

Early Life Conditions and Later Life (65+) Co-Morbidity Trajectories: The Utah Population Database Linked to Medicare Claims Data

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Abstract

We examine the association between one of the earliest life conditions, one's family history of longevity. Family longevity history is often used to predict an individual's mortality risk but here we consider co-morbidity dynamics using the Charlson Co-Morbidity Index, a construct that summarizes nearly all serious illnesses afflicting older individuals. We identify distinctive co-morbidity trajectories (e.g., some individuals remain healthy through their older years while others have rising levels of morbidity) using PROC TRAJ in SAS and then estimate how familial longevity affects an individual's chances of being a member of a trajectory type. The data used are based on linkages between the Utah Population Database and Medicare claims data that hold medical diagnoses data for the Medicare population in Utah. We show how persons with a history of advantageous familial longevity are not only living longer but are also living longer while experiencing the most healthful disease trajectories.

Introduction

There is wide variation in individual and health status over the life course. The source of this variation may be present very early in an individual's life. The goal of this analysis is to estimate the association between earlier life conditions (ELCs) and later life health based on new and extensive additions to a premier longitudinal, familial health database.

The research question we address in this analysis asks whether conditions present early in life are associated with their morbidity profile as an elder. In particular, we will examine the role of central traits and exposures when an individual was young including exposures occurring prenatally, at birth, or during childhood. These exposures include low parental SES, deaths in the family (parents, siblings), large family sizes, high birth orders, and very young and very old parental age. A trait that is frequently overlooked as the earliest of life conditions is an individual's family history of disease or longevity. **For this abstract, we report on the association between family history of longevity and co-morbidity trajectories after age 65.** In the full paper, our family history of longevity will be complemented by the inclusion of the aforementioned early exposures.

The health endpoints for this analysis are co-morbidity trajectories. These trajectories will be derived empirically and depict common health patterns that occur among older populations. In particular, some older individuals have almost no co-morbid episodes with no serious illnesses or doctor visits. Others are quite ill and continue to experience repeated illnesses that lead to frequent diagnoses or early death. Naturally, there are many other patterns and dynamics that comprise the health histories of older individuals that we will identify.

The motive for looking at co-morbid trajectories is the need to move beyond death or specific causes of death. While these outcomes are important to examine, focusing on morbidity profiles is equally critical and may shed light on mechanisms that link early conditions to delayed health effects later in life. The specific consideration of co-morbidity *per se* is based on the simple fact that older individuals face a range of health challenges and quite often multiple organ systems and pathologies are involved and they arise with increasing frequency after age 65. Our analysis of co-morbidity trajectories acknowledges this complexity and therefore attempts to address a larger range of critical medical diagnoses.

Data

This study utilizes data drawn from the Utah Population Database (UPDB). The UPDB is one of the world's richest sources of linked population-based information for demographic, genetic, and epidemiological studies. UPDB has supported biodemographic studies as well numerous important epidemiological and genetic studies in large part because of its size, pedigree complexity, and linkages to numerous data sources. The majority of life-span epidemiological studies, such as that presented here, examine health influences of ELCs with relatively modest sample sizes. The UPDB now contains data on nearly 7 million individuals due to longstanding and on-going efforts to add new sources of data and update records as they become available (e.g., including death records and Medicare claims from 2011) . We have identified hundreds of thousands of members of birth cohorts from the first half of the 20th century, individuals for whom early and midlife conditions are measured and who are linked to their adult medical records generated decades later. It is these complex data links that provide

unparalleled data quality and depth that focus on families (nuclear, multigenerational, full pedigrees) and health outcomes that span entire life spans of individuals and their relatives.

The models we estimate here are computationally intensive and the large sample sizes at our disposal present estimation challenges. Accordingly, in this abstract, we rely on selected ages to demonstrate the technique and illustrate the associations of interest. We use three specific ages in 1992, the first year in which we have Medicare data: ages 66, 76, and 86. Subjects who were eligible were then followed for 11 years (to 2002, our last year of Medicare data or until death). For males, the sample sizes for the three ages respectively are 1,805, 1,523, and 278. The comparable figures for the female samples are 1,932, 1,897, and 597. The complete analysis will examine all eligible subjects in the database.

Key Measures

The family history of longevity is measured here using Familial Excess Longevity (FEL), a statistic developed using deep genealogical data of multigenerational pedigrees drawn from the UPDB. We have published the development of this statistic [1] and have applied it to other life-span studies using UPDB [2]. At its foundation, the FEL is based on the assumption that family history of longevity follows Mendelian patterns of inheritance. To construct familial excess longevity we first measure individual level excess longevity, defined as the difference between an individual's attained age and the age to which that individual was expected to live according to a model that incorporates basic predictors (gender, birth year). Expected longevity is estimated from an accelerated failure time (AFT) model and excess longevity (l) is simply the

difference between expected and attained age. Expected longevity is based on the lognormal distribution and the AFT model was used because it provides a simple point estimate for duration and that fits the observed data. Excess longevity is then extended to blood relatives who reached the age of 65 for each individual. Averaging the excess longevity of all kin over 65 for each ego, with the appropriate weighting scheme, generates a point estimate of familial excess longevity. The kinship coefficient, the probability that an individual shares a particular allele with another individual, is used as a weight in calculating familial excess longevity.

We are able to observe morbidity episodes from Medicare claims collected over time for each individual. There are groups of individuals in a population with similar trajectories with respect to their morbidity patterns. These clusters of individuals can then be identified by their shared health experiences over time as measured by the Charlson comorbidity index which, in its original form, was created using clinical records [3]. The Charlson index was adapted for use with ICD-9 codes by Deyo et al [4] and Romano et al [5]. Deyo et al adapted the index for use with ICD-9 diagnosis and procedure codes. Romano et al included some diagnoses that were not in the original Charlson index. Both modifications were intended for use with the Medicare Part A records [6]. Klabunde and colleagues [7] created two indices, one for Medicare Part A records and one for Medicare Part B records. Introducing information from physician claims data significantly enhanced the index's predictive value for the risk of mortality. In the present study, we have adopted this variant of the Charlson co-morbidity index based on the SEER-Medicare co-morbidity SAS macros. A second SEER-Medicare macro calculates the co-morbidity index with respect to cancer based on the Deyo adaptation of Charlson co-morbidity index. Given that cancer originally was the index disease, it was not included as a co-morbid condition in this SEER-Medicare program. The Deyo version of the Charlson index uses ICD-9-

CM codes and two classifications not included in the Deyo version are cancer and metastatic carcinoma. They were not included because this macro is assuming that co-morbidities are also relative to cancer. Accordingly, we have added cancer and as a co-morbid disease. We can identify specific episodes of the following 17 major morbidities conditions occurring during the interval 1992-2002 that form the basis of the Charlson Co-Morbidity Index:

1. Myocardial Infarction
2. Congestive Heart Failure
3. Peripheral Vascular Disease
4. Cerebrovascular disease
5. Dementia
6. Chronic pulmonary disease
7. Rheumatologic disease
8. Peptic Ulcer Disease
9. Mild Liver Disease
10. Diabetes (mild to moderate)
11. Diabetes with chronic complications
12. Hemiplegia or paraplegia
13. Renal (kidney) disease
14. Any malignancy
15. Moderate or severe liver disease
16. Metastatic Solid Tumor
17. AIDS

We seek to determine how familial longevity affects the likelihood of having a particular later life morbidity trajectory. Assessment of co-morbidity trajectories are accomplished through the application of a finite mixture modeling approach that is currently available as a SAS procedure, called PROC TRAJ through the work of Dr. Daniel Nagin and his colleagues [8-10]. The group-based modeling approach for determining health trajectories has advantages over other approaches of developmental modeling, such as growth-curve modeling. Notably, growth-curve modeling requires that growth be determined by a mean pattern of increase or decline, with deviations from the mean being explained by selected determinants. This assumption does not seem to fit the health experiences we are considering as outcomes in the current project, which involves distinctive qualitative groupings that may not be linearly related. Moreover, growth-curve modeling is ill suited for dealing with mortality since the probability of death is not randomly distributed across morbidity trajectories. PROC TRAJ provides a mechanism to account for mortality.

Results

We have tested our models based on samples from the UPDB where we have both a measure of familial longevity and linked Medicare data from which to determine trajectories. For this abstract, we estimate models for those who were aged 66, 76, and 86 in 1992. The models are estimated by gender.

In Figures 1 and 2, we find that the best fitting models (based on BIC criteria) for females (Figure 1) is one that contained seven trajectories and for men (Figure 2) five. The vertical axis for all figures is the Charlson co-morbidity index where higher numbers are associated with more co-morbidities. The horizontal axes identify the number of years since 1992. In Figure 1, there are Trajectories 1, 2, and 3 that comprise 37%, 11% and 15%, respectively (see legend in Figure 1) and all begin with nearly zero levels of co-morbidities but then group 2 rises in the later years. All of the figures will have similar appearances in that there are 3-7 distinct groups that are detected using PROC TRAJ. Trajectories 5 and 6 begin the interval with higher levels of co-morbidities which then increase with time, especially group 6 although this group includes very few subjects (<2%). The central point of Figure 1 is that for those with the most favorable family history of longevity, they are the least likely to be represented in trajectories that generally have the most disadvantageous patterns (see inset on hazard rate ratios).

Figures 1 and 2 here

For Figure 2 (males), we observe similar patterns (though there is one less trajectory group). Here, Trajectory 5, the group with the steepest increase in co-morbidities, is the least likely to be represented with persons with a strong family history of longevity.

Figures 3 and 4 repeat the analyses for 76 year olds. The male results in Figure 4 again show the benefits of a favorable family longevity history. Inexplicably, the female findings in Figure 3 show (three) distinct trajectories but the influence of family longevity history is not found as it was for females in the 66 year old analysis.

Figures 3 and 4 here

Finally, the patterns for the 86 year olds are, perhaps not surprisingly, more complex. For females (Figure 5), the weak influence of family longevity history persists at this more advanced age. More analysis will need to be conducted to explore this apparent sex-specific finding. For males (Figure 6), we again detect that the least desirable trajectory is associated with those with the most favorable familial longevity but we now see Trajectory 2 (rising co-morbidities) is associated with a beneficial longevity background.

Figures 5 and 6 here

Conclusion

These analyses represent our initial efforts to estimate the association between early life conditions with later life morbidity trajectories, an advance over outcomes focused only on mortality or a single morbid outcome. These models will be expanded by the inclusion of additional measures of early life circumstances that are measured in the UPDB. In general, these analyses illustrate how persons, particularly men, benefitted by a history of advantageous familial longevity appear to be more likely to live their later years not only living longer but also living longer healthier.

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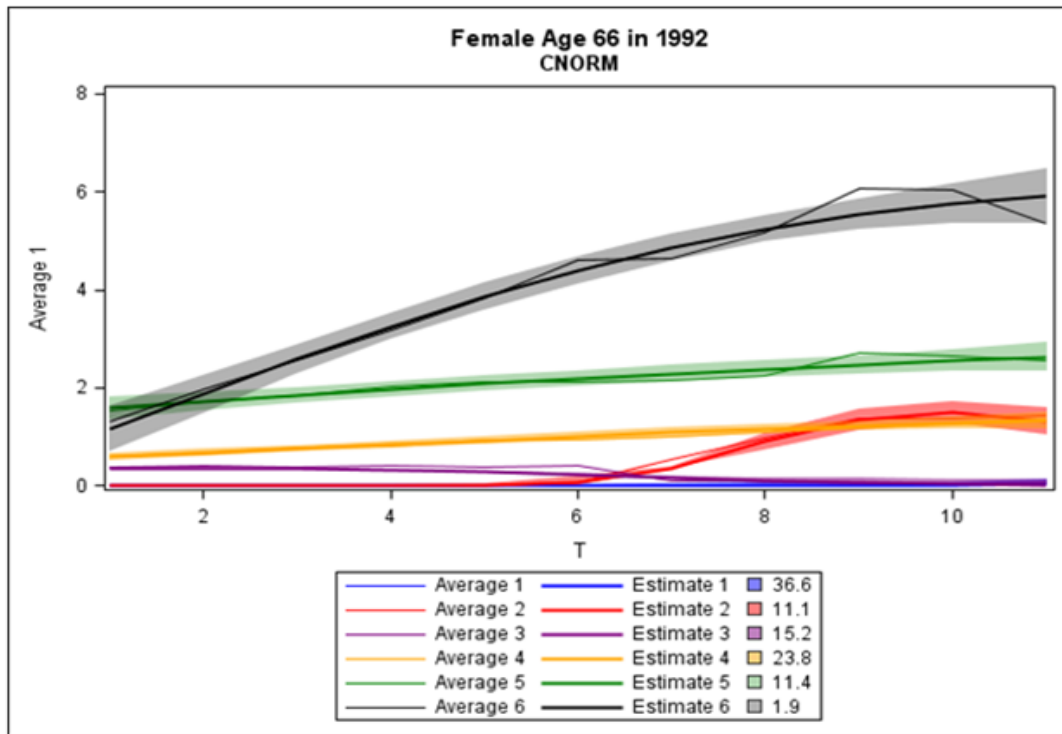
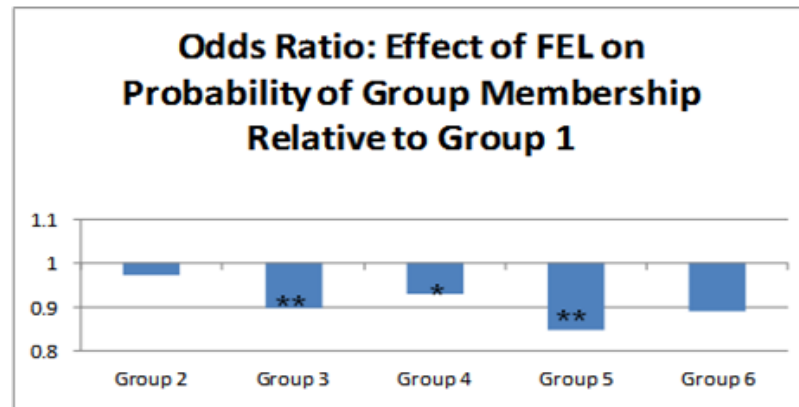


FIGURE 1



* p<0.1
 ** p<0.05
 *** p<0.0001

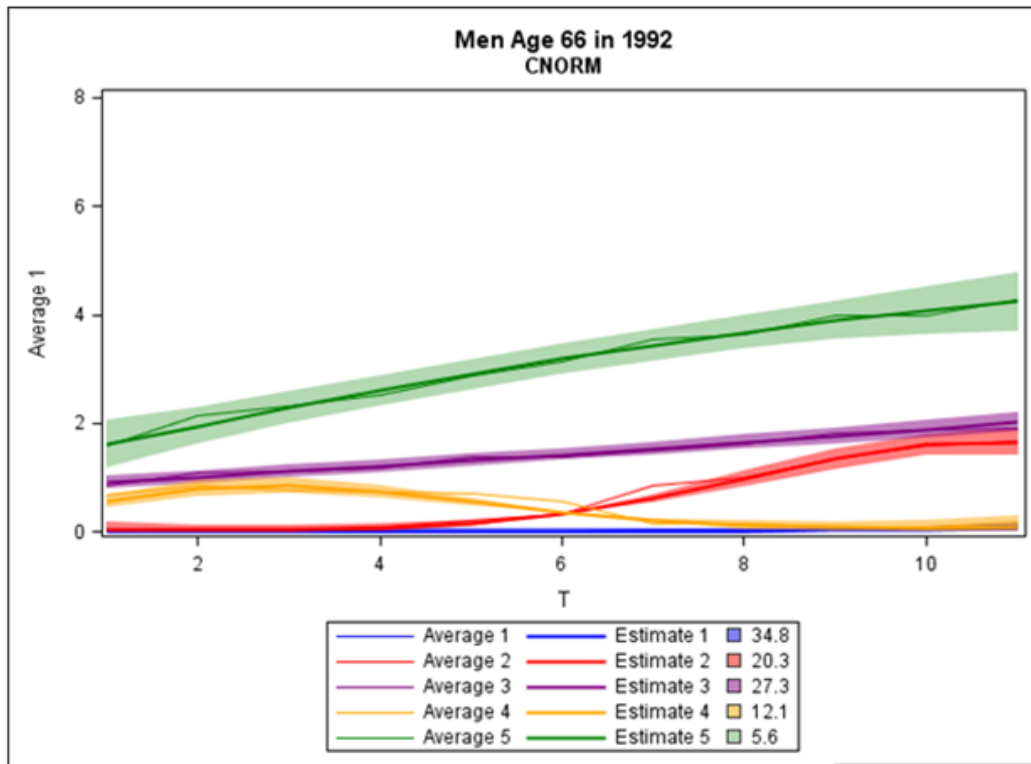
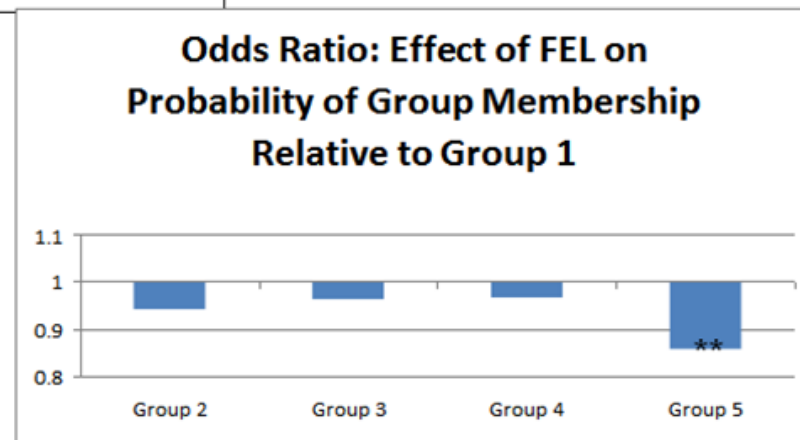


FIGURE 2

* $p < 0.1$
 ** $p < 0.05$
 *** $p < 0.0001$



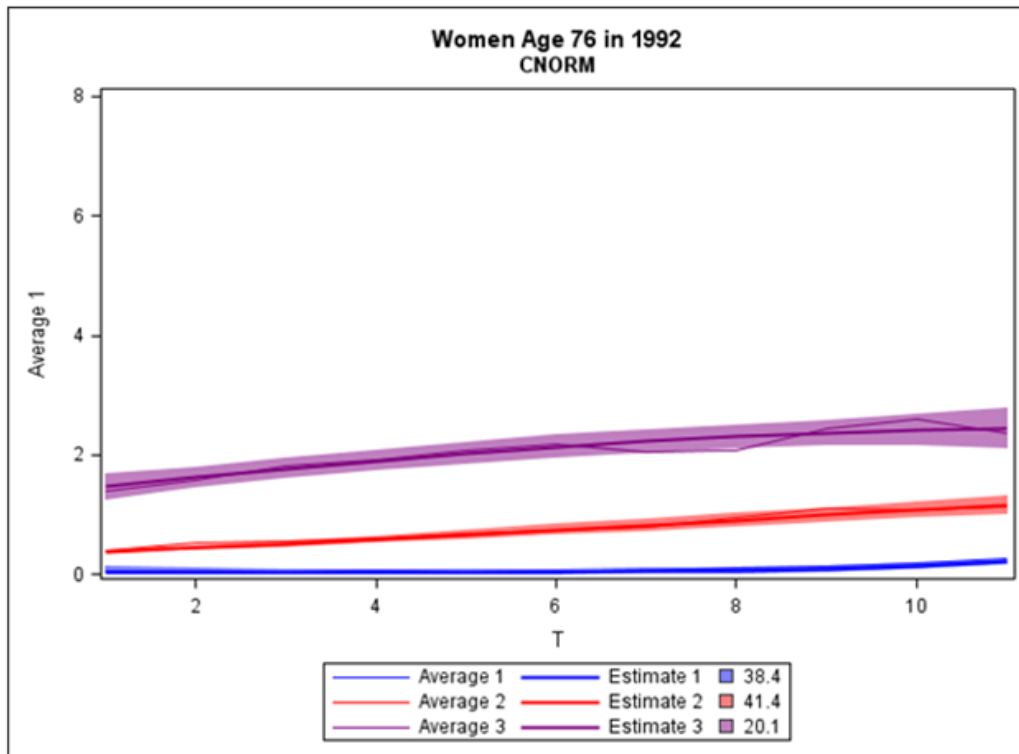
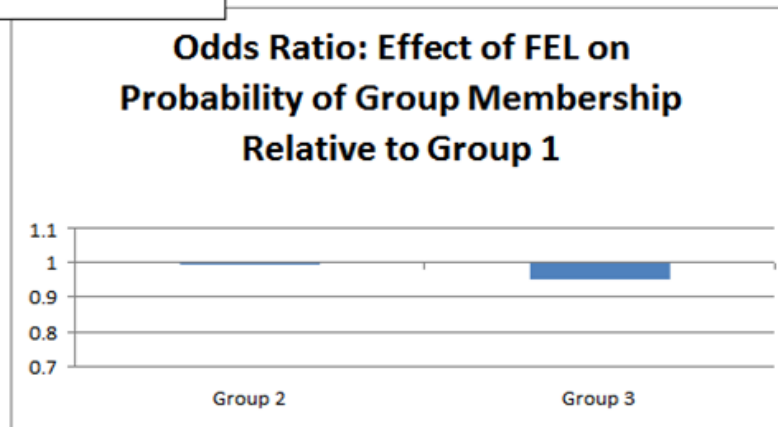


FIGURE 3



* p<0.1
 ** p<0.05
 *** p<0.0001

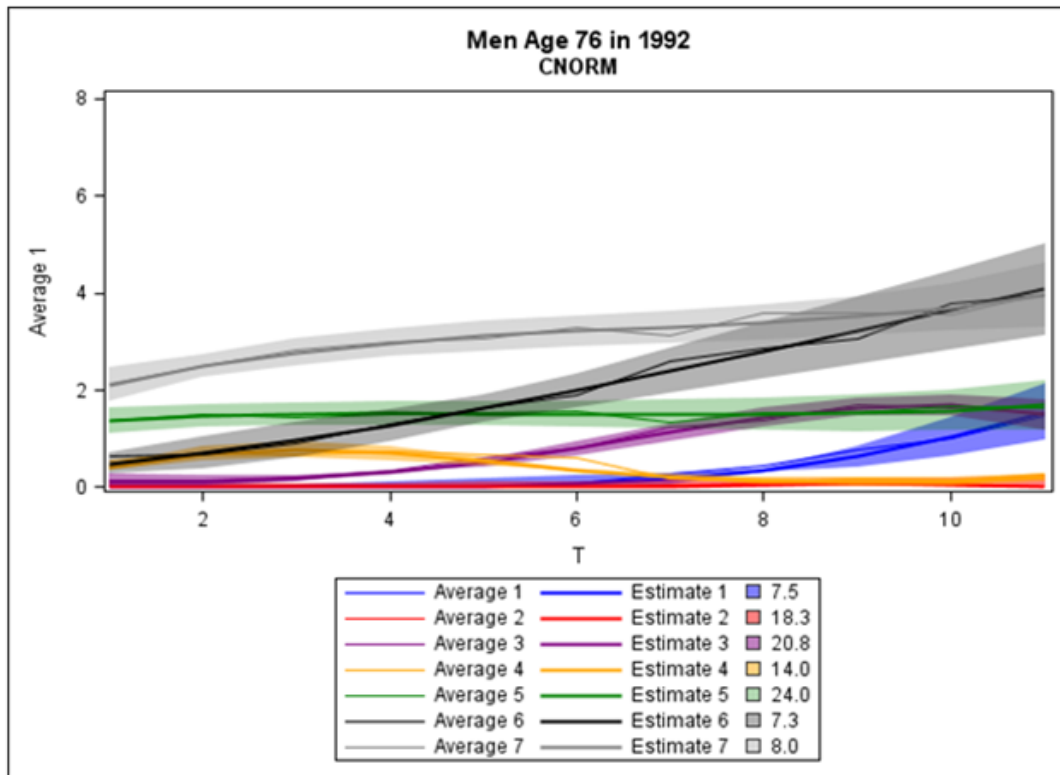
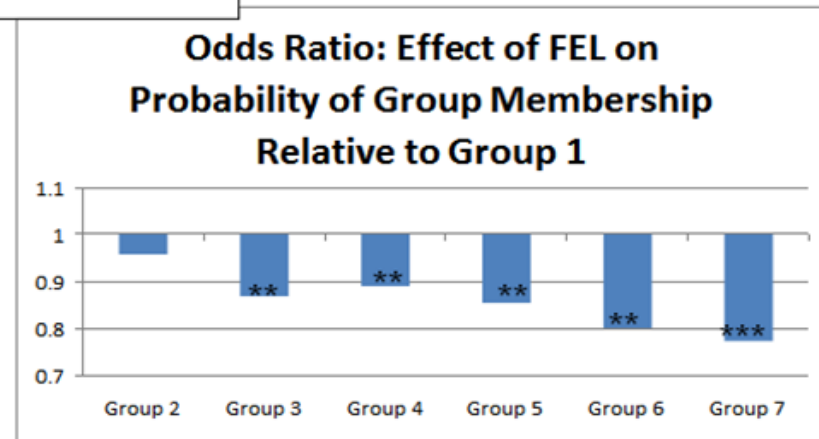


FIGURE 4

* $p < 0.1$
 ** $p < 0.05$
 *** $p < 0.0001$



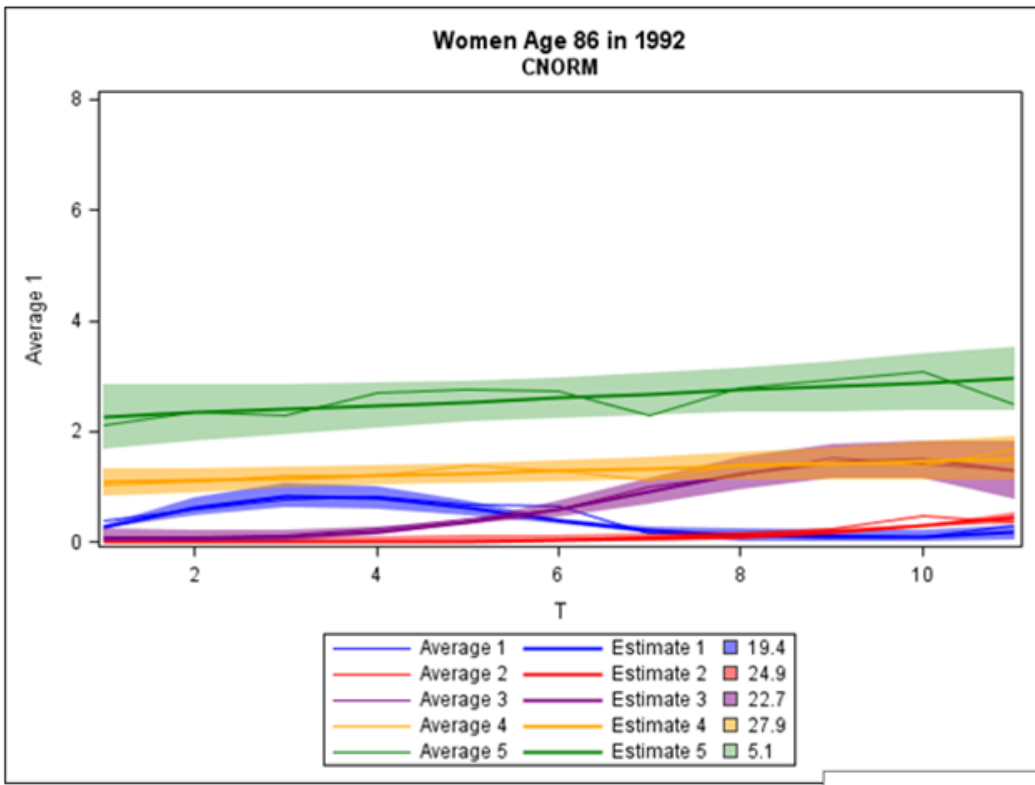
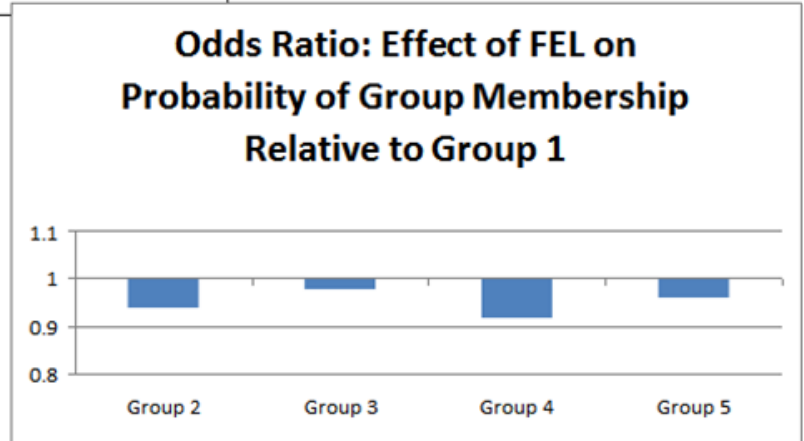


FIGURE 5



* p<0.1
 ** p<0.05
 *** p<0.0001

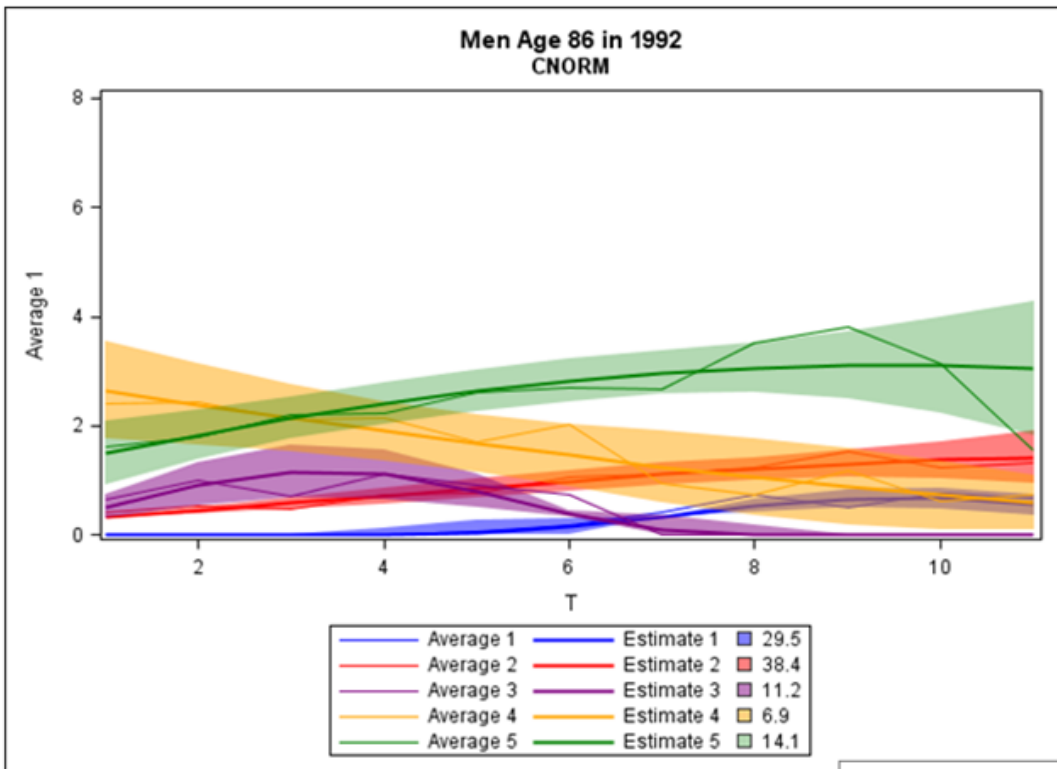


FIGURE 6

* p<0.1
 ** p<0.05
 *** p<0.0001

