

Sample of Recent Work/Publication

*This document is the first analytic study of my dissertation. It is a slightly reworded draft for the purposes of submission to **Demography** for publication. Dr. Tim Bruckner is my advisor and Dr. Ken Smith from the University of Utah provided suggestions as the former director of the Utah Population Database.*

Religion, Policy Change, and Older-Age Mortality in 19th and 20th Century

Utah

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Abstract

Birth, reproduction, and death are of primary interest to both public health and religion. Both institutions maintain beliefs and practices surrounding these events meant to shape personal behavior that in turn influence demographic dynamics. We examine how mortality outcomes respond to a sudden change in religious policies related to tobacco use. Using the Utah Population Database, we compare life expectancy and tobacco-related health outcomes to the Church of Jesus Christ of Latter-day Saints' (LDS) smoking policy change in 1921 among Utah's 1880 to 1920 birth cohorts. Active LDS males and females who are born after 1900 exhibit an increase in life expectancy (Females = 3.644 years, Males = 5.177 years; $P < .0001$) compared to their inactive counterparts. Additionally, less-active LDS males and females born after 1900 (and exposed to the policy change before adulthood) manifest a dramatic increased Hazard Risk Ratio (HRR) in tobacco related death (Female HRR= 3.046, Male HRR= 3.865) compared to their LDS active peers. Similar differences do not exist in pre-1900 cohorts nor do the tobacco-related findings replicate in non-tobacco related cancers. These results suggest that in early 20th century Utah, abrupt changes in behavioral norms dictated by religious policy may have reduced mortality.

Keywords: religion, public health, morbidity, mortality, health behavior

Introduction

Both public health and religious institutions maintain practices that induce population expansion. While public health research approaches reductions in morbidity and mortality as a fundamental ethic (Kass, 2001), beliefs and rules often emerge from religion to influence fertility and longevity (Baumard & Boyer, 2013; Radnitsky & Bartley, 1987). The Church of Jesus Christ of Latter-day Saints' (LDS) smoking-abstention policy of 1921 provides a modern example of the application of institutional rules to mortality-reducing behavior. As such, this early 20th century health code offers an opportunity to consider whether the interests of religion may indirectly precede improvements in public health.

Previous research establishes a firm health advantage for Latter-day Saints across a range of diseases and medical conditions (Bartz et al. 2010; Bush, 2022; Daniels, 2004; Hawkes et al. 2007; S. J. Lyon, 2013; Norton et al. 2010). Although research attributes multiple factors to this phenomenon (Lindahl-Jacobsen et al. 2013; Merrill, 2004), many credit an LDS culture that eschews tobacco and alcohol (Enstrom & Breslow, 2008; Merrill & Lyon, 2005; Merrill & Thygeson, 2001). Despite these associations, to postulate that religious policy invokes positive behavioral change requires one to address the counterargument that persons unwilling to conform to institutional codes are more likely leave the faith (Dengah et al., 2019; Francis & Katz, 2000; Jokela & Laakasuo, 2023; Scheitle & Adamczyk, 2010). Consequently, we set out to examine religion's employment of rules in shaping health behavior by leveraging the quasi-experimental setting provided by the nature of the early 20th century policy (Campbell & Stanley, 2011). To do so, we offer a formal test of the introduction of LDS tobacco abstention to changes in all-cause and cause-specific morbidity and mortality.

Background

Religion and Demography

Social theory holds that the supernatural essence of religion's beliefs and rules fuels its authority to direct behavior (Azra et al., 2010; Durkheim, 2016; Fitouchi & Singh, 2022; Singh et al., 2021). If expansion ranks as a key objective of a religious body, leadership must consider the most efficient behaviors to meet this ambition. These behaviors would presumably need to support demographic fundamentals that conceive population change as a function of the number of individuals who enter the population through birth and immigration compared to those who exit through emigration and death (Coale, 1989; Coale & Trussle, 1996). Consequently, we may find artifacts of religious beliefs surrounding fertility and mortality behavior within highly religious societies.

Health as LDS Identity

Both the LDS Church's history of assimilation with outside world and its theology paved the way for the 1921 anti-tobacco policy. In 1847, three years after the killing of church founder Joseph Smith, the Latter-day Saints migrated westward to escape persecution and find independence (Arrington, 2005; Brown, 1980). Once in Utah their long-fought seclusion collided with the railroad's 19th century expansion, transforming the isolated Mormon colony into an inter-mountain crossroads (Farmer, 2015; Kucharski, 2017). The territory's industrialization left church leaders struggling to protect the unique LDS identity against cosmopolitanism. Scholarship now argues this cultural struggle created vacillations between national assimilation and institutional orthodoxy, out of which a 20th century Latter-day Saint identity emerged (Bowman, 2012; Mauss, 1994a, 1994b). Polygamy and external conflict, casualties of national pressure and the bid for statehood, ceded to teetotaling and patriotism.

The theology of LDS health is found in “The Word of Wisdom” (WoW), introduced by Joseph Smith as a revelation in 1833. The initial teaching stood as advice on healthy living that promised physical stamina and greater spirituality (Hoskisson, 2012; *The Doctrine and Covenants*, Section 89). Yet, until the 20th century authoritative WoW interpretations varied, leaving tobacco and alcohol both taboo and ubiquitous within the church (Alexander, 1981; Eddington, 2023; Peterson, 1972). By the early 1900s, emerging social forces such as the evangelic temperance movement, a post-polygamy desire for acceptance within the American mainstream, and the exponential rise in cigarette uptake among the faith’s youth produced the circumstances to solidify the Word of Wisdom (Alexander, 1996). In 1921, the church settled upon complete abstention from tobacco, alcohol, coffee, and tea as a prerequisite to temple ceremony participation, rituals at the heart of its theology, 30 years prior to the epidemiologic evidence concerning cigarettes (Cornfield et al., 1959; Doll & Hill, 1950; Khuder, 2001). For the next two decades church leaders solidified this new paradigm through speeches and printed media aimed at LDS youth. By 1940 obeying the WoW was a matter of orthodox identity.

Religion and Health

Much literature finds a relation between religious identity and lower tobacco use. Identity measured through church service attendance (Bowie et al. 2017; Q. L. Brown et al. 2014; Gillum, 2005; Hofstetter et al., 2010), belief in God (Elkalmi et al. 2016; Gmel et al. 2013; Sanchez et al. 2011), prayer (Alzyoud et al. 2015; McFadden et al. 2011), and religious network (Andres-Sanchez et al. 2021; Bailey et al. 2015) display inverse associations with tobacco consumption. Latter-day Saints display similar findings. LDS members exhibit lower levels of smoking uptake (Koenig et al. 1998; Merrill & Thygeson, 2001), lower levels of all-cause and

tobacco-specific cancers (Enstrom, 1975, 1978, 1980; Enstrom & Breslow, 2008; Gardner & Lyon, 1982; Lyon et al. 1980; Merrill, 2004; Merrill & Lyon, 2005), and increased life expectancy (Lindahl-Jacobsen et al. 2013; Mineau et al. 2004) compared to non-LDS populations, even when controlling for church attendance.

Despite the enormity of evidence that indicates LDS membership and activity is associated with reduced tobacco use, the work is limited in two ways. First, none of this research rules out the plausible rival that persons inclined to smoke tobacco exit LDS membership rather than conform. This non-random selection of persons into and out of the LDS faith limits causal inference. In addition, we know of no work that examines the relation between the enactment of 1921 WoW policy and the *change* in health of Latter-day Saint cohorts. Given the recent and abrupt nature of the policy, this circumstance provides a unique opportunity to examine whether a change in religious norms could affect health behavior and mortality.

Two empirical considerations require careful attention when investigating the WoW as health policy: defining LDS religiosity and establishing temporal order. Regarding religiosity, research often employs self-reported survey responses of church service attendance (Chatters, 2000; Hall et al. 2008; Kim & VanderWeele, 2019; T. W. Smith, 1998; VanderWeele, 2017). For the LDS faith, however, health researchers benefit from the faith's belief in, and practice of, meticulous record keeping (Snow, 2019). For epidemiologic and demographic studies, this high-quality record keeping often permits operationalization of church activity in the form of LDS member/non-member status (Lyon et al. 1994; Williams et al. 1979) or rank in the lay priesthood (Enstrom & Breslow, 2008; Gardner & Lyon, 1982). Yet, these tactics have their own shortcomings. For instance, the LDS church determines membership by baptism status, which occurs at age 8 (median age= 8.54). As such, measurement of religiosity by LDS membership

would precede early-life behavioral decisions for many. The alternative, advancement in the male lay priesthood, allows for adult comparisons of health behaviors, but precludes females. Consequently, both member/non-member status and rank in the male-priesthood have limited value when addressing a church-wide policy.

As prior literature concerns LDS lifestyle characteristics rather than policy effects, it does not consider temporal events (Badanta et al. 2020; de Diego Cordero & Badanta Romero, 2017; Merrill, 2004; Merrill & Salazar, 2002). While these efforts illuminate religious experience as a determinant of health, they do not offer a mechanism that explains institutional function. For example, Merrill (2004) investigates how much of LDS increased life expectancy during 1994 to 1998 in Utah is due to decreased smoking. The author determines that decreased tobacco consumption accounts for only 1.5 years of the additional 7.3 years of period life-expectancy found amongst LDS males compared to non-LDS males. Although this study offers strong evidence regarding the health quality of LDS culture during the mid-1990s, it does not establish a clear link between how institution objectives cause individual behavior changes. As such, the need exists for a study that isolates whether, and to what extent, specific church rules precede improvements in health outcomes.

Therefore, we extend previous LDS research by investigating the WoW as precursor to health behavior among LDS members. We consider how cohort rates of smoking-related disease and death respond to the 1921 LDS anti-tobacco policy according to church activity. We extend past efforts within religion and health through the application of a historically informed theoretic framework paired with an extensive genealogic dataset that allows for both within-religious and across-time comparisons.

Predicted Outcomes

We anticipate that active church members born between 1900 to 1920 will experience greater life expectancy past age 50, and a lesser hazard of tobacco related cancer and death compared to inactive LDS members of the same birth cohorts. For individuals from the 1880 to 1899 birth cohorts, we expect no difference in these outcomes according to religious practice. We employ the year 1900 as a nexus point given that authorities in 1921 were most concerned with changing tobacco patterns in LDS youth, rather than older adults for whom they made allowances (Peterson, 1972). Psychology and substance-abuse literature that identifies adolescence as a key age for identity and decision making offers additional support for this age choice (Amos & Bostock, 2007; Arnett, 1997, 2000; Currie et al., 2004; Sowden & Stead, 2003). We further test our hypotheses through a falsification test that employs prostate and breast cancers as health outcomes. For these tests, we expect to see no difference in non-tobacco related cancer diagnoses rates based upon religious activity.

Methods

Data

The Utah Population Database (UPDB), one of the world's most comprehensive computerized genealogies, serves as the primary data source. The UPDB links population information of over 11 million individuals from the late 1700s until today using LDS genealogical data matched to census, vital records, hospital records, and more (Smith & Mineau, 2021). For a given individual, UPDB lists all known residential, occupational, marriage, children, baptism, and endowment records. Thus, the ability to match social and health information over many generations stands as a key strength of the UPDB. We know of no other

data set of its kind, in terms of historical accuracy and completeness dating back to the 19th century, in the United States.

Population

Our population consists of 138,739 baptized members of the LDS church, born in Utah between 1880 to 1920, who have a place of death in Utah, and survived to at least age 50. These cohorts enjoy advantages in the UPDB as 1907 marks the initial use of death certificates in Utah, and 1975 is the last year of religious participation recorded in the UPDB. Further, the 1880 to 1920 cohorts would have been between the ages of zero to 41 during both the 1921 policy and the early 20th century boom in cigarette smoking (Brandt, 2007; Jackson, 1950). To ensure accuracy of records and similarity of exposure to church culture, we include only baptized LDS individuals and those whose birth and death states were Utah.

We considered four factors based upon exploratory data and literature to justify our inclusion criteria of survival to age 50. First, 90% of endowments among Utah birth cohorts from 1880 to 1920 occurred prior to age 50. As 1975 constitutes the last year of recorded endowments in the UPDB, an individual born in 1920 and then endowed at age 50 sits five years prior to this threshold. Second, 97% of all tobacco-related deaths occur after age 50 among this UPDB cohort. Third, the requirement of survival to age 50 accounts for the high prevalence of unintentional injuries among younger ages that may dilute life expectancy differences between active and inactive LDS members (*WISQARS Leading Causes of Death Visualization Tool*, 2023). Fourth, research demonstrates that a male who quits smoking at age 50 will return to at or near baseline lung cancer risk in their 70s (Halpern et al. 1993; Peto et al. 2000; Saito et al. 2017). Our age inclusion requirement allows sufficient time for one to be endowed and forsake

or cease smoking, renders persons less likely to suffer accidental death, and yet still captures nearly all tobacco-related deaths.

Variables

Our dependent variables are life expectancy in years past age 50, risk of smoking related death and risk of lung and bronchus cancer. We focus upon tobacco because it serves as the majority cause of a specific class of disease (aerotract cancers), unlike other substances prohibited in the WoW (Hall et al. 2008; Kuper et al. 2002; United States Office on Smoking and Health, 1979; Zhou et al. 2021). Further, cigarette smoking during this time exponentially grew. Without an intervention, we would expect tobacco related disease and mortality to climb in tandem among all groups (Gershon, 2022). We define tobacco-related deaths as deaths whose primary cause is either Chronic Obstructive Pulmonary Disease (COPD) or smoking specific cancers of the lung, bronchial, trachea, or esophagus (*appendix: Table A2*). When measuring morbidity, we use the individual classification provided by the UPDB “lung or bronchus” cancer diagnosis, as our primary endpoint. As a test of falsification, we consider diagnosis of colorectal, prostate and breast cancers.

We deviate from previous LDS tobacco studies by considering religious activity as a modifying factor between policy timing and health outcomes. Consequently, our exposure variable is whether one was born from 1880 through 1899 (coded “0” as “unexposed pre-20 years old”), or from 1900 through 1920 (coded “1” as “exposed” pre-20 years old). We reason that an LDS individual born on or after 1900 fell within the policy’s target age in 1921 at or before age 20. Furthermore, by confining our exposure to a pre-adult age, our measurement of religiosity through recorded participation in the endowment allows for a clear means to identify

those who likely conformed to the new policy. Consistent with past UPDB studies, we defined our modifying covariate, “active LDS,” as those with an endowment participation date recorded within the UPDB and “inactive LDS” as those with only a baptism date (Lindahl-Jacobsen et al. 2013; Mineau et al. 2004; K. R. Smith et al. 2002). The endowment ceremony constitutes a temple ritual generally performed in one’s 20s (median age = 25.12) involving promises of loyalty to church teachings and leaders (*About the Temple Endowment*, n.d.). For simplicity of interpretation, we stratify our models by sex.

We also employ birth cohort sex ratio (the ratio of males to females born in each birth cohort) as a measure of in utero environmental harshness that influences old age longevity and disease susceptibility. Prior literature demonstrates lower than expected sex ratios often follow harsher prenatal conditions that cull weaker fetuses, who generally are male (Bruckner, 2018; Catalano & Bruckner, 2006; Schacht et al. 2021). Thus, birth cohorts with higher-than-expected sex ratios may produce weaker males that in-turn display increased hazard of disease and mortality. We also include urban or rural born as much Utah based literature reveals worse health effects in rural areas post-industrialization (Blackburn et al., 2019; Koric et al., 2023; Ou et al., 2018; Rogers et al., 2020). 29 counties in Utah were categorized as “urban” or “rural” based upon both past UPDB precedent and SEER*Stat definitions (Park et al., 2018; Rogers et al., 2020; *Rural-Urban Continuum Code - SEER Datasets*, 2014).

Models

For tests of life expectancy, we applied Ordinary Least-Squares Regression techniques to examine life expectancy Y_{ij} from age 50 years until death. We include a vector of individual background characteristics X_i that may predict the outcome but are not caused by the Word of

Wisdom policy, including birth cohort sex ratio and urban or rural status. We code sex ratio as a continuous variable. Urban or rural status is coded being born in an urban county (1) or born in a rural county (0).

Whether one was born between 1880 to 1899 (0) or 1900 to 1920 (1) is our key independent variable $WoWc_t$; β_1 is our coefficient of interest. In our second model, we add background characteristics X_i , and religious participation $ActiveLDS_j$. We code religious participation as a binary variable that measures whether the LDS member was endowed (1) or not endowed (0). In our third model we add an interaction term between $WoWc_t$ and $ActiveLDS_j$. These models take the following form:

$$1: Y_t = \alpha + \beta_1 * WoWc_t + \epsilon_t$$

$$2: Y_{itj} = \alpha + \beta' * X_i + \beta_1 * WoWc_t + \beta_2 * ActiveLDS_j + \epsilon_{itj}$$

$$3: Y_{itj} = \alpha + \beta' * X_i + \beta_1 * WoWc_t + \beta_2 * ActiveLDS_j + \beta_3 * WoWc_t \times ActiveLDS_j + \epsilon_{itj}$$

We employ Cox Proportional Hazard models to examine cause-specific mortality λ_{1itj} from age 50 years until death. Let (a) denote age. We define mortality as death from a tobacco related disease (1) or death from any other cause (0). Our independent variables are similar to our life expectancy models. We express the hazard rate $\lambda_{1itj}(a)$ of tobacco related death in the following three models:

$$4: \lambda_{1t}(a) = \lambda_{0t}(a) \exp(\beta_1 * WoWc_t + \epsilon_t)$$

$$5: \lambda_{1itj}(a) = \lambda_{0itj}(a) \exp(\beta' * X_i + \beta_1 * WoWc_t + \beta_2 * ActiveLDS_j + \epsilon_{itj})$$

$$6: \lambda_{1itj}(a) = \lambda_{0itj}(a) \exp(\beta' * X_i + \beta_1 * WoWc_t + \beta_2 * ActiveLDS_j + \beta_3 * WoWc_t \times ActiveLDS_j + \epsilon_{itj})$$

The test for lung and bronchus cancer diagnosis and our falsification tests using colorectal, breast, and prostate cancer use the same models save for the fact that the outcome variables differ.

Results

Tables 1 and 2 present our descriptive results. Our population of 138,739 includes 33,569 inactive LDS individuals who were 49.19% female and 105,170 active individuals of which 52.11% were females. The 74.58% of inactive LDS individuals compared to 48.84% of active LDS individuals were born after 1900. Active LDS were slightly more represented in urban births.

[Table 1 Here]

[Table 2 Here]

Figure 1 provides visual evidence in support of health effect modification due to LDS church activity. There is striking difference between active and inactive LDS individuals' rates of tobacco related death and lung cancer. These same trends do not appear in breast and prostate cancers. Additionally, these charts support 1900 as an inflection point to a limited degree. Overall and male-only life expectancy, female tobacco related death, female lung cancer, and male prostate cancer appear to show clear effect modification occurring near 1900. Other outcomes, such as male tobacco related death and female life expectancy, appear to differentiate between 1890 and 1895 birth cohorts.

[Figure 1 here]

Table 3 offers the results from our OLS regression analysis. Active LDS males born after 1900 possessed 5.177 extra years of life past age 50 compared to inactive males. For females,

the difference was only 3.644 years. For those born prior to 1900, active LDS members demonstrated less life expectancy than inactive members— 2 years less for females and 9 months less for males. These life expectancy differences, though lower than those reported by Enstrom (1978) and Merrill (2004), seem reasonable given that we employ differences among LDS sub-groups rather than comparisons between members and non-members.

[Table 3 here]

Tables 4 and 5 displays the results from our Cox Proportional Hazard models. These finding demonstrate significant differences cohort and religious effects among both sexes, although religion has the greatest modifying effect for males born 1900 to 1920. For the 1900 to 1920 cohorts, both sexes show significant increase in hazard of tobacco related death (Female HRR= 2.367, Male= 1.660) and lung cancer (Female HRR= 3.048, Male= 2.414). Yet when accounting for religiosity, its inactive LDS individuals who see the greatest increase of risk post 1900 with inactive females and males reporting increased risk in tobacco related death (Female HRR= 3.046, Male HRR 3.865) and lung cancer (Female HRR= 2.427, Male= 3.773). These findings echo previous research that suggests religious influences may modify male risk taking (Byrnes et al., 1999; Friedl et al., 2020; Robbins & Martin, 1993). Our covariates, birth cohort sex ratio and urban birth status, show some statistical significance but do not provide a substantial change in risk.

When testing for breast and prostate cancers, differences upon LDS activity lines either disappear or reverse. Our models for breast cancer show no significant difference according to LDS activity no matter the birth cohort. A difference in prostate cancer risk appears after 1900 with active LDS males demonstrating a 12% increase in risk of diagnosis, though not statistically significant. This small difference in prostate cancer risk, seen both in our exploratory charts and

statistical analysis, is likely an artifact of lengthened exposure time due to the increased longevity among active LDS males (*appendix: Table A1 and Figure A1*).

[Table 4 here]

[Table 5 here]

Figure 2 displays relevant survival ratios by sex, according to church activity and birth year relation to 1900. These charts echo the same between group differences found in exploratory charts concerning tobacco related death.

[Figure 2 here]

Discussion

The WoW provides a unique religious policy that tied cigarette abstention to higher level LDS ceremony participation, 30 years prior to epidemiological evidence of the risk of tobacco. As such, we tested the 1921 policy's relation to changes in all-cause and cause-specific morbidity and mortality as modified through participation in the LDS endowment. This test was strengthened by our choice in data. The UPDB allowed a population comparison of morbidity and mortality according to endowment status due to its composition of LDS genealogical data matched to census, vital records, hospital records. Our tests of reveal that LDS individuals who were endowed prior to age 50, enjoyed both decreased tobacco related death and lung/ bronchus cancer life expectancy, and increased life expectancy compared to LDS individuals who were not endowed. These results did not carry over into non-tobacco related health outcomes such as breast and prostate cancers. Therefore, our findings support the inference that religion may act in ways that support mortality reduction in populations.

Despite religious differences in our findings, all post-1900 born individuals displayed increased cancer incidence and risk of tobacco caused death when compared to pre-1900 cohorts. This increase seems plausible given the growing popularity of smoking at the time. Yet the rise in tobacco related death and disease among those who participated in temple ceremonies appears counterintuitive. Possible explanations include increased environmental exposures, religious disillusionment post-endowment, and/or selective uptake of church teachings (Mumford et al., 1987; Öberg et al., 2011; Scheitle & Adamczyk, 2010).

The results from our falsification tests reveal health benefits from temple ceremony participation were not universal across outcomes. Providing further clarity to past findings that report lower breast cancer among LDS women compared non-LDS women (Daniels, 2004), we found that within group comparisons of breast cancer diagnoses revealed no meaningful difference based upon church activity. These new results, when combined with previous studies, may point to a line of demarcation as to where the Word of Wisdom did influence health, and where other factors such as identity and social support play a more meaningful role.

Among active Latter-day Saint men, those born post-1900 displayed a 12% greater risk of prostate cancer compared to inactive Latter-day Saint men-- a difference not found in pre-1900 birth cohorts. The age-dependent nature of prostate cancer diagnosis helps explain these results and offers confirmation to the hypotheses. As lower risk of tobacco related deaths among active LDS males contributed to higher life expectancy compared to inactive males, the exposure time for prostate cancer increased in tandem (*appendix: Table A3, Figure A1*). In sum, the total evidence suggests that those who obeyed tobacco prohibitions of the LDS church lived long enough to be diagnosed with prostate cancer late in life.

Limitations and Future Directions

Limitations include the questionable external validity of our results to other religions. We do not argue that religion in every circumstance improves mortality, nor specifically does the LDS faith. As such, further examinations within the UPDB considering LDS policies of fertility, marriage, sexual morality, and economics may yield positive and negative health outcomes. Previous studies reporting negative health aspects among LDS people support this approach (Bodson et al., 2017; Merrill & Thygeson, 2001) Replication studies to health outcomes among groups outside of the LDS faith may show differing relations between religion and health.

We also note measurement error in LDS activity given that a single timepoint of endowment participation does not ensure a lifetime of LDS church activity. New research might glean additional timepoints of participation from linked offspring's baptism and endowment dates. Additionally, a network analysis of sibling's, cousins, and parental temple participation might offer a clearer picture of family faith dynamics.

Our focus upon tobacco allows for similar investigations of alcohol concerning life expectancy and digestive tract diseases. We also did not consider socioeconomic status as smoking uptake during the time of the policy was sign of modernism and spreading to all levels of society (Brandt, 2007: pg. 61; Gardner, 2013; Gershon, 2022). But economic circumstance may have application in survival given a cancer diagnosis. As such, further research analyzing hazard rate ratio differences between tobacco-related cancer and tobacco related deaths by religion or socio-economic status may prove useful. Finally, although demographic theory holds decreased mortality at older ages partially determines population expansion, its contribution is relatively small compared to mortality reductions in pre-fertile ages. Therefore, while the Word of Wisdom likely did not alter Utah's population numbers in a substantial manner, these results do suggest that religion can support demographic expansion through mortality reductions.

Conclusions

Despite our highly consistent results, we cannot claim with certainty that the 1921 policy caused believing Latter-day Saints to forgo tobacco. While our results provide clear support for the hypothesis that the 1921 policy initiated a behavioral boundary followed by the faithful, the possibility remains that the WoW drove smoking inclined youth away from the church who may have remained in absence of the ban. The extent to which this rival explanation seems plausible would require additional work on rates of change in LDS membership over time. We, however, note descriptively that LDS membership only increases post-WoW policy (Bennion & Young, 1996).

An exploration of the Word of Wisdom as policy must recognize its historical context. The 1921 policy preceded scientific evidence regarding the harms of tobacco by almost 30 years. Intriguingly, this circumstance likely indicates that this policy had no explicit goal to improve mortality among LDS members. Thus, the nature of this research suggests that considerations of emergent beliefs and practices as specific health policies could strengthen arguments in favor of religion as a social determinant of health (VanderWeele & Chen, 2020). We encourage other research using both historical and contemporary cohorts to further illuminate the extent to which religious policies—deliberately or inadvertently—could affect population health.

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Table 1. Descriptive population characteristics of Utah births, 1880 to 1920, who also died in Utah, according to LDS church activity.

	All (n = 138739)		Inactive LDS (n = 33569)		Active LDS (n = 105170)		
	<u>N</u>	<u>%</u>	<u>N</u>	<u>%</u>	<u>N</u>	<u>%</u>	
Sex							
	Female	70317	50.68%	16513	49.19%	54804	52.11%
	Male	68422	49.32%	17056	50.81%	51366	48.84%
Birth cohort							
	1880 to 1899	53931	38.87%	8532	25.42%	45399	43.17%
	1900 to 1920	84808	61.13%	25037	74.58%	59771	56.83%
Birth Cohort Sex Ratio							
	Below 1.02	19197	13.84%	4403	13.12%	14794	14.07%
	1.02 to 1.03	37764	27.22%	9549	28.45%	28215	26.83%
	1.04 to 1.05	27675	19.95%	7917	23.58%	19758	18.79%

	1.06	18520	13.35%	3734	11.12%	14786	14.06%
	1.07 and above	35583	25.65%	7966	23.73%	17617	16.75%
Urban or rural county born							
	Urban	76633	55.24%	18147	54.06%	58486	55.61%
	Rural	62106	44.76%	15422	45.94%	46684	44.39%

Table 2. Descriptive population characteristics of Utah births, 1880 to 1920, who also died in Utah, according to LDS church activity and status of tobacco related death or lung/bronchus cancer diagnosis.

	All (n = 138739)		Inactive LDS (n = 33569)		Active LDS (n = 105170)		
	N	%	N	%	N	%	
Those who died from a tobacco related death							
N	3487		1643		1844		
Sex							
	Female	867	24.86%	400	24.35%	467	25.33%
	Male	2620	75.14%	1243	75.65%	1377	74.67%
Birth cohort							
	1880 to 1899	802	23.00%	207	12.60%	595	32.27%
	1900 to 1920	2685	77.00%	1436	87.40%	1249	67.73%
Those who died without a tobacco related death							
N	134863		31845		103018		
Sex							
	Female	69264	51.36%	16075	50.48%	53189	51.63%
	Male	65599	48.64%	15770	49.52%	49829	48.37%
Birth cohort							
	1880 to 1899	52869	39.20%	8293	26.04%	44576	43.27%
	1900 to 1920	81994	60.80%	23552	73.96%	58442	56.73%
Those with a lung or bronchus cancer diagnosis							
N	1992		961		1031		
Sex							
	Female	509	26.28%	212	22.06%	297	28.81%
	Male	1483	76.56%	749	77.94%	724	70.22%
Birth cohort							
	1880 to 1899	357	17.92%	99	10.30%	258	25.02%
	1900 to 1920	1635	82.08%	862	89.70%	773	74.98%
Those without a lung or bronchus cancer diagnosis							
N	136747		32608		104139		
Sex							
	Female	69808	51.05%	16301	49.99%	53507	51.38%
	Male	66939	48.95%	16307	50.01%	50632	48.62%
Birth cohort							
	1880 to 1899	53574	39.18%	8433	25.86%	45141	43.35%
	1900 to 1920	83173	60.82%	24175	74.14%	58998	56.65%

Table 2. Estimated regression coefficients for life expectancy after age 50 as a function of birth cohort sex ratio (SSR), individual participation in the LDS Church, and the interaction between LDS membership and cohort indicator of exposure to LDS tobacco policy.

Variable	Female (n=70317)		
	Model 1	Model 2	Model 3
Constant	29.582*** (0.067)	28.112*** (1.633)	31.012*** (1.640)
Born between 1900 to 1920 (a)	2.753*** (0.086)	2.848*** (0.088)	-0.124 (0.202)
Birth cohort sex ratio ¹		0.001 (.016)	0.004 (0.015)
Urban born		-0.308** (0.084)	-0.328*** (0.084)
Active LDS member (b)		0.598*** (0.010)	-2.038*** (0.190)
Interaction between a and b			3.644*** (0.223)

Variable	Male (n = 68422)		
	Model 1	Model 2	Model 3
Constant	25.269*** (0.070)	26.096*** (1.711)	30.250*** (1.714)
Born between 1900 to 1920 (a)	2.072*** (0.090)	2.416*** (0.092)	-1.689*** (0.198)
Birth cohort sex ratio ¹		-0.029 (0.016)	-0.040* (0.016)
Urban born		-0.301** (0.088)	-0.328** (0.088)
Active LDS member (b)		2.870*** (0.102)	-0.749*** (0.185)
Interaction between a and b			5.177*** (0.222)

*P = <.05, ** P = <.001, *** P = <.0001

¹ Birth sex ratios scaled to .01

Table 4. Estimated hazard rate coefficients for tobacco related death as a function of the individuals birth year's relation to the LDS tobacco policy changes, and individual participation in the LDS Church.

Variable	Female (n = 70,317)					
	Model 1		Model 2		Model 3	
	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)
Born between 1900 to 1920 (a)	0.862*** (0.08874)	2.367 (1.989 - 2.817)	0.715*** (0.091)	2.045 (1.712 - 2.442)	1.433*** (0.200)	4.191 (2.832 - 6.203)
Birth cohort sex ratio ¹			-.001 (.013)	0.999 (0.974 - 1.025)	-.012 (.013)	0.988 (0.963 - 1.014)
Urban born			-0.004 (0.068)	0.996 (0.871 - 1.139)	0.001 (0.068)	1.001 (0.875 - 1.144)
Active LDS member (b)			-0.976*** (0.069)	0.377 (0.329 - 0.431)	-0.114 (0.212)	0.892 (0.589 - 1.352)
Interaction between a and b					-1.000*** (0.225)	0.368 (0.237 - 0.572)
Variable	Male (n = 68,422)					
	Model 1		Model 2		Model 3	
	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)
Born between 1900 to 1920 (a)	0.507** (0.045)	1.660 (1.519 - 1.814)	0.423*** (0.046)	1.527 (1.394 - 1.672)	0.859*** (0.081)	2.361 (2.014 - 2.767)
Birth cohort sex ratio ¹			2.271* (0.763)	9.69 (2.171 - 43.251)	2.376* (0.764)	10.759 (2.406 - 48.116)
Urban born			-0.086* (0.039)	0.917 (0.849 - 0.991)	-0.080* (0.040)	0.923 (0.854 - 0.998)
Active LDS member (b)			-1.193*** (0.040)	0.303 (0.281 - 0.328)	-0.652*** (0.088)	0.521 (0.438 - 0.619)
Interaction between a and b					-0.700*** (0.099)	0.497 (0.409 - 0.602)

*P<.05, **P = <.001, *** P = <.0001

¹ Birth sex ratios scaled to .01

Table 5. Estimated hazard rate coefficients for female lung and breast cancer diagnoses as a function of the individuals birth year's relation to the LDS tobacco policy changes, and individual participation in the LDS Church.

Variable	Lung Cancer Diagnoses					
	Model 1		Model 2		Model 3	
	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)
Born between 1900 to 1920 (a)	1.114** (0.124)	3.048 (2.388 - 3.890)	1.033** (0.127)	2.810 (2.192 - 3.603)	2.246*** (0.387)	9.450 (4.426– 20.177)
Birth cohort sex ratio ¹			0.017 (.017)	1.017 (0.983 – 1.052)	0.018 (0.018)	1.018 (0.983 – 1.055)
Urban born			0.106 (0.089)	1.112 (0.935 - 1.324)	0.111 (0.089)	1.118 (0.939 - 1.331)
Active LDS member (b)			-0.749** (0.091)	0.473 (0.396 - 0.565)	0.629 (0.397)	1.875 (0.861 – 4.084)
Interaction between a and b					-1.516** (0.408)	0.219 (0.098- 0.489)
Variable	Breast Cancer Diagnoses					
	Model 1		Model 2		Model 3	
	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)
Born between 1900 to 1920 (a)	0.897** (0.039)	2.453 (2.274 - 2.646)	0.913** (0.039)	2.492 (2.308 - 2.692)	0.929** (0.095)	2.547 (2.110 - 3.075)
Birth cohort sex ratio ¹			0.011 (0.006)	1.011 (0.999 – 1.023)	0.011 (0.006)	1.011 (0.999 – 1.023)
Urban born			0.078* (0.030)	1.081 (1.019 - 1.147)	0.078* (0.030)	1.081 (1.019 - 1.147)
Active LDS member (b)			0.027 (0.035)	1.028 (0.960 - 1.101)	0.044 (0.097)	1.045 (0.864 - 1.264)
Interaction between a and b					-0.019 (0.104)	0.981 (0.800 - 1.203)

* P <.05, ** P <.001, ***P < .0001

¹ Birth sex ratios scaled to .01

Table 6. Estimated hazard rate coefficients for male lung/bronchus and prostate cancer diagnoses as a function of the individuals birth year's relation to the LDS tobacco policy changes, and individual participation in the LDS Church.

Variable	Lung/Bronchus Cancer Diagnoses					
	Model 1		Model 2		Model 3	
	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)
Born between 1900 to 1920 (a)	0.881*** (0.066)	2.414 (2.120 - 2.749)	0.819*** (0.067)	2.268 (1.986 - 2.590)	1.110*** (0.112)	3.034 (2.866 - 3.213)
Birth cohort sex ratio ¹			.050*** (.010)	1.051 (1.031 – 1.072)	.051*** (.010)	1.052 (1.031 – 1.073)
Urban born			-0.025 (0.052)	0.976 (0.881 - 1.081)	-0.022 (0.052)	0.979 (0.883 - 1.084)
Active LDS member (b)			-1.240*** (0.052)	0.289 (0.261 - 0.321)	-0.844*** (0.127)	0.430 (0.335 - 0.552)
Interaction between a and b					-0.484** (0.140)	0.616 (0.468 - 0.811)
Variable	Prostate Cancer Diagnoses					
	Model 1		Model 2		Model 3	
	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)	Coefficient (SE)	Hazard Ratio (95% CI)
Born between 1900 to 1920 (a)	0.791*** (0.029)	2.205 (2.081 - 2.335)	0.802*** (0.030)	2.104 (2.104- 2.365)	0.700*** (0.070)	2.014 (1.756 - 2.310)
Birth cohort sex ratio ¹			.004 (.005)	1.005 (0.995 – 1.015)	.004 (.005)	1.005 (0.995 – 1.015)
Urban born			-0.012 (0.024)	0.989 (0.943 - 1.036)	-0.012 (0.024)	0.988 (0.942 - 1.035)
Active LDS member (b)			0.104** (0.030)	1.110 (1.047 - 1.176)	0.002 (0.070)	1.002 (0.874 - 1.149)
Interaction between a and b					0.123 (0.077)	1.130 (0.972 - 1.315)

* P <.05, ** P <.001, ***P < .0001

¹ Birth sex ratios scaled to .01

Figure 1. Descriptive charts of Lung and Bronchus, Colorectal, Breast, and Prostate cancers.

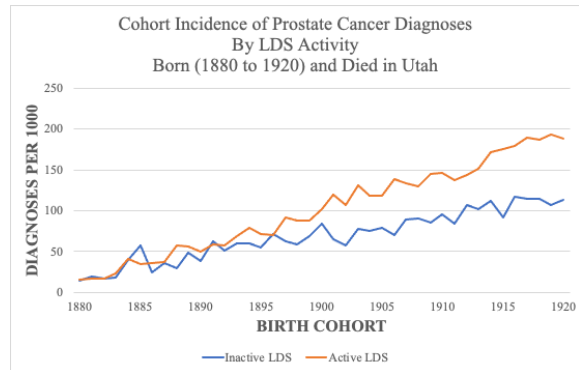
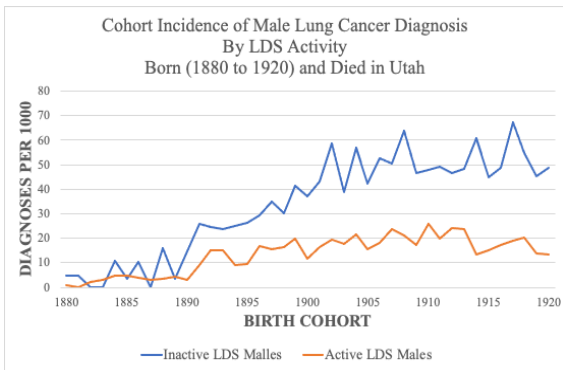
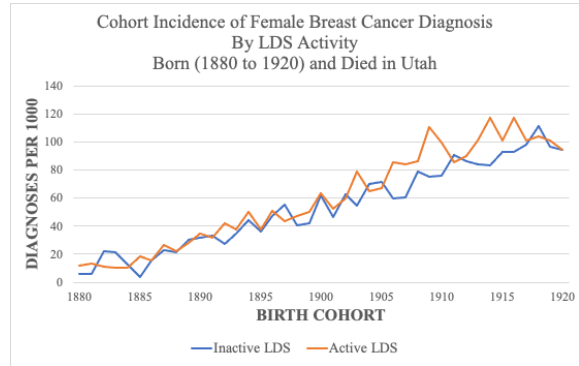
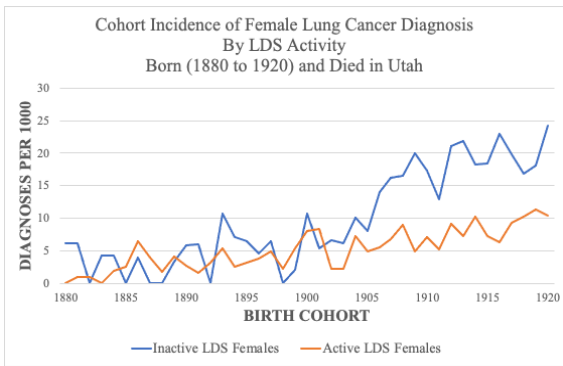
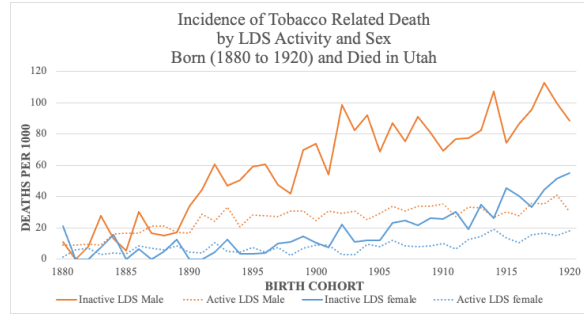
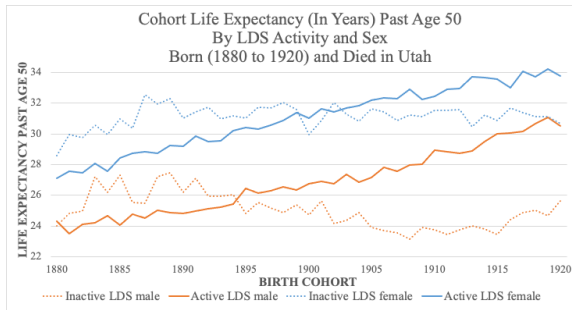
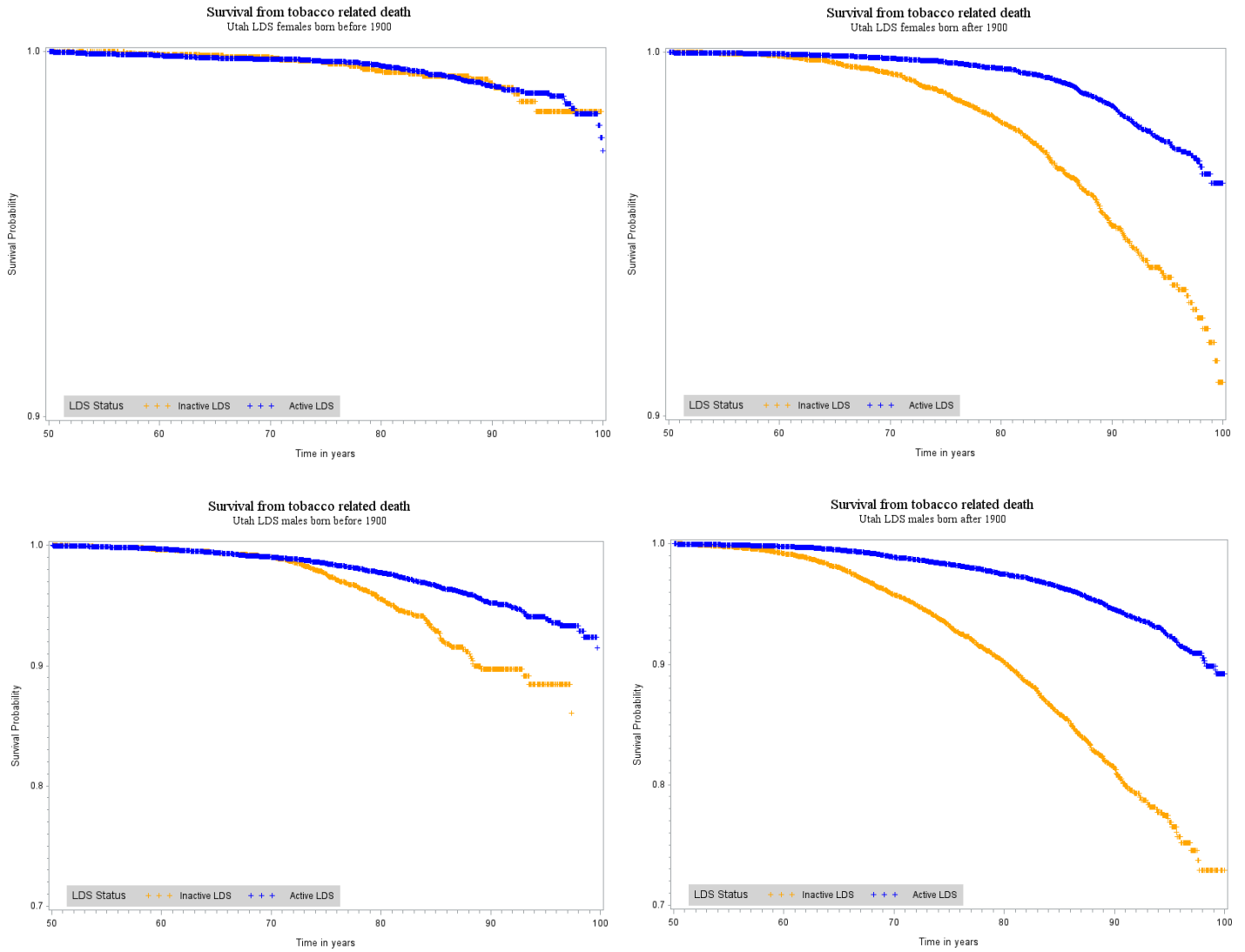


Figure 3. Survival plots, risk of tobacco related death, by sex and LDS activity status. Females appear in the top row, and males appear in the bottom row.



Online Appendix

Demographic Expansion in Utah: Religion, Policy, and Mortality

Jason P. Bonham, Ken R. Smith, Tim A. Bruckner

Supplementary Tables and Figures.

Table A1. Distribution of age at prostate cancer diagnosis by LDS activity level

	Active LDS Men	Inactive LDS Men
100% Max	101	102
99%	94	93
95%	89	88
90%	86	86
75% Q3	82	81
50% Median	77	76
25% Q1	72	71
10%	67	65
5%	64	62
1%	58	57
0% Min	39	47

Table A2. ICD Codes by disease group and version.

Disease Group	ICD Version: Code
Esophagus	ICD 6: 150 ICD 7: 150 ICD 8: 150 ICD 9: 150, 150.0, 150.1, 150.2, 150.3, 150.4, 150.5, 150.8, 150.9 ICD 10: C15, C15.0, C15.1, C15.2, C15.3, C15.4, C15.5, C15.8, C15.8, C15.9
Trachea/Bronchus/Lung	ICD 6: 162, 163 ICD 7: 162, 162.1, 162.0, 162.1, 162.8 ICD 8: 162, 162.0, 162.1, 162.8 ICD 9: 162, 162.0, 162.2, 162.3, 162.4, 162.5, 162.8, 162.9 ICD 10: C33, C34, C34.0, C34.1, C34.2, C34.3, C34.8, C34.9
COPD	ICD 8: 490, 491, 492 ICD 9: 491, 491.0, 491.1, 491.2, 491.20, 491.21, 491.8, 491.9, 492, 492.0, 492.8 ICD10: J40, J41.0, J41.1, J41.8, J42, J43, J43.0, J43.1, J43.8, J43.9, J44, J44.0, J44.1, J44.8, J44.9

Figure A1.

