(+3,8)	1.0	1	1
HALF-SIBLINGS	$\frac{1}{4}$	DAVE(+1,8) to JANE(+1,9) thru PATTI(+1,12)	0.5	0	0
NIECES and NEPHEWS	$\frac{1}{4}$	LIZ(0,1) thru BECKY(0,5) to MYRON(-1,3)	0.5	2	3
GRAND-NIECES and -NEPHEWS	$\frac{1}{8}$	FRED(+2,4) thru KAREN(+2,6) to SANDY(0,8)	0.25	4	3
FIRST COUSINS	$\frac{1}{8}$	LIZ(0,1) thru BECKY(0,5) and LUCY(0,11) thru JILL (0,15) to PETER(0,7) and SANDY(0,8)	0.25	9	9
		INCLUSIVE FITNESS[f]		14.0	11.0

a. Due to limitation in genealogical data, $r_D \geq \frac{1}{8}$. (Cf. Wright 1922, Coefficients of Relationship)

b. Degree of relatedness is doubled in order to make units of offspring equivalents comparable to units of fertility: children have 50% of each parent's genes ($r_D=\frac{1}{2}$) but as measures of fertility each child=1.0.

c. JOAN(+2,8) and DAN(+2,9) are also half-grand-niece and -nephew, respectively, to PETER(0,7) and SANDY(0,8); in a complete measure of inclusive fitness each would contribute an additional 0.125 ($r_D=\frac{1}{16}$) to PETER(0,7) and SANDY(0,8).

d. JACK(+3,11), CHLOE(+3,12) and JAN(+3,13) are also half-great-grand-nephew and -nieces, respectively, to PETER(0,7) and SANDY(0,8); in a complete measure of inclusive fitness each would contribute an additional 0.0625 ($r_D=\frac{1}{32}$) to PETER(0,7) and SANDY(0,8).

e. MAX(+3,14), NAN(+3,15) and AL(+3,16) are also related to SANDY(0,8) through CHAS.(+1,2), MIKE(0,2), DAVID(-1,2), MINNIE(-2,1)=JOSEPH(-2,2) and MYRON(-1,3); in a complete measure of inclusive fitness each would contribute an additional 0.03125 ($r_D=\frac{1}{64}$) to SANDY(0,8).

f. Inclusive fitness measure is abbreviated due to limitations in genealogical data

TABLE 1. FREQUENCIES OF EACH BIRTH COHORT BY GENDER AND MARITAL STATUS

ALL COHORTS	1650-1699	1700-1749	1750-1799	1800-1849	1850-1899	1900-1949
N=2156	N=25 (1.2%)	N=105 (4.9%)	N=408 (18.9%)	N=740 (34.3%)	N=830 (38.5%)	N=47 (2.2%)
F=950 (44%)*	F=10 (40%)	F=43 (41%)	F=185 (45%)	F=337 (45%)	F=348 (42%)	F=27 (57%)
M=1206 (56%)*	M=15 (60%)	M=62 (59%)	M=223 (55%)	M=403 (55%)	M=482 (58%)	M=20 (43%)
W=1855 (86%)**	W=22 (88%)	W=94 (90%)	W=351 (86%)	W=638 (86%)	W=712 (86%)	W=37 (79%)
U=301 (14%)**	U=3 (12%)	U=11 (10%)	U=57 (14%)	U=102 (14%)	U=118 (14%)	U=10 (21%)

F=FEMALES
M=MALES
W=MARRIED
U=NEVER MARRIED

* Birth cohort by gender differences are not significant (X^2=7.2452, df=6).
** Birth cohort by marital status differences are not significant (X^2=3.4625, df=6).

TABLE 2. MEAN LONGEVITY BY BIRTH COHORT AND GENDER

ALL COHORTS	1650-1699	1700-1749	1750-1799	1800-1849	1850-1899	1900-1949
$\bar{X}$=51.3	$\bar{X}$=58.6	$\bar{X}$=60.6	$\bar{X}$=54.2	$\bar{X}$=56.1	$\bar{X}$=33.9*	$\bar{X}$=9.6⊛
s=26.4	s=22.0	s=22.7	s=26.3	s=25.0	s=22.2	s=7.6
N=1239	N=19	N=84	N=319	N=571	N=234	N=12
	$\bar{x}_m$=57.7, n=14	$\bar{x}_m$=58.7, n=52	$\bar{x}_m$=57.6, n=179	$\bar{x}_m$=56.2, n=334	$\bar{x}_m$=35.6*, n=136	$\bar{x}_m$=11.0⊛, n=7
	$\bar{x}_f$=60.1, n=5	$\bar{x}_f$=63.8, n=32	$\bar{x}_f$=49.8, n=140	$\bar{x}_f$=55.9, n=237	$\bar{x}_f$=31.5*, n=98	$\bar{x}_f$=7.6⊛, n=5

*Maximum possible =74 due to publication of genealogy in 1925.
⊛Maximum possible =24 due to publication of genealogy in 1925.

TABLE 3. MEAN ADULT LONGEVITY (aged 17+) BY BIRTH COHORT AND MARITAL STATUS

ALL COHORTS	1650-1699	1700-1749	1750-1799	1800-1849	1850-1899	1900-1949
$\bar{X}$=58.6	$\bar{X}$=61.8	$\bar{X}$=64.9	$\bar{X}$=60.8	$\bar{X}$=61.2	$\bar{X}$=44.6*	$\bar{X}$=20.0⊛
s=20.5	s=17.7	s=17.0	s=20.9	s=20.1	s=16.3	s=2.0
N=1064	N=18	N=78	N=279	N=518	N=168	N=3
	$\bar{x}_u$=37.0, n=2	$\bar{x}_u$=61.5, n=4	$\bar{x}_u$=45.8, n=22	$\bar{x}_u$=54.2, n=50	$\bar{x}_u$=46.1*, n=25	
	$\bar{x}_m$=64.9, n=16	$\bar{x}_m$=65.1, n=74	$\bar{x}_m$=62.0, n=257	$\bar{x}_m$=61.9, n=468	$\bar{x}_m$=44.4*, n=143	$\bar{x}_m$=20.0⊛, n=3

*Maximum possible =74 due to publication of genealogy in 1925.
⊛Maximum possible =24 due to publication of genealogy in 1925.

TABLE 4. MEAN FERTILITY BY BIRTH COHORT AND BY LONGER-LIVED VERSUS SHORTER-LIVED SUBJECTS.

ALL COHORTS*	1650-1699	1700-1749	1750-1799	1800-1849	1850-1899	1900-1949
$\bar{X}$=3.85	$\bar{X}$=7.17	$\bar{X}$=6.81	$\bar{X}$=5.74	$\bar{X}$=3.61	$\bar{X}$=2.43	$\bar{X}$=2.00
s=3.09	s=4.10	s=2.80	s=3.52	s=2.82	s=1.92	s=1.41
N=1039	N=18	N=68	N=195	N=376	N=378	N=4
$\bar{X}'$=4.53	$\bar{X}'$=6.67	$\bar{X}'$=6.95	$\bar{X}'$=5.84	$\bar{X}'$=3.76	$\bar{X}'$=2.33	
s'=3.36	s'=3.81	s'=2.76	s'=3.62	s'=2.96	s'=1.82	
N'=616	N'=15	N'=59	N'=168	N'=298	N'=76	
	$\bar{x}_L$=7.30	$\bar{x}_L$=7.00	$\bar{x}_L$=6.18	$\bar{x}_L$=4.25	$\bar{x}_L$=2.62	
	$\bar{x}_S$=5.40	$\bar{x}_S$=6.88	$\bar{x}_S$=5.06	$\bar{x}_S$=2.72	$\bar{x}_S$=1.86	
	Diff: n.s.	Diff: n.s.	Diff: p=.065	Diff: p .001	Diff: p=0.79	

$\bar{x}_L$ = mean for subjects longer-lived than mean for that birth cohort.
$\bar{x}_S$ = mean for subjects shorter-lived than mean for that birth cohort.
Diff = t-test for significance of difference between $\bar{x}_L$ and $\bar{x}_S$.

* Married subjects only.
' Married subjects with reliable longevities only.

FIGURE 1

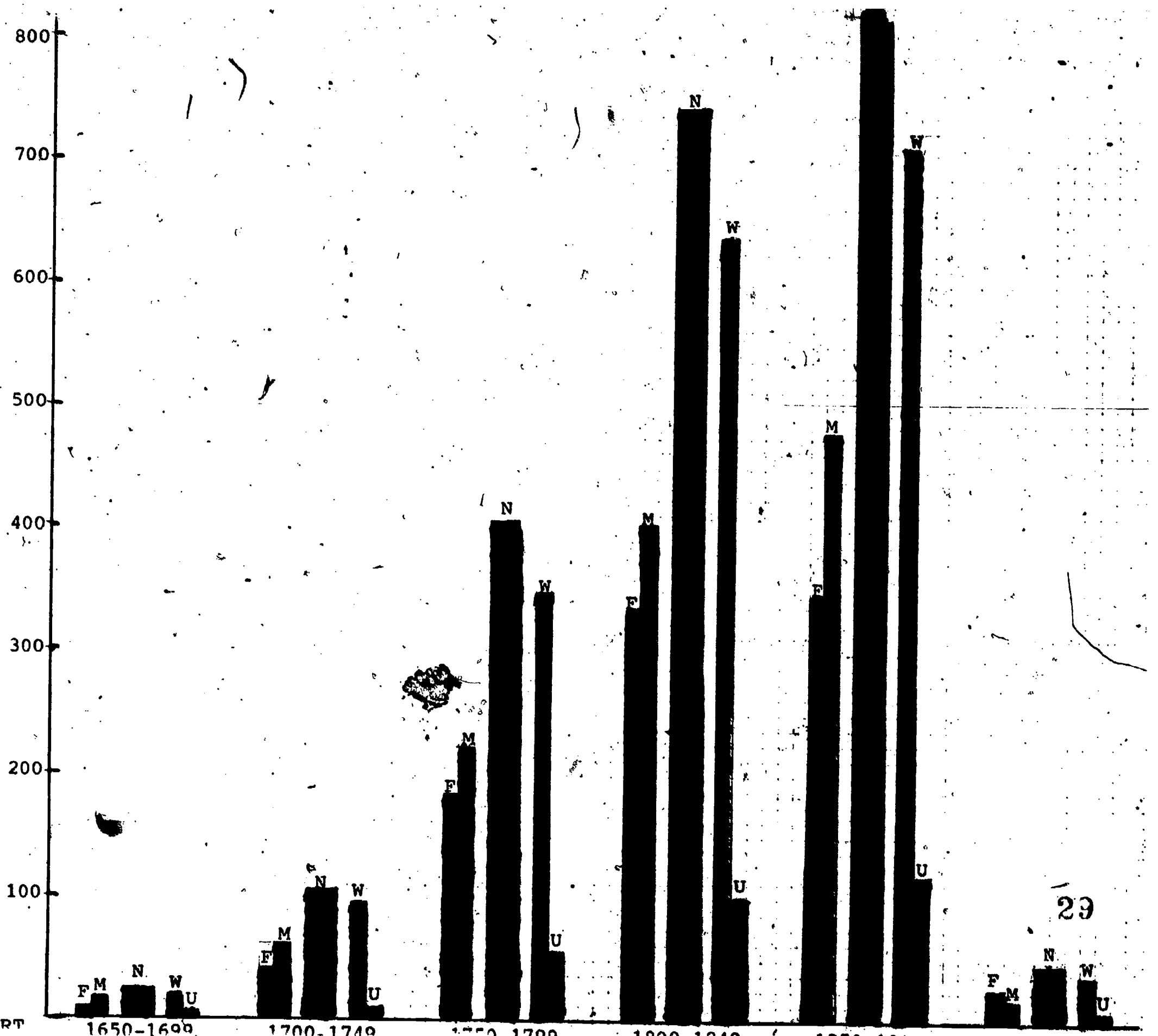

800
700
600
500
400
300
200
100
FREQUENCIES
N=BIRTH COHORT
F=FEMALES
M=MALES
W=MARRIED
U=NEVER MARRIED
1650-1699
1700-1749
1750-1799
1800-1849
1850-1
BIRTH COHORT

FIGURE 2

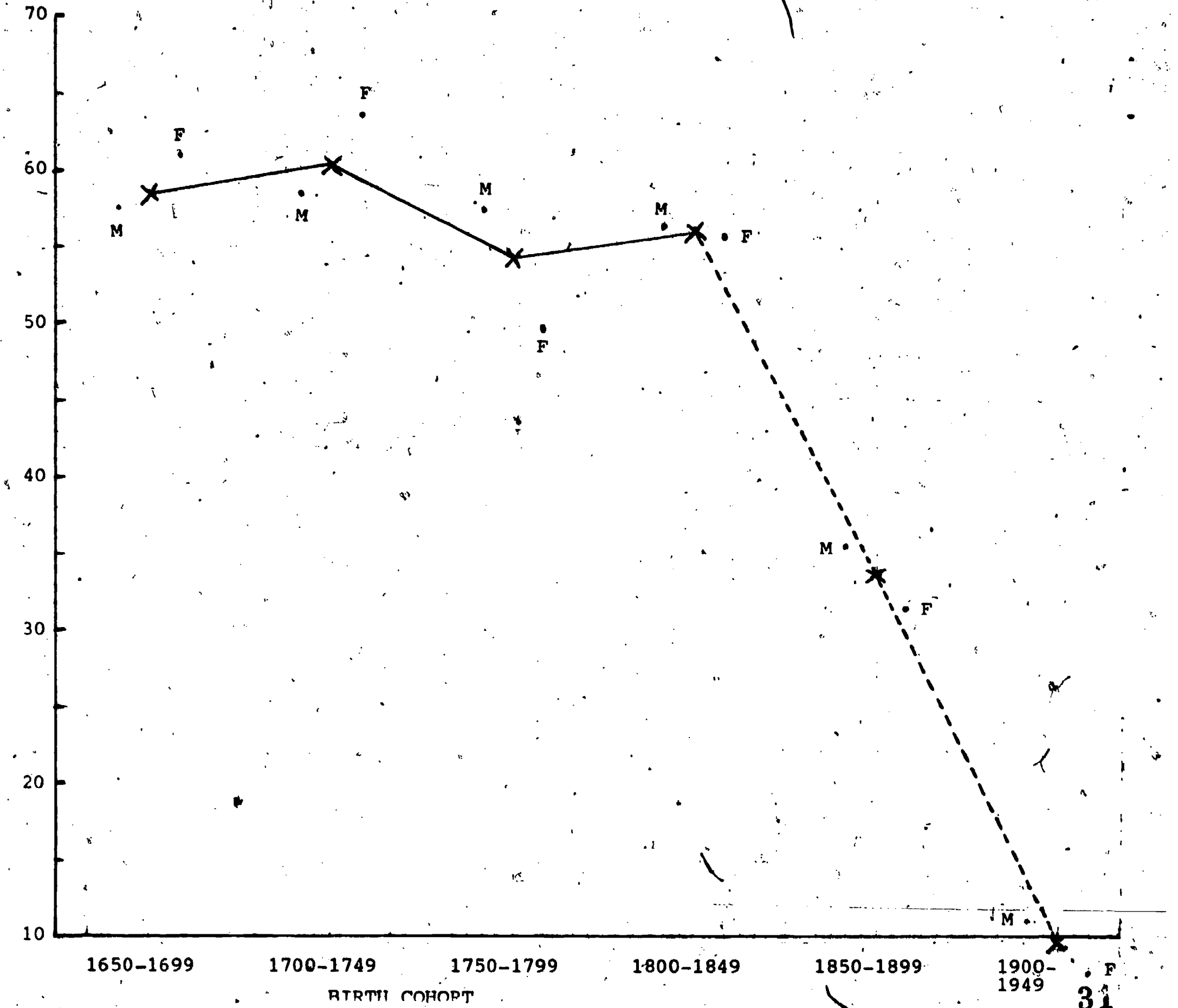

70
60
50
40
30
20
10
$\bar{X}$ LONGEVITY
M=MALE
F=FEMALE
M
F
M
F
M
F
F
M
F
M
F
1650-1699
1700-1749
1750-1799
1800-1849
1850-1899
1900-1949
BIRTH COHORT

FIGURE 3

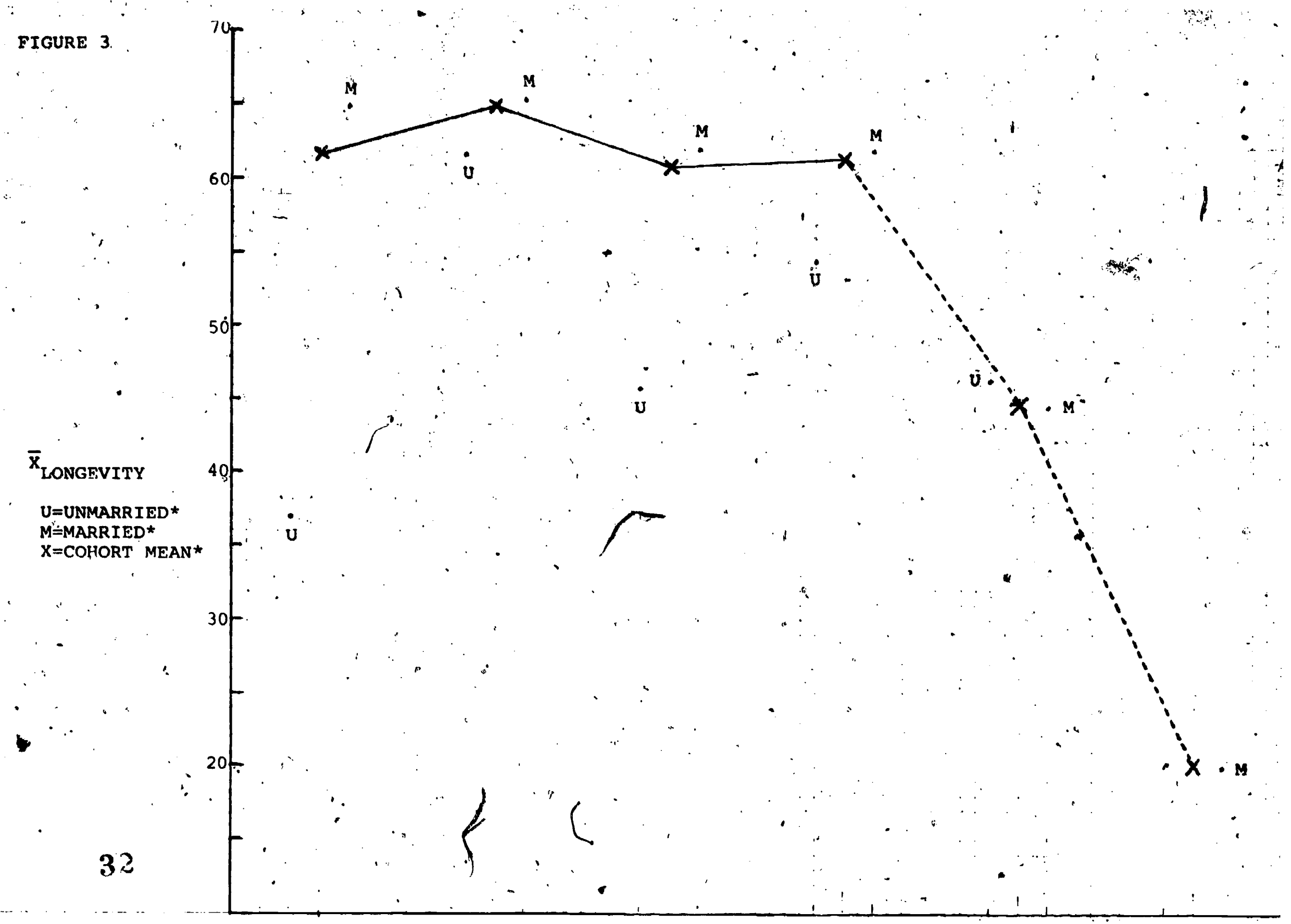
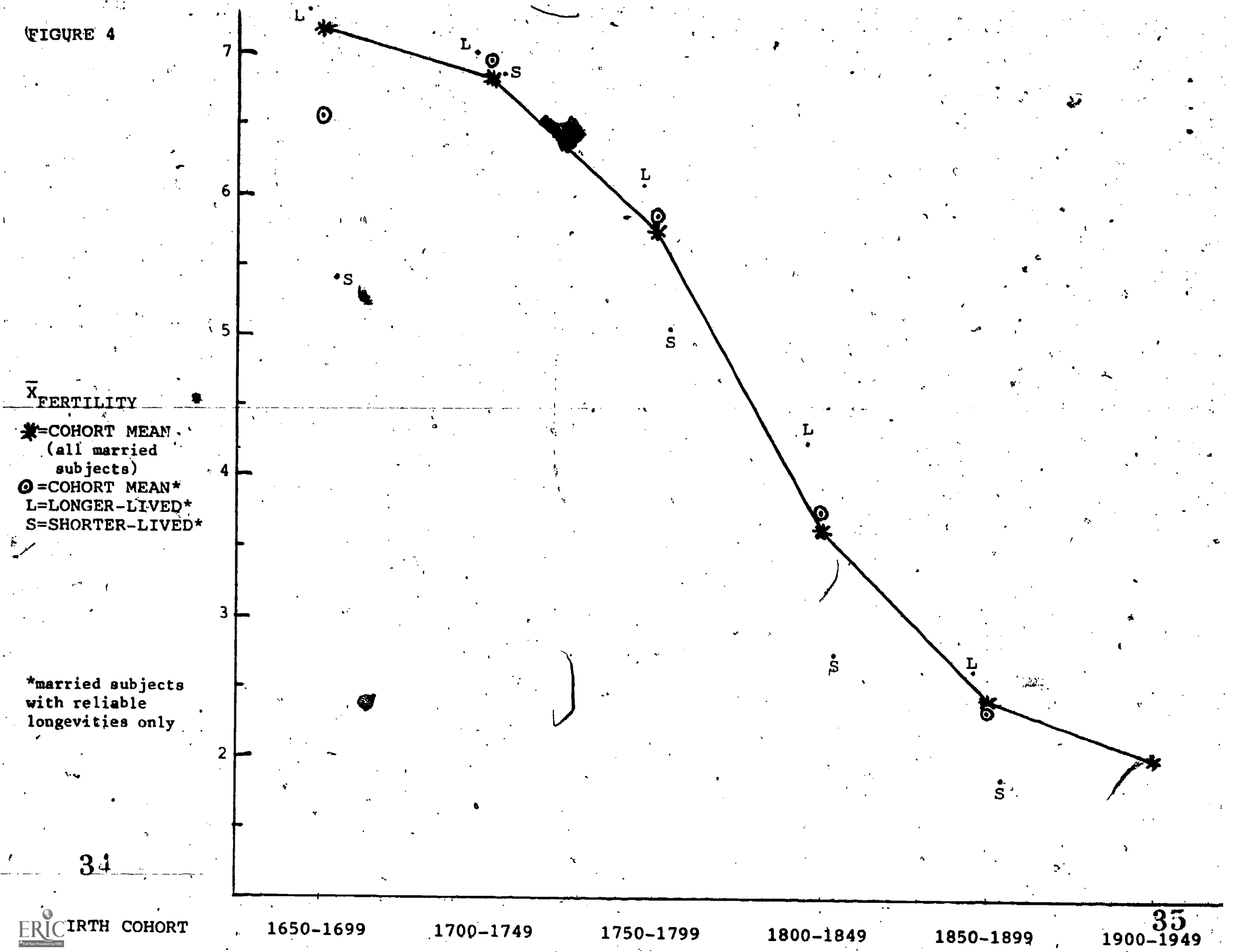

70
60
50
40
30
20
M
M
M
M
M
U
U
U
U
X
$\bar{X}_{LONGEVITY}$
U=UNMARRIED*
M=MARRIED*
X=COHORT MEAN*
*Adults only, aged 17+
1650-1699
1700-1749
1750-1799
1800-1849
1850-1899
1900-1949
BIRTH COHORT

FIGURE 4



✱=COHORT MEAN (all married subjects)
⊙=COHORT MEAN*
L=LONGER-LIVED*
S=SHORTER-LIVED*

*married subjects with reliable longevities only

BIRTH COHORT 1650-1699 1700-1749 1750-1799 1800-1849 1850-1899 1900-1949

TABLE 5. THE PARDEE FAMILY: SOME DEMOGRAPHIC DATA

VARIABLE	FREQUENCY	PERCENTAGE*
GENDER		
Female	950	44%
Male	1206	56%
(missing cases)	(2)	
MARITAL STATUS		
Never married	301	16%
Married	1613	84%
(missing cases)	(244)	
MIGRATION STATUS		
Did not migrate (place of birth= place of death)	806	75%
Did migrate (place of birth=place of death)	263	25%
(missing cases)	(1089)	
PLACES OF BIRTH (by state)		
Connecticut	766	45%
New York	390	23%
Ohio	160	9%
Michigan	93	5%
Pennsylvania	85	5%
Wisconsin	34	2%
All other states combined	187	11%
(missing cases)	(443)	

* Based on subjects with complete data for that variable; missing cases excluded.

			BIRTH COHORT											
GENERATION	N	%	1600-1649	1650-1699	1700-1749	1750-1799		1800-1849		1850-1899		1900-1949	
1[a]	1	0%	1 (100%)										
2	9	0%		9 (100%)									
3	28	1%		16 (57%)	12 (43%)								
4	90	4%			76 (84%)	14	(16%)						
5	210	10%			17 (8%)	184	(88%)	9	(4%)				
6	404	19%				207	(51%)	196	(49%)	1	(0%)		
7	525	24%				2	(0%)	404	(77%)	117	(22%)	1	(0%)
8	605	28%				1	(0%)	131	(22%)	465	(77%)	7	(1%)
9	254	12%								225	(89%)	29	(11%)
10[b]	32	2%								22	(69%)	10	(31%)
	2158		1	25 1%	105 5%	408 19%		740 34%		830 39%		47 2%	

a. First person was born in 1624.
b. Last persons were born in 1924.

TABLE 6. THE PARDEE FAMILY: SOME DATA ON LONGEVITY*

	N	MEAN	STD. DEV.	STD. ERR.	MIN.	MAX.
Subjects	1239[a]	51.3	26.3	0.75	2	100
First Spouse[b]	391	64.0	17.7	0.89	18	97
Second Spouse[b]	48	69.0	15.7	2.26	30	99
Parent[b] (not in Pardee family)	918	66.8	16.6	0.55	20	97

* Total subsample with reliable longevities.
a. 57.5% of total Pardee family.
b. Spouses and parent of subjects in this subsample.

REGION OF BIRTH⊛	N	%	MEAN
New England (Conn., Maine, Mass., N.H., R.I., Verm.)	572	58%	53.4
Middle Atlantic (Del., Md., N.J., N.Y., Pa., Wash. D.C.)	268	27%	57.6
Great Lakes and Plains (Ill., Ind., Iowa, Kan., Mich., Minn., Mo., Neb., N. Dak., Ohio, S. Dak., Wisc.)	132	13%	43.3
Southeast (Ala., Ark., Fla., Ga., Ken., La., Miss., N. Car., S. Car., Tenn., Va., W. Va.)	7	1%	42.7
West and Southwest (Az., Cal., Colo., Id., Mont., Nev., N. Mex., Ok., Ore., Tx., Ut., Wash., Wyo.)	7	1%	19.7
	986		52.9

⊛ Subjects with reliable longevities and complete data; 46% of total Pardee family.

TABLE 7. THE PARDEE FAMILY: SOME CORRELATIONAL DATA

Bivariate Correlations of Independent Variables with Longevity[a]

	N	r
Fertility	617	.30***
First Spouse's Longevity[a]	391	.14*
Second Spouse's Longevity[a]	48	.06[ns]
Parent's Longevity[a] (not in Pardee family)	916	.13***[b]
Birth Order	1218	.02[ns,c]
Year of Birth	1240	-.26***
Year of Death	1240	.33***

a. Reliable longevities only.
b. Correlation of this parent with longevity of first spouse =.06 (ns).
c. Correlation is the same for non-parametric statistic.

*p<.01 ***p<.001

Partial Correlations[a]

Zero order partials with longevity[b]	r	MEAN	STD. DEV.
Fertility	.22***	5.2	3.3
First Spouse's Longevity	.10*	63.4	17.9
Parent's Longevity (not in Pardee family)	-.02[ns]	67.8	16.2
Birth Order	-.07[ns]	4.2	2.7

Higher order partials of fertility with longevity

First order, controlling for effects of:	
First Spouse's Longevity	.22***
Parent's Longevity (not in Pardee family)	.22***
Birth Order	.22***
Second order, controlling for effects of:	
First Spouse's Longevity and Parent's Longevity (not in Pardee family)	.22***
First Spouse's Longevity and Birth Order	.22***
Birth Order and Parent's Longevity (not in Pardee family)	.22***
Third order, controlling for effects of:	
First Spouse's Longevity, Birth Order, and Parent's Longevity (not in Pardee family)	.22***

a. N=290 for all analyses.
b. For longevity $\bar{x}$=67.1, s=15.3. (Reliable longevities only)

*p<.05 ***p<.001