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The Nature-Nurture Debate is Over, and Both Sides Lost! Implications for Understanding Gender Differences in Religiosity*

Matt Bradshaw and
University of North Carolina at Chapel Hill

Christopher G. Ellison
University of Texas at Austin

In their article, “A Power-Control Theory of Gender and Religiosity,” Collett and Lizardo (2009) seek to address an important, and largely unanswered, question: Why do women tend to be more religious than men? Drawing on power-control theory, they attempt to test a socialization-based explanation for this phenomenon. The motivation for their study was based, in part, on the author’s desire to refute Miller and Stark’s (2002; Stark 2002) contention that biological differences between women and men—specifically concerning their propensities toward risky behavior—may offer a better explanation for the gender gap in religiosity than the dominant sociological theory: differential sex-role socialization. In fact, the authors forcefully argue that Miller and Stark made “a premature concession to biology,” and that their “emphasis on the biological basis of the higher religiosity of women is misplaced.”

In an attempt to support their argument, Collett and Lizardo show that patriarchal versus egalitarian family backgrounds and structures—as tapped by mother’s socioeconomic status (SES)—affect levels of religiosity among daughters, but not sons, and that this accounts for observed gender differences. Specifically, they find that women raised by high-SES mothers (a proxy for a household that is more egalitarian than patriarchal, and thus has lower levels of gender-role socialization) tend to be less religious than women raised by mothers with lower levels of SES (a proxy for a patriarchal family structure that is characterized by high levels of gender-role socialization). For men, levels of religiosity do not appear to be contingent upon the structure of their rearing environment, at least as tapped by mother’s (or father’s) SES. Based on these findings, the authors conclude that they have identified a socialization-based explanation that accounts for gender differences in religiosity.

The purpose of this commentary is not necessarily to refute Collett and Lizardo. Their study is a significant contribution to our understanding of a complex, ill-explained phenomenon, and it is certainly worthy of publication. Instead, this is a broad-based discussion of five issues surrounding current debates on the biological and / or environmental causes of gender differences in religiosity; these include: (1) the fallacy of nature “versus” nurture; (2) the presence (or absence) of biological influences on religious life; (3) biological influences on the predictors of religious participation; (4) issues of causality and confounding; and (5) growing interdisciplinary endorsement of models of biology-environment interplay. These issues are important not only for Collett and Lizardo, but for everyone else involved in debates in this area as well (e.g., Miller and Stark 2002; Miller and Hoffman 1995; Stark

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*Direct correspondence to: Matt Bradshaw, Carolina Population Center, University of North Carolina at Chapel Hill, University Square, CB# 8120, 123 West Franklin Street, Chapel Hill, NC 27516. (817) 946-1165. drmmattbradshaw@yahoo.com..

2002; Sullins 2006). Thus, this is more of a commentary on the current state of the literature, than it is a specific critique of Collett and Lizardo's study.

ISSUE #1: THE FALLACY OF NATURE “VERSUS” NURTURE

The problem with theories and empirical research that take a nature “versus” nurture approach is simple: they are inadequate, and possibly even incorrect, in most cases. Recent advances in the biological, psychological, and social sciences provide strong evidence for the “ubiquitous partial heritability thesis” (Freese 2008:S2)—the fact that virtually all measurable outcomes are the products of both biological and environmental influences, not an either-or dichotomy (Bouchard and Loehlin 2001; Freese 2008; Guo, Roettger, and Cai 2008; Kendler and Baker 2007; Kendler and Prescott 2006; Shanahan and Hofer 2005). The truth is, all living organisms, even human beings, are the product of a “...unique interaction between the genes they carry, the temporal sequence of external environments through which they pass during life, and random events...” (Lewontin 2000:23). In essence, genetic or other biological influences do not specify outcomes in completely deterministic ways, and environments do not unidirectionally influence individuals. Instead, biological influences vary depending upon environmental contexts, and environments are constructed by individuals and the genes that they carry. The widespread endorsement of this reality signals the end of the so-called nature-nurture debate.

That said, not everyone agrees. Sociology's focus on environmental influences means that it almost always endorses some form of environmental determinism. Biology, in contrast, is guilty of making erroneous arguments from both sides of the debate (Lewontin 2000). More specifically, developmental models in biology focus on the set of biological mechanisms that are common to all individuals of a species. Given this search for “law-like processes” that affect everyone in similar ways, such a model is inherently a form of biological determinism. In contrast, variational models in biology, which are based on Darwinian theory, do not assume that all individuals undergo parallel development, but instead focus on the fact that there is variation among individuals, and that some variants survive and leave more offspring than others (in large part due to their fit within the environment). This scenario is, in reality, a form of environmental determinism, since environments ultimately select which individuals survive and pass on their [biology-based] characteristics to future generations.

Such models are becoming increasingly marginalized, however, and rising from the ashes of biological and environmental determinism are coming treatises on “gene-environment interplay,” “bioecological models,” “gene-environment interaction,” “biosocial influences,” and “biodemographic approaches,” among many others (Guo, Roettger, and Cai 2008; Rutter, Moffit, and Caspi 2006; Shanahan and Hofer 2005). This work, which is endorsed by a rapidly growing number of scientists from diverse fields of study, suggests that human social life, including religious participation, is a biosocial phenomenon that cannot be reduced to either nature or nurture. Let us look at some of the evidence.

ISSUE #2: EVIDENCE FOR BIOLOGICAL (AND ENVIRONMENTAL) INFLUENCES ON RELIGIOUS LIFE

Although rarely mentioned by social scientists, there is a small but rapidly growing literature examining biological influences on religious life. Much of this work has been conducted within the framework of behavior genetics, which typically employs data on multiple family members with known and differing levels of genetic relatedness (i.e., monozygotic and dizygotic twins, full and half siblings, etc.) in order to estimate the heritability of religious outcomes. Findings in this area suggest that genetic factors explain 20-30 percent of the

variation on the most commonly-examined aspect of religious life: organization-based religious practices such as church attendance (Boomsma et al. 1999; Bradshaw and Ellison 2008; D'Onofrio et al. 1999; Kendler, Gardner, and Prescott 1997; Kirk et al. 1999). With respect to more private dimensions of religious life—e.g., personal religious devotion (Kendler, Gardner, and Prescott 1997), intrinsic versus extrinsic religious orientations (Bouchard et al. 1999), personal religiosity (Winter et al. 1999), and subjective religiousness (Bradshaw and Ellison 2008), among others—research suggests that genetic differences account for roughly a third of the variation. Research also indicates sizable genetic and environmental effects on other religious outcomes as well, including spirituality, conservative ideologies, and coping (Bradshaw and Ellison 2008; D'Onofrio et al. 1999). These findings certainly do not preclude the importance of environmental influences, and evidence indicates that social factors also account for a considerable proportion of the variation on all of these outcomes.

In addition to behavior genetic approaches, which provide heritability estimates, a handful of molecular genetic studies (i.e., research designs that measure genetic differences at the level of DNA) have also been published. These studies, which have focused almost exclusively on a personality trait referred to as self-transcendence, suggest that measurable genetic differences in known polymorphic genes correspond to individual variation on this aspect of religious life. Specifically, different versions (alleles) of the DRD4, 5-HTTLPR, AP-2 β , and 5-HT $_2A$ genes—which are involved with the serotonin and dopamine systems—have been correlated with different levels of self-transcendence (Comings et al. 2001; Ham et al. 2004; Nilsson et al. 2007).

Biological influences other than genetic differences—e.g., brain structure and function—have also been linked with religious outcomes. For example, imaging studies (e.g., MRI, PET, etc.) have shown that certain areas of the brain are “activated” during religious activities such as scripture reading (Azari et al. 2001) and meditation (Newberg, d'Aquili, and Rause 2002). In addition, individual differences in serotonin receptor density in the brain have been linked with spiritual experiences, at least among males (Borg et al. 2003).

Of critical importance to this commentary are findings suggesting that genetic effects on religious outcomes may vary by gender. For example, one study reported a larger genetic effect on individual-level variation among women compared with men on two aspects of religious involvement: church attendance and conservative religious ideologies (D'Onofrio et al. 1999). Another study of religious attendance found that proportional genetic influences were 21 percent for women and 0 percent for men, with environmental influences explaining the remaining 79 and 100 percent, respectively (Truett et al. 1992). In a study of religious affiliation, a moderate genetic effect was found for females (but not males) who did not live with their twin siblings (Eaves, Martin, and Heath 1990).

Overall, then, there is reason to believe that both biological and environmental influences play a role in religious life, and that both may also contribute to gender differences in religiosity. If true, this could pose profound implications for social scientific research on this topic. There are, however, other issues to consider as well. As the next section will show, biological factors also appear to influence many of the predictors of religious participation.

ISSUE #3: EVIDENCE FOR BIOLOGICAL (AND ENVIRONMENTAL) INFLUENCES ON THE PREDICTORS OF RELIGIOUS LIFE

With respect to the correlates of religious life, there are known biological differences between women and men (e.g., different chromosomes, variable levels of hormones such as testosterone and estrogen, etc.), and these have been shown to manifest themselves in many

different social outcomes, including aggressiveness, dominance, nurturance, mating behavior, sociality, parent-child bonds, and risk aversion, among many others (Lippa 2005; Taylor 2002). Importantly, hormonal differences appear to predict variation both within, and across, the sexes—i.e., they help to explain why some women are different from other women, why some men are different from other men, and why women are different from men.

Research on SES—a key “environmental” variable in Collett and Lizardo’s manuscript—suggests sizable genetic effects (possibly explaining up to half of the variation) on both educational attainment (Heath et al. 1985; Tambs et al. 1989; Vogler and Fulker 1983) and monetary income (Rowe, Vesterdal, and Rodgers 1998). A recent study even found significant genetic effects on both grade-point average and college aspirations among adolescents and young adults (Nielsen 2006). Importantly, SES has been linked with multiple aspects of religious life, and in Collett and Lizardo’s study, mother’s SES was taken as a proxy for an environmental variable: patriarchal versus egalitarian household structures and socialization.

Research also suggests that biological factors play a role in another major predictor of religious participation: family life. For example, three different domains of parent-child relations—positivity (e.g., warmth, empathy, etc.), negativity (e.g., disputes, anger, etc.), and control (e.g., monitoring, knowledge of activities, etc.)—all appear to be influenced by both genetic and environmental factors (Plomin et al. 1994). These outcomes, although not exclusively modeled by Collett and Lizardo or other researchers in this area, are potentially important given the current focus on family environments and socialization. Romantic and marital relations have also been examined, and there is evidence for significant genetic effects on this aspect of family life as well (Jerskey et al. 2001; Spotts et al. 2004). Given that religious socialization takes place primarily in the family environment, these findings may pose profound implications for research in this area.

The explanation for gender differences in religiosity advocated by Collett and Lizardo, among others, is that being irreligious is risky (due to the possibility of eternal damnation), and that since women tend to be more risk-averse than men, this partially accounts for why they are also more religious. As Miller and Stark (2002) reported, the literature contains numerous studies reporting biological effects on risk-taking, with evidence coming from behavior genetic designs and molecular biology (Guo, Tong, and Cai 2008). Research on personality, which suggests that some individuals are more risk averse / prone (i.e., impulsive, open to experience) than others, is important here as well given the strong evidence for biological influences on virtually all personality characteristics (Bouchard and Loehlin 2001; Jang, Livesley, and Vernon 1996). Hormonal differences also appear to influence risky behavior, with testosterone being implicated in many different outcomes (Lippa 2005). Importantly, research on the biological basis of risk has been published in elite sociology journals, including the *American Sociological Review* and the *American Journal of Sociology* (Guo, Roettger, and Cai 2008; Guo, Tong, and Cai 2008).

In sum, current evidence suggests that virtually all “environmental” variables are influenced, to at least some degree, by biological factors (Freese 2008; Kendler and Baker 2007; Plomin et al. 1994). This appears to be true not only for religious involvement, but also for many of the predictors of religious life as well (i.e., sex differences, SES, family background, risk aversion, etc.). This begs the question: What implications might this pose for social scientific research that is attempting to make arguments for the importance of socialization?

ISSUE #4: CAUSALITY AND CONFOUNDING IN SOCIAL SCIENTIFIC RESEARCH

Survey research—as exemplified by Collett and Lizardo, Miller and Stark, etc.—employs variables (to gauge theoretical constructs) that are measured without regard their underlying “causes.” To understand the causes, researchers either employ independent variables as predictors, or assume one or more specific influences (e.g., biological, environmental, etc.). Since the causes of independent variables in survey research are almost never actually empirically examined, it is routine practice in social science to assume that these variables are purely environmental in origin and nature. This is exactly what Collett and Lizardo have done.

Looking at Figure 1, Collett and Lizardo argued that observed sex differences in levels of religiosity (Pathway A) are contingent upon the moderating effects of family-environmental influences (i.e., patriarchal versus egalitarian household structures, as gauged by mother’s SES; see Pathway B). Assuming purely environmental causes of each of the three key variables in their model, they found that gender differences in religiosity were smaller in families where the mother had high SES (i.e., egalitarian environments), and larger in ones where she had low SES (i.e., patriarchal environments). It was therefore concluded that differential sex-role socialization occurs in the latter type of household, and that this accounts for gender differences in religiosity.

Even though Collett and Lizardo’s interpretations are almost certainly true to at least some degree, our knowledge on this topic is still extremely limited, and their conclusions should not be taken as the final word. At the present time, it is important to ask: What potential implications do the findings reviewed above—i.e., that biological factors predict religious outcomes, sex differences, SES, family life, and risky behavior, among others—pose for their study, as well as others that use similar variables and methods? To address this question, let us look at three scenarios that are guided by Figure 2 (a graphical depiction of the literature reviewed above).

First, based on the evidence that biological factors influence levels of religiosity (shown as Pathway b1 in Figure 2), it would seem reasonable to include these potential confounders as control variables in empirical studies, especially ones like Collett and Lizardo’s that attempt to refute biological explanations. Unfortunately, this is easier said than done (although not necessarily impossible), and no research to date, including Collett and Lizardo’s study, has done this. Thus, it is not known what their findings would look like if such controls were included. If biological influences on religious involvement function the same for both women and men (i.e., if B1 and B2 are not correlated), they would probably not pose much of a dilemma, but if they do not, this could be problematic. This issue must be addressed before causal arguments concerning environmental influences can realistically be made.

Second, in addition to influencing levels of religiosity, biological factors also appear to differentiate women and men to at least some degree (Pathway b2 in Figure 2). Given this, it is important to ask two elemental questions: Are the biological factors that influence levels of religiosity the same ones that contribute to the differences between women and men (i.e., are B1 and B2 correlated)? If so, does this account for the fact that women and men display different levels of religiosity—i.e., does it explain Pathway A? Miller and Stark (2002; Stark 2002) suggested that the answer to both of these questions may be yes, and Collett and Lizardo attempted to disprove them. Unfortunately, Collett and Lizardo did not include any measures of biological influences in their model (neither did Miller and Stark for that matter), which prevents them from ruling out this alternative explanation. If there actually are biological differences between women and men that ultimately influence their levels of

religiosity—e.g., predispositions toward not only risk aversion, but also emotionality, sociality, attachment relations, etc. (Hrdy 1999; Taylor 2002)—including controls for these in their model would have functioned to reduce the gender differences they observed. In this case, biological influences would take the form of confounding influences (or “Z” variables, as they commonly called in methods and statistics textbooks), and the failure to control for these effects would bias all findings. For example, assuming that risk aversion is indeed associated with higher levels of religiosity, if women actually are more biologically predisposed toward (instead of socialized into) risk aversion than men, the failure to control for this potentially confounding influence could lead to a spurious correlation between the “environmental” variable of risk aversion, and [gender differences in] religiosity. No research to date has addressed this issue, which means that biological predispositions remain a viable, and untested, explanation for gender differences in religiosity.

Third, in addition to influencing religiosity and sex differences, biological factors also appear to influence family environments (as reviewed above, and as shown as Pathway b3 in Figure 2). What implications might this pose for social scientific research like Collett and Lizardo’s study, which assumed that these were completely environmental in origin and nature? To begin with, even if we could somehow address the two previous issues—i.e., rule out biological influences on religiosity and sex differences as explanations for the gender gap in religiosity (something that has yet to be done)—it would still be possible for biological factors to play a role via their effects on the family environment. The literature reviewed above showed that SES, including that of mothers, is influenced to at least some degree by biological influences, and that many other aspects of family life are as well. This suggests that biological influences (particularly those of the mother) could be at least partially responsible for the fact that some households are patriarchal, while others are egalitarian. More specifically, if biological factors predispose some mothers (more than others) toward traditional family life—i.e., if some have more of a “maternal nature” or “tending instinct” than others (Hrdy 1999; Taylor 2002)—this would almost certainly lead to lower levels of educational attainment, as well as a more patriarchal family environment (note: in this case, B2 and B3 in Figure 2 would be correlated). Thus, Collett and Lizardo’s assumption that a patriarchal versus egalitarian family structure is an “environmental variable” may not be entirely correct, and this could pose profound implications for their arguments. There is, however, even more to this story. Given the connection between family life and religious participation, biological predispositions toward family life are also likely to be associated with higher levels of religious involvement. This sets up a scenario where biology influences the “religiously-relevant characteristics” of mothers (including, but not limited to, patriarchy), who subsequently: (a) create environments that satisfy these innate predispositions; and (b) pass on both their genes “and” their environments to their children (particularly their daughters). Thus, the religious socialization of daughters by patriarchal mothers, the key to understanding gender differences in religiosity according to Collett and Lizardo, is almost certainly confounded by biological influences to at least some degree. They did not take this possibility into consideration, but instead assumed that patriarchy was an entirely environmental phenomenon driven by the socioeconomic attainment, and thus gender-role ideology, of mothers.

Overall, these issues are extremely complex, and no single study can rule out all potential confounders. It would obviously be difficult for Collett and Lizardo, or anyone else for that matter, to address all of the concerns raised here. That said, scientific progress requires, at the very least, that we: (a) control for influences that we seek to refute; (b) recognize the limitations of our findings; and (c) acknowledge the true complexities of all human outcomes, including religious participation. The first two issues have already been addressed, so the remainder of this commentary deals with the last one by discussing models of biology-environment interplay.

ISSUE #5: MODELS OF BIOLOGY-ENVIRONMENT INTERPLAY

Social scientific research on gender differences in religiosity—including Collett and Lizardo’s study—represents a style of thinking indicative of the old nature-nurture debate. Even though there may be examples of outcomes that truly are the products of either biological or environmental influences, the current literature suggests that these instances are rare, at best, and the gender gap in religiosity is almost certainly not one of these. A more fruitful approach to understanding religious life, therefore, lies in models of biology-environment interplay. Based on existing research, these two influences appear to work in both correlated and interactive manners, and applying these ideas to religious outcomes will almost certainly provide greater insight than reductionist biological or sociological models. (Note: Research in this area uses the term “gene-environment interplay” instead of “biology-environment interplay,” so this terminology is employed here as well. It should be noted, however, that genetic and biological are not necessarily synonymous.)

Gene-Environment Correlation

The first and simplest way that biological and environmental factors might interconnectedly influence religious outcomes is through “gene-environment correlation,” which comes in three forms: passive, evocative, and active (Plomin, Defries, and Loehlin 1977; Scarr and McCartney 1983). The passive variety occurs through the environments supplied to individuals by genetically-related individuals, particularly their parents, who also provide genetic dispositions related to these environments. For example, if religious involvement is at least partially heritable, religious individuals will tend to have religious parents who provide them with both gene-based predispositions toward religiosity, as well as social environments that facilitate these inherited characteristics. In this situation, the two influences—genes and social environments—naturally occur together, and disentangling the unique influences of each is quite difficult (for other examples of passive gene-environment correlation, see: Jaffee and Price 2007; Reiss et al. 2000). The main question raised by this possibility is: Do parents pass on religion to their children via genetic factors, environmental influences, or both? The answer is almost certainly both, but investigating this question could facilitate our understanding the mechanisms by which religion is transmitted from one generation to another. This may even help us to understand gender differences in religiosity, especially if women are indeed more predisposed toward religion or any of the predictors of religious life than men.

Evocative gene-environment correlation occurs through the responses evoked from others in one’s social environment that are due to the genetic makeup of the evoking individual. Empirical examples for religious phenomena are not yet available, but research on other topics may help to illustrate this scenario. For example, scholarship on corporal punishment has shown that genetic factors are at least partially responsible for antisocial behavior in children, which subsequently “evokes” a response from parents in the form of corporal punishment (Jaffee et al. 2004). Similarly, research on another family outcome, marital quality, suggests that gene-based predispositions toward depression tend to “evoke” negative responses from spouses, which, in turn, has harmful consequences for marital relations (Jaffee and Price 2007). Given these findings, it is easy to envision scenarios that are relevant to the study of religious life. For example, genetic predispositions toward undesirable outcomes such as psychopathology or an antisocial personality may evoke negative reactions from religious others, thereby making it difficult to participate in a religious organization. Likewise, other biologically-influenced characteristics, including extraversion, may elicit favorable responses from religious others, thus facilitating religious group formation and activities. A more concrete example might involve persons with innate musical or teaching abilities, which evoke the support of religious leaders, and therefore enhance the religious experiences of everyone involved. Countless other examples could be

offered, but the important point is this: possibilities such as these can only be understood with an integrated biology-environment approach. Given that there are known gender differences in many different genetically-influenced “evocative traits” (e.g., women report more symptoms of psychopathology than men, and they also tend to be more neurotic, agreeable, extraverted, and conscientious, on average; Kendler and Prescott 2006; Schmitt et al. 2008), this could help us to understand the gender gap in religiosity.

Active gene-environment correlation functions through the purposive selection or “niche-picking” of environments by individuals based on their genetically-predisposed motivations (Scarr and McCartney 1983). For example, some distressed individuals might consciously seek out religious participation or a connection with God based on an innate need for social attachment or feelings of security in a stressful world (Hamer 2004; Newberg, d’Aquili, and Rause 2002). We know that women report higher levels of distress than men, and that biological factors influence distress, so taken together, these two pieces of the puzzle could help us to understand gender differences in religiosity. Another example might involve individuals who are biologically-predisposed toward agreeableness, which motivates them to participate in religious organizations that emphasize selflessness, altruism, and prosocial activities. We know that women tend to be more agreeable than men, so this could be extremely insightful. Further, to the extent that worldviews and exclusivist beliefs are influenced by biology, some individuals might consciously seek out conservative religions while others look toward more mainstream or liberal ones. It is important to note that active gene-environment correlation is most likely to occur when individuals have freedom of choice and environmental opportunities to express their innate needs, orientations, or inclinations. Although not the focus of this commentary, this idea corresponds nicely with the rational choice theories that are currently popular in the scientific study of religion (see Stark and Finke 2000). They may even enhance these models by providing the biological underpinnings of purposive action and agency.

Gene-Environment Interaction

In addition to correlated effects, there is at least one other way (two forms) in which biological and environmental factors might work together to influence religious participation: “gene-environment interaction.” Essentially, research on many different outcomes suggests: (a) that genetic factors may be either more or less pronounced depending upon environmental influences; and (b) that environmental influences may have different effects based on the genetic makeup of individuals (Boomsma et al. 1999; Jaffee et al. 2005; Shanahan and Hofer 2005).

When thinking about the study of religious life, this could be an extremely important idea. For example, we know that religious socialization takes place primarily from parents to children, but it is also true that some individuals from religious households end up being religious themselves, while others do not. Why? Social science would say that these individuals experienced alternative environmental influences that ultimately led to different paths and outcomes in their religious lives. This is likely true to some extent, but it could also be the case that religious socialization in the family is contingent upon biological differences among individuals. If some individuals are indeed more biologically predisposed toward religious participation than others, socialization may accentuate these propensities, but for those who do not possess such innate inclinations, socialization may not have much an impact. This could possibly be functioning in the creation of gender differences in religiosity—i.e., socialization might have a bigger impact on the religiosity of women partially because they are more genetically-predisposed toward it than men. Biology-environment interactions could also explain religious conversions. For example, if an atheist who was raised in a non-religious family actually carries an inherited biological

predisposition toward religious participation, this may be expressed when this individual encounters a triggering environmental stimulus, whereas it may remain dormant otherwise.

Essentially, then, the interaction between biology and social life suggests that the effects of one are contingent upon the other. Given the evidence for both biological and environmental influences on many different religious outcomes, it does not seem controversial to assume that these factors are interacting with each other in some way. To date, however, no studies of gene-environment interaction have been published using religious involvement as the outcome of interest. For those interested in making a substantial contribution to our understanding of religious life, this is an area of inquiry that deserves serious consideration.

SUMMARY AND CONCLUSION

This commentary began by applauding Collett and Lizardo for their effort to provide insight into an important, and ill-understood, phenomenon: the empirical fact that women tend to be more religious than men. While not necessarily disputing their findings—but raising several issues surrounding current debates on this topic—it was then argued that the authors have not done what they set out to do: refute the biological explanation proposed by Miller and Stark (2002). The reason: they did not measure anything biological. Not only does this prevent them from ruling out biology as an alternative explanation, it also means that their findings should be interpreted with caution due potential confounding by theoretically important biological influences.

In essence, there is mounting evidence that all aspects of human existence, including religious participation, are the products of both biological and environmental influences, particularly the interplay of these two factors (Bradshaw and Ellison 2008; Freese 2008; Guo et al. 2008; Heath et al. 1985; Loehlin and Martin 2000; Plomin et al. 1994). If both influences really do play a role in religious life, scholars should not be arguing for, or against, one or the other, but should instead be striving to understand how they work together in correlated and interactive ways to produce religious life, including gender differences in religiosity. Thus, there is no such thing as conceding to biology (or sociology for that matter); both are important.

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FIGURE 1.

Explaining sex differences in levels of religiosity via the moderating effects of family environment (Collett and Lizardo's model).

FIGURE 2.

Explaining sex differences in levels of religiosity via the moderating effects of family environment, with biological (b) and environmental (e) influences specified for all variables (a biologically-informed model).