



SECTION 5

HOW RELIGION INFLUENCES MORBIDITY AND HEALTH:
REFLECTIONS ON NATURAL HISTORY, SALUTOGENESIS
AND HOST RESISTANCE*

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Abstract—This paper surveys the field that has come to be known as the epidemiology of religion. Epidemiologic study of the impact of religious involvement, broadly defined, has become increasingly popular in recent years, although the existence, meaning and implications of an apparently salutary religious effect on health have not yet been interpreted in an epidemiologic context. This paper attempts to remedy this situation by putting the “epidemiology” into the epidemiology of religion through discussion of existing empirical findings in terms of several substantive epidemiologic concepts. After first providing an overview of key research findings and prior reviews of this field, the summary finding of a protective religious effect on morbidity is examined in terms of three important epidemiologic concepts: the natural history of disease, salutogenesis and host resistance. In addition to describing a theoretical basis for interpreting a religion–health association, this paper provides an enumeration of common misinterpretations of epidemiologic findings for religious involvement, as well as an outline of hypothesized pathways, mediating factors, and salutogenic mechanisms for respective religious dimensions. It is hoped that these reflections will serve both to elevate the status of religion as a construct worthy of social–epidemiologic research and to reinvigorate the field of social epidemiology. Copyright © 1996 Elsevier Science Ltd.

Key words—religion, epidemiology, natural history of disease, salutogenesis, host resistance

This paper provides a look back on the development of a body of scientific research that has come to be known as the “epidemiology of religion” [1] from the vantage point of more than a dozen years of involvement in this field. The study of the epidemiologic impact of religious involvement, broadly defined, has become an increasingly popular area of research in recent years, although the existence, meaning and implications of a possible effect of religion on health status have not yet been satisfactorily placed in epidemiologic context. That is, while sociologists, psychologists, gerontologists, physicians and social epidemiologists, among others, have by now provided ample empirical evidence of a salutary religious effect on morbidity and mortality, there has been little in the way of interpretation of these findings expressed in terms of substantive epidemiologic concepts.

The present paper seeks to remedy this situation through, in simple terms, putting the “epidemiology” into the epidemiology of religion. Specifically, after providing an overview of key research findings and prior reviews of this field, principally those of the

present author and his colleagues, the findings of a salutary effect of religious involvement will be examined in light of at least three important epidemiologic concepts: the natural history of disease, salutogenesis and host resistance. While these concepts are familiar to many epidemiologists in general, they are not widely considered by social epidemiologists, despite classic expositions by Antonovsky [2] and Cassel [3]. These concepts are essential, however, for meaningfully interpreting an epidemiologically protective effect of religious involvement. Centerpieces of this discussion will be: an enumeration of what existing findings both imply and do not imply (as their radical misinterpretation is a continuing problem), and an outline of possible salutogenic mechanisms by which respective dimensions of religious involvement may protect against morbidity and influence health. It is hoped that this discussion will serve as a springboard both for elevating religiosity to a more mainstream status as a social–epidemiologic construct and for rejuvenating social epidemiology as a field.

OVERVIEW OF PRIOR RESEARCH

In 1987, two literature reviews were published almost simultaneously that for the first time provided summary overviews of a vast body of previously

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unreviewed empirical studies on the health effects of religious involvement. In one paper [4], we provided a comprehensive review of over 200 published studies, dating back to the 19th century, in which measures of religious involvement, broadly defined, had been examined in association with a very wide range of epidemiologic outcomes, in terms of both morbidity and mortality. These included studies of cardiovascular disease, hypertension and stroke, cancer (overall and of numerous sites, especially the uterus and cervix), gastrointestinal disease, overall health status and symptomatology, and overall and cause-specific mortality [4]. Studies tended to be of two types: those in which rates of morbidity or mortality were compared across categories of religious affiliation (i.e. religions or religious denominations), and those in which measures of religious involvement (e.g. frequency of religious attendance) were associated with indices of health status or morbidity. In the other paper [5], many of the former type of studies were reviewed, separately by categories of religious affiliation, and an excellent discussion offered a context for interpreting these findings. In general, results pointed to a mostly salutary or protective epidemiologic effect of religiosity, regardless of the religious measure used or the outcome under study, and this relationship manifested in study populations regardless of age, sex, race, ethnicity, nationality, study design or the period of time during which the study was conducted [6]. The apparent consistency of this relationship led us to propose that research on the epidemiology of religion would seem to represent a vital and worthy intellectual challenge for social epidemiologists [1].

Building on this work, we published a series of essays in which we sought to lend some focus and direction to this field. In 1987 [1], we reviewed and critiqued those studies in which the principal independent variable was a measure of religious attendance. A total of 27 epidemiologic studies were found, in 22 of which more frequent religious attendance was associated with less of the particular type of morbidity or mortality under study. The review included both a brief primer on religion for epidemiologists and a serious methodological critique of this body of literature, which included consideration of epistemological and conceptual issues, problems related to study design and research methods, limitations of data analysis, and biases and misconceptions inherent in how epidemiologists tended to measure religious attendance. It was concluded that, in light of possible methodological limitations, there was currently insufficient evidence to claim that these studies definitively "proved" a protective effect of religious attendance. The consistency of findings, however, was highly suggestive that frequent religious attendance was conducive to better health and that this topic thus warranted further investigation.

Following these efforts to review and critique this

literature, we published another paper in 1989 [7] in which, alongside reviewing the subset of this literature investigating hypertension, we proposed a dozen alternative hypotheses for a protective epidemiologic effect of religious involvement. This paper represented an attempt to answer the "why" question: why are such findings present? That is, how can we account for or explain the presence of salutary religion-health associations? Three of the proposed hypotheses were methodological in nature, implicating potential conceptual, psychometric or analytical flaws in existing studies as responsible for the presence of such findings (important considerations in reviewing the effects of any "new" epidemiologic factor). The other nine hypotheses were substantive; they sought to identify those specific functions, characteristics, expressions or manifestations of being religious or practicing religion that are known or believed to be health-related. These included the promotion of health-related behavior (i.e. studies show that the religious, on average, smoke and drink less), the engendering of social support (e.g. through religious fellowship), and the psychodynamic benefits of ritual (e.g. psychophysiological responses to positive emotions during worship or prayer), belief (e.g. the consonance of particular theological worldviews with particular salutary health beliefs), and faith (e.g. as in the fostering of learned optimism). In addition, hereditary transmission was proposed as a possible explanation for findings in which heavily intramarrying religious groups were at either excess or lower risk for particular diseases (e.g. Tay Sachs in Ashkenazi Jews). A multifactorial hypothesis was also proposed—one in which the correct explanation for a particular finding was more likely "all of the above" (e.g. as in Seventh day Adventists, who are about the healthiest religious denomination in North America, and who, on average, practice healthy behaviors, are integrated into supportive networks, have health-promoting patterns of worship, belief and faith, and tend to intramarry). Finally, for the sake of completeness, two additional hypotheses were proposed: a "super-empirical" explanation (i.e. an esoteric or paranormal force or energy, believed in by many religious traditions but not yet proven to the consensus acceptance of Western biomedical science) and a supernatural explanation (i.e. the possibility that a God who is transcendent, or "resides" at least partly outside of the natural universe, blesses the faithful in ways that are, by definition, unverifiable by science).

The next step in synthesizing this literature was a 1992 book chapter [8] written for an applied audience in which, after providing a detailed history of epidemiologic research on religion, we outlined implications of these studies' findings for the prevention of illness. Many possible public health and social work interventions were suggested, including pastoral roles in encouraging health promotion and behavioral change; congregational activities such as outreach programs, screening, and

church- or synagogue-based clinics; and, encouragement of supplemental forms of communal fellowship focusing on self-actualization, study and healing. The chapter's final passage noted emphatically that

Once secular educators, planners, and policymakers approach religious phenomena with greater objectivity and circumspection, they may discover new means and sources of power for fostering salutary health-related beliefs, attitudes, and behaviors and, eventually, for promoting and improving health (pp. 98–99).

Following these reviews with their respective bibliographic, methodological, theoretical and applied emphases, a paper was published in 1994 [6] that sought to evaluate this apparent relationship between religion and health according to the criteria by which all proposed epidemiologic effects are (ideally) evaluated. This consisted of asking three questions: (1) is there an association, (2) is it valid, and (3) is it causal? The answers were deemed to be (a) a guarded "yes", (b) "probably", and (c) "maybe". First, the presence of more than a couple hundred published studies whose findings are consistently positive, as described earlier, was taken as sufficient evidence that, yes, an association is apparently present. Second, validity was a tougher hurdle, as its constituent components—including bias and confounding—can never be absolutely ruled out through research based solely on observational epidemiologic study designs. The weight of evidence reviewed, however, argued that this association was probably valid. Third, the literature was evaluated in terms of Hill's well-known nine features of a causal epidemiologic association (strength, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment and analogy). For certain of these features, the literature seemed to support causality, for other features there was insufficient evidence, and still others did not seem to apply. In light of this incomplete yet mostly positive evaluation, but coupled with the perspectives of those epidemiologists of the falsificationist school who assert that causality can never be conclusively proven in epidemiologic research, a "maybe" seemed the most prudent answer.

The next contribution to this series of reviews and critiques of the literature on the epidemiologic effects of religion was a chapter [9] from the present author's book, *Religion in Aging and Health: Theoretical Foundations and Methodological Frontiers* [10], in which barriers to empirical research in this field were outlined. Potential and existing barriers to the conduct of epidemiologic research on the effects of religious involvement include the belief that "religion is unimportant" (i.e. the belief of some clinicians and scientists that because they are not religious, religion must not matter to the lives and well-being of patients or of the public at large), that "religion is not real" (i.e. that, although it may indeed matter and be important to people, it is entirely reducible to, for example, a delusion or personality disorder and is

thus unworthy of serious study), that "this is bad science" (i.e. that all existing studies are or must be fatally flawed—a belief principally held by clinicians and scientists who are unfamiliar with the extensive literature in this field), that "this goes against my training" (i.e. that the salience of religious involvement for health was not taught in medical or graduate school, so it therefore cannot be important), and that "this will only encourage the clergy" (i.e. that even if this association is real, it ought not be publicized or even discussed lest a wider role for pastors in health care be encouraged) [9].

The most recent contribution to this series of reviews was an article published in 1994 that noted several key research considerations which ought to be addressed in subsequent studies of the epidemiology of religion [11]. These considerations for future research include the necessity of longitudinal designs (whether prospective cohort investigations or multi-wave panel studies), the use of multidimensional measurement instruments for assessing religious involvement (such as the three-dimensional indices widely used in gerontological and geriatric research), the positing of multifactorial theoretical or etiologic models (such as those reviewed at the recent NIH Conference on Methodological Approaches to the Study of Religion, Aging, and Health [12]), the study of whole or randomly sampled populations (as opposed to small, nonprobabilistic samples of convenience, which are all too common in clinical research), and greater reliance upon more sophisticated multivariate analysis procedures (such as covariance-structure modeling, multiple logistic regression, and proportional hazards modeling) [11].

RELIGIOUS PROTECTION AND THE NATURAL HISTORY OF DISEASE

The skepticism and even derision expressed by physicians and biomedical scientists toward epidemiologic research on "the R word" [13] is misplaced but not entirely unexpected. After all, medical professionals, untrained in religious studies and perhaps unfamiliar with religious phenomena, may tend toward a reductionistic view of the definition of religion and thus misperceive the intent of this body of research as promoting something necessarily supernatural and thus outside the purview of science [1]. Naturally, this would be problematic for many Western scientists and physicians who are "still wrestling with a body-mind dualism that defies consensus" and thus for whom "any resolution of a body-mind-spirit pluralism is simply beyond consideration" (pp. 590–591). Understanding a statistically significant association between, for example, frequency of religious attendance and diastolic blood pressure in a population-based cohort, however, does not require the invocation of "divine" forces. As noted above, there are numerous biobehavioral,

psychological and sociological pathways by which religious involvement may influence health.

A more unanticipated barrier to discourse on the epidemiology of religion has been the dramatic misinterpretation of these findings by often sympathetic physicians and scientists (and especially by the media). Over the past few years, as this body of findings has become more widely known through the review essays discussed above and through the work of other notable scholars (e.g. Drs Harold G. Koenig, David B. Larson, Ellen L. Idler, Christopher G. Ellison and several others), the presence of an apparently protective effect of religious involvement for subsequent morbidity has metamorphosed into, for example: "The 'spiritual factor in health' that Dr Levin mentions is *the* critical factor in all healing"; "The evidence strongly suggests that faith in God truly is linked to a long, healthy life"; "Epidemiologist Jeffrey Levin is making headlines with old news: Prayer works"; and, "Norfolk doctor believes that God can heal". (To protect the guilty, the sources of these quotes have been omitted.) In fairness, it should be noted that, as a religious believer, the present author has an abiding faith that each of these statements is true. The obvious objection, of course, is that existing epidemiologic research on the effects of religious involvement, however, in *no* way supports or even begins to address such issues in any way, shape or form. Nor have any of the review papers discussed above even broached such possibilities. It is an impossibly long way from observational findings suggesting that infrequent public religious behavior seems to be associated with elevated risk in terms of subsequent rates of overall morbidity in healthy populations to the assertion that such findings demonstrate that prayer heals. Such gross misinterpretations, however, are understandable in light of the widespread unfamiliarity among both lay-people and medical professionals with an important public health concept: the natural history of disease.

The natural history of disease

In their classic explanation of the levels of prevention, Leavell and Clark [14] describe how disease, as manifested in human populations, is expressed over a natural history that contains various stages or periods. These include the period of prepathogenesis, in which components of the well-known "epidemiologic triangle"—agent, host and environment—interact to produce noxious stimuli in otherwise normal, healthy populations. Next is the period of pathogenesis, which covers the "course of a disorder in man from the first interaction with disease-provoking stimuli to the changes in form and function which result, or until equilibrium is reached or recovery, defect, disability, or death ensues" (p. 17). Within this period is a clinical horizon—a dividing line between presymptomatic and symptomatic disease. Further, at each of these stages, respective preventive or therapeutic strategies

are indicated—strategies that would be inappropriate or impossible at other stages. During prepathogenesis, primary prevention is indicated, encompassing either health promotion efforts for normal, healthy populations or specific protection for at-risk populations. Throughout pathogenesis, there are various critical points [15] at which respective secondary and tertiary preventive strategies are indicated (e.g. early diagnosis, normal treatment, disability limitation, rehabilitation). For example, during the asymptomatic phase of prepathogenesis, early diagnosis is possible through screening [16]. Once symptoms emerge, prompt treatment can produce a cure; if tissue changes have ensued, disability limitation is called for; when anatomic and physiologic changes have been stabilized, rehabilitation is indicated [14].

An important heuristic feature of the natural history concept is that different factors may be pathogenically or salutarily important at different stages of the natural history of disease. Because "[m]any of the current and major health problems have been shown to have a presymptomatic but detectable early development phase.... [i]ntervention at this stage may arrest or minimize the disease process" [17] (p. 99). This underlies the current search for epidemiologic "risk factors"—aspects of agent, host or environment that are associated with an increased probability (i.e. risk or odds) of a subsequent occurrence of disease and whose modification through intervention efforts may reduce the probability of disease or rate of morbidity in a population [18]. Through primary prevention targeting risk factors, morbidity can be prevented before it occurs in particular individuals. At the population-wide level, lower levels of a particular risk factor at a given point in time will, in theory, be associated with lower morbidity rates (e.g. cumulative incidence) for the subsequent occurrence of a respective disease entity or condition. The charge of epidemiologic research in this regard is to identify risk factors which emerge on average, across populations and *caeteris paribus*, or all things being equal [11]. In other words, "epidemiology cannot and does not tell us whether it was smoking that caused Uncle Fred's emphysema... but it can and does tell us that among people who smoke the incidence of emphysema is considerably elevated" (p. 13). These are basic principles familiar to any epidemiologist.

The implications of this concept are that interventions targeting the reduction of particular risk factors or the promotion of particular protective factors are inappropriate if applied at the wrong stage of the natural history of disease. Several hypothetical examples should illustrate this point. First, a primary preventive measure may not be helpful as a therapeutic or rehabilitative measure: as specific protection, seatbelt-wearing behavior is rightly encouraged among drivers, since low levels of this behavior represent a risk factor for fractured bones; once an automobile accident has occurred, however,

the buckling of one's seatbelt is not a useful secondary or tertiary preventive treatment for orthopedic morbidity. Second, a treatment measure may not be helpful as primary prevention: an antivenin injection is life-saving as a prompt treatment for snakebite as it will prevent some pathogenic changes, reverse others and promote recovery; as a health promotion intervention for normal, healthy populations, it will not likely influence the future behavior of ravenous snakes. Third, a tertiary preventive measure may not be helpful in primary or secondary prevention: range-of-motion therapy is used by occupational therapists to aid recovery of function from nerve damage to the arm; as specific protection or treatment, such therapy is unlikely to prevent the pathogenic changes resulting from a subsequent weightlifting accident or to engender the surgical repair of acutely damaged muscle tissue. Finally, a secondary preventive measure may also be useful for tertiary prevention but not be helpful for primary prevention and may even increase risk: debriding necrotic tissue is an important treatment for enabling wound healing from a brown recluse spider bite; cutting away healthy tissue in a normal, healthy person creates a wound.

The way in which the natural history concept interacts with the notion of population-level risk is also key for understanding epidemiologic findings. The relationship between a given risk factor and a respective disease is expressed as a rate of morbidity across a population and manifests irrespective of its severity or clinical impact on typical members of the population. That is, a factor that substantially increases the rate of disease occurrence in a particular population may not be responsible for much personal risk at all—it may even fail to cross the clinical horizon in most members of the population and thus be seen as negligible. In other words, as Rose [19] has noted, “a large number of people exposed to a small risk may generate many more cases than a small number exposed to a high risk” (p. 24). By limiting one's focus to individual-level manifestations of serious disease, medical professionals may miss opportunities to prevent considerable amounts of morbidity in the community; by asking the wrong questions, such morbidity may remain largely invisible [20]. As the associated risks for the developed world's most highly prevalent chronic, degenerative diseases are legion and tend to interact in complex multifactorial fashion (*à la* the well known “web of causation” [21] or the more recent “ecosocial framework” [22]), a proper understanding of the nature of risk and protection and its manifestation in populations is essential for both public health and clinical medicine.

Natural history and social epidemiology

For social epidemiology, these are especially important considerations. The study of social,

psychosocial and behavioral factors in morbidity and mortality is complicated by a lack of dynamic models which integrate these factors with biomedical variables in ways that clearly and convincingly implicate etiologic mechanisms and describe pathogenic processes to the consensus of most scientists [23]. Psychophysiological, neuroendocrine and psychoneuroimmunological processes are indeed becoming more widely understood, but explicit multifactorial models incorporating their constituent structures and functions in interaction with constructs such as Type A behavior, social support, self-esteem, learned optimism and health locus of control, for example, do not yet exist. The integration of biological, psychosomatic and “sociosomatic” processes into coherent, testable models is a central charge of social epidemiology [24] but one that has not yet been fulfilled. As Taylor [25] has noted, “Research that examines whether or not psychological and social factors are involved in health and illness has largely made its point” (p. 46). Yet comprehensive theoretical models that integrate sociological and psychology theory, biomedical science and population dynamics in detailing why and how this is so have not been forthcoming. Perhaps this is due to the ascendancy of discrete health-related behaviors as a principal focus of sociomedical research [26]; the rise in behavioral epidemiology has brought a concomitant decline in the more context-sensitive psychosocial and social (i.e. sociological) branches of social epidemiology.

This failure is disappointing but undeniable. In 1964, the late Dr John Cassel [27], social epidemiology's most important figure, called for

the joint efforts of social and health scientists...to develop a conceptual scheme which, by indicating the social and cultural processes of potential relevance to health, will provide leads as to the characteristics to be selected for study and help interpret associations that are discovered (p. 1484).

He acknowledged the centrality of the natural history concept to this task, noting that “greater attention must be given to the possibility that those sets of ‘causes’ which are responsible for the onset of conditions may be very different from those responsible for the lack of recovery from those conditions” (p. 1486), while bemoaning the fact that this idea “has not yet found general acceptance” (p. 1486). As he further noted [28], the concept of multifactorial causation is inadequate for understanding risk and protection without integrating the natural history of disease:

Even the acceptance of a multicausal theory...is not sufficient for our purposes in trying to understand the causes of any disease. We have to pose the further question: why do we want to understand causes? Are we concerned with the causes for the onset of the disease or with the causes for recovery? These factors, those causing onset and those causing recovery may be, and frequently are, different (p. 438).

The centrality of the natural history concept to the

theoretical perspective of Cassel is usually overlooked in assessing his legacy, obscured as it is by his overarching "belief in the importance of the social environment as a determinant of the distribution of health in populations" [29] (p. 2). The natural history of disease was, nonetheless, a vital component in Cassel's theories of psychosocial risk and protection, an influence carried over from the work of his mentor, the late Dr Sidney L. Kark, a founder of the field of social medicine and his predecessor as Chairman of the Department of Epidemiology at the University of North Carolina [30]. This same consideration of the interplay of natural history, disease etiology, the stages of pathogenesis, and risk and protective statuses was reflected in his program of empirical research, conducted both alone [31] and with his North Carolina colleagues [32], on the epidemiologic effects of social and cultural change, social disorganization and social-structural stress [33]. It is also visible in the perspectives of later social epidemiologists, such as Graham [34], Syme [35], Berkman [36], Kaplan [37] and especially Jenkins [38].

In his Presidential Address to the American Psychosomatic Society in 1984, Dr C. David Jenkins [38], a colleague of Cassel and a pioneer of psychosocial epidemiology, posited a comprehensive, integrated theoretical model for the field of psychosomatic medicine. Squarely in the tradition of the epidemiologic triangle, he described a multidimensional matrix by which aspects of environment (physical, biological, social and cultural-ideological) interacted with personal characteristics and processes (biological, psychological and behavioral) in complex and unique patterns to influence morbidity related to particular diseases in particular populations. His schema was meticulously outlined (e.g. personal or host factors included a category of psychological characteristics, which itself included the four categories of perceptions, emotions, cognitive functions and ego functions, each with several constituent constructs). Jenkins noted that variations of this model, while less articulated, had been implicit in social-epidemiologic research for many years. He expanded his model, however, to encompass the natural history of disease, a dimension largely ignored in previous studies. He stated:

Here is another dimension across which psychosomatic medicine could profitably expand its horizons. We clearly need much greater attention to the psychosomatic processes in managing active clinical disease, assisting in rehabilitation, and promoting full recovery of function and general sense of well-being (p. 10).

Jenkins noted that only 10% of published studies in a recent year of *Psychosomatic Medicine* bothered to consider the role of the natural history of disease in interpreting results of analyses.

What existing epidemiologic findings on religion do not tell us

Given these caveats about the importance of the natural history of disease, as well as of related concepts that characterize the complicated interplay among stage of pathogenesis, population-level risk, multiple causation and psychosocial protection, the nature and source of misconceptions about the meaning of epidemiologic findings on religion can now be explored. This body of findings has been misinterpreted in many subtle and not-so-subtle ways. Many types of misinterpretations have frequently been observed, and they result from fundamental confusion over issues such as the definition and nature of religion; the role of social, psychosocial and behavioral risk factors; the ways in which multiple causation manifests; and the differentiation of individual-level and population-level risk. More often than not, a common denominator for these misinterpretations has been a lack of awareness of the natural history of disease. Several common misinterpretations of epidemiologic findings on religion are described below; for each one, a correction is noted along with the principal source(s) of error. These misinterpretations are summarized in Table 1.

Misinterpretation 1: Religious involvement promotes healing. No, religious involvement seems to prevent morbidity. The wealth of epidemiologic data that implicate the importance of religious affiliation or religious attendance for health are by and large derived from observational studies in which the association is gauged between a particular measure of religious involvement (e.g. religious attendance) and the subsequent incidence of disease in a population. These data suggest that religious involvement is a protective factor in healthy populations and thus apparently acts in a primary preventive fashion. These studies have nothing to say about "healing", which implies a therapeutic or secondary preventive action among individuals in whom the clinical horizon has been breached and symptomatic pathogenesis has occurred. The clinical value of simply being religious has not been studied, and its therapeutic efficacy as a specific strategy for curing diseases seems implausible.

Misinterpretation 2: Religious people don't get sick. No, religious involvement seems to be associated with lower risk or odds of morbidity. This relationship, as with all epidemiologic relationships

Table 1. Common misinterpretations of epidemiologic findings on religion

Religious involvement promotes healing.
Religious people don't get sick.
Spirituality is a protective factor.
Prayer heals.
Religion is the most important factor in health.
There is empirical evidence of a supernatural influence on health.
Other factors explain away a religion-health association.

between a protective factor and an outcome, is not universal, but rather a probabilistic statement that accounts for the almost infinite diversity of ways in which host factors (e.g. human constitution) and environmental factors (i.e. the context for human lives) interrelate in given populations. The epidemiologic literature on the effects of religious involvement suggests that the incidence and/or prevalence of certain diseases or conditions in certain populations varies as a function of levels of certain measures of religious behavior or religious identification in mostly unadjusted analyses. This is still an interesting and provocative finding, given its consistency as noted earlier, but it does not mean what many people think it means. Religious involvement also appears to be a protective factor with respect to mortality, and in terms of adjusted rates as well, but this certainly does not mean that most or even any religious people do not die. Religious people most assuredly do get sick—almost all of them, in fact—and they all die.

Misinterpretation 3: spirituality is a protective factor. No, these findings suggest that religious involvement is a protective factor, not “spirituality”, whatever that may be. As usually meant by those who use the term, spirituality is taken to refer to a general religious sense that is not linked to or identified with organized religious institutions or practices. It is also frequently used by individuals who dislike the term “religion” but still wish to express religious-like sentiments. Spirituality, as commonly conceived, may well exert protective effects on morbidity and mortality, but no one has yet successfully developed an operational definition or a usable, valid index. More importantly, the epidemiologic effects of spirituality have never been studied—just those of discrete religious behaviors, attitudes, feelings and statuses. The present author once lectured on this topic to a metaphysically-oriented group and afterwards was questioned by an audience member who brusquely demanded to know why the narrow and intolerant term “religion” was used throughout instead of the more inclusive and apparently more acceptable term “spirituality”. Because religion was precisely what was being studied, it was explained—not spirituality. The hundreds of epidemiologic, psychiatric, gerontological, sociological and behavioral studies of the effects of religious involvement on health and other psychosocial outcomes are studies of decidedly *religious* indicators—precise, often standardized measures of church or synagogue attendance, adherence to religious doctrines, subjective self-ratings of religiosity, bible or scripture study, saying grace, tithing, volunteering for religious organizations, watching religious TV, going to religious schools, holding a church office, reporting a religious affiliation—not of an ineffable or undefined spirituality. There would be nothing wrong with studying the epidemiologic effects of spirituality—it just has not yet been done.

Misinterpretation 4: Prayer heals. No, religious involvement seems to protect against morbidity. While there is, in fact, a substantial and growing body of scientific evidence that seems to support the therapeutic efficacy of prayer and directed intention [39], that is an entirely separate literature and research tradition than what is being considered here. That epidemiologic studies of religious involvement even speak to the healing power of prayer is a perplexingly common misinterpretation, and is utterly wrong on several counts. First, the therapeutic effects of an intervention (such as prayer) are determined via clinical trials or intervention studies, and the epidemiologic literature on religion consists of observational studies that, by definition, are inappropriate for, and thus not used for, determinations of clinical efficacy. Second, the epidemiologic literature on religion includes but a small handful of studies in which a prayer variable was even included, and in all cases this was some type of survey item in which respondents reported their involvement with prayer as an indicator of personal religious behavior. Third, to investigate whether or not prayer heals requires not only the appropriate study design to answer such a question (the first point) and not only the actual operationalization of prayer as an intervention (the second point)—and neither of these requirements has been met even remotely in epidemiologic studies of religion—but also the study of people who are currently in a diseased state and thus in need of healing. The individuals who were included in most of the epidemiologic studies reviewed, on the other hand, were typically members of normal populations or groups who were healthy at baseline and whose static exposure classification was based on host characteristics (e.g. religious involvement) reflecting prepathogenic status. In short, these studies of the epidemiology of religion do not and cannot address the healing effects of prayer—and never intended to—as they examine neither healing nor prayer, and thus no conclusions about this issue one way or the other could possibly be inferred from this literature.

Misinterpretation 5: Religion is the most important factor in health. No, religious involvement appears to be a statistically significant factor for morbidity and mortality. As correlates, predictors or protective factors go, occasional associations and relative risks for religiosity have been quite strong, but most have been relatively moderate in magnitude. Further, as with most protective factors, the salutary effect of religious involvement sometimes attenuates (or even disappears) in stratified, adjusted or other multivariate analyses. In no way can it be stated that being religious or practicing religion is the most important means of promoting health or preventing morbidity. Not only is evidence lacking, but this proposition is implausible on its face. Is religious involvement really more important than smoking cessation, than sound nutrition, than good genes? Is it more important than

other much-studied social, psychosocial and behavioral protective factors, such as social support? More significantly, the literature on the epidemiology of religion is, technically speaking, a body of findings pertinent to disease prevention, with results expressed in such a way as to elucidate whether or not a factor exerts a protective role in preventing morbidity. These are not studies of the salience of religion for promoting health—something entirely different. Epidemiologic studies of health promotion, by classical definition [14], involve following normal, healthy populations in order to determine the proportion of a cohort that attains “wellness”—a higher-level, more optimal state than normal health. This is the inverse of the disease prevention mode in which epidemiologists typically operate—the more typical framework in which the “protective” category of an exposure variable (e.g. high religiosity) is taken to represent a preventive factor with respect to a clinically defined outcome which is usually a disease entity or condition. In health promotion research, according to classical definition, the goal is not to identify factors that prevent normal, healthy populations from declining into morbidity but to identify those factors that elevate normal, healthy populations into wellness. This clearly has not been the focus of epidemiologic studies of religious involvement.

Misinterpretation 6: There is empirical evidence of a supernatural influence on health. No, there is empirical evidence of a religious influence on health. This misinterpretation is often made by sympathetic religious partisans, and the accusation that such findings are promoted as such is often made by skeptics. That epidemiologic findings on the effects of religious involvement necessarily imply the operation of a supernatural or “divine” force is not true, that reviews of these findings encourage such a perspective is also not true, and, moreover, that any empirical findings could demonstrate such a thing is simply impossible. The supernatural, by definition, encompasses what is outside of or beyond nature, and the scientific enterprise enables systematic investigation of only naturalistic phenomena. So even if there really is a supernatural domain or the possibility of a supernatural mechanism of healing, these are not things that can be proven by empirical science [39]. As for published findings in the epidemiology of religion, notably those from the dozens of studies of religious effects on cancer morbidity and mortality [4], the various hypothesized social, psychological, biological, and especially behavioral pathways by which religious involvement influences health, described previously [7], likely can account for most of the observed associations [40, 41]. The notion that such findings automatically imply something supernatural is a common error of both partisans and skeptics due to reliance upon reductionistic definitions of religion

as an otherworldly phenomenon. “Religion” as a phenomenon that can be assessed in individual people is a domain of human life, a meta-construct encompassing institutional affiliations, roles, beliefs, values, attitudes, behaviors, experiences, personal states and traits, etc. All of these aspects of personal religious expression can be studied empirically, and positive findings can be interpreted in terms of other naturalistic human phenomena.

Misinterpretation 7: Other factors explain away a religion–health association. No, other factors may elucidate the pathways and mechanisms by which religion is related to health. It is a common misinterpretation to state that positive findings linking religion and health are unimportant because, after all, religious involvement is, by nature, just a proxy for some other construct or variable, the most often cited being social support. This contention “appears to be the central, unspoken assumption of epidemiologists working with religion variables” [42] (p. 32), and it is not supported by empirical evidence. This misinterpretation speaks to a deeper misinterpretation as to the nature of “explanation” and is elucidated by reference to the distinction made by epidemiologists between indirect and confounding associations.

A confounding association is one in which an apparent effect of an exposure on an outcome is due to at least one other factor that also exerts an effect on the outcome and is co-incident or correlated with and may cause, but is not caused by, the exposure. For example, a study might determine that the incidence of coronary artery disease is higher among owners of expensive automobiles. This presumed risk, however, would likely be spurious due to the confounding effects of other factors (e.g. sedentary lifestyle, less exercise, refined diet, obesity, job stress, Type A behavior) believed to heighten risk for coronary heart disease and also, on average, likely correlated with, but not caused by, the exposure variable. In contrast, an indirect association is one in which a particular exposure has an impact on an outcome because the exposure leads to one or more other factors which, possibly in a chain-like sequence or through synergism or in some other fashion, lead eventually to morbidity. For example, a study might determine that the rate of mortality is higher among those experiencing downward social mobility into poverty. While entering into a lower social class category does not magically and instantaneously alter physiological parameters to produce death, it may set in motion a process whose component factors (e.g. lower access to medical care, less income available for food and shelter, greater crowding and exposure to communicable disease agents, increased social stress) are believed to be associated with elevated risk for morbidity and subsequent mortality. A poverty–mortality relationship is thus not an artifact of proxy effects, as is the relationship between driving an expensive car and

developing coronary artery disease, but rather representative of an indirect association.

For the epidemiology of religion, then, the general effects of religious indicators on morbidity rates and health outcomes are no more an artifact of confounding than are the epidemiologic effects of other psychosocial factors, such as social support or bereavement. Mortality rates are known to be higher among lonely widows, for example, but it is not of much use in preventing this circumstance to state that bereavement is reducible to certain physiological and biochemical processes. As noted in a prior discussion of confounding [6],

Granting exploratory primacy to one particular level of the human system (cultural, social, psychological, organ systems, cellular, molecular, etc.) is arbitrary; human biology is itself "explained" by the activity of molecules and, ultimately, to paraphrase Democritus, everything is just atoms and empty space. Yet no one would suggest that research in subatomic physics will yield the best approach for improving the life expectancy of lonely or bereaved people (p. 1477).

In sum, other factors such as social support do not "explain away" the health effects of religious involvement, but rather elucidate the pathways and mechanisms by which being religious and practicing religion seem to benefit health.

SALUTOGENIC MECHANISMS FOR A PROTECTIVE RELIGIOUS EFFECT

The natural history concept describes the "career" of disease in individuals and human populations. It represents a conceptual map of the stages of the pathogenic process and depicts the evolution of disequilibrium disruption of health from a disease-free state to a pathogenic state to recovery, stabilization or death. Epidemiologic research thus typically involves the search for factors that increase the risk or odds of attaining a pathogenic status. The flipside of risk is protection. For a given exposure variable (e.g. religious attendance), one category may denote the presumed risk-enhancing effect (infrequent attendance) and the opposite category or categories may signify the presumed protective or preventive or salutary effect (frequent attendance). The typical way to describe the effects of a protective factor are to state that it protects against or prevents morbidity. This implies that the force of this effect is somehow to "hold back" the tide of pathogenesis. Another way to conceive of this same effect is as an active agent that reverses the course of the pathogenic process and moves an individual or population "back" to the prepathogenic stage of normal health. The natural history of disease, viewed in this way, could be characterized as a natural history of health. This would require a framework to conceptualize the pathways and mechanisms by which a protective factor enhances the likelihood or probability of

health, much as etiologic hypotheses exist for how risk factors initiate and shepherd pathogenesis.

Salutogenesis vs pathogenesis

The most powerful counterpart to the concept of pathogenesis has been the late Dr Aaron Antonovsky's [2] concept of "salutogenesis". According to Antonovsky, a preeminent medical sociologist from Israel, salutogenesis "is not just the other side of the coin from the pathogenic orientation, but, rather, is radically different" (p. 2) and based on different assumptions: (1) there is a dynamic "health ease/dis-ease continuum" along which all living people fall, not a simple dichotomy of healthy vs diseased, and thus all people are to some extent healthy; (2) epidemiologic research should focus on people's "story" and not their disease in order to identify those factors that help to maintain the health they possess or to move them in the healthy direction; (3) factors that initiate and enable salutogenesis are often not just the "opposite" categories of respective risk factors for pathogenesis, but rather entirely different factors altogether; (4) "stressors" are ubiquitous and just as likely salutary as pathological, in that they can make demands on an organism which can lead eventually to positive health consequences; (5) epidemiologic research should focus on factors that facilitate adaptation, rather than on factors that are etiologic and can be diagnosed and targeted with magic bullets; and (6) studies should examine the "deviant" cases—the smokers who do not develop lung cancer, the Type A persons who do not develop coronary heart disease, the African Americans who do not develop hypertension—rather than ignoring them to focus on pathogenic cases.

Antonovsky [2] summed up the significance of his salutogenic orientation by stating, "Thinking salutogenically not only opens the way for, but compels us to devote our energies to, the formulation and advance of a theory of coping" (p. 13). Central to his own theory is his concept of the "sense of coherence" [43]. This concept was seen to comprise three component elements, which he termed comprehensibility (the extent to which internal psychological and external environmental stimuli make cognitive sense), manageability (the extent to which available resources are adequate), and meaningfulness (the extent to which challenging events are seen as worthy of emotional investment and engagement). Antonovsky noted that the sense of coherence develops over the course of life, echoing Jenkins' [38] assertion that the salience and interaction of host and environmental factors vary across life stages and that this should be taken into account by epidemiologists. He also urged that research on salutogenesis begin by identifying "pathways" leading to coping and health.

The impact of positive psychological states in preventing and reducing morbidity has been studied [44], but their influence on enhancing or enabling salutogenesis is largely undocumented and

untheorized. Cassel [45] also called for research that would offer "a fuller explanation of the potential role of psychosocial factors" (p. 478), but realized that this would require "a second set of processes" (p. 478) different from pathogenesis. These he saw as rooted in "the strength of the social supports provided by the primary groups of most importance to the individual" (p. 478). Kaplan *et al.* [37], in their classic review of the health effects of social support, identified religious rituals, beliefs and values as salient pathways through which a salutary supportive mechanism can function, a view that Kaplan [46] had expressed previously with respect to coronary heart disease. Antonovsky saw our work on the social epidemiology of religion [1] as consonant with his views that religious commitment represented a "concrete expression of the sense of coherence" and told us he believed that findings in the epidemiology of religion would someday fit into a subsuming "theoretical model of the relationship between Weltanschauung and health" [47].

Religion and salutogenesis

In a recent editorial, Holland [48] noted that

many epidemiological papers and workers appear to neglect the need for medical-biologic knowledge.... [I]t is important to remember that epidemiologic studies are based on sound medical-biologic knowledge, not only mechanical analysis of available data. Most important advances in our subject have been made as a result of research that tested plausible biologic hypotheses (p. 616).

Therefore, in proposing how it is that religious involvement benefits health, it is important to keep in mind the need to identify pathways whose effects are mediated by specific factors that operate according to mechanisms that are known to be, have been proposed to be, or could reasonably be hypothesized to be salutogenic.

A starting point for this endeavor is our list of alternative explanations for a salutary religious effect briefly enumerated in *Social Science & Medicine* in 1989 [7]. A more elaborated matrix of religious dimensions and hypothesized associated pathways, mediating factors and salutogenic mechanisms is presented in Table 2. This presentation is meant to stimulate discussion and research; it is not intended that these suggested linkages be taken as conclusive pronouncements or "proven" facts. Both superempirical and supernatural pathways are included for the sake of completeness, as they are believed to exist by many respective scientists and religious individuals. Their inclusion is not meant to endorse their existence; indeed, the presence of the former is a point of great contention among scientists, and "proof" of the latter through scientific means is necessarily impossible. Even for many of the other hypothesized connections in Table 2, much of the necessary supportive research remains to be conducted.

In Table 2, eight dimensions of religious involvement are listed, along with the ways in which they

Table 2. Hypothesized pathways by which dimensions of religious involvement influence health

Religious dimensions	Pathways	Mediating factors	Salutogenic mechanisms
Religious commitment	Health-related behavior and lifestyles	Avoidance of smoking, drinking, drug use, poor diet, unprotected sex, etc.	Lower disease risk and enhanced well-being
Religio-ethnic identity	Hereditry	Phenotype	Hereditary transmission
Religious involvement and fellowship	Social support	Social relationships, supportive networks, and friends and family	Stress-buffering, coping and adaptation
Religious worship and prayer	Psychodynamics of ritual	Relaxation, hope, forgiveness, catharsis, empowerment, love, contentment and positive emotions	Psychoneuroimmunology, psychoneuroendocrinology and psychophysiology
Religious and theological beliefs and worldviews	Psychodynamics of belief	Salutary health beliefs, personality styles and behavioral patterns	Consonance between religious and health-related cognitions
Religious faith	Psychodynamics of faith	Optimism and positive expectation	Placebo effect
Religious, spiritual and mystical or numinous experience	Superempirical effects	Activation or invocation of healing bioenergy or a life force and experience of altered states of consciousness	Naturalistic subtle energy and nonlocal effects
Religious obedience (via faith, behaviour, worship or prayer)	Supernatural effects	Divine blessing	Supernatural intercession

might lead to health. These dimensions represent different aspects of being religious or practicing religion. They include religious commitment; religious-ethnic identity; religious involvement and fellowship; religious worship and prayer; religious and theological beliefs and worldviews; religious faith; religious, spiritual and mystical or numinous experience; and religious obedience (via faith, behavior, worship or prayer).

The hypothesized associated pathways represent those specific functions, characteristics, expressions or manifestations of respective ways of being religious or practicing religion that are either known or believed by some to be health-related. These include health-related behavior and lifestyles, heredity, social support, psychodynamics of ritual, psychodynamics of belief, psychodynamics of faith, superempirical effects and supernatural effects. For example, it is hypothesized that religious commitment (e.g. high subjective religiosity or observance) benefits health through promoting health-related behavior. It is also hypothesized that religious involvement (e.g. frequent religious attendance) benefits health through the provision of social support. Similar hypotheses could be framed for the other religious dimensions in Table 2.

The hypothesized associated mediating factors are those variables that are either known or believed by some to operationalize the respective pathways to health. These include avoidance of smoking, drinking, drug use, poor diet, unprotected sex, etc.; phenotype; social relationships, supportive networks, and friends and family; relaxation, hope, forgiveness, catharsis, empowerment, love, contentment and positive emotions; salutary health beliefs, personality styles and behavioral patterns; optimism and positive expectation; activation or invocation of healing bioenergy or a life force and experience of altered states of consciousness; and divine blessing. For example, it is hypothesized that religious commitment benefits health through promoting health-related behavior by way of encouraging and discouraging respective prescribed and proscribed behaviors related to smoking, drinking, diet, hygiene, etc. It is also hypothesized that religious involvement benefits health through the provision of social support by way of facilitating integration in supportive social networks.

Finally, the hypothesized associated salutogenic mechanisms contain the "medical-biologic knowledge" mentioned earlier [48], which may explain the effects of the mediating factors in terms that are either known or believed by some to operate in the world. These include lower disease risk and enhanced well-being; hereditary transmission; stress-buffering, coping and adaptation; psychoneuroimmunology, psychoneuroendocrinology and psychophysiology; consonance between religious and health-related cognitions; placebo effect; naturalistic subtle energy and nonlocal effects; and supernatural intercession.

For example, it is hypothesized that religious commitment benefits health through promoting health-related behavior by way of encouraging and discouraging respective prescribed and proscribed behaviors related to smoking, drinking, diet, hygiene, etc., which operate to lower disease risk and enhance well-being. In addition, it is also hypothesized that religious involvement benefits health through the provision of social support by way of facilitating integration in supportive social networks, which operate to buffer stress, enhance coping and engender adaptation.

The most immediately striking aspect of Table 2 is that most of the right half of the table represents a comprehensive inventory of the prominent theories, variables and mechanisms of the sociomedical sciences. That is, many of the hypothesized pathways, mediating factors and salutogenic mechanisms for a religious effect on health involve the same constructs and processes central to the work of social-epidemiologic researchers in various disciplines and fields, such as health psychology, medical sociology, behavioral medicine, psychosomatic medicine, health behavior and health education, social psychology and psychophysiology. The epidemiology of religion, then, may represent a valuable focus for social and behavioral research, as its further exploration will require and encourage greater integration among sociomedical fields and, ideally, may lead to the "subsuming theory" for social epidemiology hoped for by Antonovsky [47].

RELIGION, EPIDEMIOLOGY AND HOST RESISTANCE

The late Dr Reuel Stallones [49], visionary founding Dean of the School of Public Health at the University of Texas, recalled how he and Dr John Cassel had often discussed the attractiveness of developing a disease classification system for noninfectious diseases analogous to similar systems in use by epidemiologists with respect to infectious diseases (e.g. water-borne, air-borne, food-borne, tick-borne). Such categories of infectious disease "may be anathema to a microbiologist or clinician" (p. 77) because they are not etiologically meaningful, Stallones noted, but schemata based on portal of entry or exit or mode of transmission serve a useful public health purpose due to their utility for disease prevention efforts. For noninfectious diseases, he believed that "epidemiological characteristics provide a basis for grouping and differentiating diseases that is supplementary to and as valid as those derived from pathology and clinical medicine" (p. 77).

In his Wade Hampton Frost Lecture to the American Public Health Association in 1975, Cassel [3] agreed that for psychosocial exposures especially, etiologically based disease classifications (e.g. "stress-related diseases") are not likely to be helpful. With few exceptions, Cassel noted, fundamental social-epi-

demologic exposures such as social-environmental disruption and social disorganization do not exhibit etiologic specificity, but rather exert general etiologic effects on humans. Further, these noxious effects are not invariant in their pathogenic action but "idiosyncratic" (p. 111) in that their impact depends upon life situations. Cassel [2] recognized that for the social sciences, as for the biological and medical sciences, there was a pressing need "to modify the mono-etiological model to one which recognizes that factors which may be causal under certain circumstances may under other circumstances be neutral or perhaps even beneficial" (p. 1484).

In essence, Stallones and Cassel together reaffirmed the importance of the old, familiar epidemiologic triangle—the agent–host–environment paradigm—for understanding disease-making and health-making processes, and, as such, its superiority to the ICD-style system of disease classification based on etiology and pathologic processes. Stallones [49] criticized biomedical scientists and clinicians for this latter method of classification that relies so strongly on "etiologic doctrines" (p. 76) and thus fails to account for "social and environmental causes of disease distributions" (p. 76). Cassel [3] criticized epidemiologists and social scientists for their tacit conceptualization of social and environmental phenomena that might bear on morbidity as direct pathogenic agents etiologically responsible for a supposedly unique category of outcomes known as "stress diseases". In theorizing a place for psychosocial exposures in epidemiology, Cassel looked not to a role as etiologically specific agents nor to a role as invariant, agent-like environmental stimuli, but looked instead to a role as enhancers of the resistance of the third member of the epidemiologic triangle—the host.

Host resistance in social epidemiology

In reasoning out a role for psychosocial factors in social epidemiology, Cassel [3] marshaled scientific evidence from both human and animal research, such as the experimental finding that "variations in the social milieu are indeed associated with profound endocrine changes in the exposed subjects" (p. 109). In the years since, additional findings pointing both to an impact of social environment on human psychology (e.g. the theory of person–environment fit [50]) and to linkages between human psychology and elements of the nervous, endocrine and immune systems (e.g. the field of psychoneuroimmunology [51]) serve to confirm Cassel's belief that psychosocial factors play a significant role in the manifestation of health and disease by impacting the resistance of the human host, for better or for worse, depending upon circumstances. In Cassel's view, a

the endocrine balance in the body, increase the susceptibility of the organism to direct noxious stimuli, i.e., disease agents. The psychosocial processes thus can be envisaged as enhancing susceptibility to disease.... Presumably, the disease manifestations will also be determined by constitutional factors, which in turn are a function of genetic endowment and previous experience (pp. 109–110).

Other social epidemiologists and health psychologists have picked up on this theme and theorized as to how psychosocial factors influence the impact of the social environment on human physiological changes by way of altering host resistance. For example, Kiritz and Moos [52] noted succinctly "that one can distinguish different types or dimensions of social environmental stimuli; that these dimensions can have distinctive influences on physiological processes; and that the effects may differ from one individual to another" (p. 96). They further noted that "the social stimuli associated with the relationship dimensions of *support*, *cohesion*, and *affiliation* generally have positive effects—enhancing normal development and reducing recovery time from illness, for example" (p. 109). Bahnson [53] suggested that "secondary physiological effects of emotion may produce strain on selected tissues, thus predetermining the development of later somatic symptoms in these tissues" (p. 1037). Berkman [36] outlined several pathways by which life circumstances may impact on host resistance. Social and cultural circumstances may influence behavioral processes that are either beneficial (e.g. exercise) or harmful (e.g. smoking) to the host; social circumstances may engender psychological processes that then lead to emotional changes (e.g. depression) or cognitive changes (e.g. in coping or appraisal) that compromise the host; and, circumstances may possibly produce direct physiological changes (e.g. as in evidence linking loneliness to mortality [54]). Matarazzo [55] outlined what he termed "behavioral pathogens" (e.g. Type A behavior, alcohol abuse, smoking and overeating) and "behavioral immunogens" (e.g. the well-known Alameda County favorable health practices). The latter, he noted, were most prominent among certain religious denominations (e.g. Mormons and Seventh-day Adventists), whose favorable epidemiologic profiles underscore the role of behavioral immunogens as means "to better insure a healthy body" (p. 15). Finally, Henry [56] presented a very sophisticated step-sequential theoretical model progressing from social–environmental events to perceptions of these experiences and interactions to neuroendocrine responses to pathophysiological changes to chronic disease. He further noted that this whole chain of processes can be short-circuited given a proper "system of psychologic defenses and coping devices" (p. 376) that control vulnerability to pathophysiological breakdown and enhance the capacity to respond to demands. One of these host defenses, he noted, was religious beliefs.

reasonable formulation would hold that psychosocial processes acting as "conditional" stressors will, by altering

Religion as host resistance

According to the above summary, psychosocial factors may act pathogenically by increasing susceptibility to disease and thus weakening the host. Other psychosocial factors (or the same psychosocial factors in different contexts or different populations) may act salutogenically by decreasing susceptibility to disease and enhancing overall constitution and thus strengthening the host. It is significant that many of the hypothesized pathways, mediating factors and salutogenic mechanisms for an epidemiologic effect of religiosity, identified earlier, involve the same psychosocial and physiological constructs and processes thought to be central to host resistance. Religious involvement, broadly defined, may then represent a salutogenic force that acts to strengthen host constitution, thus serving to decrease general susceptibility to pathophysiological changes by heightening resistance to pathogenic agents and nonsupportive social and cultural environments. This action may also serve to "free up" psychological and physiological resources, which, rather than mobilizing to recover from the onset of pathogenesis, can be put to better use for purposes of self-actualization, leading the host in a salutogenic direction along a natural history of health to a higher-level state of wellness.

In considering the possibility that religious involvement can heighten the host resistance of human populations, it may be instructive to explore whether such religiously-based resistance to morbidity can be imparted throughout a population by the mechanism of herd immunity. This concept is most often invoked in discussions of group-wide resistance to infectious disease attack, and almost always in relation to the efficacy of immunization in populations [57]. It is not typically discussed in relation to chronic disease morbidity, and virtually never in relation to psychosocial protection. In most discussions, herd immunity "has to do with the protection of populations from infection which is brought about by the presence of immune individuals" [58] (p. 265). In other words, herd immunity implies that "the presence of immune individuals could provide indirect protection to others" (p. 266). As Fine [58] noted, "The concept has a special aura, in its implication of an extension of the protection imparted by an immunization program beyond vaccinated to unvaccinated individuals and in its apparent provision of a means to eliminate totally some infectious diseases" (p. 265), a phenomenon that might appear to be "magic" (p. 266). Even within infectious disease epidemiology, this concept is controversial; there has been considerable discussion over what it means and even over "whether it exists at all" (p. 265).

Several complex theories of herd immunity have been proposed [58]. One theory is based on the "mass action principle", whereby the number of trans-

missions (i.e. incident cases) of a disease is a function of the number of susceptibles (i.e. sufficiently vulnerable hosts) in a population. The future incidence of a disease is thus a function of the current prevalence of the disease multiplied by the number of current susceptible hosts. A second theory is based on "case reproduction rates", whereby for "an infection to persist, each infected individual must, on average, transmit that infection to at least one other individual. If this does not occur, the infection will disappear progressively from the population" (pp. 270-271). The potential of an infection to spread throughout a population is thus a function of the mechanism of transmission and the patterns of interaction among hosts. These proposed processes are not dissimilar from many discussions of host resistance and susceptibility that appeared in the social-epidemiologic literature of 20-35 years ago, and they do not implicitly contradict the views, for example, of Cassel or of many medical sociologists and social psychologists who study the influence of patterns of social interaction on health status and rates of morbidity in populations. The possibility of herd immunity as a salutogenically enhancing process for human populations also seems consonant with the view of Stallones [49] who stated, "Variations in the frequency of human disease occur in response to variations in the intensity of exposure to etiologic agents or other more remote causes, or to variations in the susceptibility of individuals to the operation of such causes" (p. 80), an axiom he affirmed was "substantially invariant...[and] not limited to any special kind of epidemiology" (p. 80).

This concept raises some very provocative and intriguing questions. Can psychosocial factors that promote salutogenesis and strengthen host resistance impart something akin to herd immunity throughout a population? Can this concept of population-wide resistance as imparted by a subset of constitutionally protected or invulnerable hosts be extended to the domain of the chronic, degenerative diseases most highly incident in the developed world? If both of these questions can be answered affirmatively, then it must also be asked whether religious involvement can impart herd immunity for chronic disease morbidity. Perhaps herd immunity represents an underlying conceptual framework for explaining the salutogenic effects of religious involvement within particular populations. Religious involvement may serve to bolster the personal competency and emotional well-being of individual hosts, as well as to strengthen the cohesiveness, functioning, security and thus "constitution" of collective bodies such as families, churches and synagogues, and other primary non-kin networks [59]. The presence of enough of these less vulnerable hosts in a community or population may impart a sort of herd immunity which would be seen in otherwise inexplicably lower rates of morbidity (e.g. as in the case of Bruhn and Wolf's [60] account of the wonderfully mysterious "Roseto story").

IMPLICATIONS FOR SOCIAL EPIDEMIOLOGY

The present paper has offered reflections on the apparent influence of religious involvement on morbidity and health status. Its principal objective, as noted earlier, has been to put the "epidemiology" into the epidemiology of religion. It has also sought to elevate the status of religion as a construct worthy of social-epidemiologic research and to propose that study of the epidemiology of religion may serve to reinvigorate the field of social epidemiology. It has been suggested that important epidemiologic concepts such as the natural history of disease, salutogenesis and host resistance are required in order to make sense of a protective effect of religion, and, further, that the study of religious effects on health can be a challenging and fruitful means of reintroducing these concepts into social epidemiology and thus fostering a renaissance of this field.

The present author is not alone in envisioning this future for social epidemiology and the epidemiology of religion. At the 1995 meeting of the Gerontological Society of America, Dr Linda K. George [61], the prominent sociologist and psychiatric epidemiologist, remarked that social scientists who study the impact of religion on health would be well advised to begin engaging real epidemiologic concepts. Health status is substantively different from and much more complex than the psychosocial outcome variables typically studied in fields such as medical sociology, and investigation of the causes, predictors or correlates of health requires some familiarity with both clinical realities and the substantive concepts and tools of epidemiologic research. She added that consideration of one such concept, "length of exposure", was critical to understanding how an exposure such as religious involvement might impact the incidence of disease. Recent empirical evidence suggests that frequent religious attendance, for example, has lagged effects on certain dimensions of mental health and contemporaneous effects on others [62]. This type of pattern of effects for a given exposure is not at all an unusual circumstance for certain domains of epidemiology, but may be a less familiar idea to many sociomedical scientists and religious researchers. The use of sophisticated cohort, case-control and ambidirectional designs for longitudinal analysis may be beyond the experience of researchers trained in cross-sectional survey research or the conduct of experiments and quasi-experiments. Likewise, sociologists and psychologists of religion may not be used to thinking of religious involvement as an "exposure" variable with constituent properties such as entry point, mode of transmission, latency period, intensity and duration of exposure, vector, communicability, and so on. To best study the epidemiologic impact of religion, however, social scientists and religious scholars who are untrained in either public health or medical fields may need to carefully reconsider how religion is

operationalized and how it is expected to influence human bodies.

A final hope is that social-epidemiologic engagement of religion will lead to consideration of other important psychosocial constructs, which, operationalized, might meaningfully be explored as potential protective factors for morbidity. This would prove to be a boon for social epidemiology, as the litany of constructs conventionally investigated is becoming rather stale. A closer examination of the role of religion in human life may serve as a radix for the discovery of new constructs that might be studied epidemiologically, whether in conjunction with religion or in and of themselves. In seeking to understand just how it is that religious involvement seems to benefit health, several contributing constructs recently have been proposed, including religiously motivated expressions such as hope [63], forgiveness [64], faith [7], altruism [65], empathy [66] and love [67]. It may be that these human expressions serve to strengthen the host resistance of individuals and communities and thus represent important and epidemiologically significant contributors to the health of populations.

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