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Religious Involvement and Mortality: A Meta-Analytic Review

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A meta-analysis of data from 42 independent samples examining the association of a measure of religious involvement and all-cause mortality is reported. Religious involvement was significantly associated with lower mortality (odds ratio = 1.29; 95% confidence interval: 1.20–1.39), indicating that people high in religious involvement were more likely to be alive at follow-up than people lower in religious involvement. Although the strength of the religious involvement–mortality association varied as a function of several moderator variables, the association of religious involvement and mortality was robust and on the order of magnitude that has come to be expected for psychosocial factors. Conclusions did not appear to be due to publication bias.

Key words: religion, mortality, survival, longevity, meta-analysis

Substantial numbers of Americans engage in religious activity. More than 90% of American adults are affiliated with a formal religious tradition (Kosmin & Lachman, 1993). Nearly 96% of Americans believe in God or a universal spirit, 42% attend a religious worship service weekly or almost weekly, 67% are members of a local religious body, and 60% feel that religion is "very important" in their lives (Gallup, 1995).

Could such religious activities and beliefs confer physical health benefits? Some research suggests that religious involvement is favorably associated with measures of physical health such as high blood pressure (Levin & Vanderpool, 1989), cancer (Jarvis & Northcott, 1987), heart disease (Friedlander, Kark, & Stein, 1986), stroke (Colantonio, Kasl, & Ostfield, 1992), and suicide (Kark, Shemi et al., 1996). Other studies suggest that religious involvement might help to buffer the impact of stress on physical and mental health (Kendler, Gardner, & Prescott, 1997; Krause & Van Tran, 1987; Pressman, Lyons, Larson, & Strain, 1990).

Hypothetically, these associations of religious involvement and health might lead to longer life. Several recent studies (Goldbourt, Yaari, & Medalie, 1993; Hummer, Rogers, Nam, & Ellison, 1999; Kark, Shemi, et al., 1996; Oxman, Freeman, & Manheimer, 1995; Strawbridge, Cohen, Shema, & Kaplan, 1997) have found that religious involvement—variously operationalized as religious attendance, membership in religious kibbutzim, finding strength and comfort from one's religious beliefs, and religious orthodoxy—is associated with lower mortality.

Potential Moderators of the Association of Religious Involvement and Mortality

However, the association of religious involvement and mortality is unlikely to be unequivocal; it is probably influenced not only by the quality of research methods used to examine the association but also by several characteristics of the research samples under study in individual investigations. For example, a century of sociological theory and research suggests that the association of religious involvement and physical health might be more closely tied to the psychosocial resources that religion provides rather than any positive psychological states engendered specifically by more private forms of religious expression (Durkheim, 1912/1995; Idler & Kasl, 1997a). For this reason, measures of public religious involvement (i.e., religious attendance) may be more strongly related to health outcomes than are measures of private religiousness (e.g., self-rated religiousness, frequency of private prayer, or use of religion as a coping resource). However, this relation is complicated by a possible confound: Healthy persons might be more likely than unhealthy persons to attend public religious

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activities. Thus, the association between religious involvement and mortality is likely to be stronger for measures of public as compared with private religiousness, and effect sizes for studies using public measures of religious involvement should be moderated also by statistical control of physical health.

Second, two studies of patients with cancer (Kune, Kune, & Watson, 1992; LoPrinzi et al., 1994) found that religious involvement was not associated with mortality, whereas many of the studies finding favorable associations of religious involvement and mortality involved basically healthy, community-dwelling adults (Goldbourt et al., 1993; Kark, Shemi, et al., 1996; Strawbridge et al., 1997). Because the health benefits of religiousness may be mediated in part by lifestyle choices and coping behaviors that have their effects over a number of years, the association of religious involvement and mortality might be stronger in basically healthy, community-dwelling samples than in samples of clinical patients.

Third, some data suggest that the association of religious involvement with mortality might be stronger in women than in men (House, Robbins, & Metzner, 1982; Strawbridge et al., 1997). If so, then studies with mostly female samples should yield more favorable associations of religious involvement and mortality than would studies with mostly male samples.

Finally, measures of religious involvement could be associated with, confounded with, or mediated by a variety of other demographic, psychosocial, and physiological variables, such as (a) age, (b) gender, (c) race–ethnicity, (d) general social support, (e) psychological well-being, (f) health practices such as exercise and smoking, and (g) physical health. To the extent that this is the case, the association of religious involvement with mortality would be more favorable in studies that controlled for fewer of these variables than in studies that controlled for large numbers of potential confounds and mediators (Idler & Kasl, 1997a, 1997b).

Although reviews of the relationship between denominational affiliation and mortality (Jarvis & Northcott, 1987; Troyer, 1988) and between religious involvement and physical health (Craigie, Liu, Larson, & Lyons, 1988; Levin & Vanderpool, 1989) have been published, no researchers to date have used meta-analytic methods to examine the association of religious involvement and all-cause mortality. To address this gap in the literature, we conducted a meta-analysis of the research on religious involvement and mortality.

Method

Literature Search

The literature search involved three steps. First, we searched six electronic databases relevant to medicine (Medline), psychology (PsycINFO), sociology (Sociofile), nursing (Cumulative Index of Nursing and Allied Health Literature [CINAHL]) and education (Education Resources Information Center [ERIC], Dissertation Abstracts) to find published and unpublished studies on religious involvement and mortality through June 1999. We crossed multiple search terms related to religious involvement (*religion, religiousness, religiosity, religious*) with multiple search terms related to mortality (*mortality, fatality, death, survival*) and leading causes of death (e.g., *cardiovascular, cancer*). Second, we examined reference sections of retrieved studies to identify additional studies. Third, we examined previous reviews of the literature and consulted with three experts in the field to identify fugitive studies. We excluded studies that used religious affiliation or denomination (e.g., Christian, Jewish) as the sole measure of religion.

Relevant Studies

We identified 41 research reports in which a measure of religious involvement was examined as a predictor of all-cause mortality. Of these reports, 5 (Berkman & Syme, 1979; Enstrom, 1975; Seeman, Kaplan, Knudsen, Cohen, & Guralnik, 1987; Strawbridge et al., 1997; Wingard, 1982) were based on the Alameda County data set, 5 (Comstock & Lundin, 1967; Comstock & Partridge, 1972; Comstock, Shah, Meyer, & Abbey, 1971; Comstock & Tonascia, 1977; Helsing & Szklo, 1981) were based on the Washington County data set, 2 (Idler & Kasl, 1991, 1992) were based on the Yale Health and Aging Project, 2 (Koenig, 1995; Koenig et al., 1998) were based on a cohort of male patients at a Veterans Administration Hospital, 2 (Bryant & Rakowski, 1992; Goldman, Korenman, & Weinstein, 1995) were based on the *National Health Interview Survey: Longitudinal Study of Aging, 70 Years and Over, 1984–1990* (Kovar, Fitti, & Chyba, 1990), and 2 (Ringdal, 1996; Ringdal, Gotestam, Kaasa, Kvinnsland, & Ringdal, 1995) were based on a cohort of cancer patients at the University Hospital of Trondheim, Norway. To satisfy the assumption of statistical independence that underlies meta-analytic research, effect size estimates for data sets yielding more than one report were based on the report that used (a) the longest observation period and (b) the largest number of cases, as is standard meta-analytic practice (e.g., Miller, Smith, Turner, Guijarro, & Hallet, 1996). Thus, 42 effect sizes were extracted from 29 (noted in reference section by an asterisk) of 41 research reports.

Computation of Effect Size Estimates

Most studies reported the association of religious involvement and all-cause mortality in relative risk, relative hazard, or odds ratio metrics. Typically, these measures of association were adjusted for one or more covariates. Despite its ease of interpretability (Davies, Crombie, & Tavakol, 1998; Laird & Mosteller, 1990), the relative risk (and by extension, the relative hazard) is not ideal for meta-analysis (Fleiss, 1994). Instead, most meta-analysis experts recommend using odds ratios as a standard measure of effect size for categorical data (Fleiss, 1994; Haddock, Rindskopf, & Shadish, 1998; Laird & Mosteller, 1990). The odds ratio for a fourfold table is the odds of a favorable outcome for a group of interest (i.e., the odds of survival at follow-up for highly religious individuals) divided by the odds for the comparison group (i.e., less religious individuals). For studies that included control variables (e.g., baseline physical health, alcohol or drug use), the odds ratios are likewise adjusted—they represent the relative odds of survival for religious and nonreligious individuals, controlling for the designated attributes. Odds ratios near 1.0 indicate weak or nonexistent associations between variables, whereas odds ratios greater than 3.0 (or less than 0.33, in the case of negative associations) represent strong associations between variables (Haddock et al., 1998).

For studies in which authors reported odds ratios, we used those as our effect size estimates. When only raw data (e.g., 2×2 cell frequencies) were available, we calculated odds ratios and variances using standard formulas (e.g., Fleiss, 1994). When study authors reported relative risks or relative hazards and measures of sampling variability (e.g., standard errors, variances, or 95% confidence intervals [CIs]), we estimated the corresponding odds ratios by reconstructing the implied fourfold tables. Odds ratios are always of slightly larger magnitude than their corresponding relative risks (Davies et al., 1998). As would be expected, our estimated odds ratios were also slightly larger (i.e., 6% larger on average) than their corresponding relative risk and relative hazard values.

Some authors (e.g., Janoff-Bulman & Marshall, 1982; Kune et al., 1992; Spiegel, Bloom, & Gottheil, 1983; Yates, Chalmer, St. James, Follansbee, & McKegney, 1981) reported effect sizes in other metrics (e.g., correlation coefficients, survival time). Details on how we derived odds ratio estimates for these effect sizes are available from Michael E. McCullough.

Log transformation. Because odds ratios are asymmetrical (negative associations can vary from 0 to 1.0, whereas positive associations can vary from 1.0 to $+\infty$), they are customarily subjected to a natural log transformation for use in meta-analyses (Fleiss, 1994; Haddock et al., 1998). Log odds ratios are distributed around zero with a theoretical range of ($-\infty$ to $+\infty$). Negative values indicate negative associations, and positive values indicate positive associations. This transformation is ideal when within-study sample sizes are large (Shadish & Haddock, 1994), as was the case for the present meta-analysis. An additional advantage of using log odds ratios for meta-analysis is that their variances are independent of the magnitude of association between the variables and are easily estimated from the cell frequencies in the fourfold table (Fleiss, 1994). We present the results of the present study in log odds ratios and odds ratios (derived by taking the antilog of the log odds ratio) to facilitate interpretation.

Multiple effect sizes in a single study. Five studies (Janoff-Bulman & Marshall, 1982; Krause, 1998; Oxman, Freeman, & Manheimer, 1995; Idler & Kasl, 1992; Yates et al., 1981) examined the association of mortality with two or more measures of religious involvement. We computed the mean effect size across all measures of religious involvement for these five studies. Several studies also reported an effect size for the association of religious involvement and all-cause mortality both (a) before adjusting for other variables and (b) after adjusting for other variables. In such studies, we used the more stringently controlled effect size. Thus, each study contributed a single effect size to the meta-analysis, with the exception of nine studies in which we were able to derive independent effect sizes for multiple subsamples (e.g., men and women), yielding a total of 42 independent effect sizes for analysis.

Moderator Coding

Along with effect sizes, we coded each study for three classes of potential moderator variables: variations in research design, variations in sample characteristics, and variations in how religious involvement was operationalized. To understand the implications of research design, we coded each study for (a) statistical controls (i.e., number and types of variables for which the religious involvement–mortality association was adjusted) and (b) length of follow-up period in months. Sample characteristics of interest were (c) percentage of males, (d) whether the sample was drawn from a community or clinical population, and (e) mean age of participants at baseline. To examine the effect of variations in measurement practices, we created a categorical variable called (f) measure type (public, private, a combination of public and private, or missing—i.e., the authors indicated that religiousness was measured, but they did not indicate how). Interjudge agreement for the coding of the above-mentioned categorical variables was evaluated with Cohen's kappa (κs $> .85$). Interjudge reliabilities for ratings of continuous variables were estimated using Shrout and Fleiss's (1979) formula for the intraclass correlation coefficient (3, 1). The mean intraclass correlation coefficient for all coded variables was .97, with intraclass correlation coefficients ranging from .78 to 1.0.

Analyses

To generalize beyond the sample of studies actually reviewed (i.e., to claim that their results reflect the likely magnitude of effects for other, future samples of studies in the research domain), meta-analytic researchers should use random-effects models to aggregate effect sizes and estimate the reliability of these aggregates (Hedges & Vevea, 1998). This strategy was clearly desirable for the present meta-analysis: Our belief that the above variables serve as moderators of the observed association between religion and mortality implies that the studies reviewed estimate different population effect sizes. Random-effects models take such between-studies variation into account, whereas fixed-effects models do not (Mosteller & Colditz, 1996).

Hierarchical linear modeling is a useful tool for conducting random-effects meta-analysis with multiple moderator variables (Bryk & Raudenbush, 1992; Haddock et al., 1998). Estimates of within-study variances are supplied by the investigator, with between-studies (random-effects) variance estimated using a program such as HLM (Bryk, Raudenbush, & Congdon, 1996). Moderator effects are then examined using regression models, with categorical variables dummy coded (Haddock et al., 1998).

The analyses presented here were conducted using the HLM software program (Bryk et al., 1996). We first determined the weighted mean effect size across all studies and then examined whether variation among effect sizes was greater than expected by chance. Second, we examined the impact of the theoretically derived moderator variables on effect size. Third, we examined whether statistical control of specific demographic, psychosocial, and medical variables influenced effect size (to explore which variables might be confounds or mediators of the association of religious involvement and mortality). Fourth, we conducted sensitivity analyses to evaluate the validity of our meta-analytic findings and their tolerance to future null results.

Results

We computed a total of 42 independent effect sizes representing 125,826 participants. Effect size estimates (odds ratios) and characteristics associated with each effect size appear in Table 1.

Omnibus Analysis

In the omnibus analysis, no moderator variables were modeled, and the observed effect sizes were presumed to constitute a representative sampling of the study populations of interest. Effect size estimates were subject to both between-studies variance (because the true effect sizes differ for different classes of studies) and within-study variance (due to sampling error). The aggregate log odds ratio for the omnibus analysis ($k = 42$, $N = 125{,}826$) was $\gamma_0 = .26$, $SE = .036$, $p < .001$. The γ_0 of .26 corresponds to an odds ratio of 1.29 (95% CI: 1.21–1.39), indicating that across all studies, highly religious individuals had odds of survival approximately 29% higher than those of less religious individuals. These effect sizes were heterogeneous. Between-studies variance was significantly greater than zero: $\tau = .0206$, $\chi^2(41) = 91.62$, $p < .001$. The corresponding Birge ratio (Haddock et al., 1998) was 2.23, suggesting that between-studies variation was 123% greater than expected due to sampling error alone. We therefore estimated other models that incorporated the moderator variables to determine the study characteristics to which between-studies variation in effect size could be attributed.

Moderator Analyses

Moderator analyses can be conducted in HLM using random-effects regression models with prediction equations of the form:

$$ES_j = \gamma_0 + \gamma_1 W_{1j} + \gamma_2 W_{2j} + \ \ldots \ + \gamma_S W_{Sj} + u_j + e_j, \quad (1)$$

where ES_j is the effect size for study j, W_{1j} to W_{Sj} are S predictor (moderator) variables, γ_1 to γ_S are regression weights associated with each of these predictors, u_j represents systematic variability in study j not captured by the S predictors, and e_j represents sampling error for study j. In this model, the intercept (γ_0) is the estimated effect size for studies with a value of zero on all moderator variables, and the remaining regression weights indicate the amount of expected variation in this effect size for a one-unit change on each moderator. We centered continuous predictors around their means and coded the two categorical moderators so

Table 1
Effect Size Estimates (Odds Ratios) and Sample and Methodological Characteristics Associated With Each Effect Size

Reference	Sampling	*N*	% male	Mean age at baseline	Length of follow-up (months)	Measures of religious involvement	Variables controlled in effect size[a]	Adjusted effect size (95% CI)
Abramson, Gofin, & Peritz (1982)	83% sample of all men, age > 60, from a Jewish neighborhood in Jerusalem	387	100	67.35	60	Missing	1, 3, 6, 8, 13	1.00 (0.60–1.66)
Baugher, Burger, Smith, & Wallston (1989/1990)	Subsample of terminally ill people from National Hospice Study	300	48.5	68.10	0.25	Rating of religion as the greatest source of strength in one's life	—	1.93 (1.21–3.06)
Comstock & Tonascia (1977)	98% sample of all adults, age > 16.5, in Washington County, MD	38,839	47.3	48	96	Frequency of church attendance	1, 2, 3, 5, 11, 16	1.38 (1.29–1.46)
Devins, Mann, Mandin, & Paul (1990)	50% random sample of patients receiving hemodialysis or renal transplant in Calgary, Alberta, Canada	97	57.7	40.3	46	Missing	1, 3, 4, 5, 8, 11, 16, 17	1.00 (0.37–2.73)
Enstrom (1989)	Representative sample of community-dwelling adults in Alameda County, CA	364	100	39.8	168	Frequency of church attendance	1, 2, 3, 11	2.20 (1.13–4.31)
Enstrom (1989)	Representative sample of community-dwelling adults in Alameda County, CA	627	0	42.5	168	Frequency of church attendance	1, 2, 3, 11	1.26 (0.77–2.05)
Goldbourt et al. (1993)	Stratified random sample of male Israeli civil servants and municipal employees	9,245	100	50.3	276	Three-item measure of religious orthodoxy	1, 3, 17	1.16 (1.06–1.26)
Goldman et al. (1995)	National probability sample of noninstitutionalized adults, age > 70	2,847	100	76.8	72	Religious service attendance in past 2 weeks	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 16	1.43 (1.23–1.66)
Goldman et al. (1995)	National probability sample of noninstitutionalized adults, age > 70	4,631	0	77.7	72	Religious service attendance in past 2 weeks	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 16	1.42 (1.25–1.62)
House et al. (1982)	10% random sample of community-dwelling adults in Tecumseh Community Health Study	1,398	0	48	126	Frequency of church attendance in past year	1, 3, 8	1.15 (0.73–1.80)
House et al. (1982)	10% random sample of community-dwelling adults in Tecumseh Community Health Study	1,301	100	48	126	Frequency of church attendance in past year	1, 3, 6, 8, 11, 17	1.02 (0.79–1.32)
Hummer et al. (1999)	Nationally representative sample in National Health Interview Survey	21,204	47.5	43.2	102	Frequency of religious attendance	1, 2, 3, 4, 5, 6, 7, 9, 10, 11, 12, 13, 16	1.20 (1.09–1.32)
Idler & Kasl (1992)	Stratified probability sample of noninstitutionalized adults, age > 65, in New Haven, CT	1,166	100	73.9	72	Two-item measure of public religiousness (α = .63); two-item measure of private religiousness (α = .71)	1, 6, 8	1.00 (0.71–1.41)
Idler & Kasl (1992)	Stratified probability sample of noninstitutionalized adults, age > 65, in New Haven, CT	1,646	0	74.9	72	Two-item measure of public religiousness (α = .63); two-item measure of private religiousness (α = .71)	1, 2, 3, 4, 5, 6, 8	1.00 (0.67–1.50)
Janoff-Bulman & Marshall (1982)	24% convenience sample of residents from a nursing home in western Massachusetts	25	32	75.4	30	Self-rated religiousness; perceived increase in religiousness since youth	1, 3, 5, 7, 8, 14, 19	0.42 (0.08–2.15)
Kark, Shemi, et al. (1996)	All adults from 11 religious and 11 secular kibbutzim in Israel	1,996	100	53.6	192	Membership in religious vs. secular kibbutz	1, 3	1.84 (1.32–2.56)

Kark, Shemi, et al. (1996)	All adults from 11 religious and 11 secular kibbutzim in Israel	1,904	0	53.2	192	Membership in religious vs. secular kibbutz	1, 3	2.79 (1.73–4.51)
Koenig et al. (1998)	Consecutive sample of male admissions to Veterans Administration Medical Center in Durham, NC	1,010	100	67.8	109.5	Three-item measure of religious coping (α = .82)	1, 2, 3, 4, 5, 6, 8, 9, 12, 14, 16	1.00 (0.77–1.30)
Koenig et al. (1999)	Probability sample of community-dwelling adults, age > 65, in North Carolina	1,387	100	74.35	75.5	Frequency of attending religious meetings or services	1, 2, 3, 5, 6, 7, 8, 9, 11, 12, 14, 16	1.31 (1.04–1.65)
Koenig et al. (1999)	Probability sample of community-dwelling adults, age > 65, in North Carolina	2,581	0	75.1	75.5	Frequency of attending religious meetings or services	1, 2, 3, 5, 6, 7, 8, 9, 11, 12, 14, 16	1.86 (1.56–2.20)
Krause (1998)	Probability sample of adults, age > 65, in Health Care Finance Administration Medicare Beneficiary Eligibility List	819	40.5	73.8	48	Three-item measure of organizational religiosity (α = .86); two-item measure of nonorganizational religiosity (α = .68); three-item measure of religious coping (α = .97)	1, 3, 5, 6, 7, 16, 17	1.00 (0.71–1.40)
Kune et al. (1992)	Consecutive sample of new cases of adenocarcinoma of the colorectum in Melbourne, Australia	695	54.1	65	62	Self-rated religiousness	—	1.37 (1.02–1.85)
LoPrinzi et al. (1994)	Consecutive sample of new patients with advanced cancer being treated by North Central Cancer Treatment Group	1,077	55.8	62.2	24	Personal involvement in organized religion; role of faith in personal life	—	1.00 (0.61–1.63)
Musick, House, & Williams (1998)	National probability sample of adults, age > 25	3,617	47	47.1	93	Frequency of religious service attendance	1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 15, 16, 17	1.39 (1.16–1.66)
Oman & Reed (1998)	Community-dwelling residents, age > 55, of Marin County, CA	1,931	42.2	74.4	59	Frequency of religious service attendance	1, 3, 8, 10, 11, 12, 13, 14, 15, 16	1.38 (1.11–1.71)
Oxman et al. (1995)	Consecutive sample of adults, age > 55, undergoing elective heart surgery by one of four Dartmouth surgeons	232	75	69.9	6	Frequency of attendance at religious services; strength and comfort derived from religion; number of people known in congregation; self-rated religiousness	—	3.45 (1.22–9.75)
Reynolds & Nelson (1981)	Convenience sample of chronically ill veterans from two medical centers in Los Angeles, CA	180	100	70.1	41	Missing	1	0.71 (0.38–1.35)
Ringdal (1996)	Convenience sample of cancer patients, University of Trondheim, Norway	216	55	57	21.25	Two-item measure of private religiousness (α = .71)	1, 3, 6, 14	1.25 (0.73–2.13)
Rogers (1996)	National probability sample of adults, age > 55, in National Health Interview Survey	15,938	43	69.42	90	Church or temple attendance in past 2 weeks	1, 2, 3, 4, 5, 6, 7, 9, 10, 16	1.30 (1.15–1.47)
Rosenblatt (1996)	Type II diabetes patients undergoing dialysis in Dallas and Bexar County, TX	333	58.3	60.8	35.9	Four-item measure of religiousness (item factor loadings > .73)	1, 3, 5, 8, 9	1.09 (0.71–1.68)

(*Table continues*)

Table 1 (*continued*)

Reference	Sampling	*N*	% male	Mean age at baseline	Length of follow-up (months)	Measures of religious involvement	Variables controlled in effect size[a]	Adjusted effect size (95% CI)
Schoenbach, Kaplan, Fredman, & Kleinbaum (1986)	Random sample of White males, ages 15–39, in Evans County, GA	605	100	51.3	144	Spending spare time in church activities	1, 2, 3, 4, 8, 9, 11, 13, 15, 16	1.43 (0.99–2.07)
Schoenbach et al. (1986)	Random sample of White females, ages 15–39, in Evans County, GA	719	0	52.5	144	Spending spare time in church activities	1, 2, 3, 5, 8, 9, 11, 13, 15, 16	1.11 (0.77–1.62)
Schoenbach et al. (1986)	Random sample of Black males, ages 15–39, in Evans County, GA	297	100	53.6	144	Spending spare time in church activities	1, 2, 3, 4, 8, 9, 11, 13, 15, 16	0.71 (0.44–1.15)
Schoenbach et al. (1986)	Random sample of Black females, ages 15–39, in Evans County, GA	438	0	53.5	144	Spending spare time in church activities	1, 2, 3, 5, 8, 9, 11, 13, 15, 16	1.40 (0.90–2.17)
Spiegel et al. (1983)	Consecutive sample of women with metastatic carcinoma of the breast	54	0	54.5	12	Four-item measure of family moral–religious orientation	1	0.70 (0.22–2.27)
Strawbridge et al. (1997)	Random sample of adults, ages 21–65, in Alameda County, CA	2,497	100	39.8	336	Frequency of religious attendance	1, 2, 3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16	1.14 (0.91–1.42)
Strawbridge et al. (1997)	Random sample of adults, ages 21–65, in Alameda County, CA	2,789	0	39.8	336	Frequency of religious attendance	1, 2, 3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16	1.58 (1.09–2.29)
Yates et al. (1981)	Sample of patients at Vermont Regional Cancer Center in Burlington, VT	36	39.4	59	12	Ten-item measure of religious beliefs; affiliation with a church (yes–no); importance of church; frequency of church attendance; feelings of closeness to God or nature	—	1.39 (0.37–5.17)
Zuckerman, Kasl, & Ostfield (1984)	Men in poor physical health, age > 62, in New Haven, CT	52	100	72	24	Three-item measure of public–private religiousness	1, 6, 8	3.07 (0.83–11.28)
Zuckerman et al. (1984)	Women in poor physical health, age > 62, in New Haven, CT	151	0	72	24	Three-item measure of public–private religiousness	1, 6, 8	2.03 (0.72–5.73)
Zuckerman et al. (1984)	Men in good physical health, age > 62, in New Haven, CT	77	100	72	24	Three-item measure of public–private religiousness	1, 6, 8	1.05 (0.26–4.24)
Zuckerman et al. (1984)	Women in good physical health, age > 62, in New Haven, CT	118	0	72	24	Three-item measure of public–private religiousness	1, 6, 8	1.45 (0.20–10.57)

Note. Dashes indicate that no variables were controlled. CI = confidence interval.

[a] 1 = sex; 2 = race; 3 = age; 4 = income–socioeconomic status; 5 = education; 6 = functional health; 7 = global subjective health appraisals; 8 = clinical or biomedical measures of physical health; 9 = social support; 10 = social activities; 11 = smoking; 12 = alcohol use; 13 = obesity–body mass; 14 = mental health, mood, or affect; 15 = exercise; 16 = marital status; 17 = employment status.

that zero represented the value for a typical study (0 = community sample, 1 = clinical sample) or a study whose measurement of religion would be expected to capture the most health-relevant variance (0 = public measure of religious involvement, 1 = other measures).

Study characteristics. Table 2 shows the regression coefficients and associated standard errors for the theory-derived moderators. The fact that the coefficient for the intercept (γ_0) is significant ($p < .001$) indicates that it is unlikely that the population effect size for our "typical" study is 0 (log odds). On the contrary, in a study with a score of zero on all moderator variables, we should expect to find a positive association between religiousness and longevity—the log odds of .3650 corresponds to an odds ratio of 1.44 (95% CI: 1.31–1.58), or a 44% higher odds of survival in the religious as compared with the less religious group.

The regression weights for the moderator variables indicate the extent to which each of these study characteristics would be expected to influence the observed effect size. Of the two study design characteristics, only the number of statistical adjustments was related to the size of the observed effect: Better-controlled studies (i.e., those including more covariates or copredictors) had smaller log odds ratios. This result is as predicted: Adjusted effect sizes (after controlling for mediators or confounds) are expected to be smaller than zero-order (unadjusted) effect sizes. Of the sample characteristics variables, the proportion of males in the sample was significantly related to effect size: As the proportion of males in a sample increased, the expected association between religiousness and mortality decreased. This result suggests that religious involvement might be a stronger protective factor for women than for men.

The type of measure used to assess religious involvement was also significantly associated with observed effect size. Because we regarded public measures of religious involvement as most likely to capture health-relevant variance in religiousness, we dummy coded this four-category variable so that public measures would fall into the 0 category on each dummy variable. All regression weights are negative, indicating that use of other measure types is likely to reduce the observed effect size. To clarify this relation, we repeated the analysis with a single indicator of measure type: a contrast between public measures (0) and all other measure types (1). All other theory-derived moderators were in the regression equation as before. The regression weight for measure type in this latter analysis was $\gamma = -.3179$, $SE(\gamma) = .1041$, $p = .005$. A study using a nonpublic measure of religious involvement is predicted to have a substantially lower effect size, corresponding to an odds ratio of 1.04, compared with an odds ratio of 1.43 for studies indexing religious involvement by self-reports of public religious behaviors.

Table 2
Random-Effects Regression Weights for Design Characteristics Associated With 42 Effect Sizes

Parameter	γ	$SE(\gamma)$	p
Intercept	.3650	.0470	<.001
Length of follow-up (months)	.0006	.0005	.252
No. of statistical adjustments	−.0180	.0085	.041
% male	−.0018	.0008	.043
M age at baseline	.0043	.0029	.149
Community (0) vs. clinical (1)	−.0010	.1737	.995
Measurement of religiousness[a]			
Private (1) vs. others (0)	−.1435	.2053	.489
Mixed (1) vs. others (0)	−.3077	.1070	.007
Missing (1) vs. others (0)	−.4369	.2238	.059

[a] Each religion measure was coded into one of four categories (public, private, mixed, and missing). For the regression analyses, these four categories were converted into three dummy variables (measures of private religious involvement, measures that combined public and private measures of religious involvement, and measures that were insufficiently described) so that public measures would fall into the 0, or other, category for each dummy variable.

Substantial between-studies variance remained unaccounted for by the theoretical moderators, $\tau = .0087$, $\chi^2(35) = 55.41$, $p = .015$. This corresponds to a Birge ratio of 1.58 (i.e., 58% more between-studies variance than would be expected by chance in contrast to a Birge ratio of 2.23 for the omnibus model), indicating a substantial reduction in unexplained effect size variation. The chi-square difference test comparing this model with the omnibus model shows a significant increase in explanatory power, $\Delta\chi^2(6) = 36.21$, $p < .001$, with the moderators accounting for 58% of the random-effects variance among the 42 effect sizes.

Exploratory analyses on the effect sizes for public measures. The strong effect of type of religious measure in the preceding moderator analyses suggests that the positive association of religion and mortality is derived largely from (public) participation in religious organizations rather than from (private) religious attitudes and beliefs alone. To examine the association of public religious involvement and mortality more carefully, we conducted exploratory analyses with the ($k = 21$) effect sizes ($N = 107{,}910$) involving public measures of religiousness. To avoid extremely high Type II error rates in these exploratory analyses, we chose to tolerate an increased risk of Type I errors and interpreted as marginally significant any moderator effect with a probability greater than or equal to .20. In an unconditional model involving the 21 effect sizes involving measures of public religiousness, the intercept was $\gamma_0 = .3121$, $SE(\gamma_0) = .0404$, $p < .001$, odds ratio = 1.37.

Then, we examined the moderating effects of study characteristics as we did with all 42 effect sizes. We excluded the dummy variable contrasting community and clinical samples because all of the studies using public measures of religious involvement involved community samples. For obvious reasons, we also excluded the three dummy variables representing the types of measures of religious involvement. The only study characteristic that was associated with effect size was percentage of males in the sample, $\gamma = -.0020$, $SE(\gamma) = .0009$, $p = .046$. For a study with a gender breakdown typical of these samples (i.e., 56% males), the intercept was $\gamma_0 = .3045$, $SE(\gamma_0) = .0359$, $p < .001$, odds ratio = 1.36.

Given the diversity of covariates and copredictors of mortality included in the primary studies, we set out to compare the effect sizes from studies that controlled for each of 15 variables (race, income, education, employment status, functional health, global health appraisals, clinical or biomedical measures of physical health, social support, social activities, marital status, smoking, alcohol use, obesity–body mass index, mental health or affective distress, and exercise) with effect sizes from studies that did not control for each respective variable (0 = controlled, 1 = not

controlled). We conducted 15 separate moderator analyses. In these analyses, we entered the percentage male variable simultaneously with individual control variables into a series of moderator models. Among the 21 effect sizes, obesity–body mass index was the only control variable that was associated even marginally with effect size, $\gamma = .1156$, $SE(\gamma) = .0706$, $p = .118$. A study that controlled for obesity–body mass index in a sample that was 56% male would be expected to yield an odds ratio of 1.26, whereas a similar study that did not control for obesity–body mass index would be expected to yield an odds ratio of 1.42.

At a reviewer's request we also examined the aggregate effect size when all 15 control variables were controlled simultaneously. The purpose of these analyses was to address whether the relation between public religious involvement and mortality could be attributed to some combination of sociodemographic differences, initial health status differences, differences in health behaviors, and differences in social support between religious and nonreligious groups.

We conducted a series of four regression models in which classes of control variables (i.e., sociodemographics, physical health, health behaviors, and social support) were added systematically. We encountered problems with multicollinearity among these control variables, but we included as many control variables within each class as was empirically possible. The predictor-to-case ratio increased threefold (i.e., from a 4-to-21 to a 12-to-21 ratio) from the first to the fourth model. As a result, each successive model yielded coefficients with larger standard errors and, consequently, lower statistical power. Nevertheless, these analyses are helpful for modeling how the association of public religious involvement and mortality might change as greater numbers of possible confounds and mediators of the association are controlled statistically.

The intercept (γ_0) in each model reflects the expected log odds ratio for a study with 56% males, controlling for all included moderators. The first model, including percentage male, race, income, and education, yielded $\gamma_0 = .2650$, $SE(\gamma_0) = .0623$, $p = .001$, corresponding to an odds ratio of 1.30. No sociodemographic control variable was associated with effect size (all $ps > .20$). The second model including (a) the sociodemographic variables entered in the previous model and (b) functional and clinical–biomedical measures of physical health yielded $\gamma_0 = .2298$, $SE(\gamma_0) = .0870$, $p = .020$, corresponding to an odds ratio of 1.26. None of the control variables was associated with effect size (all $ps > .20$). The third model including (a) the sociodemographic control variables and health variables included in the previous model and (b) smoking, alcohol use, and obesity–body mass yielded $\gamma_0 = .1886$, $SE(\gamma_0) = .0990$, $p = .083$, corresponding to an odds ratio of 1.21. In this model, control for smoking ($\gamma = -.2700$) and alcohol use ($\gamma = -.2833$) were marginally associated with effect size ($ps = .144$ and .104, respectively). Studies that did control for smoking and alcohol use yielded larger effect sizes than studies that did not control for smoking and alcohol use. This finding is counterintuitive and probably reflects sampling variation rather than any substantive effects. The fourth model including (a) the sociodemographic, health, and health behavior control variables included in the previous model and (b) social support, social activities, and marital status yielded $\gamma_0 = .2031$, $SE(\gamma_0) = .1853$, $p = .306$, corresponding to an odds ratio of 1.23.

Although the power of the significance tests in these analyses was low due to the small number of effect sizes, it appears that these general classes of variables account for part of the religion–mortality association. A study that controlled sociodemographics, physical health, health behaviors, and social support would be expected to demonstrate a smaller, but still substantial, association between public religious involvement and mortality.

Publication Bias and Sensitivity Analyses

The studies that are practically available for inclusion in a meta-analysis (i.e., those studies obtainable by the meta-analysts) may not be a representative sample of the studies conducted in the research domain. Indeed, the most easily obtained studies (i.e., those available in journals) tend to be biased toward positive results (Becker, 1994). This creates the potential for publication bias, also called the file drawer problem (Begg, 1994; Rosenthal, 1979).

We used several methods for evaluating the possible impact of publication bias on our findings. First, we examined a graphical display of the effect sizes as a function of their sample size. A roughly funnel-shaped display suggests that the meta-analytic data points represent an unbiased, representative sample from the population of relevant studies (Begg, 1994). The funnel-shaped distribution should occur because studies with small sample sizes have greater sampling variability, and thus, greater interstudy variability in their estimates of the population effect size, whereas studies with larger sample sizes have less sampling variability and, thus, should estimate more accurately the population effect size. By contrast, a graph that is skewed (to the right) toward more positive effect sizes for smaller sample studies suggests bias due to overreliance on published studies; the presumption here is that a number of small-sample studies that exist with less favorable effect sizes are missing from the meta-analytic sample. The display of effect sizes (log odds ratios) as a function of sample size conformed to a funnel shape (see Figure 1).

Second, we used the formulas presented in Begg (1994) to examine the correlation between the ranks of standardized effect sizes and the ranks of their sampling variances. Using the Spearman rank correlation coefficient, $r_s(42) = -.07$, $p > .30$, one-tailed. Using Kendall's rank correlation coefficient, $\tau(42) = -.06$, $p > .25$, one-tailed. These near-zero rank correlations also suggest little or no publication bias.

Third, we calculated Rosenthal's (1979) fail-safe N, which estimates the number of file drawer studies, averaging null results, that would be required to overturn an observed pattern of meta-analytic results (i.e., if the file drawer studies had been included). We calculated a fail-safe N for the omnibus analysis ($k = 42$ effects) based on formulas given in Begg (1994), which is a function of the z values associated with each of the effect sizes included in the meta-analysis. This revealed that 1,418 effect sizes with a mean odds ratio of 1.0 (i.e., literally no relationship of religious involvement and mortality) would be needed to overturn the significant overall association of religious involvement and mortality (i.e., to render the resulting mean effect size nonsignificant, $p > .05$, one-tailed) that we found in our omnibus analyses.

Begg (1985) also noted that publication bias is most likely in meta-analyses of research domains that consist of many studies with small sample sizes. In contrast, our search for relevant studies

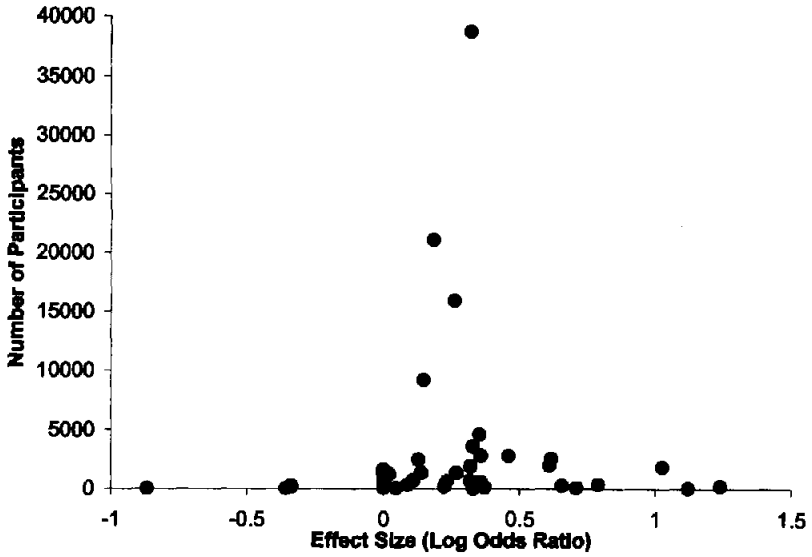


Figure 1. Relationship between effect size (log odds ratio) and number of participants for 42 effect sizes.

yielded only 42 effect sizes with a mean sample size of 2,996. These converging lines of evidence suggest that our conclusions are relatively safe from publication bias. However, readers are invited to send unpublished or published study results that were not included in the present review to Michael E. McCullough. Submitted data will be included in a future update to the present review and will help in ruling out publication bias as an explanation for the present results.

Discussion

In the course of an extensive literature search, we identified 42 independent effect sizes based on samples of nearly 126,000 people that represented the association of religious involvement and all-cause mortality. Most (k = 23) of these effect sizes were based on single-item measures of religious attendance or subjective religiousness with limited reliability, even though superior tools for assessing religious involvement are widely available (Hill & Hood, 1999). Unreliability attenuates the association of the measured variable with other variables of interest (e.g., mortality), yielding smaller effect sizes than would be observed had variables been measured without error (Hunter & Schmidt, 1990). Thus, the effect sizes reported here should be considered conservative estimates of the association of religious involvement and mortality.

Association Between Religious Involvement and All-Cause Mortality

Despite such psychometric limitations, the meta-analysis indicated that the odds of survival for people who scored higher on such measures of religious involvement (after statistical control) were 129% of the odds of survival for people who scored lower on such measures. An odds ratio of this size is equivalent to a tetrachoric correlation of .10 (Davidoff & Goheen, 1953). This effect size is considered small by Cohen's (1988) rules of thumb for the behavioral sciences. Nonetheless, the religious involvement–mortality association may have considerable practical significance given the importance of the criterion variable (i.e., mortality) and the number of people in the population who are potentially exposed to religion (Rosenthal, 1990). Although the strength of the association varied as a function of several moderator variables, the basic finding was robust: Religious involvement is associated with higher odds of survival (or conversely, lower odds of death) during any specified follow-up period. These findings could not be attributed to publication bias.

Moderator Variables: Explaining the Association of Religious Involvement and Mortality

Our moderator analyses helped to clarify the nature of the relation between religious involvement and mortality. The following explanations are offered with circumspection, however, because they are derived by interpreting multivariate correlational data gleaned from a fairly small sample of studies (Hedges, 1994; Hunter & Schmidt, 1990).

Study characteristics. As expected, studies exerting the greatest statistical control yielded the least favorable associations of religious involvement and mortality. This finding suggests that the association of religious involvement and mortality can be explained in part as a function of other demographic, psychosocial, or health-related variables. For example, studies that failed to control for obesity–body mass yielded more favorable effect size estimates than did those that did control for obesity–body mass. There is some evidence that people with high levels of religious involvement are less obese (Baecke, Burema, Frijters, Hautvast, & van der Wiel-Wetzels, 1983), suggesting that people who are religious might avoid early death in part via lower obesity (but cf. Strawbridge et al., 1997). Therefore, researchers should include obesity–body mass index in their models to estimate the extent to which religious involvement obtains its association with mortality through obesity–body mass.

Sample characteristics. The percentage of males in the study sample was the only characteristic we examined that was related to

effect size. Every 1% increase in males within a study sample is expected to yield a reduction of 0.0018 in the observed log odds ratio. Thus, a sample with 100% males (44 percentage points higher than the mean of 56%) would be expected to yield an effect size of $0.3650 - (44 \times 0.0018) = 0.2858$, or an odds ratio of 1.33, compared with a sample of 100% females, with a predicted effect size of $.3650 + (56 \times 0.0018) = 0.4658$, or an odds ratio of 1.59. Thus, the favorable association of religious involvement and mortality appears to be considerably greater for women than for men. This gender difference might be due to differences in the psychosocial resources that men and women receive from religious involvement. Because women live longer than men and tend to be more religious than men (Levin & Chatters, 1998; Levin & Taylor, 1997), researchers should control for sex statistically or estimate models separately for men and women to prevent confounding.

Measures of religious involvement. Studies using public measures of religious involvement yielded larger effect sizes than did those using other types of measures of religious involvement. This finding is consistent with speculations that the health-related effects of religious involvement are due partially to the psychosocial resources derived from frequent attendance at religious services, membership in religious groups, or involvement with other (religious) people (Goldbourt et al., 1993; Idler & Kasl, 1997a).

The particularly favorable association of public religious involvement and mortality might also be, in part, due to what Levin and Vanderpool (1987) identified as a *proxy effect* (i.e., a confounding of public religious involvement with physical functioning). Although we found no evidence that the association of religious involvement and mortality was stronger in studies that did not control for physical health, researchers should take care to control baseline physical health functioning in future research, lest the true association of religious involvement and mortality be overestimated. Indeed, researchers who investigate religion and mortality in the future should endeavor to control for all of the sociodemographic, social, and health variables that are known to be risk factors for early death. Some of these variables (e.g., race, gender, age, and probably physical health status) are confounds of the relationship between religious involvement and mortality. Others (including social support, social activities, and health behaviors) could be confounds or mediators of the religion–mortality relationship. In either case, researchers will paint an accurate picture of the religion–mortality association only when they are careful to measure and model these potential confounds and mediators adequately.

Conclusion

Although the correlational nature of the data prohibit causal inferences, religious involvement has a nontrivial, favorable association with all-cause mortality. This association is stronger in studies in which women constitute the majority of participants, there is inadequate control of other covariates of mortality, and measures of public religious involvement are used. Although part of the religious involvement–mortality association may be a product of confounding, much of the association may be substantive, perhaps mediated by health-promotive behaviors, such as maintaining a healthy body mass.

Given these conclusions—based on a meta-analytic sample representing nearly 126,000 participants—future researchers interested in these issues should probably not focus exclusively on exploring whether an association exists but should also explore the mechanisms through which religious involvement obtains a favorable association with mortality. To advance this research agenda, researchers should use more reliable measures of multiple dimensions of religious involvement (e.g., public religious involvement, private religious activities, religious beliefs, religious motivations, and religious coping). In addition, more sophisticated statistical methods (i.e., structural equation modeling) should be used to model the mechanisms (including substantive mechanisms, such as psychosocial or physiological pathways, as well as methodological mechanisms such as confounding) by which religious involvement could obtain its associations with mortality. Potential confounds that should be modeled include age, race, gender, and physical health. Potentially substantive pathways might include reductions in risky behaviors such as smoking, drug use, alcohol use, obesity, and unsafe sexual practices (e.g., see Benson, 1992); improvements in social support and marital–family stability (Ellison & George, 1994); and positive attitudes and emotions that are associated both with physical health and with religious involvement (e.g., Kark, Carmel, Sinnreich, Goldberger, & Friedlander, 1996; Myers & Diener, 1995; Witter, Stock, Okun, & Haring, 1985).

Given the large numbers of people who are religiously active, the favorable association of religious involvement and mortality is a health phenomenon with some relevance for a substantial proportion of the American population. Elucidating the nature of this robust but poorly understood association could be a fruitful topic for future research at the interface of psychology and health.

References

References marked with an asterisk indicate studies included in the meta-analysis.

*Abramson, J. H., Gofin, R., & Peritz, E. (1982). Risk markers for mortality among elderly men—A community study in Jerusalem. *Journal of Chronic Disease, 35,* 565–572.

Baecke, J. A., Burema, J., Frijters, J. E., Hautvast, J. G., & van der Wiel-Wetzels, W. A. (1983). Obesity in young Dutch adults: I. Sociodemographic variables and body mass index. *International Journal of Obesity, 7,* 1–12.

*Baugher, R. J., Burger, C., Smith, R., & Wallston, K. (1989/1990). A comparison of terminally ill persons at various time periods to death. *Omega, 20,* 103–115.

Becker, B. J. (1994). Combining significance levels. In H. Cooper & L. V. Hedges (Eds.), *Handbook of research synthesis* (pp. 215–230). New York: Russell Sage Foundation.

Begg, C. B. (1985). A measure to aid in the interpretation of published clinical trials. *Statistics in Medicine, 4,* 1–9.

Begg, C. B. (1994). Publication bias. In H. Cooper & L. V. Hedges (Eds.), *Handbook of research synthesis* (pp. 399–409). New York: Russell Sage Foundation.

Benson, P. (1992). Religion and substance use. In J. F. Schumaker (Ed.), *Religion and mental health* (pp. 211–220). New York: Oxford University Press.

Berkman, L., & Syme, L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology, 109,* 186–204.

Bryant, S., & Rakowski, W. (1992). Predictors of mortality among elderly African-Americans. *Research on Aging, 14,* 50–67.

Bryk, A. S., & Raudenbush, S. W. (1992). *Hierarchical linear models: Applications and data analysis methods.* Newbury Park, CA: Sage.

Bryk, A. S., Raudenbush, S. W., & Congdon, R. T. (1996). HLM (Version 4) [Computer software]. Chicago: Scientific Software International.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Colantonio, A., Kasl, S. V., & Ostfield, A. M. (1992). Depressive symptoms and other psychosocial factors as predictors of stroke in the elderly. *American Journal of Epidemiology, 136,* 884–894.

Comstock, G. W., & Lundin, F., Jr. (1967). Parental smoking and perinatal mortality. *American Journal of Obstetrics and Gynecology, 98,* 708–718.

Comstock, G. W., & Partridge, K. (1972). Church attendance and health. *Journal of Chronic Disease, 25,* 665–672.

Comstock, G. W., Shah, F., Meyer, M., & Abbey, H. (1971). Low birth weight and neonatal mortality rate related to maternal smoking and socioeconomic status. *American Journal of Obstetrics and Gynecology, 111,* 53–59.

*Comstock, G. W., & Tonascia, J. A. (1977). Education and mortality in Washington County, Maryland. *Journal of Health and Social Behavior, 18,* 54–61.

Craigie, F. C., Liu, I. Y., Larson, D. B., & Lyons, J. S. (1988). A systematic analysis of religious variables in the *Journal of Family Practice* 1976–1986. *Journal of Family Practice, 27,* 509–513.

Davidoff, M. D., & Goheen, H. W. (1953). A table for the rapid determination of the tetrachoric correlation coefficient. *Psychometrika, 18,* 115–121.

Davies, H. T. O., Crombie, I. K., & Tavakol, M. (1998). When can odds ratios mislead? *British Medical Journal, 316,* 989–991.

*Devins, G., Mann, J., Mandin, H., & Paul, L. (1990). Psychosocial predictors of survival in end-stage renal disease. *Journal of Nervous and Mental Disease, 178,* 127–133.

Durkheim, E. (1995). *The elementary forms of religious life* (K. E. Fields, Trans.). New York: Free Press. (Original work published 1912)

Ellison, C. G., & George, L. K. (1994). Religious involvement, social ties, and social support in a Southeastern community. *Journal for the Scientific Study of Religion, 33,* 46–61.

Enstrom, J. (1975). Cancer mortality among Mormons. *Cancer, 36,* 825–841.

*Enstrom, J. (1989). Health practices and cancer mortality among active California Mormons. *Journal of the National Cancer Institute 1989, 81,* 1807–1814.

Fleiss, J. L. (1994). Measures of effect size for categorical data. In H. Cooper & L. V. Hedges (Eds.), *Handbook of research synthesis* (pp. 245–260). New York: Russell Sage Foundation.

Friedlander, Y., Kark, J. D., & Stein, Y. (1986). Religious orthodoxy and myocardial infarction in Jerusalem: A case-control study. *International Journal of Cardiology, 10,* 33–41.

Gallup, G. (1995). *The Gallup poll: Public opinion 1995.* Wilmington, DE: Scholarly Resources.

*Goldbourt, U., Yaari, S., & Medalie, J. H. (1993). Factors predictive of long-term coronary heart disease mortality among 10,059 male Israeli civil servants and municipal employees. *Cardiology, 82,* 100–121.

*Goldman, N., Korenman, S., & Weinstein, R. (1995). Marital status and health among the elderly. *Social Science & Medicine, 40,* 1717–1730.

Haddock, C. K., Rindskopf, D., & Shadish, W. R. (1998). Using odds ratios as effect sizes for meta-analysis of dichotomous data: A primer on methods and issues. *Psychological Methods, 3,* 339–353.

Hedges, L. V. (1994). Fixed effect models. In H. Cooper & L. V. Hedges (Eds.), *Handbook of research synthesis* (pp. 285–299). New York: Russell Sage Foundation.

Hedges, L. V., & Vevea, J. L. (1998). Fixed- and random-effects models in meta-analysis. *Psychological Methods, 3,* 486–504.

Helsing, K., & Szklo, M. (1981). Mortality after bereavement. *American Journal of Epidemiology, 114,* 41–52.

Hill, P. C., & Hood, R. (1999). *Measures of religiosity.* Birmingham, AL: Religious Education Press.

*House, J. S., Robbins, C., & Metzner, H. L. (1982). The association of social relationships and activities with mortality: Prospective evidence from the Tecumseh Community Health Study. *American Journal of Epidemiology, 116,* 123–140.

*Hummer, R. A., Rogers, R. G., Nam, C. B., & Ellison, C. G. (1999). Religious involvement and U.S. adult mortality. *Demography, 36,* 273–285.

Hunter, J. E., & Schmidt, F. L. (1990). *Methods of meta-analysis: Correcting for error and bias in research findings.* Newbury Park, CA: Sage.

Idler, E. L., & Kasl, S. (1991). Health perceptions and survival: Do global evaluations of health status really predict mortality? *Journal of Gerontology: Social Science, 46B,* S55–S65.

*Idler, E. L., & Kasl, S. (1992). Religion, disability, depression, and the timing of death. *American Journal of Sociology, 97,* 1052–1079.

Idler, E. L., & Kasl, S. V. (1997a). Religion among disabled and nondisabled persons. I: Cross-sectional patterns in health practices, social activities, and well-being. *Journal of Gerontology: Social Science, 52B,* S294–S305.

Idler, E. L., & Kasl, S. V. (1997b). Religion among disabled and nondisabled persons. II: Attendance at religious services as a predictor of the course of disability. *Journal of Gerontology: Social Science, 52B,* S306–S316.

*Janoff-Bulman, R., & Marshall, G. (1982). Mortality, well-being, and control: A study of a population of institutionalized aged. *Personality and Social Psychology Bulletin, 8,* 691–698.

Jarvis, G. K., & Northcott, H. C. (1987). Religion and differences in morbidity and mortality. *Social Science & Medicine, 25,* 813–824.

Kark, J. D., Carmel, S., Sinnreich, R., Goldberger, N., & Friedlander, Y. (1996). Psychosocial factors among members of religious and secular kibbutzim. *Israel Journal of Medical Sciences, 32,* 185–194.

*Kark, J. D., Shemi, G., Friedlander, Y., Martin, O., Manor, O., & Blondheim, S. H. (1996). Does religious observance promote health? Mortality in secular vs. religious kibbutzim in Israel. *American Journal of Public Health, 86,* 341–346.

Kendler, K. S., Gardner, C. O., & Prescott, C. A. (1997). Religion, psychopathology, and substance use and abuse: A multimeasure, genetic–epidemiologic study. *American Journal of Psychiatry, 154,* 322–329.

Koenig, H. G. (1995). Use of acute hospital services and mortality among religious and non-religious copers with medical illness. *Journal of Religious Gerontology, 9,* 1–21.

*Koenig, H. G., Hays, J. C., Larson, D. B., George, L. K., Cohen, H. J., McCullough, M. E., Meador, K. G., & Blazer, D. G. (1999). Does religious attendance prolong survival? A six-year follow-up study of 3,968 older adults. *Journal of Gerontology: Medical Sciences, 54A,* M370–M376.

*Koenig, H. G., Larson, D. B., Hays, J. C., McCullough, M. E., George, L. K., Branch, P. S., Meader, K. G., & Kuchibhatla, M. (1998). Religion and survival of 1,010 male veterans hospitalized with medical illness. *Journal of Religion and Health, 37,* 15–29.

Kosmin, B. A., & Lachman, S. P. (1993). *One nation under God.* New York: Harmony.

Kovar, M. G., Fitti, J. E., & Chyba, M. M. (1990). The longitudinal study of aging: 1984–90. *Vital Health Statistics, Series 1, No. 28* (DHHS Publication No. PHS 92-1304). Hyattsville, MD: U.S. Department of Health and Human Services.

*Krause, N. (1998). Stressors in highly valued roles, religious coping, and mortality. *Psychology and Aging, 13,* 242–255.

Krause, N., & Van Tran, T. (1987). Stress and religious involvement among older Blacks. *Journal of Gerontology, 44,* S4–S13.

*Kune, G., Kune, S., & Watson, L. (1992). The effect of family history of

cancer, religion, parity and migrant status on survival in colorectal cancer. *European Journal of Cancer, 28A,* 1484–1487.

Laird, N. M., & Mosteller, F. (1990). Some statistical methods for combining experimental results. *International Journal of Technology Assessment in Health Care, 6,* 5–30.

Levin, J. S., & Chatters, L. M. (1998). Religion, health, and psychological well-being in older adults. *Journal of Aging and Health, 10,* 504–531.

Levin, J. S., & Taylor, R. J. (1997). Age differences in patterns and correlates of the frequency of prayer. *Gerontologist, 37,* 75–88.

Levin, J. S., & Vanderpool, H. Y. (1987). Is frequent religious attendance *really* conducive to better health? Toward an epidemiology of religion. *Social Science & Medicine, 24,* 589–600.

Levin, J. S., & Vanderpool, H. Y. (1989). Is religion therapeutically significant for hypertension? *Social Science & Medicine, 29,* 69–78.

*LoPrinzi, C. L., Laurie, J. A., Wieand, H. S., Krook, J. E., Novotny, P. J., Kugler, J. W., Bartel, J., Law, M., Bateman, M., Klatt, N. E., Dose, A. M., Etzell, P. S., Nelimark, R. A., Mailliard, J. A., & Moertel, C. G. (1994). Prospective evaluation of prognostic variables from patient-completed questionnaires. *Journal of Clinical Oncology, 12,* 601–607.

Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin, 119,* 322–348.

Mosteller, F., & Colditz, G. A. (1996). Understanding research synthesis (meta-analysis). *Annual Review of Public Health, 17,* 1–23.

*Musick, M. A., House, J. S., & Williams, D. R. (1998). *Attendance at religious services and mortality in a national sample.* Manuscript submitted for publication.

Myers, D. G., & Diener, E. (1995). Who is happy? *Psychological Science, 6,* 10–19.

*Oman, D., & Reed, D. (1998). Religion and mortality among the community-dwelling elderly. *American Journal of Public Health, 88,* 1469–1475.

*Oxman, T. E., Freeman, D. H., & Manheimer, E. D. (1995). Lack of social participation or religious strength and comfort as risk factors for death after cardiac surgery in the elderly. *Psychosomatic Medicine, 57,* 5–15.

Pressman, P., Lyons, J. S., Larson, D. B., & Strain, J. J. (1990). Religious belief, depression, and ambulation status in elderly women with broken hips. *American Journal of Psychiatry, 147,* 758–760.

*Reynolds, D., & Nelson, F. (1981). Personality, life situation, and life expectancy. *Suicide and Life-Threatening Behavior, 11,* 99–110.

*Ringdal, G. (1996). Religiosity, quality of life and survival in cancer patients. *Social Indicators Research, 38,* 193–211.

Ringdal, G., Gotestam, K., Kaasa, S., Kvinnsland, S., & Ringdal, K. (1995). Prognostic factors and survival in a heterogeneous sample of cancer patients. *British Journal of Cancer, 73,* 1594–1599.

*Rogers, R. G. (1996). The effects of family composition, health, and social support linkages on mortality. *Journal of Health and Social Behavior, 37,* 326–338.

*Rosenblatt, M. W. (1996). *Predictive value of social support on survival in Type II diabetic patients with end stage renal disease.* Unpublished doctoral dissertation, St. Mary's University, San Antonio, TX.

Rosenthal, R. (1979). The "file drawer problem" and tolerance for null results. *Psychological Bulletin, 86,* 638–641.

Rosenthal, R. (1990). How are we doing in soft psychology? *American Psychologist, 45,* 775–777.

*Schoenbach, V., Kaplan, B., Fredman, L., & Kleinbaum, D. (1986). Social ties and mortality in Evans County, Georgia. *American Journal of Epidemiology, 123,* 577–591.

Seeman, T., Kaplan, G., Knudsen, L., Cohen, R., & Guralnik, J. (1987). Social network ties and mortality among the elderly in the Alameda County study. *American Journal of Epidemiology, 126,* 714–723.

Shadish, W. R., & Haddock, C. K. (1994). Combining estimates of effect size. In H. Cooper & L. V. Hedges (Eds.), *Handbook of research synthesis* (pp. 261–281). New York: Russell Sage Foundation.

Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: Uses in assigning rater reliability. *Psychological Bulletin, 86,* 420–428.

*Spiegel, D., Bloom, J. R., & Gottheil, E. (1983). Family environment as a predictor of adjustment to metastatic breast carcinoma. *Journal of Psychosocial Oncology, 1,* 33–44.

*Strawbridge, W. J., Cohen, R. D., Shema, S. J., & Kaplan, G. A. (1997). Frequent attendance at religious services and mortality over 28 years. *American Journal of Public Health, 87,* 957–961.

Troyer, H. (1988). Review of cancer among 4 religious sects: Evidence that life-styles are distinctive sets of risk factors. *Social Science & Medicine, 26,* 1007–1017.

Wingard, D. (1982). The sex differential in mortality rates. *American Journal of Epidemiology, 115,* 205–216.

Witter, R. A., Stock, W. A., Okun, M. A., & Haring, M. J. (1985). Religion and subjective well-being in adulthood: A quantitative synthesis. *Review of Religious Research, 26,* 332–342.

*Yates, J. W., Chalmer, B. J., St. James, P., Follansbee, M., & McKegney, F. P. (1981). Religion in patients with advanced cancer. *Medical and Pediatric Oncology, 9,* 121–128.

*Zuckerman, D., Kasl, S., & Ostfield, A. (1984). Psychosocial predictors of mortality among the elderly poor: The role of religion, well-being, and social contacts. *American Journal of Epidemiology, 119,* 410–423.