Intergenerational Correlation in Mortality: The Role of Chronic Conditions

Dan Zeltzer, Liran Einav, Joseph Rashba,

Nicholas Scott-Hearn, Ran Balicer*

July 22, 2024
PRELIMINARY - DO NOT CITE

Abstract

This paper examines the contribution of intergenerational transmission of chronic conditions to the established yet under-explored correlation in mortality across generations. Utilizing comprehensive administrative claims data from Israel's largest healthcare system, we analyze the morbidity and mortality of individuals and their parents. We first estimate the elevated morbidity hazard associated with a family history of medical conditions, and the subsequent elevation in mortality hazard following the onset of each condition. We then embed these estimates into a model in order to quantify the contribution of various chronic conditions to the overall intergenerational correlation in mortality. Results show that parental history of common chronic conditions can decrease life expectancy by several months. The intergenerational transmission of these conditions accounts for about 10% of the intergenerational correlation in mortality.

JEL Code: I11.
Keywords: health mobility, chronic conditions, life expectancy

*Zeltzer (corresponding author): Tel Aviv University and Stanford University dzeltzer@tauex.tau.ac.il; Einav: Stanford University and NBER. Rashba: Tel Aviv University and Clalit Health Services. Scott-Hearn: Stanford University. Balicer: Clalit Health Services and Ben Gurion University. We thank ASHEcon participants for helpful comments.

1 Introduction

The persistence of health and health inequality across generations has long been a focus of economics research (Black et al., 2011; Currie and Moretti, 2007; Currie, 2011; Lu and Vogl, 2023). The length of life, perhaps the most fundamental measure of health, is well known to be significantly correlated across generations (Beeton and Pearson, 1901; Pearl, 1931; Black et al., 2023). However, the underlying drivers of this correlation in mortality remain largely unexplored. Understanding the role of family histories in shaping individual health outcomes is important for understanding the fundamental aspects of inequality and social mobility.

In this study, we take advantage of a unique data opportunity from Israel in order to quantify the role of chronic conditions in the intergenerational correlation in mortality. The analysis is facilitated by linking detailed morbidity patterns of a large, diverse population from administrative healthcare records with vital statistics data on mortality. Existing work on intergenerational correlation in longevity traditionally uses data on linked generations that lacks morbidity information. Our key contribution lies in linking morbidity and mortality records across generations, which allows for novel insights into how intergenerational correlations in morbidity and mortality are related.

We analyze detailed and comprehensive medical data covering about half of the Israeli population from 2008–2017. The data come from Clalit Health Services, the largest of four non-profit health maintenance organizations (HMOs) in Israel that provide all residents universal, tax-funded health insurance. The data include detailed chronic condition information and date of death. We link these data across generations based on information from the population registry on family relations.

The longitudinal nature of the data provides an average observation period of several years per individual, allowing us to track their health trajectories, including the onset of a rich set of chronic conditions and, in some cases, mortality. Utilizing this dataset, we observe two critical risk processes: the age-specific incidence of different chronic conditions for individuals with and without a family history of the condition and the age-specific mortality rates for individuals with and without each condition.

To quantify the contribution of chronic conditions to the intergenerational correlation in mortality, we integrate morbidity and mortality hazard estimates into a lifecycle model. We use the descriptive evidence to parameterize the model, estimate its parameters, and then use the estimated model to quantify the intergenerational correlation in mortality that is attributable to chronic conditions and to assess the importance of each condition to this overall correlation.

Our results indicate that cardiovascular and endocrine conditions are the strongest con-

tributors to intergenerational correlation in mortality. Individuals with a family history of these conditions have life expectancies shorter by 0.45 and 0.3 years, respectively. Overall, chronic conditions predict the intergenerational correlation in mortality to be 0.016, about 11% to 17% of the total intergenerational correlation in mortality, which a rich existing literature estimates to be between 0.09 and 0.14. The contribution is primarily due to conditions that are common, significantly elevate mortality risk, and are transmissible across generations.

Our study contributes to the broader understanding of intergenerational health mobility. It adds to a growing body of recent literature focusing on intergenerational mobility in different aspects of health. (Johnston et al., 2013) documents intergenerational correlation in mental health... (Halliday et al., 2021) in self-reported health status... (Thompson, 2014, 2017; Jia et al., 2019) aims to separate nature from nurture...

It also adds to a demographic literature that explores inherited drivers of morbidity (Mahmood et al., 2014; Polubriaginof et al., 2018) and mortality (Vaupel, 1988; Ruby et al., 2018; Slagboom et al., 2018; Christensen et al., 2020).

The rest of this paper continues as follows. Section 2 describes the data and presents descriptive patterns, Section 3 lays out the lifecycle model and its estimation, and Section 4 reports the main results. The last section concludes.

2 Data and descriptive evidence

2.1 Data source

Our data are sourced from Clalit Health Services (in short, Clalit), the largest among Israel's four non-profit health maintenance organizations (HMOs) that provide universal, mandatory, tax-funded healthcare coverage to all Israeli residents from birth, in accordance with the National Health Insurance Law (1995). Under Israeli health insurance, covered services are fully funded by government capitated payments.

The data cover a large and stable population. Clalit serves over half of the Israeli population, with approximately 5 million enrollees at the end of our sample period. All four HMOs offer identical coverage and share the same hospital networks but have distinct outpatient provider networks. Individuals can switch HMOs twice a year while retaining their universal coverage. However, the annual switching rate is remarkably low (around 1%), and newborns are assigned to their mother's HMO by default. Thus, most individuals remain enrolled with Clalit throughout their lifetime, and it is common for children and parents to be enrolled with the same HMO.

The data provide a comprehensive record of the population's chronic conditions. It has three main advantages in that respect. First, Electronic Health Record (EHR) systems were adopted well before the start of our study period, so data quality is generally high. Second, by law, Clalit and all other HMOs maintain a registry of over one hundred conditions for care management and reimbursement purposes (for example, certain chronic conditions qualify for a different maximum out-of-pocket cap on drug spending). If individuals switch insurers, the law mandates transferring their chronic condition registry records, so even new enrollees have a record of any previously-diagnosed chronic conditions. Third, under-diagnosis is likely minimal, thanks to universal coverage and free primary care.

We augment these data with detailed and complete vital statistics data on mortality and family links, sourced from the Israeli population registry and available to us through Clalit. We observe birth and death dates for any individual who has been a Clalit enrollee at some point of their life. These data also include the identification numbers of all children of Clalit enrollees, regardless of the child's current coverage status. We use these unique identifiers to link Clalit enrollees to their children, when the latter are also Clalit enrollees.

2.2 Sample construction and key variables

Sample We begin by sampling all Clalit enrollees who are 30 years old or older with at least 12 months year of continuous coverage during the study period, 2009-2022.[1] We use this *general sample* of the Clalit population to estimate the overall mortality and morbidity hazard rates.

For all other analyses, we use the *linked-generations sample* by restricting attention to Clalit enrollees for whom we observe at least one parent in our data.[2] Appendix Figure A1 describes the age distribution of children and adults in our linked generations sample.

Outcomes Our analysis focuses on mortality, chronic conditions, and their intergenerational relationships. In order to avoid small cells related to less common conditions, we group all the chronic conditions into 15 broader categories; Appendix Table A1 provides the full list of conditions in each group (for brevity, in what follows we refer to each such group as "condition").

We measure the overall prevalence of each condition in both the general sample and the linked-generations sample. In the linked-generations sample, we also estimate the age-

[1] We focus attention on adults aged 30 and older because most chronic conditions appear after this age.

[2] Because we observe very few such individuals older than 80 years old, we only include in this sample Clalit enrollees who are 80 years old or younger, and do not analyze morbidity and mortality rates at older ages. Restricting the analysis to adults aged 30–80 means that our results abstract from intergenerational correlation that is driven by mortality in very young or very old ages.

specific hazard rate of obtaining each condition; that is, the probability of being diagnosed with the condition at a certain age, conditional on reaching that age and not having this condition earlier. We estimate this hazard rate separately for individuals with and without a family history of the condition, where a family history of a given condition is a binary indicator that is turned on if at least one of the individual's parents has ever been diagnosed with the condition. Similarly, we estimate the age-specific hazard rate of mortality rate, overall and separately for individuals with and without each condition.

2.3 Descriptive evidence

Sample composition Appendix Table A2 shows the prevalence of chronic conditions at age 75 for our different samples. The most common conditions at this age are endocrine (71%) and cardiovascular (65%), followed by musculoskeletal (45%). Reassuringly, the prevalence is similar among parents in our linked-generations sample, suggesting selection into this sample is limited.

Figure 1 displays the age-specific hazards for the onset of various conditions among individuals with and without a family history of each condition. As expected, the hazard for most conditions increases with age. The figure also shows a consistent difference, across all conditions, between individuals with and without family history, indicating that family history increases the likelihood of developing these conditions. Appendix Figure A3 shows the difference in log hazard associated with family history by condition. This difference remains relatively stable across ages for most conditions, suggesting that a family history leads to a proportional increase in the hazard of developing a condition. The degree of this increase in hazard varies by condition, with some conditions (e.g., hematological) more influenced by family history than others (e.g., genitourinary).

Figure 2 presents the mortality rates by age for individuals with and without each condition. As may be expected, individuals with chronic conditions exhibit higher mortality rates. Appendix Figure A4 focuses on the differences in mortality rates between individuals with and without each condition. It reveals that the elevation in mortality rates associated with all conditions is more pronounced at younger ages and diminishes with age, with the decrease showing an approximately linear trend.

Taken together, the descriptive evidence presented in this section shows that the incidence rates of nearly all chronic conditions are higher among individuals with a family history. Moreover, the onset of these conditions is associated with increased mortality rates. These findings suggest that parents with chronic conditions are both more likely to die at a younger age and, at the same time, to have children with chronic conditions who are therefore also more likely die at a younger age; that is, intergenerational correlation in mortality. Yet, a

more complete framework is required in order to quantitatively connect these various hazard rates – by age and across conditions – to the intergenerational correlation in mortality, which is our primary object of interest. This is what we turn to next.

3 Model and estimation

To quantify the contribution of chronic conditions to the intergenerational correlation in mortality, we embed morbidity and mortality hazard estimates into a life-cycle model.

Consider a parent-child pair, and denote by T_c and T_p the child's and parent's realized age at the time of death, respectively. The intergenerational correlation in mortality is governed by the joint distribution of T_c and T_p, and in particular by how the child's expected life varies as a function of the parent's age at death, $E(T_c|T_p)$. Within this framework, our key focus is on the contribution of chronic conditions to this correlation.

Model If $m_{a,i}$ denotes the mortality hazard rate of individual i at age a, her probability of dying at age T_i is given by

$$Pr_i(T_i = t) = m_{t,i} \prod_{a=a_0}^{t-1} (1 - m_{a,i}). \tag{1}$$

and her life expectancy is given by

$$E[T_i] = \sum_{a=a_0}^{\bar{a}} a Pr_i(T_i = a), \tag{2}$$

where a_0 and $\bar{a} > a_0$ define the relevant age range for the analysis.

We now consider J chronic conditions indexed by $j = 1, 2, ..., J$. We denote by $G_{a,i} \subseteq J$ the set of chronic conditions individual i has at age a, and we make the strong assumption that individual i's mortality hazard rate is only a function of their age and the chronic conditions they have, that is $m_{a,i} = m(a, G_{a,i})$.

Let $h^j_{a,i}$ be the hazard rate of individual i attracting condition j at age a, so her probability that the onset of condition j occurs at age a (conditional on survival) is given by

$$Pr_i(S^j_i = s) = h^j_{s,i} \prod_{a=a_0}^{s-1} (1 - h^j_{a,i}). \tag{3}$$

Collecting all these objects together, expected life is then given by applying equation (2) and integrating over the stochastic arrival rates of the J chronic conditions.

Parameterization and estimation We parameterize the model by making several tractable assumptions, which are motivated by the descriptive patterns we presented in Section 2.3.

First, we assume chronic conditions arrive independently of each other. That is, $h^j_{a,i}$ does not depend on which other conditions individual i already has, and the arrival of each condition, conditional on the hazard rate, is independent of the arrival of the others.

Second, the mortality hazard rate can be written by

$$m_{a,i} = f(a) \prod_{j \in G_a, i} \gamma_j, \tag{4}$$

which implies that each condition affects the mortality rate multiplicatively, by a factor that is linear in age, $\gamma_j = \gamma^0_j + \gamma^1_j a$.

Similarly, we assume that family history also enters multiplicatively with an age-invariant parameter into the hazard rate of the arrival of each condition. Formally, let H^j be the set of individuals with a family history of j. Then, for all $i \in H^j$ and $i' \notin H^j$:

$$h^j_{a,i} = \beta_j h^j_{a,i'}. \tag{5}$$

That is, there is an age-invariant ratio between the hazard rates for attracting condition j for individuals with and without a family history of condition j. We can therefore abstract from $h^j_{a,i}$ and only consider h^j_{a,f^j_i}, where f^j_i is a dummy variable that indicates whether individual i has family history of condition j.

Taken together, the model parameters are given by four components: (i) the baseline, age-specific mortality hazard rate, $m(a, \emptyset)$; (ii) the age-specific hazard rate of the arrival of each chronic condition j for individuals without a family history of the condition, $h^j_{a,f^j_i=0}$; (iii) the impact of each chronic condition on mortality, γ_j; and (iv) the impact of family history on the arrival rate of each chronic condition, β_j. Loosely, these objects are closely related to the descriptive analysis in 2.3.

We non-parametrically estimate age-specific mortality rates and condition hazards. Initially, for each age a and condition j, we estimate the mortality rate for individuals with and without the condition. We also estimate the hazards $h^j_{a,f^j_i=0}, h^j_{a,f^j_i=1}$ of being diagnosed with condition j with and without a family history of the condition. For each condition j, we derive β_j, and $\gamma_j = (\gamma^0_j, \gamma^1_j)$ from these non-parametric estimates of m and h.

4 Results

Table 1 presents our estimates of the key model parameters, quantifying, for each chronic condition, its expected prevalence, transmissibility across generations, and association with mortality risk. Column 1 displays the expected prevalence of each condition by age 80.[3] Conditions vary greatly in their prevalence (from less than 1% for hematological conditions to most of the older population for endocrine conditions (which include diabetes and thyroid disorders). Prevalence is an important factor in determining the influence of chronic conditions on intergenerational correlation in mortality because very rare conditions, by definition, can, at most, affect only a small part of the population.

Column 2 of Table 1 further shows the increased risk of developing a condition for individuals with a family history of that condition (β), derived from fitting the difference between the nonparametric hazard rates of onset for these groups. Essentially, this is the difference between the nonparametric curves in Figure 1 (a difference that is also directly depicted in Appendix Figure A3). Conditions exhibit considerable variability in their association with family history. For example, genitourinary conditions show the weakest correlation with family history, where having such a history is linked to only a modest 9.4% proportional increase in the hazard of developing these conditions across all ages. Conversely, a family history of hematological conditions—the most strongly correlated across generations—increases the hazard of onset by a factor of 15.

Columns 3 and 4 of Table 1 show the heightened mortality risk associated with having the condition (γ_0) and its change with age (γ_1). These parameters are based on linearly fitting the difference between the mortality risk at various ages between individuals with and without each condition, as depicted by the difference between the fitted curves in Figure 2, explicitly plotted in Appendix Figure A4). The increase in mortality risk varies substantially across conditions. Considering the age-dependent nature of this risk elevation, we present a comparison at age 65, an age when many conditions are already manifest. At this age, the mortality risk for individuals with musculoskeletal conditions less than doubles, whereas it increases more than eightfold for those with neoplasms, encompassing all types of cancerous tissue growth.

Such wide variation in prevalence, risks associated with family history, and association with mortality underscores the diverse roles different conditions play in intergenerational mortality correlation. To clarify how prevalence and other risk factors are interrelated,

[3]This is a hypothetical measure of prevalence in a population comprised of individuals with no family history of the condition and are immune to mortality. This measure correlates strongly with the actual prevalence of conditions in our sample.

we calculate the *difference in life expectancy between individuals with and without a family history of each condition*, denoted as $\Delta LE_j = E[T_i|f_i^j = 1] - E[T_i|f_i^j = 0]$. Typically negative, this measure captures the decrease in life expectancy associated with a family history of the condition, encapsulating both the morbidity risk linked to family history and the elevated mortality risk each condition carries. It is an aggregate measure of a condition's intergenerational significance, combining both β and γ. Figure 3 illustrates the association of ΔLE_j and condition prevalence across different conditions. Notably, endocrine and cardiovascular conditions stand out as both highly prevalent and strongly interconnected across generations, being significantly associated with both prior family history and increased mortality risks. Consequently, a family history of cardiovascular conditions is associated with a reduction in life expectancy of 0.45 years, while endocrine conditions correlate with a decrease of 0.3 years. In contrast, family histories of less common or less hereditary conditions, such as otolaryngological (ear, nose, and throat) disorders, have only a negligible link to life expectancy changes.

Comparing cardiovascular diseases, neoplasms, and hematological diseases highlights the importance of all three factors: prevalence, intergenerational association in risk, and association with mortality. Cardiovascular diseases, aside from being very prevalent (affecting most of the population by age 80) and correlated across generations (a history of heart diseases increases the risk of onset by 1.5 times), substantially elevate mortality risk. They are associated with a 4.5-fold increase in mortality hazard at age 65.[4] Conversely, while a family history of neoplasms elevates mortality risk to a greater extent, it is linked to a lesser reduction in life expectancy (0.19 year), attributed to its lower baseline prevalence and more modest transmissibility. Hematological diseases, despite their lower prevalence (3% at age 80 in the absence of a family history), still significantly impact life expectancy (0.20 years reduction), mainly due to their considerable mortality impact and the notable increase in risk due to family history (by a factor of 15). As these examples show, the transmissibility of mortality across generations is not driven by a single factor but by an interplay of factors.

To more comprehensively quantify the significance of chronic conditions in the intergenerational correlation of mortality and to incorporate the impact of various factors discussed previously, we employ simulation analysis using the model and estimates derived from our sample. Specifically, we simulate morbidity and mortality within a population of N families, each consisting of a parent and a child. Utilizing the nonparametric estimates for the baseline morbidity hazard for each condition, $h^j_{a,f_i^j=0}$, we determine for every parent the risk profile over their lifetime for catching different conditions and simulate the age at onset of

[4]Cardiovascular diseases in our sample are associated with an increase of $\exp \gamma_0 + \gamma_1 \cdot a = \exp\{3.253 - 0.27 \cdot 65\} = 4.473$.

any conditions they acquire (up to age 80) by drawing realizations from this risk process. Based on this realized morbidity, we then calculate the mortality hazard for each parent at every age ($m_{a,i}$ from equation 4) and draw their age of death from this mortality risk profile. Subsequently, we assign each child the vector f_i^j, which documents the conditions their parent would have encountered had they lived to 80. Utilizing this family history, we calculate each child's morbidity risk for every condition over their life and draw the child's age of onset from that risk profile. Finally, drawing on the child's actual morbidity, we evaluate their individual mortality risk process and subsequently draw their age of death. All draws are independent across families. Using the simulated population, we calculate different statistics related to the intergenerational correlation between child and parent mortality.

The intergenerational correlation in the age of death within the simulated population, driven exclusively by the transmission of chronic conditions, quantifies the degree to which these conditions contribute to the intergenerational correlation in mortality. To evaluate the contribution of specific chronic conditions to the intergenerational correlation in mortality, we use a variant of these simulations, where we selectively neutralize the intergenerational transmission of one condition at a time in our simulations (by setting $\beta_j = 0$ and recalculating the condition risk of onset among children with a family history of condition j) while holding constant the mortality impact of condition j and the realized onset and influence on mortality of all other conditions. To explore the age-related heterogeneity in the relationship of different chronic conditions to the correlation in mortality, we reproduce the same simulation analyses using a different assumption about the age range of the analysis, replacing the baseline range of 30–80 with ranges that increasingly focus on younger ages for both parents and children, by capping the mortality process at age 50, 60, or 70 instead of 80.

Figure 4 presents the results of these simulation-based estimates for the intergenerational correlation in the length of life and its association with chronic conditions from a simulated population of $N = 10^7$ families. The darkest column in the group labeled "No Exclusion" shows the correlation when all conditions are considered, which is 0.0173. Given that prior research estimates intergenerational lifespan correlations to be between 0.09 and 0.14 (Black et al., 2023), our findings indicate that approximately 11%–17% of the intergenerational correlation in mortality can be attributed to the transmission across generations of chronic conditions.

The other column groups in Figure 4 show the (reduced) correlation in the duration of life when we neutralize the transmissibility across generations of specific conditions. In line with findings from Figure 3, cardiovascular and endocrine conditions emerge as the most substantial contributors to the intergenerational correlation in the length of life, as reflected in the fact that when they are neutralized, the correlation decreases sharply. In particular,

neutralizing the transmission of cardiovascular and endocrine conditions reduces the intergenerational correlation in the length of life by 35% and 17%, respectively (from 0.0173 to 0.0113 and 0.0140, respectively). These conditions are prevalent, transmissible across generations, and have a significant impact on mortality. Psychiatric conditions, neoplasms, and neurological conditions have moderate importance, and neutralizing their transmission reduces the intergenerational correlation in the length of life by 9–12%. Other conditions result in smaller reductions, suggesting they account for only a small part of the overall correlation in the length of life.

The lighter-colored bars in each of the groups in Figure 4 show the results when the age cap in the simulation is reduced. Notably, reducing the maximal age of death from 80 to 70, 60, and 50 decreases the intergenerational correlation in the age of death to 0.0124, 0.0056, and 0.0011, respectively. Namely, most of the association in the age of death that is explained by chronic conditions is driven by variation in the age of death after the age of 60, which is consistent with late onset of most such conditions. There is only a negligible variation in the age of death at 50 or below.

5 Conclusion

We leveraged comprehensive administrative claims data from Israel's largest healthcare system to explore the role of chronic conditions in intergenerational mortality correlation. Our findings reveal that chronic conditions, specifically cardiovascular and endocrine diseases, are significant contributors to the intergenerational transmission of mortality risk, together explaining about 10% of the observed intergenerational correlation in mortality. Our results suggest that chronic conditions not only reduce life expectancy but also perpetuate health disparities across generations.

References

Beeton, Mary and Karl Pearson, "On the Inheritance of the Duration of Life, and on the Intensity of Natural Selection in Man.," *Biometrika*, 1901, *1* (1), 50–89.

Black, Sandra E, Neil Duzett, Adriana Lleras-Muney, Nolan G Pope, and Joseph Price, "Intergenerational correlations in longevity," Technical Report, National Bureau of Economic Research 2023.

_ **, Paul J Devereux et al.**, "Recent Developments in Intergenerational Mobility," *Handbook of Labor Economics*, 2011, *4*, 1487–1541.

Christensen, Kaare, Mary K Wojczynski, Jacob K Pedersen, Lisbeth A Larsen, Susanne Kløjgaard, Axel Skytthe, Matt McGue, James W Vaupel, and Michael A Province, "Mechanisms underlying familial aggregation of exceptional health and survival: a three-generation cohort study," *Aging Cell*, 2020, *19* (10), e13228.

Currie, Janet, "Inequality at birth: Some causes and consequences," *American Economic Review*, 2011, *101* (3), 1–22.

_ **and Enrico Moretti**, "Biology as destiny? Short-and long-run determinants of intergenerational transmission of birth weight," *Journal of Labor economics*, 2007, *25* (2), 231–264.

Halliday, Timothy, Bhashkar Mazumder, and Ashley Wong, "Intergenerational mobility in self-reported health status in the US," *Journal of Public Economics*, 2021, *193*, 104307.

Jia, Gengjie, Yu Li, Hanxin Zhang, Ishanu Chattopadhyay, Anders Boeck Jensen, David R Blair, Lea Davis, Peter N Robinson, Torsten Dahlén, Søren Brunak et al., "Estimating heritability and genetic correlations from large health datasets in the absence of genetic data," *Nature Communications*, 2019, *10* (1), 5508.

Johnston, David W, Stefanie Schurer, and Michael A Shields, "Exploring the intergenerational persistence of mental health: Evidence from three generations," *Journal of Health Economics*, 2013, *32* (6), 1077–1089.

Lu, Frances and Tom Vogl, "Intergenerational persistence in child mortality," *American Economic Review: Insights*, 2023, *5* (1), 93–109.

Mahmood, Syed S, Daniel Levy, Ramachandran S Vasan, and Thomas J Wang, "The Framingham Heart Study and the epidemiology of cardiovascular disease: a historical perspective," *The Lancet*, 2014, *383* (9921), 999–1008.

Pearl, Raymond, "Studies on human longevity. IV. The inheritance of longevity. Preliminary report," *Human Biology*, 1931, *3* (2), 245.

Polubriaginof, Fernanda CG, Rami Vanguri, Kayla Quinnies, Gillian M Belbin, Alexandre Yahi, Hojjat Salmasian, Tal Lorberbaum, Victor Nwankwo, Li Li, Mark M Shervey et al., "Disease heritability inferred from familial relationships reported in medical records," *Cell*, 2018, *173* (7), 1692–1704.

Ruby, J Graham, Kevin M Wright, Kristin A Rand, Amir Kermany, Keith Noto, Don Curtis, Neal Varner, Daniel Garrigan, Dmitri Slinkov, Ilya Dorfman et al., "Estimates of the heritability of human longevity are substantially inflated due to assortative mating," *Genetics*, 2018, *210* (3), 1109–1124.

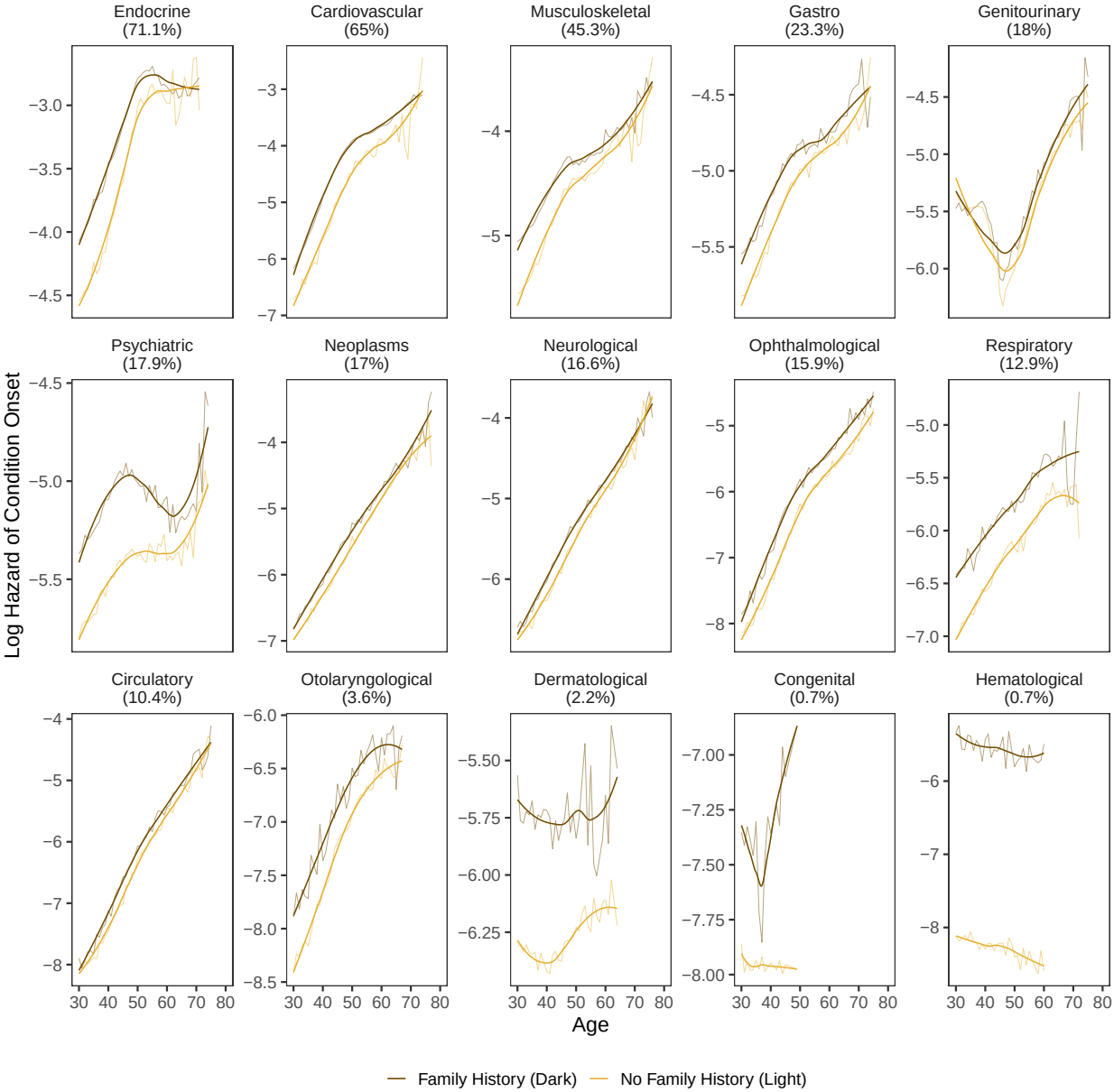
Slagboom, P Eline, Niels van den Berg, and Joris Deelen, "Phenome and genome based studies into human ageing and longevity: An overview," *Biochimica Et Biophysica Acta (BBA)-Molecular Basis of Disease*, 2018, *1864* (9), 2742–2751.

Thompson, Owen, "Genetic mechanisms in the intergenerational transmission of health," *Journal of Health Economics*, 2014, *35*, 132–146.

_ , "Gene–Environment Interaction in the Intergenerational Transmission of Asthma," *Health Economics*, 2017, *26* (11), 1337–1352.

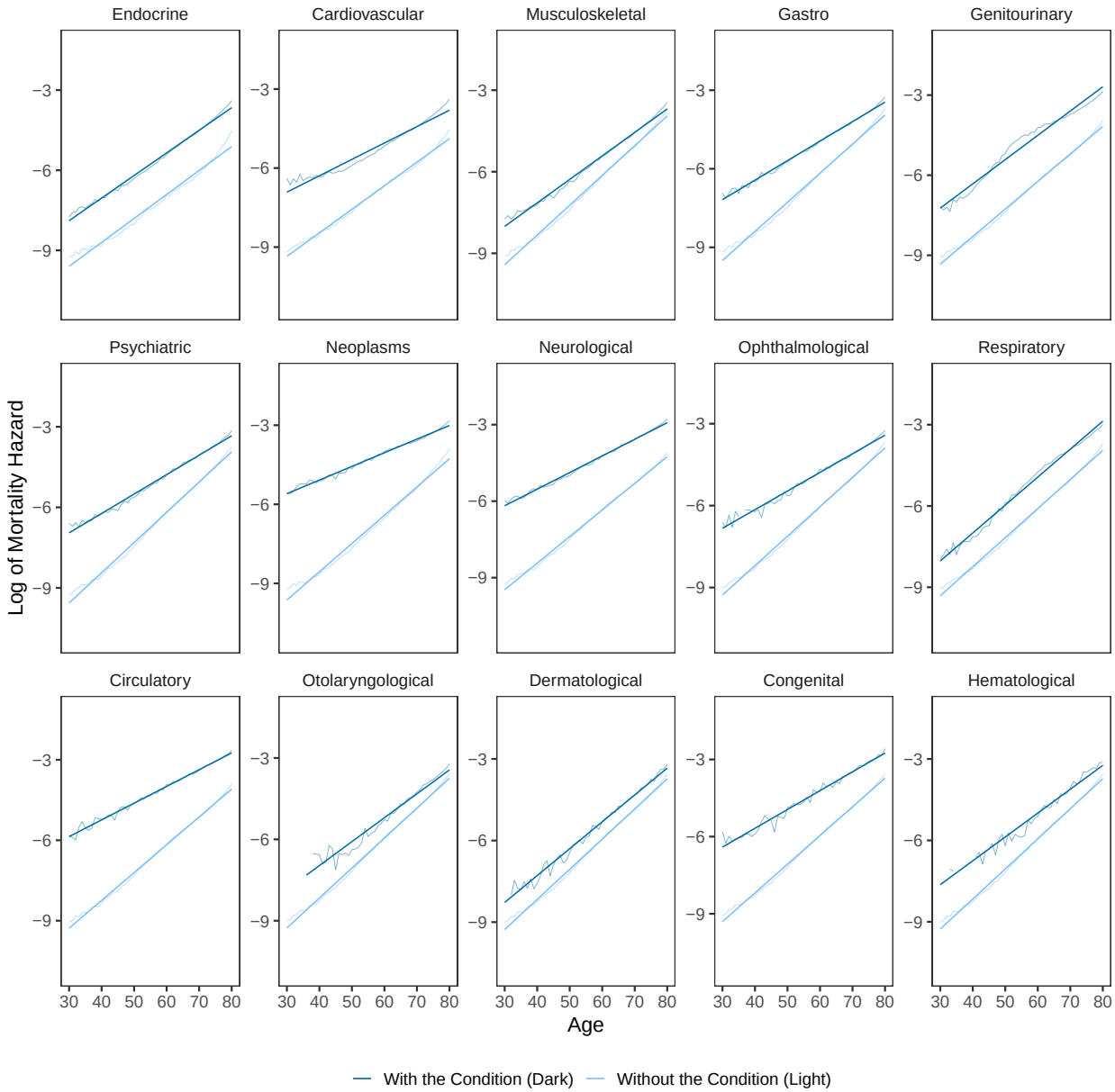
Vaupel, James W, "Inherited frailty and longevity," *Demography*, 1988, *25* (2), 277–287.

Figure 1: Morbidity Risk and Family History



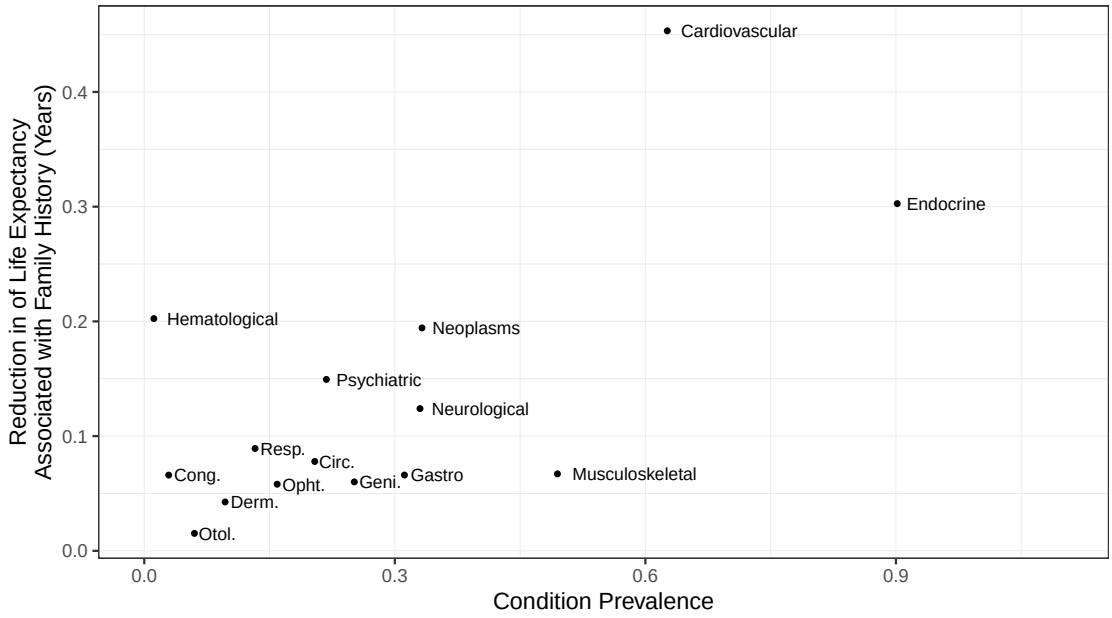
The figure shows nonparametric estimates of the hazard for the onset of various chronic conditions in individuals with and without a family history of each condition. Each panel corresponds to a different condition group, with its sample prevalence shown in parentheses. The x-axes show age in years. The y-axes show the hazard of obtaining each condition, conditional on survival, with and without a family history of the condition. Estimates are overlaid with a loess smoother.

Figure 2: Mortality Hazard with Different Chronic Condition



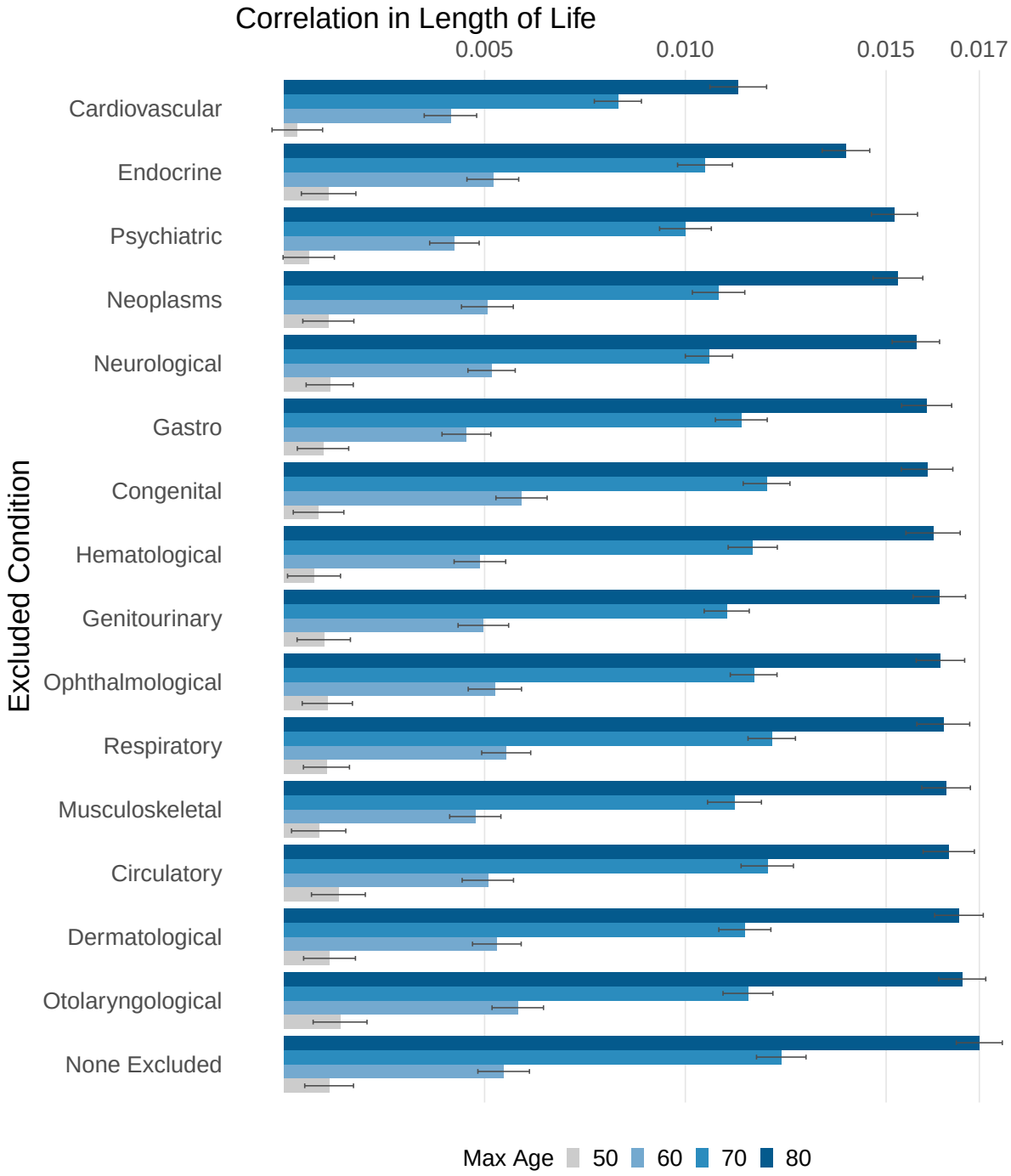
The figure shows nonparametric estimates of age-specific mortality rates for individuals diagnosed with each condition versus those not diagnosed. The y-axes show age-specific mortality rates with and without each condition. Nonparametric estimates are overlaid with a linear fit.

Figure 3: Reductions in Life Expectancy with Family History and Prevalence of Different Chronic Conditions



The figure shows the prevalence of various chronic conditions and the associations of family history and life expectancy across different groups. On the x-axis, the proportion of individuals with no family history diagnosed with a condition by age 80 is displayed for a hypothetical population immune to mortality. The y-axis quantifies the disparity in life expectancy, measured in years, between individuals with and without a family history of each condition.

Figure 4: Simulated Intergenerational Correlations in Length of Life



This figure shows the intergenerational correlations in mortality from simulated cohorts of parents and children, all of whom are healthy at the age of 30 and eventually die at or before a maximal age $\bar{a}$. Each simulated individual is exposed to morbidity risk based on their family history and to mortality risk based on their current health status. Each group of columns presents estimates obtained by neutralizing the effects of family history on the morbidity risk associated with one condition while keeping all other factors constant. The group labeled "None Excluded" represents the baseline scenario where no conditions are neutralized. Different colored bars within each group show results under different maximum age caps $\bar{a}$. 95% confidence intervals shown are based on bootstrapped standard errors.

Table 1: Comparative Risks by Condition: Prevalence, Family History, and Mortality

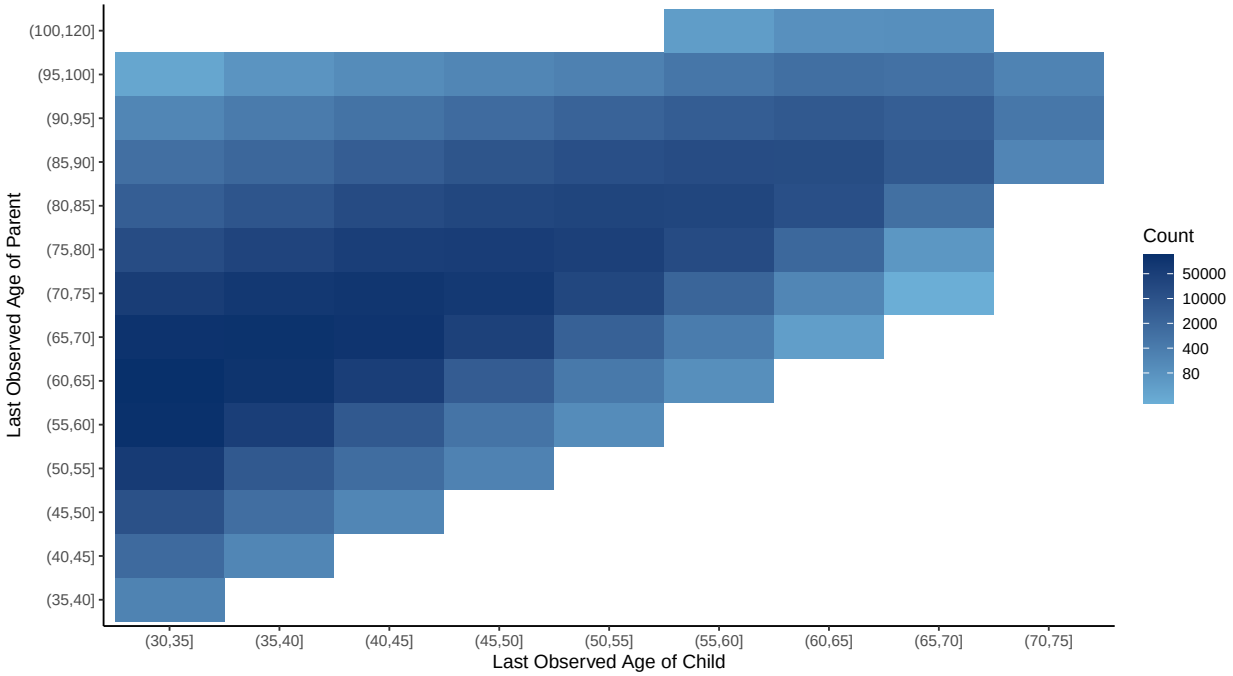
	Prevalence	Morbidity Risk Change w/ Family History (β)	Mortality Risk Change w/ the Condition	
			Initial Risk (γ_0)	Age-related Change (γ_1)
	(1)	(2)	(3)	(4)
Endocrine	0.902	1.244	1.862	-0.005
Cardiovascular	0.626	1.532	3.252	-0.027
Musculoskeletal	0.495	1.272	2.105	-0.023
Neoplasms	0.333	1.205	5.699	-0.055
Neurological	0.330	1.152	4.468	-0.039
Gastro	0.312	1.171	3.426	-0.037
Genitourinary	0.251	1.094	2.469	-0.012
Psychiatric	0.218	1.386	3.820	-0.040
Circulatory	0.204	1.184	4.659	-0.041
Ophthalmological	0.159	1.451	3.635	-0.040
Respiratory	0.133	1.502	1.409	-0.004
Dermatological	0.097	1.679	1.387	-0.012
Otolaryngological	0.060	1.484	2.374	-0.027
Other	0.055	2.845	2.861	-0.013
Congenital	0.029	1.942	4.048	-0.038
Hematological	0.011	15.341	2.421	-0.024

This table presents parameter estimates for different chronic conditions. For each condition, j, Column 1 shows the expected prevalence at age 80 in a hypothetical population that is immune to mortality and has no family history of condition j. Column 2 provides estimates for β_j, the parameter representing the multiplicative increase in the onset hazard for each condition when there is a family history. Columns 3 and 4 offer estimates for the change in mortality risk associated with having condition j. This increase is parameterized linearly, with $\gamma_{0,j}$ capturing the intercept (at age 0) and $\gamma_{1,j}$ capturing the slope of the hazard change with age.

On-Line Appendix for Intergenerational Correlation in Mortality: The Role of Chronic Conditions

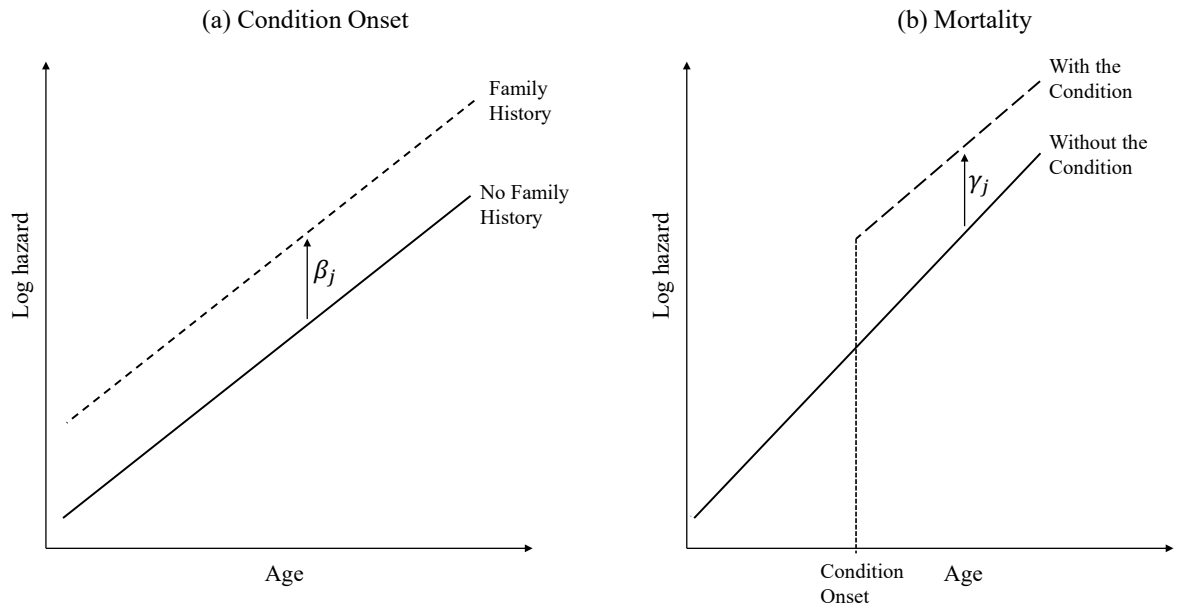
Appendix Figures and Tables

Appendix Figure A1: Joint Age Distribution of Linked Generations



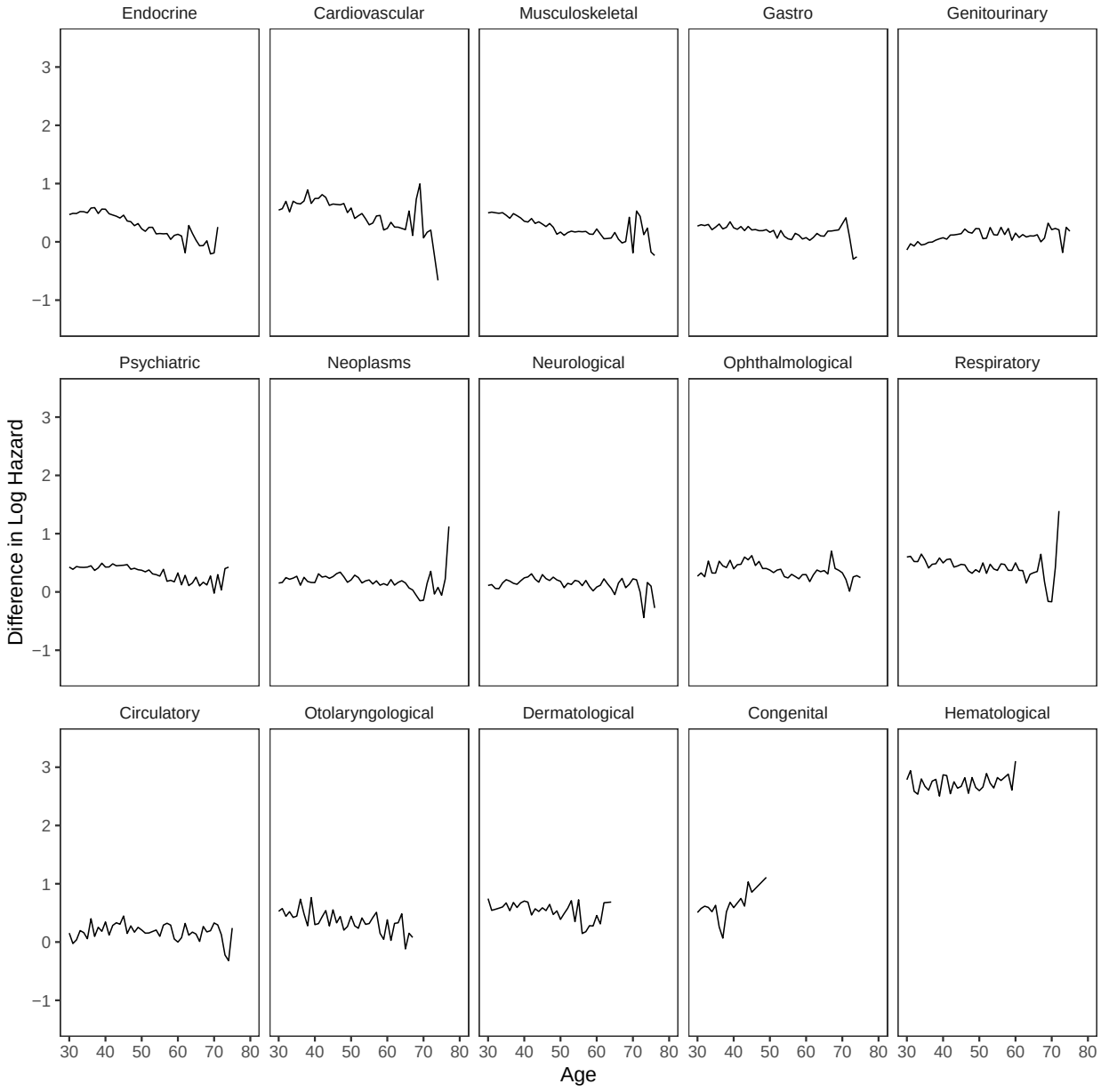
The figure presents a heatmap representing the joint distribution of the last observed ages of children and their parents in our sample. Each cell on the heatmap corresponds to a pair of 5-year age bins. To address privacy concerns, cells with fewer than 30 individuals are not plotted, and ages above 100 are categorized into broader bins. Note that the last observed ages might differ from actual age differences due to differing observation years. For instance, a child born in 1945 and last observed in 2018 at age 73, with a parent alive from 1920 to 2010 (last observed in 2010), would have a recorded age difference of 17 years in our data, which is smaller than their actual age difference of 25 years.

Appendix Figure A2: Intergenerational Relationship in Morbidity and Mortality Risk



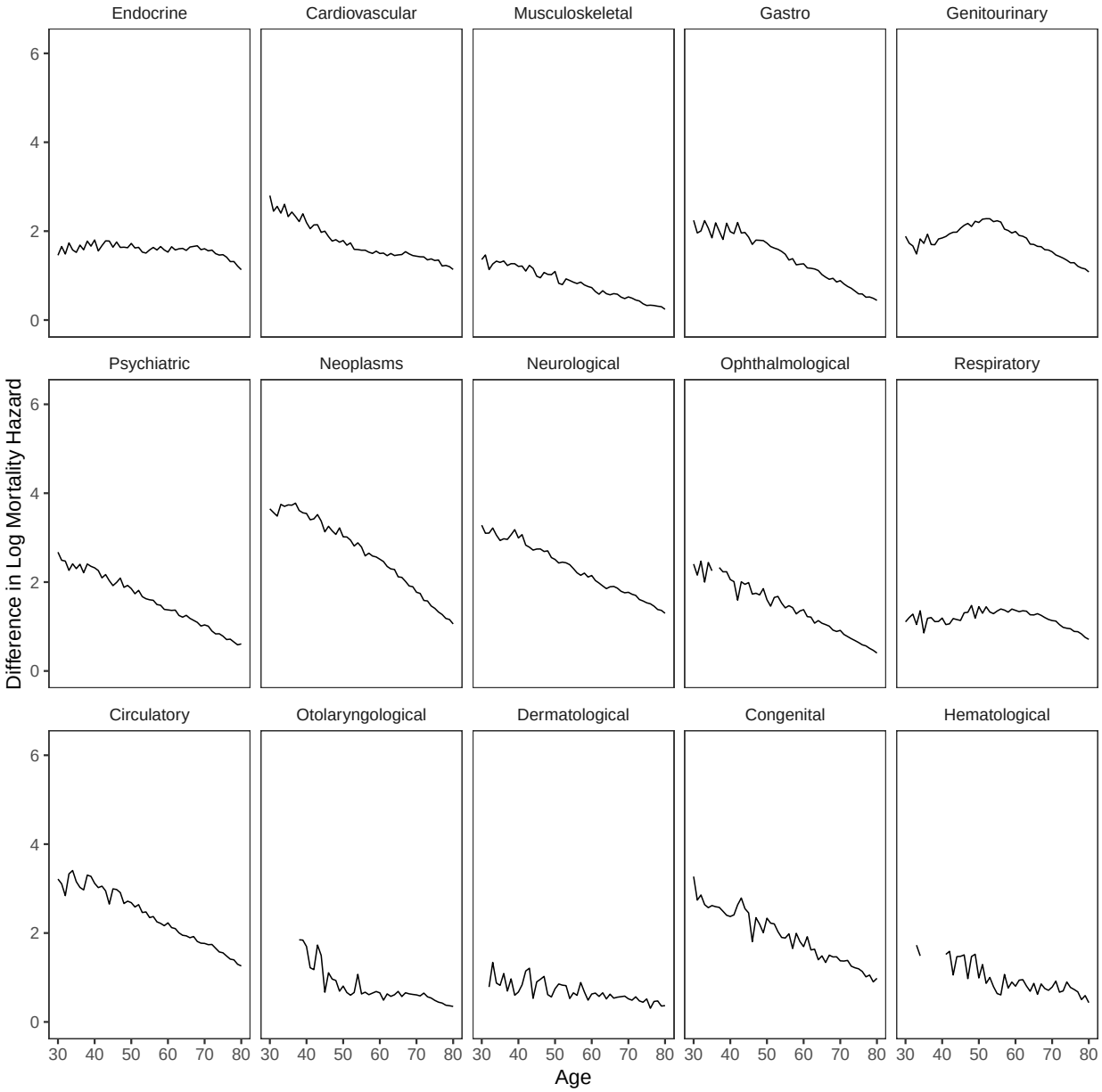
The figure shows an illustration of the key elements of our model of intergenerational correlation in mortality risk driven by intergenerational correlation in morbidity risk. Panel A shows the rate of onset of a chronic condition j, which increases proportionally at a constant rate β with a family history of the condition. Panel B shows the mortality hazard rate, which increases upon the condition's onset by a rate γ_j, which is linear in age. The baseline age-specific hazards for condition onset and mortality, depicted as linear in age, are unconstrained by the model and estimated non-parametrically for population data.

Appendix Figure A3: The Increase in Morbidity Hazard with Parental History



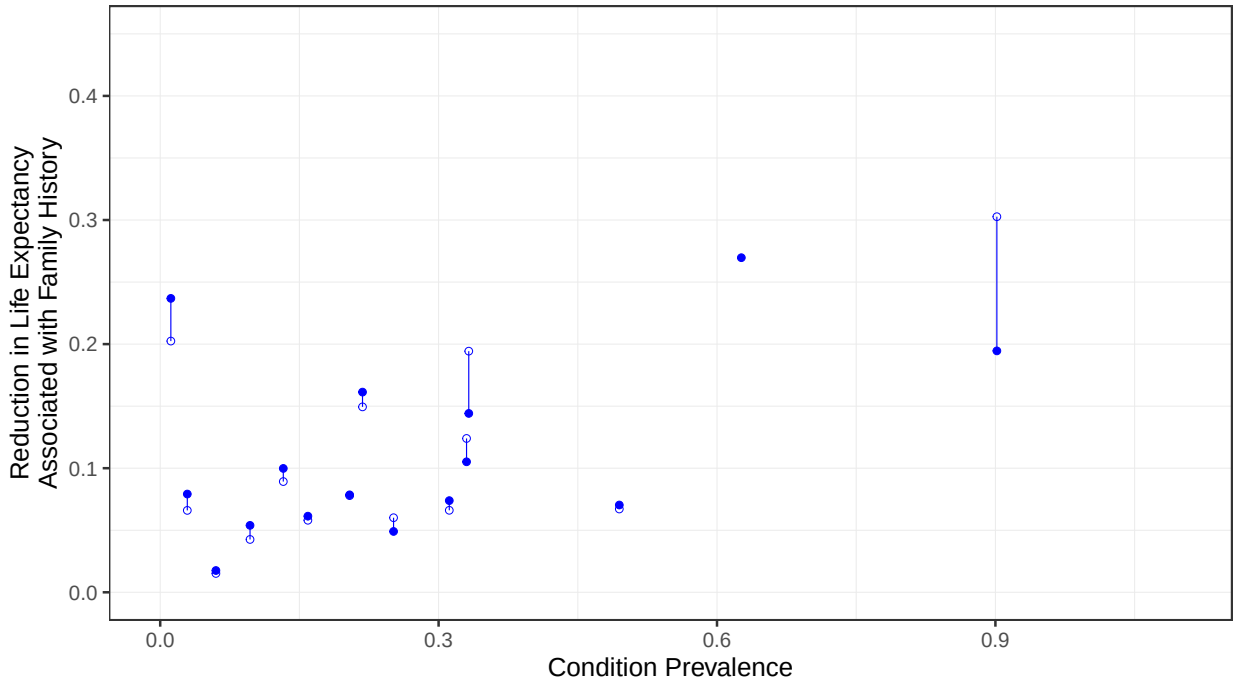
These figures show the increase in morbidity hazard between individuals with and without family history of each condition.

Appendix Figure A4: Increased Mortality Hazard with Different Chronic Conditions



These figures show the difference in the log of the age-specific mortality rates between individuals with and without each condition. Cells with fewer than individuals are suppressed.

Appendix Figure A5: Reductions in Life Expectancy with Family History and Prevalence of Different Chronic Conditions (Sensitivity to Specification)



This plot compares the "importance" of each chronic condition category in the following sense. On the x-axis we plot the estimated baseline population prevalence of each chronic condition at the age of 80, and on the y-axis we plot the difference in estimated life expectancy between individuals with and without a family history of each condition. We plot this for both non-parametric and semi-parametric estimates of prevalence and life expectancy. We also plot "iso-importance" curves each of which outlines equal multiples of prevalence and life expectancy.

Appendix Table A1: Chronic Condition Groupings

Condition Group	Specific Conditions
Endocrine	Hyperlipidemia, Obesity, Diabetes, Hypothyroidism, Hyperthyroidism, Hypo/Hyperparathyroidism, Hyperprolactinemia, Diabetes Insipidus, Addisons Disease, Cushings Disease, Acromegaly
Cardiovascular	Hypertension, IHD, Arrhythmia, Valvular Cardiac Dis (excl. MVP), CHF, PVD, Carotid Artery Disease, Pulmonary Hypertension, Cardiomyopathy, Aortic Aneurism, IHSS
Musculoskeletal	Arthropathy, Osteoporosis, Joint Replacement, Gout, Rheumatoid Arthritis, Polymyalgia Rheumatica, SLE, Scleroderma
Neoplasms	Malignancy
Neurological	Other Neurological Disease, Dementia / Alzheimers / OMS, Epilepsy, Parkinsons Disease, Cerebral Palsy, Multiple Sclerosis, Motor Neuron Disease, Myasthenia Gravis, Benign Brain Tumor, Hereditary Neurological Disease
Gastro	Reflux Esophagitis / Gastritis / Deudenitis, Peptic Ulcer, Other Liver Disease, Irritable Bowel Syndrome, Celiac Disease, Crohns Disease, Hepatitis B Carrier, Hepatitis C Carrier, Chronic Act/Per Hepatitis, Cirrhosis, Ulcerative Colitis, Gastrostomy
Genitourinary	Other kidney Disease, Chronic Renal Failure, Infertility Male/Female, Prostatic Hypertrophy, Dialysis, Kidney Transplant, Endometriosis
Psychiatric	Depression, Anxiety, Psychoses, Neuroses, Schizophrenia, Bipolar Disease
Circulatory	s/p CVA, s/p Head of Femur Fracture, s/p Pneumothorax, s/p splenectomy
Ophthalmological	Retinopathy, Glaucoma, Blindness, Retinitis Pigmentosum, Retinal vein occlusion
Respiratory	Asthma, COPD, Chronic Bronchitis, Bronchiectasis, Other pulmonary disease, Tuberculosis s/p, Tuberculosis
Dermatological	Atopic dermatitis, Psoriasis, Non-melanoma skin cancers, Hidradenitis Suppurativa
Otolaryngological	Deafness
Other	Amputation of Limb, Chronic Ventilation, Tracheostomy, Hypophysary Adenoma, Amyloidosis, Pemphigus, G-6-P-D Deficiency, Behcets Disease, Cystic Fibrosis, Other hereditary diseases, Wilsons Disease, Gaucher Disease, Mitochondrial diseases, Organ Transplantation, Sarcoidosis
Congenital	Congenital Anomalies, Mental Retardation (including Down Syndrome), Autism, Familial Mediterranean Fever
Hematological	Other Hematologic Dis (excluding Iron Def Anemia), Thalassemia, Sickle Cell Anemia, Hemophilia, ITP, Pernicious Anemia

This table reports the specific chronic conditions associated with each chronic condition group (hereafter referred to as Condition).

Appendix Table A2: Chronic Condition Prevalence

Condition	Full	Parent	Child (No History)	Child (History)
Endocrine	0.711	0.777	–	–
Cardiovascular	0.650	0.692	–	–
Musculoskeletal	0.453	0.504	–	–
Gastro	0.233	0.252	–	–
Genitourinary	0.180	0.205	–	–
Psychiatric	0.179	0.186	–	–
Neoplasms	0.170	0.179	–	–
Neurological	0.166	0.190	–	–
Ophthalmological	0.159	0.174	–	–
Respiratory	0.129	0.145	–	–
Circulatory	0.104	0.118	–	–
Otolaryngological	0.036	0.043	–	–
Dermatological	0.022	0.025	–	–
Congenital	0.007	0.008	–	–
Hematological	0.007	0.009	–	–

This is a placeholder table. This table will report the prevalence of each chronic condition category in the population (at age 75) across four relevant samples/sub-samples: the full sample, the parents in the linked sample, the children in the linked sample with no parental history, the children in the linked sample with a parental history.