Doctoral Dissertation

Bio-demographic Approaches to Economics
(経済学への生物人口学的接近)

Junji Kageyama

Graduate School of Economics,
Osaka University

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Notes

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Chapter 1

Introduction

Economics has developed its own rigorous methodology. Initiating the investigation from the assumption of rational preferences is one of them. Abstracting biological and demographic aspects, such as aging of mental and physical abilities, is another. This approach, on one hand, works as a discipline device, contributing to its successful development, but on the other, limits its own potential.

In light of the advancements in other scientific disciplines, economics can extend its potential by importing the scientific findings in related disciplines. The recent movements in behavioral economics to assimilate psychology and neuroscience demonstrate the strength of such methodology.

Along this line, this thesis incorporates bio-demographic perspectives into economics. Bio-demography provides bases of human nature and contributes to building a more concrete ground for studying human behavior and society.

The rest of the thesis is organized as follows. Chapter 2 incorporates the finding in bio-demography as to how human mortality has changed in the past decades and investigates how saving behavior is influenced by such changes. In particular, the theoretical model predicts that a greater increase in lifetime leads to greater savings because the increase in lifetime accompanies uncertainty and because the working-age cohort whose lifetime is longer saves more than the retired cohort dissaves. These hypotheses are tested empirically with cross-country data, confirming that an increase in life expectancy has a positive effect on various saving rates.

Chapters 3 and 4 employ life history theory in evolutionary biology and account for the age-trajectory of time discounting. In particular, Chapter 3 provides a basic model for connecting mortality and time-discounting be-

havior, demonstrating that both time discounting and mortality mirror the trade-off between current reproduction and survival, and Chapter 4 extends the model to incorporate childhood. Both models show that the biological rate of time discounting is equal to the age-specific mortality rate, given that the long-run population growth rate is equal to zero, and thus becomes U-shaped in age. Chapter 4 also shows that the value of survival derived in these models has an economic counterpart and argues that these two values need to be reconciled for consistency in the study of behavior.

Chapter 5 and 6 inquire into happiness, one of the fundamental issues in economics, using subjective well-being data. In particular, Chapter 5 tests the explanatory power of happiness on survival at the aggregate level. In doing so, the analysis incorporates the bio-demographic perspective that men are more fragile in a stressful situation and uses the sex difference in, rather than the level of, life expectancy as the dependent variable. In addition, it controls for the reverse causality. In the reverse direction, the life expectancy gap affects national happiness through the women's widowhood ratio. Since the widowed are, on average, less happy, an increase in the life expectancy gap between women and men raises the women's widowhood ratio and lowers women's average happiness. By taking this reverse causality into account, the results demonstrate that happiness is significant in explaining the cross-country differences in the life expectancy gap.

Chapter 6 uses this result and provides an explanation as to why the happiness level of women in former-communist European countries is particularly low. The logic is as follows. A decline in happiness influences men's mortality more than women's, widens the life expectancy gap, raises the women's widowhood ratio, and thus lowers women's average happiness. The empirical tests confirm the significance of this mechanism. These results point to the importance of controlling for the demographic composition of the population when we use aggregate happiness measures as national happiness indicators.

Chapter 7 concludes. In this chapter, I discuss the future scope of the bio-demographic approach and argue for its orthodoxy.

Chapter 2

The Effects of A Continuous Increase in Lifetime on Saving

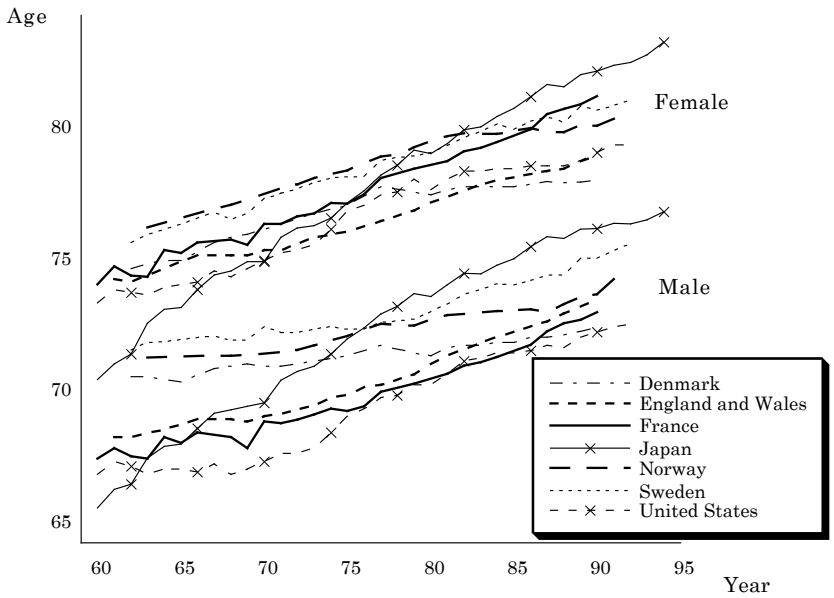
2.1 Introduction

Human longevity has increased dramatically over the past centuries. According to Levi-Bacci (1997), a newborn child in the middle of the 18th century in England and France lived to, on average, only 33 or 25 years respectively. According to recent life tables, however, a newborn child in developed countries is now expected to live for around 80 years. In Japan, life expectancy at birth was slightly over 40 years at the end of the 19th century, such that it has doubled in just one century. Figure 2.1 shows increases in life expectancy at birth for selected countries over the last half century. It can be seen that life expectancy has been rising steadily. The figure also shows that the increases differ across countries and are not necessarily smooth.[1]

This ongoing change in lifetime has significant implications for various facets of our society. In particular, it is well-known that a change in lifetime affects the allocation of wealth between consumption and saving.

This study focuses on the relationship between a steady increase in lifetime and saving. As in White (1978), Mirer (1979), and Menchik and David (1983), a number of empirical studies have pointed out that the elderly do

[1]The increase in life expectancy appears smooth for several countries. This is due to data limitations, as most of the countries do not publish life expectancy on an annual basis.



Source: Ministry of Health and Welfare of Japan (1998),*18th Life Tables*

Figure 2.1: Changes in life expectancy

not dissave as fast as the simple life-cycle hypothesis predicts. Theoretically, as been debated between Kotlikoff (1988) and Modigliani (1988), uncertainty of lifetime, or unknown date of death, and bequest motives are suspected as the prime reasons for this slow dissaving behavior. With regard to the effect of lifetime uncertainty, Yaari (1965) and Levhari and Mirman (1977) found that the shape of the survival curve reflects lifetime uncertainty and thus affects saving. Davies (1981) later showed that lifetime uncertainty reduces dissaving.

These studies indicate that an ongoing increase in lifetime which alters the shape of the survival curve should also influence saving. However, a change in lifetime has not been considered in previous studies. This study examines the effects of increasing lifetime on saving in an attempt to fill this gap.

The main finding of this study is that a greater increase in lifetime leads to greater savings. Sections 2.2 and 2.3 adopt a theoretical perspective and examine the effects on saving of a continuous increase in lifetime under the framework of the life-cycle hypothesis at the individual and aggregate

levels. In particular, Section 2.2 introduces a new lifetime indicator in a bid to overcome the limitations relating to the use of life table data. Using this indicator, the uncertainty associated with an increase in lifetime is found to depress dissaving. Section 2.3, then, shows that a greater lifetime increase results in a larger aggregate saving. This is because the working-age cohort whose lifetime is longer saves more than the retired cohort dissaves. These results are examined with aggregate data in Section 2.4, and it is confirmed that an increase in life expectancy has a positive impact on various saving rates. In particular, an increase in lifetime is found to play a very important role in explaining the high saving rates of Japan relative to other developed countries. The implications of these findings are discussed in Section 2.5.

2.2 Dissaving Behavior of the Elderly

2.2.1 Basic Model

The effect of lifetime on the dissaving behavior of the elderly was first explicitly introduced by Yaari (1965). Following his model and letting $P(x,t,j)$ denote the probability of surviving at least x more years for those born in year j and alive in year t (thus, age $t-j$), an individual born in year j and alive in year t faces the following expected intertemporal utility function,

$$E\left[V(c)\right] = \int_0^{\bar{X}(t,j)} P(x,t,j)\lambda(x,t,j)U\left[c(x,t,j)\right]dx, \tag{2.1}$$

where $\bar{X}(t,j)$ is the maximum remaining lifespan of cohort j in year t, $U[c(\cdot)]$ is the utility function, $c(\cdot)$ is consumption, and $\lambda(\cdot)$ is the subjective discount factor.[2] Then, letting $W(\cdot)$, $y(\cdot)$, and $r(\cdot)$ represent net assets, the individual's earnings (other than interest), and the expected rate of interest, the change in net assets can be written as $\dot{W}(x,t,j) = y(x,t,j) - c(x,t,j) + r(x)W(x,t,j)$. Therefore, letting $U\left[c(x,t,j)\right] = \frac{1}{1-\gamma}c(x,t,j)^{1-\gamma}$, $r(x) = r$, and $\lambda(x) = e^{-\rho x}$, and maximizing equation (2.1) under the given constraint, the change in consumption is given by

$$\frac{\dot{c}(x,t,j)}{c(x,t,j)} = \frac{1}{\gamma}\left(r - \rho + \frac{\dot{P}(x,t,j)}{P(x,t,j)}\right), \tag{2.2}$$

[2]Though his study includes bequest motives and annuity, these variables are omitted here since incorporating them does not change the result significantly except in extreme cases.

when $W(x,t,j) > 0$. Subsequently, by integrating equation (2.2), the level of consumption becomes

$$c(x,t,j) = [P(x,t,j)]^{\frac{1}{\gamma}} e^{\frac{1}{\gamma}(r-\rho)x} c(t,j) \tag{2.3}$$

where $c(t,j)$ is the consumption level at year t.

In order to focus on dissaving by the elderly, suppose that every individual retires at year t and receives no earnings other than interest after retirement. Thus, the elderly dissave from their assets accumulated during working years. With this simplification and the terminal condition that $W\left[\bar{X}(t,j)\right] = 0$, the optimal consumption level at year t becomes

$$c(t,j) = \frac{W(t,j)}{\int_0^{\bar{X}(t,j)} [P(x,t,j)]^{\frac{1}{\gamma}} e^{\frac{1}{\gamma}(r-\rho)x - rx} dx} \tag{2.4}$$

where $W(t,j)$ is the level of assets accumulated by year t.[3]

2.2.2 Estimation of Survival Curve

Equation (2.4) shows that the level of consumption, or dissaving, largely depends on the shape of the survival curve, $P(x,t,j)$ for $0 \leq x \leq \bar{X}(t,j)$. However, the true $P(x,t,j)$ is unveiled only in the future. This means that people use an estimated $P(x,t,j)$, not the true $P(x,t,j)$, to determine their level of consumption. Then, it is common to assume that people base their estimates of $P(x,t,j)$ on life table data. However, it is far from clear that the life-table estimator, say $P_{LT}(x,t,j)$, is a good estimator of the true $P(x,t,j)$. Thus, the life-table estimator is worthy of some examination.

In life tables, the construction of $P_{LT}(x,t,j)$ begins with the assumption that the age-specific mortality rate is constant across time. Under this assumption, $P_{LT}(x,t,j)$ can be calculated simply using the observed age-specific mortality rates of the older generations. These rates become proxies for the future age-specific mortality rates of the younger generations.

Practically, this approach starts by letting the realized age-specific mortality rate of cohort $j+i$ in the previous year be $m(t-1,j+i)$ and standardizing the number of newborn children in year t to $100,000$. Then, the hypothetical number of survivors in cohort j out of $100,000$, $N(t,j)$, is calculated as $100,000 \prod_{i=0}^{t-j-1} [1 - m(t-1,j+i)]$. In life tables, this hypothetical number of survivors is called the stationary population of cohort j

[3]Leung (1994) shows that wealth will be exhausted before the maximum lifetime with $y(\bar{X},t,j) > 0$ and this utility function.

in year t. The stationary population of cohort $j-x$ in year t, or x-year-older cohort, $N(t, j-x)$, can also be calculated using this method. Then, under the given assumption, the ratio of the stationary population of cohort $j-x$ to the one of cohort j, $\frac{N(t,j-x)}{N(t,j)}$, becomes the survival probability for cohort j applicable to x years later, which is $P_{LT}(x,t,j)$.

The problem with this estimator is that it does not allow for an increase in lifetime. If the direction of a change in lifetime is uncertain, this estimator would still be rational. However, lifetime increases due to a reduction in mortality in the future, and the assumption that the mortality rate remains unchanged excludes the possibility of an increase in lifetime. As a result, the life-table estimator is biased under a situation where lifetime is steadily increasing.

The reason for a secular increase in lifetime is straightforward: it is the same as the reason for economic growth. Income per capita rises due to technological progress. Similarly, lifetime increases because of technological progress in health and medicine and improvements in general living conditions. These factors ensure that the physiological process of aging is slower than actual aging as measured in calendar years. Thus, the physiological process of aging is expected to slow over time, which would correspond to an increase in lifetime.

The magnitude of an increase in lifetime can be found in Figure 2.2. The dotted lines which are constructed from the 1962 life tables correspond to the life-table survival curves of those aged 60 in 1962. The solid lines represent the realized survival curves of the same cohort. Obviously, the life-table survival curves underestimate the realized survival curves for both sexes. This illustrates the limitations inherent in using life table data to estimate lifetime.

Recognizing the existence of an increase in lifetime, the next question becomes that of how the ongoing increase in lifetime affects the survival curve. To address this question, it is necessary to determine whether the reduction in mortality is concentrated on certain ages or is common to all age groups.

The literature related to mortality reveals two key schools of thought: one is that the reduction in mortality is concentrated on the young due to the biological limit of the human species, while the other holds that the very old also experience a reduction in mortality. Vaupel and Lundström (1994) named these two positions the "limited-life-span paradigm" and the "mortality-reduction paradigm." Advocates of the limited-life-span paradigm argue that each species has its own natural, or genetic, limit of

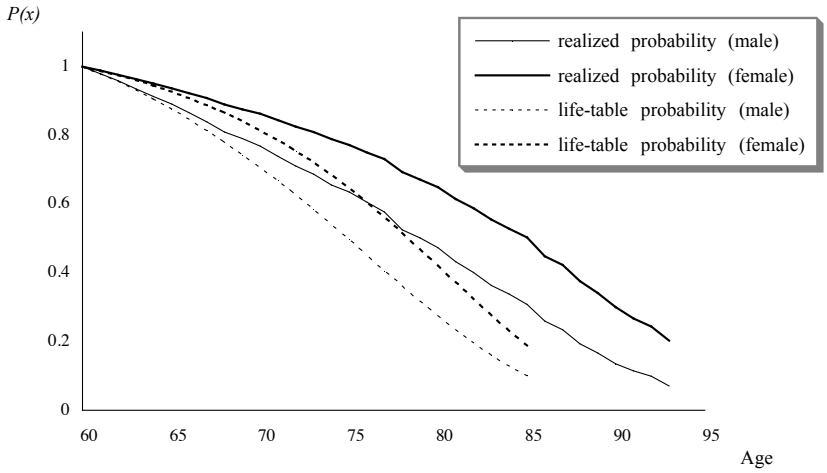


Figure 2.2: Comparison between survival curves

lifespan. Thus, the century-long rise in human life expectancy is uniquely transitional and is a result of the reduction in premature death. For example, Fries (1980) concluded that life expectancy at birth would level off at around 85 years after premature death becomes too rare to decline any further. According to this position, an increase in lifetime should stem solely from lower mortality of the young, and the remaining life expectancy of the very old should remain constant. On the other hand, proponents of the mortality-reduction paradigm such as Fogel and Costa (1997) argue that environmental and technological factors are more important than genetic factors in determining maximum lifespan. This implies that better living conditions and technological progress contribute to a reduction in mortality for all ages. Under this viewpoint, the very old as well as the young should experience an increase in remaining life expectancy.

To see which of these viewpoints is consistent with the observed lifetime increase, I use Japanese data to examine changes in the remaining life expectancy of the very old. The reason for choosing Japanese data is that its life expectancy is one of the highest in the world and is thus probably close to our biological limit if such a limit exists.

Based on life table data, Table 2.1 shows changes in remaining life expectancy and its growth rate at ages 40, 60, 80, and 90. It indicates that the very old experience an increase in remaining life expectancy as well as

the young.[4] The rate of the improvement in remaining life expectancy does not slow with age. For example, the average growth rate of remaining life expectancy for a 90-year-old female is much higher than for a 40-year-old female. These rates are about the same as for males. This indicates that the survival curve of the elderly stretches in a similar way to that of the young since remaining life expectancy is equal to the area under the survival curve.

Similar results have been reported in a number of other studies. Vaupel and Lundström (1994) and Vaupel (1998) reached the same conclusion using Nordic and American data. Curtsinger et al. (1992) examined genetic and environmental effects on the lifespan of drosophila melanogaster and found that environmental factors influence lifespan more extensively than genetic elements. These findings provide further evidence to support our view that the mortality-reduction paradigm is suitable for explaining the ongoing lifetime increase.

By applying this result, the hypothetical number of those alive x years later in cohort j can be presented by $N(t, j-x+zx)$, the population of cohort $j-x+zx$ in year t. Namely, the physiological age of cohort j after x years can be given by that of $t-j+x-zx$ whereas actual age is $t-j+x$. Therefore, the reasonable estimator of $P(x,t,j)$ when a delay in aging is taken into account, say $P_{DA}(y,t,j)$, becomes $\frac{N(t,j-x+zx)}{N(t,j)}$.

The relationship between P_{LT} and P_{DA} is presented in Figure 2.3. The solid and thick lines respectively represent the life-table survival curve and the newly constructed survival curve. The dotted line indicates the new survival curve when $z = \bar{z}$, i.e. when the uncertainty associated with an increase in lifetime is nil. It shows that a continuous increase in lifetime makes the survival curve higher and flatter.

2.2.3 Effects on Dissaving

It should now be clear that it is not appropriate to use the life-table estimator when discussing the effects of lifetime on dissaving at the individual level. The deficiencies of the life-table estimator stem from two sources. First, it does not allow for the possibility of an increase in lifetime.

Second, the uncertainty attached to a lifetime increase is not incorporated in the life-table estimator. To see this, let $P_{NU}(x,t,j)$ be $\frac{N(t,j-x+\bar{z}x)}{N(t,j)}$, i.e., the survival probability at x with a delay in aging included but without uncertainty. This estimator is the life-table estimator incorporating only a secular increase in lifetime and yields the same remaining life expectancy as

[4]The variance of the increase appears to be larger for the very old. This probably results from the smaller population sizes of older cohorts.

Table 2.1: Change in life expectancy at selected ages

		Age							
		40		60		80		90	
#	Year	Life -Exp.	Increase (%/yr.)	Life -Exp.	Increase (%/yr.)	Life -Exp.	Increase (%/yr.)	Life -Exp.	Increase (%/yr.)
male									
1	1891-98	25.70		12.80		4.80		2.6	
2	99-1903	26.03	0.214	12.76	-0.052	4.44	-1.250	2.22	-2.436
3	1919-13	26.82	0.303	13.28	0.408	4.70	0.586	2.38	0.721
4	21-25	25.13	-0.525	11.87	-0.885	3.87	-1.472	1.95	-1.506
5	26-30	25.74	0.485	12.23	0.607	4.15	1.447	2.17	2.256
6	35-36	26.22	0.266	12.55	0.374	4.20	0.172	2.14	-0.197
7		not available							
8	47	26.88	0.210	12.83	0.186	4.62	0.833	2.56	1.636
9	50-52	29.65	2.576	14.36	2.981	5.04	2.273	2.7	1.367
10	55	30.85	1.012	14.97	1.062	5.25	1.042	2.87	1.574
11	60	31.02	0.110	14.84	-0.174	4.91	-1.295	2.69	-1.254
12	65	31.73	0.458	15.20	0.485	4.81	-0.407	2.56	-0.967
13	70	32.68	0.599	15.93	0.961	5.26	1.871	2.75	1.484
14	75	34.41	1.059	17.38	1.820	5.70	1.673	3.05	2.182
15	80	35.52	0.645	18.31	1.070	6.08	1.333	3.17	0.787
16	85	36.63	0.625	19.34	1.125	6.51	1.414	3.28	0.694
17	90	37.58	0.519	20.01	0.693	6.88	1.137	3.51	1.402
18	95	37.96	0.202	20.28	0.270	7.13	0.727	3.58	0.399
	ave.(all)		0.547		0.683		0.630		0.509
	ave.(after WWII)		0.780		1.029		0.977		0.767
female									
1	1891-98	27.80		14.20		5.10		2.70	
2	99-1903	28.19	0.234	14.32	0.141	4.85	-0.817	2.36	-2.099
3	1919-13	29.03	0.298	14.99	0.468	5.26	0.845	2.61	1.059
4	21-25	28.09	-0.270	14.12	-0.484	4.41	-1.347	2.04	-1.820
5	26-30	29.01	0.655	14.68	0.793	4.73	1.451	2.24	1.961
6	35-36	29.65	0.315	15.07	0.380	4.67	-0.181	2.09	-0.957
7		not available							
8	47	30.39	0.208	15.39	0.177	5.09	0.749	2.45	1.435
9	50-52	32.77	1.958	16.81	2.307	5.64	2.701	2.72	2.755
10	55	34.34	1.198	17.72	1.353	6.12	2.128	3.12	3.676
11	60	34.90	0.326	17.83	0.124	5.88	-0.784	2.99	-0.833
12	65	35.91	0.579	18.42	0.662	5.80	-0.272	2.96	-0.201
13	70	37.01	0.613	19.27	0.923	6.27	1.621	3.26	2.027
14	75	38.76	0.946	20.68	1.463	6.76	1.563	3.39	0.798
15	80	40.23	0.759	21.89	1.170	7.33	1.686	3.55	0.944
16	85	41.72	0.741	23.24	1.233	8.07	2.019	3.82	1.521
17	90	43.00	0.614	24.39	0.990	8.72	1.611	4.18	1.885
18	95	43.91	0.423	25.31	0.754	9.47	1.720	4.64	2.201
	ave. (all)		0.600		0.778		0.918		0.897
	ave. (after WWII)		0.816		1.098		1.399		1.477

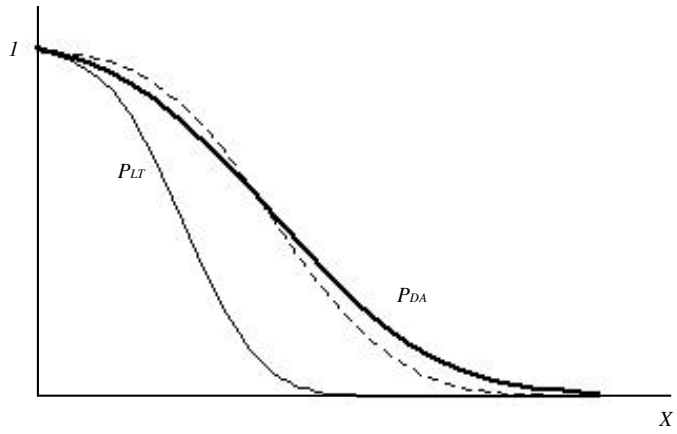


Figure 2.3: Effect of a continuous increase in lifetime on survival curve

$P_{DA}(x,t,j)$. Then, as Levhari and Mirman (1977) and Davies (1981) have shown, the difference in the level of uncertainty between these distributions results in the relationship,

$$\int_0^{\bar{X}(t,j)} [P_{DA}(x,t,j)]^{\frac{1}{\gamma}} e^{\frac{1}{\gamma}(r-\rho)x - rx} dx \tag{2.5}$$

$$\geq \int_0^{\bar{X}(t,j)} [P_{NU}(x,t,j)]^{\frac{1}{\gamma}} e^{\frac{1}{\gamma}(r-\rho)x - rx} dx,$$

with appropriate estimates of γ, r, and ρ.[5] In particular, γ needs to be larger than unity when $r = \rho = 0$. Intuitively, lifetime uncertainty makes people more cautious.

This uncertainty effect as well as the first factor makes the denominator of equation (2.4) larger. Therefore, the expected level of dissaving obtained by applying the life-table estimator will be biased upwards. An increase in lifetime and the associated uncertainty need to be incorporated in order to study dissaving behavior.

2.3 Aggregate Effect

So far, the analysis has been limited to individual behavior. The next step is to study aggregate effects. This necessitates taking those who save as

[5] Equality holds at $x = \bar{X}(t,j)$.

well as those who dissave into consideration. For this purpose, the discrete two-period over-lapping generation model is used in the following analysis.

To begin, assume that each individual lives with certainty up to the end of the first period (working-age period) but her chance of surviving the second period (retirement period) is less than certain.[6] In particular, the probability of surviving the second period for an individual born in period j is given by $P(j+1, j)$. Next, suppose that saving is entirely invested into life insurance. Then, an individual in cohort j faces the following maximization problem:

$$\begin{aligned} \max_{c(j,j),c(j+1,j)} \quad & \frac{1}{1-\gamma}c(j,j)^{1-\gamma} + \frac{P(j+1,j)}{1+\rho}\frac{1}{1-\gamma}c(j+1,j)^{1-\gamma} \\ \text{subject to} \quad & W(j,j) = c(j,j) + s(j,j), \\ & P(j+1,j)c(j+1,j) = (1+r)s(j,j) \end{aligned} \tag{2.6}$$

where $W(j, j)$, net assets, is now interpreted as individual earnings for the working-age period, and $s(j, j)$ is individual saving.[7] Then, the levels of consumption for the first and second periods and the amount of individual saving become[8]

$$c(j,j) = \frac{W(j,j)}{1 + \frac{P(j+1,j)}{1+r}\left(\frac{1+r}{1+\rho}\right)^{\frac{1}{\gamma}}}, \tag{2.7}$$

$$c(j+1,j) = \left(\frac{1+r}{1+\rho}\right)^{\frac{1}{\gamma}} c(j,j), \tag{2.8}$$

$$s(j,j) = \frac{P(j+1,j)}{1+r}\left(\frac{1+r}{1+\rho}\right)^{\frac{1}{\gamma}} c(j,j). \tag{2.9}$$

Assume further that each woman has one daughter such that fertility is constant. This means that aggregate saving is the difference between

[6] Historically, the retirement age does not rise with an increase in life expectancy as in Lumsdaine and Wise (1994). Besides, Chang (1991) shows that a rise in life expectancy does not necessarily lead to an increase in the retirement age.

[7] I continue to use r, ρ, and γ to avoid the unnecessary introduction of new symbols

[8] This shows that the amount of assets carried over to the retirement period, $(1 + r)s(j, j)$, increases with $P(j + 1, j)$ since $\frac{\partial s(j,j)}{\partial P(j+1,j)} > 0$. Therefore, the amount of assets when the retirement period starts, which is conceptually equivalent to the numerator of equation (2.4), depends positively on lifetime. This indicates that a continuous increase in lifetime makes both the numerator and the denominator of equation (2.4) larger. Although this may suggest that the effect of the increase on equation (2.4) is ambiguous, the effect is unambiguously negative. Given that lifetime income is constant, a longer lifetime, which leads to a longer retirement, necessitates a higher level of individual saving. This can be checked easily using a multiple-period, certain-longevity framework.

what the current working-age adults save and what the current old dissave, $S(j) = s(j,j) - P(j,j-1)c(j,j-1)$. Assuming that the growth rates of income and lifetime are constants, respectively g and $\hat{z}$, and standardizing $W(j-1,j-1)$ and $P(j,j-1)$ to W and P, aggregate saving can be written as

$$\begin{aligned} S(j) &= \frac{(1+\hat{z})P}{1+r}\left(\frac{1+r}{1+\rho}\right)^{\frac{1}{\gamma}} \frac{(1+g)W}{1+\frac{(1+\hat{z})P}{1+r}\left(\frac{1+r}{1+\rho}\right)^{\frac{1}{\gamma}}} \\ &\quad -P\left(\frac{1+r}{1+\rho}\right)^{\frac{1}{\gamma}} \frac{W}{1+\frac{P}{1+r}\left(\frac{1+r}{1+\rho}\right)^{\frac{1}{\gamma}}}. \end{aligned} \tag{2.10}$$

Equation (2.10) shows that aggregate saving is equal to zero when $g = r$ and $\hat{z} = 0$. This implies that the length of lifetime does not necessarily affect aggregate saving. However, the important point is not the level of lifetime, but the size of the increase in lifetime, $\hat{z}$. Differentiating equation (2.10) with respect to $\hat{z}$ reveals that a greater increase in lifetime is expected to raise aggregate saving to a greater extent. Intuitively, this is because the younger cohort saves more than the older cohort dissaves in order to prepare for a longer retirement. This indicates that the rate at which lifetime is icreasing is positively correlated with aggregate saving.

2.4 Empirical Analysis

2.4.1 Test with Household Saving Rate

Given the result in the previous sections, a greater increase in lifetime should lead to a higher saving rate. This section aims to test this result.

In recent studies, household data, especially longitudinal data, have been broadly used to examine dissaving behavior or the life-cycle hypothesis. This is because longitudinal data directly show the history of dissaving in each household. Nevertheless, aggregate data are the only practical option for testing the effects of increasing lifetime since data relating to personal expectations of the survival curve are rarely available. The only study which explicitly investigates personal expectations of lifetime is Hamermesh (1985). Hamermesh conducted a questionnaire survey in the United States and obtained the result that the life-table survival curve second-order stochastically dominates the personal expected survival curve. This result accords with the theoretical analysis in Section 2.2. However, his survey data are not related to saving. For this reason, I use aggregate data for this analysis.

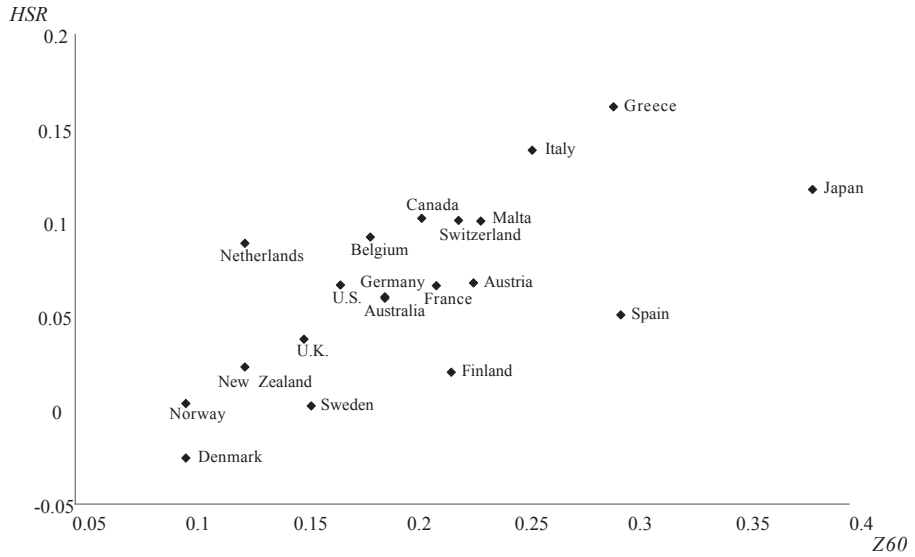


Figure 2.4: Relationship between HSR and $Z60$

The relationship between the household saving rate and an increase in lifetime is plotted in Figure 2.4 for 20 developed countries for which data on the household saving rate are available. It shows that the household saving rate and an increase in life expectancy are correlated positively, as predicted by the theoretical analysis. Here, HSR is the 1980–89 average of the household saving rate and $Z60$ is the 1960–89 average of the annual increase in life-table life expectancy at birth, the proxy for an increase in lifetime.

The 1960–89 average for the annual increase in life expectancy may appear too long compared to the 1980–89 average for the household saving rate. This is based on the assumption that forming expectations with regard to lifetime takes a long period of time. Thinking of death is not a usual thing to do in one's everyday life, and most people only think of death after a relative or friend dies, which fortunately does not occur so often. Expectations about death will be updated at such moments. I therefore assume that forming expectations about lifetime takes a long period of time. Another assumption behind $Z60$ is the linearity of an increase in life expectancy. This assumption relies on the finding, such as Harman (1991) and Lohman et al. (1992), that life expectancy rises linearly in the long run. More importantly, using $Z60$ as the proxy indicates that I can not isolate the uncertainty ef-

fect associated with an increase in lifetime. This is due to data limitations. Although we would like to use the data with regard to the survival curve on an annual basis, they are available for only a handful of countries. If such data were available, we could calculate the variance and use it to isolate the uncertainty effect. However, if the uncertainty effect becomes more significant as life expectancy grows faster, $Z60$ will also reflect the uncertainty effect.

To test the effects further, the household saving rate is regressed on a rise in life expectancy together with the economic and demographic variables which are expected to have influences on the household saving rate and which have been commonly employed in previous studies. These variables are averaged over the years between 1980 and 89 unless mentioned otherwise. Details such as definitions, sources, and sample countries, are reported in the data appendix.

With respect to the proxy for an increase in lifetime, I continue to use $Z60$. However, $Z70$ or $Z80$, the 1970–89 average or the 1980–89 average of the annual increase in life-table life expectancy at birth, is used instead of $Z60$ in some equations. This is done in order to check the effect of the length of the period over which expectations are formed. In addition, ZEX, the product of Z and EX (the level of life expectancy in life tables) representing the increase life expectancy in lifetime, is used in some equations in order to provide a comparison between the effects of ZEX and EX. If people are fully conscious of an increase in lifetime, the effect of ZEX should be greater than that of EX due to the existence of the uncertainty effect. Should the uncertainty attached to a lifetime increase be not important, the effects of ZEX and EX would be equal.

Turning to the other demographic variables, I include two sorts of dependency ratios, YNG and OLD, respectively the ratio of children to active population and the ratio of the elderly to active population.[9] As for the economic variables, the regression model includes $GYPC$, the growth rate of real GDP per capita (YPC), RDR, the real interest rate, IR, the inflation rate, and $INVY$, the inverse of YPC.[10] The reason for using $INVY$ instead of YPC is to compare the result here with previous studies that examined saving rates among developed countries.

The effects of these variables are expected as follows. YNG and OLD are

[9] Another possible demographic variable is an index of the change in retirement ages. However, no such index is available or readily constructed.

[10] Using real GDP per equivalent adult or per worker instead of YPC does not change the results significantly.

expected to have negative effects as suggested by the life-cycle hypothesis.[11] The effects of RDR and IR are ambiguous. As for $GYPC$, the effect is also ambiguous as noted by Bosworth (1993). Although it is sometimes argued that the life-cycle hypothesis clearly implies that the rate of economic growth has a positive impact on the aggregate saving rate, this need not be the case. The expectation of higher income in the future can possibly lead to an increase in current consumption at the household level. Thus, if this effect outweighs the aggregate effect, the growth rate would affect the household saving rate negatively. Empirically, however, it is common to capture a positive effect. Next, EX is expected to have a positive effect, as in Doshi (1994), since a longer lifetime will normally lead to a longer retirement. Finally, the expected effect of $INVY$ is nil under the life-cycle hypothesis.

The method of estimation is weighted least squares using the population of each country as the weighting variable. This method has been extensively used in previous studies, starting with Houthakker (1965).[12]

The results are presented in Table 2.2. In general, the equations including the Z-related variables give good $\bar{R}^2$ ranging from 0.617 to 0.800. As for the Z-related variables, the expected results are obtained. First, the coefficients always become significantly positive at the 2 percent level or better when the 1960–89 average or the 1970–89 average is used. Second, replacing $Z60$ with $Z70$ does not change the results significantly. Third, the level of significance becomes lower with $Z80$, indicating that forming expectations about lifetime takes more than one decade. Finally, the coefficients of $ZEX60$ and $ZEX70$ are always larger than the coefficients of EX.[13] These points accord with the results in the theoretical analysis.

The most striking result with respect to the other variables is that the coefficients of $GYPC$ become negative and generally significant when the Z-related variables are included. This result does not accord with previous studies such as Feldstein (1977), Modigliani and Sterling (1983), and Horioka (1989). However, in equations (2-10)–(2-12) where none of the Z-related variables is included, the coefficients become positive. Therefore, a positive effect of $GYPC$ in these previous studies may stem from the omission of an

[11] The effect of YNG can also be examined using a slight extension of the previous theoretical model. Under a three-period model which incorporates changes in fertility, YNG is expected to have a negative impact on household saving rate.

[12] The unweighted regression does not alter the results significantly.

[13] Although the null hypothesis that the coefficient of ZEX is equal to or smaller than the coefficient of EX cannot be rejected in equations (2-6) and (2-7), even at the 20 percent level of significance, this is probably due to the large standard error of EX and the small sample size.

Table 2.2: Regression results (Dependent variable: *HSR*)

eq. #	*Z60*	*Z70*	*ZEX60*	*ZEX70*	*Z80*	*ZEX80*	*EX*	*YNG*	*OLD*	*GYPC*	*RDR*	*IR*	*INVY*	*Adj. R-sq.*
(2-1)	0.4848						0.0047	-0.877	-0.621	-2.691	0.760	0.675	-0.384	0.720
	2.92						0.62	-3.73	-2.03	-1.90	1.38	2.83	-0.95	
(2-2)	0.5092							-0.861	-0.659	-2.452	0.722	0.665	-0.357	0.734
	3.24							-3.78	-2.26	-1.84	1.35	2.87	-0.91	
(2-3)	0.4320							-0.905	-0.797	-2.363	0.692	0.582		0.738
	3.28							-4.10	-3.22	-1.79	1.31	2.74		
(2-4)	0.3253							-0.919	-0.913	-1.415		0.715		0.725
	3.07							-4.06	-3.85	-1.26		3.74		
(2-5)		0.6874						-0.780	-0.377	-1.931	0.621	0.587		0.780
		3.90						-3.75	-1.30	-1.86	1.35	3.07		
(2-6)			0.0054				0.0029	-0.924	-0.770	-2.620	0.735	0.591		0.723
			2.84				0.38	-4.04	-2.98	-1.85	1.34	2.72		
(2-7)				0.0088			0.0071	-0.813	-0.290	-2.882	0.821	0.569		0.800
				3.98			1.19	-4.08	-1.03	-2.50	1.80	3.13		
(2-8)					0.3105			-0.758	-1.091	-0.170	-0.426	0.665		0.623
					1.87			-2.61	-4.13	-0.15	-0.85	2.58		
(2-9)						0.0036	0.0068	-0.808	-1.010	-0.796	-0.236	0.665		0.617
						1.57	0.79	-2.71	-3.61	-0.59	-0.43	2.56		
(2-10)							0.0098	-1.032	-1.125	0.069	-0.060	0.773	0.200	0.545
							1.06	-3.54	-3.49	0.05	-0.10	2.57	0.45	
(2-11)								-1.014	-1.265	0.909	-0.236	0.761	0.325	0.541
								-3.47	-4.30	0.83	-0.40	2.52	0.75	
(2-12)								-1.022	-1.269	0.808		0.709	0.384	0.568
								-3.61	-4.45	0.78		2.66	0.97	

All equations include a constant term. The top figure is the estimated coefficient, and the bottom figure is the *t*-statistic.

increase in life expectancy.[14]

Comparing the results in this regression model with previous studies further yields two other points to note. First, YNG has a larger effect than OLD. Although the coefficients of OLD are larger than the coefficients of YNG in previous studies as noted by Bosworth (1993), the coefficients of YNG are larger than those of OLD in this regression model. Second, the coefficients of $INVY$ are negative, but are not significantly different from 0 in this regression model. Although this result accords with the life-cycle hypothesis, Feldstein (1977) and Horioka (1989) found significant and positive coefficients of $INVY$. As noted by Horioka (1989), their result cannot be explained by either the life-cycle hypothesis or the Keynesian model.

These differences also stem from the inclusion of an increase in life expectancy. Without an adequate Z-related variable, the results become similar to the previous studies. The coefficients of OLD become larger than those of YNG in equations (2-8)–(2-12), and the coefficients of $INVY$ become positive albeit insignificant in equations (2-10)–(2-12). For these reasons, regression models without an increase in lifetime may possibly contain a specification error.

Turning to the other variables, the coefficients of RDR are positive and significant at the 10 percent level in equations (2-1)–(2-7). This may indicate that the real interest elasticity of the household saving rate is positive. Also, the coefficients of IR are significantly positive. Several reasons can be suggested for this result, such as the households' desire to maintain the real value of their financial assets, uncertainty associated with inflation as in Horioka (1989), and measurement error due to an increase in measured investment income.

2.4.2 Test with Gross Domestic Saving Rate

To test the result further, I construct a variable, GSR (1 – consumption share of GDP – government share of GDP), from the Summers and Heston data set. This variable is theoretically comparable to the gross domestic saving rate.[15] By replacing HSR by GSR, the number of countries covered

[14] In the previous studies cited above, the private saving rate, not the household saving rate, is used as the dependent variable. Besides, the sample periods, sample countries, and explanatory variables are not perfectly equal. Thus, precisely speaking, the result here is not directly comparable with previous results. Nevertheless, these two saving rates are highly correlated as noted by Horioka (1989), and the expected effects of the independent variables do not differ.

[15] A similar variable is used in Carroll and Weil (1993).

by the model increases to 126 countries. Although GSR should be less sensitive to household decisions since it is influenced by other sectors of the economy such as government and the corporate sector, results are still expected to be similar to the HSR estimation.

However, increasing the number of countries introduces other problems due to the inclusion of developing countries. The endogeneity between economic growth and saving is one example. While a country is still in the transitional period and its ability to import foreign saving is limited, its saving rate may greatly influence economic growth. This effect is likely to be more significant in developing countries. Moreover, it would not be surprising if the effects of the explanatory variables used here were to vary among the countries at different stages of development. For instance, as mentioned by Giovannini (1983), a positive real interest elasticity of saving cannot be detected easily among developing countries. The effects of the dependency ratios are sometimes difficult to identify as noted by Ram (1982).

Paying attention to an increase in lifetime, its effect may not be identical between developing and developed countries. For one thing, a greater increase in lifetime may not result in more saving in developing countries since their life expectancy is normally not very high. If people expect to die before retirement even after considering an increase in lifetime, the amount of savings needed for retirement is not affected by an increase in lifetime. Additionally, an increase in lifetime is not necessarily accompanied by more uncertainty in developing countries. This is because the reduction in mortality is relatively concentrated on young ages during the demographic transition which developing countries are generally experiencing. This means that an increase in lifetime shifts the survival curve upwards and to the right during the transitional period, not only to the right as in developed countries. In this case, an increase in lifetime could possibly lead to more certainty rather than more uncertainty.

Due to these problems, I modify the analysis in two ways. First, a dummy variable $DL5$ is included in some equations. $DL5$ is equal to 1 if YPC is at least \$5,000 and 0 otherwise. Second, weighted two-stage least squares is used in some equations to control for the endogeneity of $GYPC$. When 2SLS is employed, the lag of $GYPC$ (the 1970–79 average), a measure of openness, and the rate of population growth are used as instrumental variables.

The results are presented in Table 2.3. Generally, $\bar{R}^2$ is around 0.50 and using 2SLS does not change the results significantly. Equation (3-1) includes RDR and IR as the independent variables, equations (3-2)–(3-10) exclude

RDR and IR in order to increase the sample countries, and equations (3-11)–(3-18) limit the sample to the countries with YPC of at least \$5,000. Since RDR and IR are generally insignificant, I have chosen to omit these variables and increase the number of countries.

With respect to the effect of the Z-related variables, the expected results are again obtained.[16] The coefficients of the Z-related variables are significantly positive at up to the 1 percent level. However, the coefficients of ZEX are now smaller than the coefficients of EX in the full-sample estimation. In equations (3-6) and (3-7), the coefficients of EX become larger than those of ZEX with good t-values. This probably results from the inclusion of developing countries, as mentioned earlier. When the sample of countries is limited to those with YPC of at least \$5,000, the coefficients of the Z-related variables become larger while the coefficients of EX become insignificant and negative.[17] This possibly indicates that the variation in lifetime is more influential in developed countries.

Turning to the other demographic variables, YNG and OLD are found to be important, as in the HSR estimation. Although the coefficients of OLD are larger than those of YNG in almost all equations, the tendency towards a rise in the relative importance of YNG with the inclusion of the Z-related variables does not change. On the other hand, the effects of the economic variables, both $GYPC$ and YPC, become ambiguous. The coefficients of $GYPC$ become negative under the full-sample estimation and positive under the limited-sample estimation. Also, the level of significance depends on the regression method, yielding a higher level of significance with 2SLS. The coefficients of YPC also change in sign: they are insignificant and positive in the full-sample estimation, but negative and sometimes significant in the limited-sample estimation.

2.4.3 Case Study

The above results suggest that an increase in life expectancy has a positive impact on saving rates, and accord with the results in the theoretical analysis.

A further question relates to the explanatory power of an increase in lifetime on the saving rates. For this purpose, I apply the estimated coefficients

[16] The results with $Z80$ and $ZEX80$ are not reported in Table 2.3 because the results are similar to the ones in the HSR estimation. The coefficients of $Z80$ and $ZEX80$ become less significant.

[17] The null hypothesis that the coefficient of $ZEX60$ is equal to or smaller than the coefficient of EX can be rejected in equation (3-14) at the 15 percent level of significance.

Table 2.3: Regression results (Dependent variable: *GSR*)

eq. #	*Z60*	*Z70*	*ZEX60*	*ZEX70*	*EX*	*YNG*	*OLD*	*GYPC*	*YPC*	*DL5*	*RDR*	*IR*	*Adj. R-sq.*	# of Smpl	Method
(3-1)	0.2906					-0.568	-0.365	-1.463			-0.188	-0.053	0.654	67	WLS
	2.40					-7.66591	-1.22246	-2.38626			-0.808524	-0.551735			
(3-2)	0.1009				0.0036	-0.264	-0.571	-0.379	0.0023				0.572	126	WLS
	2.12				2.75	-4.06	-2.20	-1.41	1.02						
(3-3)	0.0974				0.0034	-0.278	-0.674	-0.336		0.035			0.597	126	WLS
	2.26				2.69	-4.25	-2.47	-1.25		1.51					
(3-4)	0.0763				0.0043	-0.262	-0.526	-0.393					0.572	126	WLS
	1.86				3.76	-4.03	-2.05	-1.46							
(3-5)		0.1030			0.0045	-0.331	-0.775	-0.431					0.576	126	WLS
		2.17			4.27	-4.68	-4.21	-1.61							
(3-6)			0.0012		0.0038	-0.257	-0.500	-0.370					0.573	126	WLS
			1.90		3.04	-3.96	-1.90	-1.41							
(3-7)				0.0019	0.0037	-0.337	-0.722	-0.442					0.580	126	WLS
				2.40	3.13	-4.79	-3.84	-1.68							
(3-8)					0.0051	-0.268	-0.865	-0.166					0.564	126	WLS
					4.89	-4.10	-4.75	-0.69							
(3-9)	0.2506				0.0012	-0.503	-0.472	-2.447					0.464	121	2SLS(W)
	2.38				0.57	-3.11	-1.55	-2.02							
(3-10)		0.1259			0.0044	-0.372	-0.866	-0.657					0.579	121	2SLS(W)
		1.97			3.65	-3.03	-3.47	-1.08							
(3-11)	0.3059					-0.524	-0.825	0.376	-0.0040				0.577	42	WLS
	3.71					-5.90	-3.06	0.91	-1.89						
(3-12)	0.3103					-0.560	-0.821		-0.0041				0.579	42	WLS
	3.77					-7.06	-3.05		-1.92						
(3-13)		0.2212				-0.559	-1.082	0.489	-0.0070				0.501	42	WLS
		2.49				-5.59	-3.99	1.09	-3.07						
(3-14)			0.0045		-0.0029	-0.527	-0.786	0.417	-0.0034				0.567	42	WLS
			3.52		-0.54	-5.10	-2.82	0.79	-1.47						
(3-15)				0.0032	-0.0005	-0.548	-1.052	0.427	-0.0068				0.487	42	WLS
				2.24	-0.09	-4.59	-3.75	0.73	-2.93						
(3-16)					0.0053	-0.439	-1.264	0.122	-0.0065				0.430	42	WLS
					0.95	-3.82	-4.54	0.20	-2.66						
(3-17)	0.2927					-0.444	-0.708	0.934	-0.0033				0.584	38	2SLS(W)
	3.39					-4.79	-2.64	1.63	-1.49						
(3-18)		0.2243				-0.481	-0.920	1.051	-0.0062				0.515	38	2SLS(W)
		2.48				-4.62	-3.40	1.76	-2.75						

Refer to Table 2.2.

Table 2.4: Effect of Z-related variables on Japanese saving rates

		GSR (%)	*HSR* (%)	
	Z (years)	Eq. (3-12)	Eq. (2-3)	Eq. (2-7)
Estimated value		32.24	11.43	11.63
Estimated value	*Z60* (*GSR*) = 0.29	29.46		
with mean *Z*	*Z60* (*HSR*) = 0.20		3.63	
	ZEX70 (*HSR*) = 16.79			1.83
Estimated value	*Z60* = 0.08 (Hungary)	22.93		
with lowest *Z*	*Z60* = 0.10 (Denmark)		-0.80	
	ZEX70 = 7.48 (Denmark)			-6.36

of the regression models to Japanese data.

First, I examine the effect of a rise in life expectancy on GSR. The results are summarized in the third column of Table 2.4. Here, the coefficients from the limited-sample estimation are used since the effect of an increase in lifetime is expected to differ between developing and developed countries. Applying the coefficients obtained from equation (3-12), which yields the highest $\bar{R}^2$ in the limited-sample estimation, the estimated GSR becomes 32.24 percent while the true GSR is 34.49 percent. Now, suppose that the increase in life expectancy declines to the mean level while the other variables remain unchanged. The estimated GSR would become 29.46 percent, which is 8.6 percent lower than the current estimated level.[18] Furthermore if the decline should reach the lowest level, that of Hungary, the estimated GSR would become 22.93 percent, which is 29 percent lower.

The effect of a rise in life expectancy is even stronger under the HSR estimation as presented in the forth and fifth columns of Table 2.4. Using the mean level of $Z60$ and the coefficients from equation (2-3), which gives the best $\bar{R}^2$ with $Z60$, the estimated HSR would drop by more than two thirds from the current estimated level of 11.43 percent, to 3.63 percent. If $Z60$ should decrease to the lowest level, that of Denmark, the estimated HSR would become negative, falling to -0.80 percent. The results are particularly striking if we use the coefficients from equation (2-7), which yields the highest $\bar{R}^2$ in this regression model. The estimated HSR, currently 11.63 percent, would fall to 1.83 percent and -6.36 percent respectively based on the mean and lowest levels of $ZEX70$.

These results indicate the important role played by an increase in lifetime

[18] The figure 8.6 percent comes from $\frac{32.24-29.46}{32.24}$.

in explaining saving rates. In particular, Japan's high saving rates relative to those of other developed countries may be attributed to its large increase in life expectancy. Japan's household saving rate is 11.75 percent, the third highest after Greece and Italy, while its increase in life expectancy has been the largest among the 20 countries.

2.5 Conclusion

The purpose of this chapter is to examine the effects on saving of a continuous increase in lifetime. Section 2.2 and 2.3 showed that, under the framework of the life-cycle hypothesis, an increase in lifetime positively affects saving. This result is tested in Section 2.4 and supported by evidence that a rise in life expectancy is accompanied by higher saving rates.

This conclusion has the following implications. First, the effect of an aging population on saving is ambiguous. This is because the two factors that cause the population to age have opposite effects on saving. On one hand, an increase in lifetime has a positive effect on saving while on the other, the aging of baby boomers has a negative effect. Therefore, studies focusing on the relative importance of these two factors are indispensable if we are to comprehend the effect of an aging population on saving.

Second, Japan's saving rates could decrease more than expected. As shown in Figure 2.1, Japan's rise in life expectancy has been remarkable. However, this trend may not continue in the future. Thus, a smaller increase in lifetime could lead to a reduction in saving, even though the level of life expectancy should remain high. In this case, both a smaller increase in life expectancy and the aging of baby boomers will have a negative impact on saving, resulting in a greater decline in saving than one might first expect.

2.6 Data Appendix

2.6.1 Definition of Variables

The 1980–89 averages are taken unless mentioned otherwise.

HSR: ratio of net household saving to household income (UN 1993)
GSR: 1– real consumption share of GDP (1985 intl. prices) – real government share of GDP (1985 intl. prices) (Summers and Heston 1998)
EX : life expectancy at birth in life tables (UN 1996)
$Z60$: average annual increase in EX between 1960 and 89 (UN 1996)
$Z70$: average annual increase in EX between 1970 and 89 (UN 1996)

$Z80$: average annual increase in EX between 1980 and 89 (UN 1996)
$ZEX60$: $Z60 * EX$ (UN 1996)
$ZEX70$: $Z70 * EX$ (UN 1996)
$ZEX80$: $Z80 * EX$ (UN 1996)
YNG: ratio of those 14 and under to those between 15 and 64 (UN 1996)
OLD: ratio of those 65 and over to those between 15 and 64 (UN 1996)
YPC: real GDP per capita in thousands of constant dollars expressed in 1985 international prices (Chain Index) (Summers and Heston 1998)
$GYPC$: annual growth rate of YPC (Summers and Heston 1998)
$INVY$: inverse of YPC (Summers and Heston 1998)
IR: rate of change in consumer price index (IMF 1998)
RDR: real interest rate (IMF 1998)
$DL5$: 1 if YPC is over $5,000$, and 0 otherwise (Summers and Heston 1998)

2.6.2 Sample Countries

HSR (20): Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Italy, Japan, Malta, Netherlands, New Zealand, Norway, Spain, Sweden, Switzerland, U.K., U.S.

GSR (67): Australia, Austria, Bangladesh, Barbados, Belgium, Botswana, Burkina Faso, Burundi, Cameroon, Canada, Central African Republic, Colombia, Congo, Costa Rica, Cyprus, Denmark, Ecuador, Egypt, Fiji, Finland, France, Gabon, Gambia, Germany, Greece, Guatemala, Guyana, Hungary, Iceland, India, Ireland, Italy, Jamaica, Japan, Jordan, Kenya, Korea, Lesotho, Malaysia, Malta, Mauritius, Morocco, Netherlands, New Zealand, Niger, Nigeria, Norway, Pakistan, Papua New Guinea, Philippines, Portugal, Senegal, South Africa, Spain, Sri Lanka, Swaziland, Sweden, Switzerland, Syrian Arab Rep., Thailand, Togo, Trinidad and Tobago, Turkey, United Kingdom, United States, Venezuela, Zimbabwe

GSR (126): GSR (42) plus Algeria, Angola, Bangladesh, Belize, Benin, Bolivia, Botswana, Brazil, Burkina Faso, Burundi, Cameroon, Cape Verde, Central African Republic, Chad, Chile, China, Colombia, Comoros, Congo, Costa Rica, Dominican Republic, Ecuador, Egypt, El Salvador, Fiji, Gabon, Gambia , Ghana, Guatemala, Guinea, Guinea-Bissau, Guyana, Haiti, Honduras, India, Indonesia, Iran, Jamaica, Jordan, Kenya, Korea, Lesotho, Madagascar, Malawi, Malaysia, Mali, Mauritania, Mauritius, Morocco, Mozambique, Myanmar, Namibia, Nicaragua, Niger, Nigeria, Pakistan, Panama, Papua New Guinea, Paraguay, Peru, Philippines, Poland, Reunion, Romania, Rwanda,

Senegal, Sierra Leone, Somalia, South Africa, Sri Lanka, Sudan, Suriname, Swaziland, Syrian Arab Republic, Thailand, Togo, Tunisia, Turkey, Uganda, Uruguay, Yemen, Zaire, Zambia, Zimbabwe

GSR (42): Argentina, Australia, Austria, Barbados, Belgium, Bulgaria , Canada, Cyprus, Denmark, Finland, France, Germany, Greece, Hong Kong, Hungary, Iceland, Ireland, Israel, Italy, Japan, Kuwait, Luxembourg, Malta, Mexico, Netherlands, New Zealand, Norway, Oman, Portugal, Puerto Rico, Qatar, Saudi Arabia, Singapore, Spain, Sweden, Switzerland, Trinidad and Tobago, U.K, U.S.A, United Arab Emirates, Venezuela, Yugoslavia

GSR (121): *GSR* (126) excluding Belize, Bulgaria, Kuwait, Qatar, United Arab Emirates

GSR (38): *GSR* (42) excluding Bulgaria, Kuwait, Qatar, United Arab Emirates

2.6.3 Data Sources

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Chapter 3

The Intertemporal Allocation of Consumption, Time Preference, and Life-history Strategies

3.1 Introduction

Intrinsic human characteristics are the end products of natural selection. The first study to formalize this notion in economics is by Hansson and Stuart (1990). Incorporating this evolutionary concept into a theory of preferences, they argued that naturally selected preferences are the preferences that maximize fitness, and that, in equilibrium, the marginal rate of substitution in utility is equal to the marginal rate of substitution in fitness.

Rogers (1994) applied this idea to explain time preference. He contended that human time preference is also in evolutionary equilibrium, and that the rate of time preference is given by the marginal rate of substitution in fitness between present and future consumption.

Sozou and Seymour (2003) further examined the relationship between time discounting and fitness by applying life history theory, or more specifically, the disposable soma theory (Kirkwood 1977; Kirkwood and Rose 1991). Life history theory is an analytical framework in biology to study species-specific life-history strategies, such as the age-trajectory of fertility, the timing of maturity, and the age-trajectory of mortality, presuming that these life-history traits are the results of adaptation to a unique environment. The disposable soma theory, in particular, suggests that the optimal

strategy is to age, and not to have an indefinite life in the natural environment in which extrinsic mortality is high. Intuitively, this is because the maintenance of the body competes with immediate reproduction for limited resources. Investing in maintenance to the level of immortality is simply too costly compared with investing in immediate reproduction.

This could be interpreted that senescence is the result of discounting the future. The future, which is uncertain in nature, is less important than the present in terms of fitness. As a result, fewer resources are allocated for the future, or maintenance of the body, and senescence becomes the optimal life-history strategy. Sozou and Seymour (2003) incorporated this idea and argued that time discounting can be measured by the rate of aging, which is equal to the sum of mortality rate and the rate of decline in fertility.[1]

This study follows these studies and examines the relationship between life-history strategies and time preference. In particular, it incorporates the transfer of resources among individuals. As humans significantly rely on the transfer of resources for their survival and reproduction, it could have had considerable impacts on human life-history traits.[2]

Deriving time preference from life-history strategies does not, however, negate the relationship between time preference and non-biological factors. Social factors, such as learning and culture, can affect time discounting

[1]Other studies that referred to the relationship between life history theory and time preference include Hill (1993) and Robson and Kaplan (2003). Acharya and Balvers (2004) also viewed time preference as the end product of natural selection, but from a different perspective. They assumed that individuals make their life-cycle consumption choices to maximize life expectancy, presuming that life expectancy would approximately mirror the expected number of offspring. Based on this assumption, they showed that time preference corresponds to mortality.

[2]Recently, Chu et al. (2010) and Robson and Samuelson (2009) have also examined time preference in the context of life history theory. Among a number of differences between Chu et al. (2010), Robson and Samuelson (2009), and the present study, the most important differences lie in model specification. Chu et al. (2010) incorporate intergenerational transfers with the assumption that the transfers require additional costs which are increasing in the amount of transfers. The present study does not assume the existence of such costs, but instead, assumes that reproduction technology is concave, not linear, in resources. Consequently, the results of Chu et al. (2010) and the present study do not necessarily match. Robson and Samuelson (2009), on the other hand, directly measure time preference using the output of reproduction, i.e., the number of offspring, without specifying how resources are utilized for reproduction. As the value of each offspring is equivalent regardless of parent's age, it is easily seen that the rate of time preference is given by the mortality rate in a stable environment. This result is equivalent to the result in the present study. However, the present study measures time preference using the input of reproduction, i.e., consumption. It seems that Robson and Samuelson (2009) use offspring as the measure of time preference in order to focus on the biological origin of hyperbolic discounting.

(Becker and Mulligan 1997). Similarly, it is not surprising if psychological and age-related factors, which may also be the end-products of life-history strategies, are related to time preference (Trostel and Taylor 2001). Furthermore, survival is not the only risk factor. This study aims to assess the bio-evolutionary basis of time preference that may be equivalent to the 'endowed discount factor' in Becker and Mulligan (1997).

The rest of the chapter is organized as follows. Section 3.2 presents human characteristics in the framework of life history theory in order to establish a common basis for applying life history theory to humans. Section 3.3 specifies the model and examines human life-history strategies. The main finding is that the age-specific mortality rate reflects the value of survival, which in turn depends on future reproductive and productive contributions. Sections 3.4 and 3.5 evaluate time discounting in the context of life-history strategies, and illustrate that time discounting reflects the age-variation in the value of survival in the same way as mortality does. In addition, the results suggest that our biologically endowed rate of time preference is positive, reaches its lowest in early adulthood and increases thereafter, and is higher when exchange transactions involve a reduction in current consumption than when they involve an increase in current consumption (intertemporal loss aversion/the sign effect). These implications do not contradict empirical findings. Section 3.6 concludes.

3.2 Life History Theory and Human Traits

Life history theory provides an analytical framework for studying the relationship between species-specific life-history strategies and fitness (see, for example, Stearns 1992). When the population reaches the upper limit of the carrying capacity and remains stationary, the measure of fitness is given by the expected number of offspring at the beginning of life;[3]

$$R_1 = \sum_{x=1}^{\infty} l_x b_x. \tag{3.1}$$

Here, l_x is the survival probability up to age x ($x \geq 1$) and b_x is the reproductive contribution at age x. This is equivalent to the reproductive value at birth under stationary population. With the pressure of natural selection, the genotypes and the associated phenotypes (strategies) that generate a higher value of R_1 have spread out and remained in the current population.

[3] Although it is technically straightforward to incorporate population growth into the model, it is theoretically difficult to justify.

More generally, the reproductive value at age j under stationary population can be written as

$$R_j = \sum_{x=j}^{\infty} \frac{l_x}{l_j} b_x = b_j + \frac{l_{j+1}}{l_j} R_{j+1} \tag{3.2}$$

where the second equality shows that the reproductive value is the sum of the current and future contributions to reproduction. The relationship between these two terms represents the essence of life history theory, i.e., the trade-off between reproduction and survival (Williams 1966). As both current reproductive contribution, b_j, and the survival probability to the next period, $\frac{l_{j+1}}{l_j}$, depend on the amount of energy allocated, spending more on one means spending less on the other.

This trade-off actually represents the trade-off between current and future reproduction. Survival is merely a means for future reproduction. Thus, if current reproduction becomes more important relative to future reproduction, more resources would be allocated to current reproduction and fewer to survival. The relative importance of current and future reproduction determines the allocation of resources between immediate reproduction and survival.

Applying life history theory to a particular species, the surrounding environment of the species needs to be taken into account. In the case of humans, the surrounding environment is considered to be the African savannah, in which humans (the genus *Homo*) existed for most of their two-million-year history. In this environment, humans lived as hunters and gatherers and created their unique society. Most of our intrinsic traits (strategies) that separate us from chimpanzees, our closest living relatives, presumably result from our adaptation to this ancestral environment.

Incorporating the surrounding environment, the mechanism connecting the environment and species-specific characteristics can be examined in the framework of life history theory. For example, Robson and Kaplan (2003) analyzed intelligence in connection with longevity, and showed that these distinctive traits have evolved together as life-history strategies to adapt in African savannahs to gather nutrient-dense food.

The significance of resource transfers between individuals is another example of human characteristics. Obviously, the transfer of energy, including parental care, allomothering, and cooperative breeding, is not limited to humans. Inter-individual transfers of energy are widely observed across species as suggested by kin selection theory (Hamilton 1964), and intergenerational

transfers, in particular, are recognized as a crucial component to shape life-history strategies (Lee 2003).

Nonetheless, the transfer of resources is still considered to be one of the most prominent features of human society. One reason for this is that resource transfers among humans are substantial in size. For example, Kaplan et al. (2000) compared the age-trajectories of consumption and production between human hunter-gatherers and chimpanzees, and showed that hunter-gatherer men produce twice as much as they consume, whereas chimpanzee males produce just as much. The surplus of energy of human males is utilized to support reproduction. The magnitude of resource transfers is one of the prominent features that separates us from chimpanzees.

Another reason suggesting the significance of resource transfers among humans is found in the variety of relationships between the donor and the recipient. Among humans, the relationship of the donor and the recipient of a transfer is not limited to a specific relationship. The donor can be a mother, a father, an aged parent, a grandparent, a child, a spouse, a sibling, a relative, or even an unrelated individual. Consequently, the type of the recipient is also diverse. On the other hand, in most of the other species, the relationship of the donor and the recipient is limited to a particular relationship, such as the mother-offspring relationship in most mammals and inter-sibling relationship among eusocial insects.[4] Thus, the diversity in relationships between the donor and the recipient is also considered to be one of our distinctive features.

For these reasons, the transfer of resources is regarded as one of the crucial factors that affect human evolutionary process in a number of studies.[5] For example, intergenerational transfers, as well as learning, play critical roles in promoting the coevolution of intelligence and longevity (Robson and Kaplan 2003). Similarly, the grandmother hypothesis (Hawkes 2003) is based on intergenerational transfers made from grandmothers to their daughters and grandchildren.

Nevertheless, the definition of resource transfers is not straightforward. In a broader sense, pregnancy, for example, can be considered to be an intergenerational transfer because the basic necessities for survival are transferred to the unborn offspring from the mother. Generally speaking, however, pregnancy, as well as other energy transfers before birth, is considered to be a part of fertility.

[4]See Carey and Gruenfelder (1997) for the roles of the elderly and intergenerational transfers in other species.

[5]The mechanism that promotes the evolution of intergenerational transfers is studied by Chu and Lee (2006).

To avoid this kind of overlap, the transfer of resources is defined in this study as the difference between production and consumption as in Kaplan (1994). This indicates that all transfers are in the form of material resources. In other words, as long as transfers consist of materials, they are not limited to the transfers from parents to offspring and can be arranged between any types of individuals. In contrast, transfers of a non-material basis are included in reproductive contribution, implying that reproductive contribution covers not only fertility but also the transfer of energy when resources are consumed and processed into energy by other individuals. This includes breast-feeding, protection, warmth, and teaching by both parents and non-parents.

The next section presents a model and solves for optimal life-history strategies when individuals intertemporally exchange resources.[6]

3.3 Optimal Life History

Focusing on life-history events after maturity, let the reproductive contribution of an individual at age x be

$$b_x = A_x V_x^{\gamma} \tag{3.3}$$

where A_x is reproductive efficiency, V_x is the energy contributed to reproduction, and γ is the parameter with $0 < \gamma < 1$. Reproductive efficiency, A_x, represents the productivity connecting energy input and reproductive output through childbirth and childcare. As it often deteriorates with senescence, A_x is expected to be higher (more efficient) when the individual is young. The parameter, γ, on the other hand, expresses the degree of concavity of reproductive output, b_x. Since the time and ability allocated for reproduction are not infinite during each period and the productivity of reproduction is expected to decline as reproductive contribution increases, b_x rises with V_x but at a decreasing rate.

Next, suppose that all death at any age occurs at the end of the period, and that the age-specific mortality rate is given by

$$m_x = e^{-qW_x} \tag{3.4}$$

[6]Another important question would be how such strategies become dominant. As these strategies require transfers between individuals, they need to overcome various difficulties such as invading free-riding mutant strains and, in a two-sex world, low and ambiguous genetic relatedness which possibly makes individuals less altruistic to offspring and which causes other free-riding problems between sexes.

where q is a parameter representing maintenance efficiency and W_x is the energy allocated for survival.[7] Equation (3.4) shows that the chance of death is certain at $W_x = 0$, and decreases with W_x. Subsequently, given that $e^{-qW_0} = 0$, the survival probability at age x becomes

$$l_x = \prod_{i=0}^{x-1} \left(1 - e^{-qW_i}\right). \tag{3.5}$$

The energy for V_x and W_x comes from consumption. At each period, the individual consumes resources and converts them into energy. Then, the energy is physiologically allocated to V_x and W_x.

On the production side, let y_x be the amount of production which possibly depends on age. Age-dependent factors, such as experience, learning, and physical strength, may affect the amount of production. Then, incorporating resource transfers between individuals, the budget constraint becomes

$$\sum_{x=1}^{\infty} l_x \left(y_x - V_x - W_x\right) \geq 0. \tag{3.6}$$

The budget constraint does not necessarily hold at each period at the individual level, but it must be met at a lifetime level so that the expected lifetime consumption does not exceed the expected lifetime income. Individuals receive resources from older generations when they are young and their reproductive values are high, but instead, they transfer resources to younger generations later in their lives.

It is worth noting that the amount of resources they repay exceeds the amount they receive. This is because survivors need to pay back for those who are dead, such as their sisters and brothers, by supporting orphans. The community works as an extended family to share mortality risk.

Equation (3.6) is also the intratemporal aggregate budget constraint of the community when the technology to store resources is not available. In this situation, x represents an individual in the community. As shown by Lee (1997), it is generally the case that older individuals transfer resources to younger individuals, such as their own children, nieces, and nephews.

Given these conditions, the next step is to solve for the optimal allocation of resources that maximizes the reproductive value, R_1 (see Taylor et al. 1974; Baudisch 2008 for the usage of fitness measures in optimization problems). Among various methods to solve this problem, the Lagrangian

[7] Although maintenance efficiency actually varies with age, it is simplified to be constant since relaxing it to age-dependent does not change the results.

method is applied here since the problem can be expressed as a simple static optimization problem in which all values are measured at the beginning of life. Substituting b_x in equation (3.3) into equation (3.1) and using equation (3.6), the Lagrangian is defined as

$$L(V_x, W_x, \phi) = \sum_{x=1}^{\infty} l_x A_x V_x^{\gamma} + \phi \left[\sum_{x=1}^{\infty} l_x \left(y_x - V_x - W_x \right) \right] \tag{3.7}$$

where ϕ is the Lagrange multiplier. Recalling that $l_x = \prod_{i=0}^{x-1} \left(1 - e^{-qW_i}\right)$, the first order conditions for any arbitrary age, j, are given by

$$\frac{\partial L}{\partial V_j} = l_j \gamma A_j V_j^{\gamma-1} - \phi l_j = 0, \tag{3.8}$$

$$\begin{aligned} \frac{\partial L}{\partial W_j} = & \sum_{x=j+1}^{\infty} \prod_{i=0}^{x-1} \left(1 - e^{-qW_i}\right) A_x V_x^{\gamma} \frac{q e^{-qW_j}}{1 - e^{-qW_j}} \\ & + \phi \sum_{x=j+1}^{\infty} \prod_{i=0}^{x-1} \left(1 - e^{-qW_i}\right) \left(y_x - V_x - W_x\right) \frac{q e^{-qW_j}}{1 - e^{-qW_j}} \\ & - \phi \prod_{i=0}^{j-1} \left(1 - e^{-qW_i}\right) = 0. \end{aligned} \tag{3.9}$$

To interpret the meaning, these equations are reorganized as follows. First, equation (3.8) can be rewritten as

$$\gamma A_j V_j^{\gamma-1} = \phi. \tag{3.10}$$

This indicates that the shadow price is equal to the marginal benefits of immediate reproductive investment, and that they are constant across ages.

Second, equation (3.9) can be rewritten as

$$q e^{-qW_j} \sum_{x=j+1}^{\infty} \frac{l_x}{l_{j+1}} A_x V_x^{\gamma} + q e^{-qW_j} \phi \sum_{x=j+1}^{\infty} \frac{l_x}{l_{j+1}} \left(y_x - V_x - W_x\right) = \phi, \tag{3.11}$$

or equivalently,

$$q e^{-qW_j} \left(R_{j+1} + \phi k_{j+1}\right) = \phi \tag{3.12}$$

where R_{j+1} and k_{j+1} are respectively equal to $\sum_{x=j+1}^{\infty} \frac{l_x}{l_{j+1}} A_x V_x^{\gamma}$, the reproductive value at age $j+1$, and $\sum_{x=j+1}^{\infty} \frac{l_x}{l_{j+1}} \left(y_x - V_x - W_x\right)$, the accumulated productive surplus that the individual is expected to obtain at age $j+1$ onwards. Here, R_{j+1} can be interpreted as the value of survival to age $j+1$

in terms of reproduction because the advantage of surviving to age $j+1$ in terms of reproduction is expressed in R_{j+1}. Similarly, k_{j+1} can be regarded as the value of survival to age $j+1$ in terms of *production*. The benefit of surviving to the next period in terms of production is presented by k_{j+1}.

However, obtaining a productive surplus is not itself the point of survival. Production is beneficial because productive output can be converted to reproductive contribution. This is why k_{j+1} is multiplied by ϕ, which is interpreted as the rate of exchange between productive surplus and reproductive contribution. Namely, ϕk_{j+1} represents the value of the accumulated productive surplus in the future in terms of reproduction.

Added together, the terms in the parentheses in equation (3.12), $R_{j+1} + \phi k_{j+1}$, can be interpreted as *the value of survival* that includes both direct (reproductive) and indirect (productive) contributions.[8] By surviving to age $j+1$, the individual currently at age j obtains $R_{j+1} + \phi k_{j+1}$. Therefore, given that qe^{-qW_j} is the marginal effect of W_j on the survival probability to the next period, equation (3.12) shows that the marginal benefit of survival investment is equal to its marginal cost represented by ϕ, and that they are constant across ages.

Finally, equating equations (3.8) and (3.9), the relationship between V_j and W_j can be expressed as

$$\gamma A_j V_j^{\gamma-1} = \frac{qe^{-qW_j} R_{j+1}}{1 - qe^{-qW_j} k_{j+1}}. \tag{3.13}$$

This shows that the marginal benefits of reproductive and survival investments must be equal intratemporally.

Equations (3.10), (3.12), and (3.13) present the optimal life-history strategies, i.e., the age-trajectories of reproductive contribution and survival. In particular, focusing on the age-trajectory of mortality, equation (3.13) can be rewritten as

$$e^{-qW_j} = \frac{\phi}{q} \frac{1}{R_{j+1} + \phi k_{j+1}}. \tag{3.14}$$

Equation (3.14) shows that the mortality rate reflects the value of survival, $R_{j+1} + \phi k_{j+1}$, given that ϕ and q are constant.

Now, assume for a moment that A_x and y_x are constant without any biological age limit. In this hypothetical case, age has no meaning, but is merely the number of years after birth. Individuals of all ages are basically

[8] They are respectively interpreted as the Hamilton and Transfer effects in Chu et al. (2008).

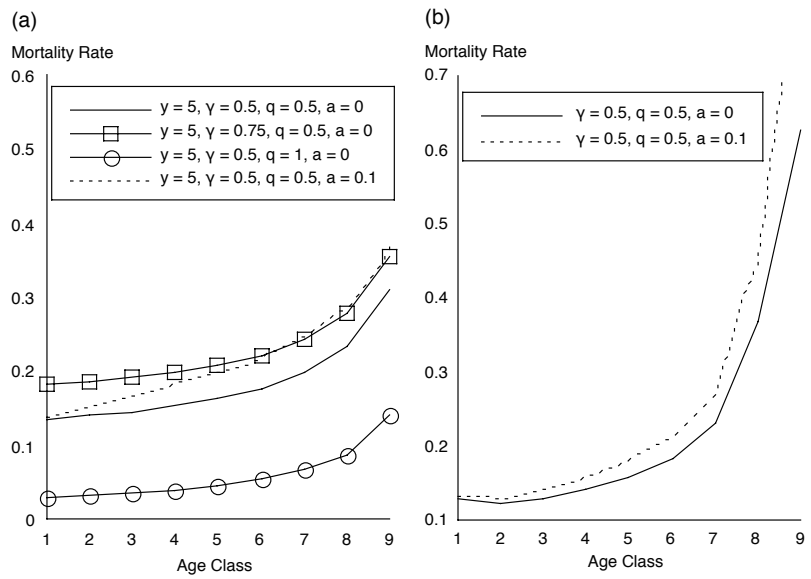
equipped with the same physiological quality. This indicates that the relative importance of the present to the future is constant across ages, and that all individuals allocate resources in the same manner. Thus, V_x and W_x are constant and the transfer of resources is equal to zero at all ages, implying that the mortality rate remains constant across ages. Consequently, R_{x+1} is constant and k_{x+1} is equal to zero for all ages. Individuals do not senesce in this hypothetical case.

However, age matters to determinate growers, including humans, that stop growing at maturity. Among determinate growers, A_x is generally expected to decrease with age after maturity. As a result, R_{x+1} often hits its peak at maturity and decreases thereafter. This is the reason why an upward mortality trend after maturity is common among determinate growers. In a similar manner, the value of k_{x+1} depends on the age-trajectory of y_x. Nevertheless, the age-trajectory of k_{x+1} is not simple for humans, as human productivity increases even after maturity. For example, human productivity in the natural environment is expected to increase until individuals reach middle age (Kaplan et al. 2000).[9] This indicates that the direction of the change in mortality rate, i.e., whether it decreases or increases, depends on the size of the change in ϕk_{x+1} relative to R_{x+1}. If the contribution of the increase in k_{x+1} outweighs the decline in R_{x+1}, the mortality rate may decrease.

To examine the significance of the age-variation in y_x as well as the influences of other parameters, the age-trajectories of mortality rates are calculated with various sets of parameter values. The results, presented in Figure 3.1, show that mortality generally increases with age. The decline in the value of survival (senescence) raises the mortality rate. The exceptional case is when the gain in k_{x+1} is significant enough to offset the decline in R_{x+1}. As shown in Figure 3.1(b), mortality can decrease in the early part of adulthood when the gain in productivity is relatively large and the value of survival increases.

This case may be close to the actual age-trajectory of human mortality in the ancestral environment. For example, Hill and Hurtado (1996) found that the age-specific mortality of the *Ache* people, a hunter-gatherer population in Paraguay, hits its lowest in early adulthood, at around 20 years of age, and increases thereafter. Hill et al. (2007) also reported a similar pattern for the *Hiwi* people, a hunter-gatherer population in Venezuela. Presuming that the

[9]Intergenerational transfers are all the more important because the changes in productivity and the reproductive value do not move together. If they move in parallel, intergenerational transfers may be simply redundant.



(a) The impacts of changes in parameter values, and (b) the impacts of the change in y (The values of y between ages 1 and 10 are, respectively, equal to $2, 4, 6, 6, 6, 6, 6, 4, 2$). In each graph, lifespan is divided into ten age-classes with one age-class corresponding to five years, assuming that the age of maturity is 15 years old and that the maximum lifespan is 65 years old. Limiting the maximum lifespan to 65 years is done merely for computational simplicity. As for A_x, it is assumed to follow the equation $A_{x+1} = A_x/(1+a)$ with $a \geq 0$ until the 10th age-class, and to be equal to zero in the 11th age-class onwards.

Figure 3.1: Simulated age-trajectories of mortality rate

age-trajectory of mortality in the ancient environment can be extrapolated from the trajectories observed in recent hunter-gatherer populations, human mortality in the natural environment is expected to reach its lowest point in early adulthood.

3.4 Resource Allocation and Preferences

As shown in the previous section, the importance of present reproduction relative to future reproduction shapes the age-trajectories of reproductive contribution and mortality. This indicates that the intratemporal and intertemporal allocation of resources is simultaneously determined by the same force.

The mechanism regulating the intratemporal allocation is expressed in equation (3.13), i.e., equalizing the marginal benefits of reproductive and survival investments. This allocation task is primarily managed by our physiology. As the neuroendocrine systems (or hormones) regulate the allocation of consumed energy, we, as humans, have virtually no means of deliberately controlling the allocation of consumed energy between reproductive contribution and survival. In this aspect, the intratemporal allocation is not related to our behavior. Although we can partly influence the allocation of energy by determining what to consume and how to behave, for example, by consuming more positional goods to increase the chances of mating instead of consuming foods to raise the chance of survival or by taking risky actions to attract the members of the opposite sex, the intratemporal allocation of energy depends more on our physiology than on our behavior.

Next, turning to the intertemporal side, the age-trajectory of consumption ($C_x = V_x + W_x$) suggests that a greater amount of resources is allocated to early adulthood. The optimal age-trajectories of consumption corresponding to the age-trajectories of mortality presented in Figure 3.1(b) are depicted in Figure 3.2. It shows that C_x decreases with age, except possibly in early adulthood. Senescence reduces the optimal level of consumption. In other words, we consume less when we are old so that we can consume more when we are young. The availability of intergenerational transfers makes this strategy possible.

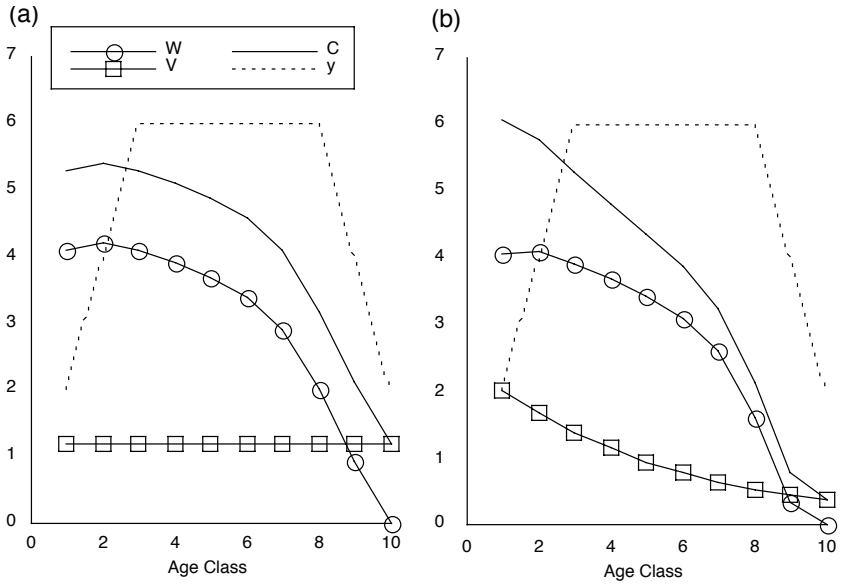
A possible rise in C_x in the early part of adulthood is due to the increase in survival investment. If the increase in W_x offsets the reduction in V_x, C_x would grow with age. As W_x reflects the value of survival, C_x is also influenced by the change in the value of survival.

The intertemporal aspect can be examined further with the intertemporal marginal rates of substitution in reproduction, $MRSR_x$, which is defined as $-\frac{dC_{x+1}}{dC_x} = \frac{\partial R_1^*/\partial C_x}{\partial R_1^*/\partial C_{x+1}}$. Here, the superscript $*$ is added to stress that this is the fitness-maximizing optimized value. Then, since C_x is allocated to either V_x or W_x so that their marginal benefits are constant, $\frac{\partial R_1^*}{\partial C_x}$ is given by ϕl_x, and $MRSR_x$ on the optimal path becomes

$$MRSR_x = \left(\frac{l_x}{l_{x+1}}\right)^* \approx 1 + m_x^*. \tag{3.15}$$

Equation (3.15) shows that $MRSR_x$ is approximately equal to $1 + m_x$.[10] This indicates that the compensation rate to give up one unit of current

[10]Consumption is not necessarily constant across ages as it is calculated on the optimal path. It coincides with $MRSR_x$ on the constant consumption path when individuals do not senesce.



The two graphs correspond to the two sets of the parameter values used in Figure 3.1 (b). In (a), A_x is constant, and in (b), A_x is decreasing.

Figure 3.2: Age-trajectories of consumption

consumption for future consumption is given by the age-specific mortality rate alone.

The intertemporal allocation is, however, not involuntary. It significantly depends on our behavior as we can deliberately control it by deciding how much to consume at present. Thus, we may possibly behave in a non-optimal way without an appropriate mechanism that coordinates our behavior.

Preferences can serve as this mechanism, as our behavior depends on them. Preferences that lead to non-optimal behavior are eliminated in the course of evolution and those that generate a higher reproductive value in the ancient environment have spread out and remained in the current population. In other words, the behavior that yields a higher reproductive value also provides a higher utility, at least in the ancient past. Consequently, the rate of time preference embedded in us needs to be the rate that prevents us from deviating from the the fitness-maximizing consumption path and must be consistent with $MRSR_x$.

In a utility-maximization model, this can be interpreted as we discount the future consumption in line with equation (3.15). Considering a typical

case that the instantaneous utility function and the rate of time preference at age x are respectively given by $u(C_x; x)$ and β_x where $u'(C_x) > 0$ and $u''(C_x) < 0$, the intertemporal marginal rate of substitution in utility, $MRSU_x$, becomes

$$MRSU_x = (1 + \beta_x) \frac{u'(C_x; x)}{u'(C_{x+1}; x + 1)}. \tag{3.16}$$

An interesting case, in particular, would be the one that instantaneous utility function mirrors the effect of consumption on the reproductive value at the corresponding age such that the marginal utilities measured at the fitness-maximizing levels of consumption are constant across ages. As the benefit of consumption on the reproductive value varies with age, the instantaneous utility function is expected to be age-dependent as well. This age-dependency induces the individual to consume more while the marginal return on reproductive value is high. This type of behavior is also consistent with our tendency to eat less as we age. In this case, the rate of time preference that equates $MRSU_x$ to $MRSR_x$ and prevents the individual from deviating is equal to the age-specific mortality rate, since $u'(C_x; x)/u'(C_{x+1}; x + 1) = 1$ at the fitness-maximizing levels of consumption. Therefore, if preferences are properly specified in the bio-evolutionary context, the individual who maximizes one's own utility can allocate resources in the fitness-maximizing manner.[11]

3.5 Implications

3.5.1 Time preference and mortality

Considering that our preferences are evolutionarily optimal, our biologically endowed time preference is expressed by mortality alone. This result differs

[11] In connection to economics, two other points need to be discussed. The first point is the age-dependency of the instantaneous utility function. When the instantaneous utility function is independent of age, the rate of time preference is not equal to the age-specific mortality rate. However, there are no biological basis to suppose that the instantaneous utility function is age-independent. The second point is the cognitive ability of one's own survival probability. If the individual is able to perceive one's own survival probability and discount future utility accordingly, the rate of time preference embedded in one's mind would be equal to 0 as long as the instantaneous utility function is age-dependent. On the other hand, when the instantaneous utility function is age-independent, the embedded time preference rate would be either positive or negative depending on the age-variation in fitness-maximizing level of consumption. However, there is also no biological foundation to suppose that we are able to correctly perceive our own survival probability.

from Sozou and Seymour (2003). In their setting, the age-variation in reproductive efficiency appears in the right-hand side of equation (3.15). In the present study, the transfer of resources absorbs the age-variation in A_x by equalizing the marginal benefits of reproductive contribution. Without the transfer of resources, it would appear in the right-hand side of the equation.

Subsequently, the age-trajectory of our biologically endowed time preference is given by that of mortality. Thus, the rate of time preference is always positive. Furthermore, since time preference is considered to be psychologically set in our minds in the evolutionary process, it is equal to the age-trajectory of mortality in the ancient past when we existed as hunters and gatherers, not the age-trajectory of mortality in the current environment. Therefore, our biologically endowed time preference is expected to hit its lowest in early adulthood and increase thereafter.

These implications are not novel from an empirical perspective. A number of studies have examined the relationship between age and time preference (Green et al. 1994; Trostel and Taylor 2001; Ventura 2003; Bishai 2004; Read and Read 2004), and Trostel and Taylor (2001) and Read and Read (2004) found that time preference increases with senescence. The difference between these two studies lies in the age at which time preference is at its lowest. The results of Trostel and Taylor (2001) suggest that time preference continues to increase during adulthood, indicating that it is lowest among individuals in their twenties. On the other hand, in Read and Read (2004), the rate of time preference is lowest among individuals in their forties. The results obtained in the present study are closer to the empirical findings of Trostel and Taylor (2001), although time preference in the present study represents only the biologically endowed time preference.

3.5.2 Time preference and empathy

Departing from the utility-maximizing framework, being endowed with the appropriate rate of time preference is not the only solution. If individuals possess the ability to share resources *intratemporally* with others so that the marginal utilities of consumption are equal, it would also generate a consumption profile that is consistent with the fitness-maximizing profile as long as the instantaneous utility function reflects the benefit of consumption on the reproductive value. This is because, given the stationarity of the population and equation (3.6) that is now regarded as the intratemporal aggregate budget constraint, allocating resources *intratemporally* so that the marginal utilities are equal across individuals within the same period is equivalent to allocating consumption *intertemporally* so that the marginal

utilities are constant across ages. This allocation strategy yields the same consumption profile as the utility-maximizing allocation, and corresponds to the fitness-maximizing allocation.

This type of behavior is indeed consistent with anthropological findings that hunter-gatherer populations share food according to their needs (see, for example, Kaplan and Gurven 2005). In this case, individuals do not need to take the intertemporal allocation of consumption into consideration, rendering the intertemporal perspective redundant.

Speculating that humans are endowed with this kind of ability and can share consumption according to our needs is justifiable if empathy, or a set of moral codes built out of empathy (Baron-Cohen 2005), is incorporated. Empathy creates affective feelings toward others by allowing the individual to infer, understand, and/or share another's emotional state. In particular, humans, and perhaps apes among primates, are known to have acute cognitive abilities to empathize. These species have the ability to take another's perspective and respond with appropriate emotion without losing self-identity (see, for example, Decety and Jackson 2004; de Waal 2008 for reviews). This ability, called empathic perspective-taking, helps an individual to understand another's specific situation and needs, and allows that individual to engage in other-regarding pro-social behaviors, such as food sharing.

Comparing time preference and empathy, it is advantageous to have time preference if there are any reasons that require intertemporal consideration. For example, when there is a seasonal or daily variation in the amount of food that individuals can possibly hunt or gather, empathy alone can not adequately cope. Since smoothing of the actual production level can contribute to one's own reproduction, it is beneficial to have time preference.

Nevertheless, the relationship between time preference and empathy may not be mutually exclusive. As discussed in psychological literature, they may be related in their origins (Posner 1995; Frederick 2003; Pronin et al. 2008). According to these studies, the present self and the future selves are virtually separate individuals, and time preferences are the reflection of the importance of the present self relative to the future selves. In particular, the experiments in Pronin et al. (2008) showed that the decisions people make for the future selves and for other people are similar. In this context, time preferences can be interpreted as one form of empathy, i.e., empathy for the future selves. In this sense, empathy and time preference share the same root.

3.5.3 Intertemporal loss aversion (Sign effect)

Equations (3.15) indicates that $MRSR_x$ is not independent of the *intratemporal* allocation of resources. The change in survival investment affects mortality rate and consequently $MRSR_x$, whereas the change in reproductive investment does not.[12] Even if the changes in W_x and V_x are of the same degree and their impacts on R_1 are the same, their effects on mortality are different. For example, let j be an arbitrary age that corresponds to the current period, and suppose that the individual gives up a unit of present consumption for next-period consumption. If the reduction in C_j leads to the reduction in W_j, it would raise mortality and $MRSR_j$, whereas they would not be affected if the reduction in C_j is fully absorbed by V_j. Thus, in this case, $MRSR_j$ would be higher if the reduction in C_j is absorbed by W_j. The compatible result holds when the change in C_j is in the opposite direction. $MRSR_j$ would be lower if the increase in C_j is absorbed by W_j.

It is not surprising if this asymmetry has influenced our preferences. Presuming that the change in C_j affects W_j, we ought to place a higher premium on the reduction than on the increase in current consumption.

This result is consistent with loss aversion (Kahneman and Tversky 1979; Thaler 1980). A number of experimental studies have shown that people place more weight on the disutility from losses than on the utility from corresponding gains (see, for example, Rabin 1998; DellaVigna 2007 for reviews). In particular, this accords with intertemporal loss aversion (Loewenstein and Prelec 1992) and the sign effect.[13] This issue has traditionally been studied in the framework of psychology and bounded rationality. The result here complements these psychological studies by providing a bio-evolutionary perspective.

3.6 Concluding Remarks

The focus of this study is the mechanism that coordinates our intertemporal choice. By incorporating a bio-evolutionary perspective, this study offers an possible explanation to our time preference and intertemporal loss aversion (sign effect).

In a broader perspective, this study looks into the biological foundation of preferences and motivation. Biological studies, including ecological studies and evolutionary game theory, investigate the relationship between evo-

[12] Technically, this is because the objective function, R_1, is not time-separable.

[13] The term "sign effect" is more widely used than "intertemporal loss aversion". See Ikeda et al. (2010) for a review.

lutionary background and behavior. Economic studies, on the other hand, focus on the relationship between preferences and behavior. Examining behavior from these two perspectives sheds light on the relationship between bio-evolutionary background and preferences. Explaining preferences with biological models and incorporating the results into economic models enrich the understanding of our decision-making mechanism.

This method is also beneficial to study anomalies found in psychology, including evolutionary psychology. If we can rationalize and formulate these anomalies with biological models, we can build economic models on solid ground.

Chapter 4

Why Are Children Impatient? Evolutionary Selection of Preferences

4.1 Introduction

The decision-making process within a household typically involves multiple individuals such as the wife, husband, and children. As a result, collective decisions made in the household depend on preferences of household members. For example, Dauphin et al. (2011) showed that children as well as parents influence household economic decisions.[1]

Nevertheless, children, who sometimes make up the larger portion of the family, seldom receive much attention in economics. Considering that even an infant is capable of tilting household decisions toward his/her preferred choices using his/her limited but powerful strategies, this gap needs to be addressed.

As a step toward this objective, this study investigates the intertemporal choice of children, focusing on the cause of their impatience. As found in Bettinger and Slonim (2007) and Steinberg et al. (2009), patience increases with age during childhood, suggesting that young children are particularly impatient.[2]

To be more specific, this study searches for the evolutionary root of the

[1] To justify the unitary model in which the unitary decision maker maximizes household utility, we often require restrictive assumptions. The rotten kid theorem (Becker 1974) is such an example. See Bergstrom (1989) for details.

[2] For other psychological studies, see Teuscher and Mitchell (2011) for a review.

impatience of children, employing a biological framework. An increasing body of literature uses this method to search for the biological basis of preferences.

The basic idea of this literature comes from the biological finding that preferences or, more broadly, genotypes and associated phenotypes (strategies), are the end-products of evolution. Since preferences that are successful in reproduction spread over the population in the evolutionary time scale, preferences in the current human population can be deduced from the preferences that maximized fitness in the environment where we evolved. Such environment is considered to be the African savannah where our genus *Homo* appeared two million years ago and stayed for most of our history as hunter-gatherers. Human-specific characteristics are considered to have evolved in this ancestral environment.

Time preference is a leading topic in this literature. Hansson and Stuart (1990) showed that the marginal rate of substitution in utility is equal to the marginal rate of substitution in fitness, suggesting that resource allocation maximizing utility corresponds to allocation maximizing fitness. Later, Rogers (1994) applied this idea to the intertemporal allocation of resources, and explained the evolutionary origin of time preference. More recently, technical similarities for studying aging in bio-demography and the intertemporal allocation in economics have allowed biologists, demographers, and economists to enter this hybrid field, leading to the examination of how time-discounting behavior relates to senescence (Sozou and Seymour 2003) and to intergenerational transfers (Chu et al. 2010), why it is hyperbolic (Robson and Samuelson 2009), the rationale for social discounting (Sozou 2009), how it depends on age (Chapter 3 in this thesis), and how it relates to extrinsic mortality (Chowdhry 2011).[3]

To investigate the impatience of children, the present study follows this literature and extends the model in Chapter 3 by incorporating childhood, i.e., the growth period to maturity. In previous studies, Chu et al. (2010) built a biological model incorporating the growth period and showed that the impatience relates to the productivity growth in childhood. Similarly, Robson et al. (2012) examined the impatience of children, but paid more attention to adulthood and did not explicitly consider the role of the growth period.

Despite these differences, both of these previous studies concluded that

[3]Acharya and Balvers (2004) examined time preference in an economic framework, assuming that utility captures the effect of consumption on mortality. Their model can also be interpreted as a biological model that assumes that reproductive success solely depends on the length of life.

the impatience of children does not relate to the absolute level of mortality. This presents a sharp contrast to the result that mortality is a major factor associated with time preference in adulthood.

The present study, on the other hand, finds that the impatience of children relates to the mortality rate, as does time preference in adulthood. The same logic applies in both childhood and adulthood.

Explaining time preference with evolutionary biology, however, does not negate the relationship between time-discounting behavior and non-biological factors. As Becker and Mulligan (1997) argue, social factors such as culture and education affect time discounting. This study aims to assess the biological basis of time preference, referred to as the 'endowed discount factor' in the above study.

The rest of the study is organized as follows. Section 4.2 examines the evolutionary optimal strategies using a biological framework. Based on the results in Section 4.2, Section 4.3 provides a biological explanation for time preference, and shows that the optimal rate of time preference is equal to the mortality rate in the entire life course. This implies that the biologically endowed rate of time preference is U-shaped in age as is the mortality rate, and that children and old adults are, by nature, less patient than young adults. Section 4.4 concludes.

4.2 The Model

4.2.1 The Basic Structure

The model in this study is based on life history theory, i.e., an analytical framework in biology to study species-specific life-history strategies such as the age-trajectories of fertility and mortality, presuming that life-history traits are the end-products of natural selection. Technically, it solves for the fitness-maximizing strategies under given constraints to deduce species-specific life-history traits.[4]

In particular, as in a standard life-history model, I consider a model with the following properties. First, the population is stationary at the upper limit of the carrying capacity. This is to follow the carrying-capacity argument that human population in our evolutionary past was confined by the environmental capacity.[5] Second, reproduction is asexual. This is simply

[4]See Stearns (1992) for the general introduction of life history theory, and Perrin and Sibly (1993) for the technical introduction.

[5]It is technically possible to examine the case that the population growth rate takes non-zero constant values. See Taylor et al. (1974).

to avoid complexities related to matching between females and males.

With these specifications, the measure of fitness is given by the expected number of offspring at the beginning of life, which is expressed as

$$R(0) = \int_0^\infty l(x)m(x)dx \tag{4.1}$$

where $l(x)$ is the survival probability to age x and $m(x)$ is the immediate reproductive output at age x.[6] With the pressure of natural selection, genotypes and associated phenotypes that generate a higher value of $R(0)$ spread over and fix in the population. Note that, in a stationary population where the population growth rate converges to zero, $R(0)$ is equivalent to the reproductive value at birth and converges to one. The lifetime expected number of offspring is, *ex post*, just sufficient to replace the current individual.

Survival and reproduction depend on age and the amount of energetic resources respectively invested in. Once consumed, resources are physiologically allocated to either survival investment or reproductive investment. Therefore, given that

$$l(x) = e^{-\int_0^x \mu(\hat{x})d\hat{x}} \tag{4.2}$$

where $\mu(x)$ is the mortality rate at age x, the dependence of survival and reproduction on resources can be expressed as $\mu(x) = \mu[w(x), x]$ and $m(x) = m[v(x), x]$ where $w(x)$ and $v(x)$ are respectively survival and reproductive investments. To avoid unnecessary technical complexity, I assume that both investments exhibit diminishing marginal returns, satisfying $\mu_w[w(x), x] < 0$, $\mu_{ww}[w(x), x] > 0$, $m_v[v(x), x] > 0$, and $m_{vv}[v(x), x] < 0$ where the subscript indicates a partial derivative, and also that the individual will certainly die without survival investment and will not have any reproductive output without reproductive investment.

In addition, reproduction depends on growth investment, $z(x)$, in the earlier stages of life. Investing in growth enhances the reproductive capacity and increases the reproductive efficiency in the later stages.

To incorporate this relationship, I focus on determinate growers, i.e., organisms that stop growing at maturity, as in the case of humans.[7] Thus, denoting the age of maturity by α, we have $v(x) = 0$ for all $x < \alpha$ and $z(x) = 0$ for all $x \geq \alpha$.

[6] Equation (4.1) does not imply that lifespan is infinite.

[7] To account for determinate growth, we can, for example, assume that the transition from the growth phase to the reproductive phase, or the other way around, incurs significant costs. This is consistent with the fact that determinate growers go through some sort of metamorphosis at maturity.

Besides, I assume that $m[v(x), x]$ can be separated into the age-dependent reproductive efficiency, $A(x)$, and the contribution of resources, $f[v(x)]$, such that

$$m[v(x), x] = A(x)f[v(x)]. \tag{4.3}$$

Note that $f[v(x)]$ is concave in $v(x)$ as is $m[v(x), x]$. In this setting, the development of the reproductive capacity during the growth period can be expressed as

$$A(x) = A(0) + \int_0^x B(\hat{x})f[z(\hat{x})]d\hat{x} \tag{4.4}$$

where $A(0)$ is the innate reproductive capacity and $B(x)$ is the age-dependent growth efficiency. Here, the contribution of resources to growth follows the same law as the one to reproduction, and, thus, both reproductive and growth investments share the same $f(\cdot)$.

Turning to the budget constraint, the model incorporates resource transfers. The extensive use of resource transfers, such as intergenerational transfers between parents and children and food sharing between households, is one of the most distinctive human characteristics.[8] In this case, the budget constraint does not necessarily hold at each point in time, and is given by the lifetime budget constraint,

$$\int_0^\infty l(x)y(x)dx \geq \int_0^\infty l(x)\left[v(x) + z(x) + w(x)\right]dx, \tag{4.5}$$

where $y(x)$ is the amount of resources that the individual obtains at age x. Note that $y(x)$ is exogenous, and that the interest rate is equal to zero since keeping resources does not generate any return.

With these conditions, we can solve for the optimal allocation of resources, expressed by $v(x)$, $z(x)$, $w(x)$, and α, using the Lagrangian method. Given the objective function (4.1), the budget constraint (4.5), and the Lagrangian multiplier, λ, the Lagrangian is defined as

$$\begin{aligned} L &= \int_0^\infty l(x)m(x)dx \\ &+ \lambda\left\{\int_0^\infty l(x)[y(x) - v(x) - z(x) - w(x)]dx\right\} \end{aligned} \tag{4.6}$$

where $l(x)$, $m(x)$, and $A(x)$ are respectively specified by the equations (4.2), (4.3), and (4.4). Using the Volterra derivative (see Ryder and Heal 1973) to

[8] See, e.g., Wiessner (1996, 2002) and Gurven et al. (2000) for the importance of food sharing in modern foraging populations.

examine the effect of changes in the investments around a particular age x, we obtain the first-order conditions:

$$\frac{\partial L}{\partial v(x)} = l(x)A(x)f_v[v(x)] - \lambda l(x) = 0 \quad \text{for } x \geq \alpha, \tag{4.7}$$

$$\frac{\partial L}{\partial z(x)} = B(x)f_z[z(x)]\eta(\alpha) - \lambda l(x) = 0 \quad \text{for } x < \alpha, \tag{4.8}$$

$$\frac{\partial L}{\partial w(x)} = -\mu_w[w(x), x]\,[R(x) + \lambda k(x)] - \lambda l_x = 0, \tag{4.9}$$

$$\frac{\partial L}{\partial \lambda} = \int_0^\infty l(x)[y(x) - v(x) - z(x) - w(x)]dx = 0 \tag{4.10}$$

where

$$\eta(\alpha) = \int_\alpha^\infty l(\hat{x})f[v(\hat{x})]d\hat{x}, \tag{4.11}$$

$$R(x) = \int_x^\infty l(\hat{x})A(\hat{x})f[v(\hat{x})]d\hat{x}, \tag{4.12}$$

$$k(x) = \int_x^\infty l(\hat{x})[y(\hat{x}) - v(\hat{x}) - z(\hat{x}) - w(\hat{x})]d\hat{x}. \tag{4.13}$$

Here, $\eta(\alpha)$ represents the benefit of an increment in the reproductive capacity, and $R(x)$ and $k(x)$ express the benefits of an increase in survival at age x respectively for reproduction and production.

At the same time, the age of maturity is determined by the returns of growth and reproductive investments. These returns are given by $B(x)f[z(x)]\eta(x)$ and $l(x)A(x)f[v(x)]$, which respectively represent the increase in the future reproduction due to a greater reproductive capacity and the increase in immediate reproduction. The individual switches from the growth phase to the reproductive phase when the return for reproduction overtakes the one for growth. Therefore, the age of maturity is implicitly given by

$$B(\alpha)\eta(\alpha) = l(\alpha)A(\alpha). \tag{4.14}$$

4.2.2 Optimal Life-history Strategies

These conditions can be interpreted as follows. From here on, I suppress the age notation when no confusion arises.

First, equation (4.7) indicates that the marginal productivity of reproductive investment, $Af_v(v)$, is equal to the shadow price of resources and is constant across ages. For example, if A depreciates after maturity, $f_v(v)$

increases and thus reproductive investment decreases with age. The decline in the reproductive efficiency is offset by the decline in reproductive investment.

Second, by rewriting equation (4.8) as

$$Bf_z(z)\frac{\eta(\alpha)}{l(x)} = \lambda, \tag{4.15}$$

equation (4.15) shows that the marginal return of growth investment conditional on survival to age x is equal to the shadow price and is constant across ages. It is conditional on survival because, while the timing of starting to reap the return on growth investment is fixed by the age of maturity that is endogenously determined, the chance of reaching maturity changes with age. This asymmetry makes the return of growth investment age-dependent. As maturity approaches, the expected return, $\frac{\eta(\alpha)}{l(x)}$, increases, and, to take advantage of the higher return, the marginal productivity, $Bf_z(z)$, decreases. For example, if B is constant across ages, $f_z(z)$ decreases and growth investment increases toward maturity.

Third, by letting

$$\bar{R}(x) = \int_x^\infty \frac{l(\hat{x})}{l(x)} A(\hat{x}) f[v(\hat{x})] d\hat{x}, \tag{4.16}$$

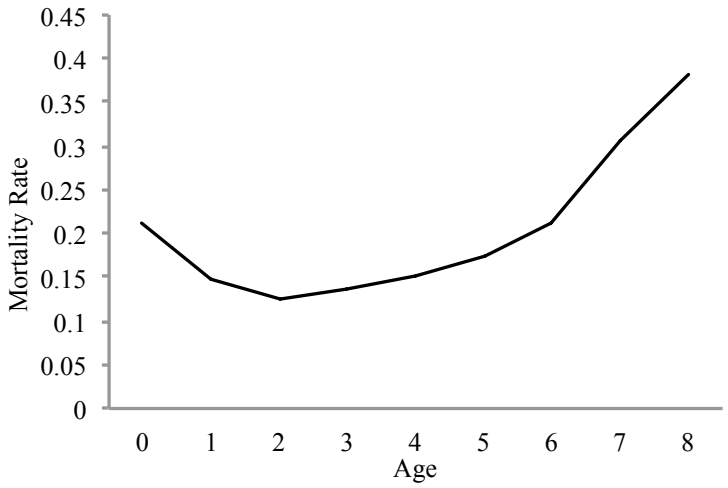
$$\bar{k}(x) = \int_x^\infty \frac{l(\hat{x})}{l(x)} [y(\hat{x}) - v(\hat{x}) - z(\hat{x}) - w(\hat{x})] d\hat{x}, \tag{4.17}$$

equation (4.9) can be rewritten as

$$-\mu_w(w,x)\left[\bar{R}(x) + \lambda\bar{k}(x)\right] = \lambda. \tag{4.18}$$

The upper bar indicates that the value is adjusted by the survival probability at the corresponding age. Thus, $\bar{R}(x)$ represents the expected reproductive output in the remaining lifetime for the individual survived to age x, and $\bar{k}(x)$ expresses the expected productive surplus in the remaining lifetime for the same individual. Therefore, given that λ is the value converter of productive surplus to reproductive contribution, the terms in the brackets in equation (4.18), $\bar{R}(x) + \lambda\bar{k}(x)$, represents the value of survival that includes both direct (reproductive) and indirect (productive) contributions. With resource transfers, the value of survival consists of not only reproductive contribution but also productive contribution.

With these results, equation (4.18) shows that the marginal benefit of survival investment is equal to the shadow price and is constant across ages.



The maximum age-class to which the individual can possibly survive is set to nine, and the functional forms and parameter values are given as follows: $\mu[w(x), x] = e^{-qw(x)}$, $A(x)f[v(x)] = A(\alpha)v(x)^{\gamma}$, $B(x)f[z(x)] = Bz(x)^{\gamma}$, $\gamma = 0.25$, $q = 0.5$, $B = 0.15$, $A(0) = 0.18$, $y(0) = 0$, $y(1) = 2$, $y(2) = 4$, $y(3) = 6$, $y(4) = 6$, $y(5) = 6$, $y(6) = 6$, $y(7) = 6$, $y(8) = 4$, and $y(9) = 4$.

Figure 4.1: Age-trajectory of the mortality rate

This implies that the marginal productivity of survival investment, $-\mu_w(w)$, depends on the change in the value of survival, and reaches its lowest level at the prime of life at which the value of survival is the highest. Assuming, for example, that $\mu(w)$ is independent of age, we can expect that the mortality rate reaches its lowest level at the prime of life.

An example of such a mortality curve is presented in Figure 4.1. It shows that the mortality rate reaches its lowest level in age-class two and increases thereafter, corresponding to the result that the value of survival hits its highest level in age-class three.

4.3 Implications on Intertemporal Allocation

4.3.1 Intertemporal Marginal Rate of Substitution

The intertemporal marginal rate of substitution (MRS) measures the importance of resources at one point in time over another. Specifically, it is defined as the rate to compensate for a loss of resources at one point in

time in exchange for resources at another point in time while keeping $R(0)$ constant. In a continuous-time setting, MRS at age x can be calculated as

$$\frac{-\partial \log R_v(0)}{\partial x} = -\frac{l_x}{l} - \frac{A_x}{A} - \frac{f_{vx}(v)}{f_v(v)}, \tag{4.19}$$

$$\frac{-\partial \log R_z(0)}{\partial x} = -\frac{B_x}{B} - \frac{f_{zx}(z)}{f_z(z)}, \tag{4.20}$$

$$\frac{-\partial \log R_w(0)}{\partial x} = -\frac{\mu_{wx}(w)}{\mu_w(w)} - \frac{\bar{R}_x + \lambda \bar{k}_x}{\bar{R} + \lambda \bar{k}} - \frac{l_x}{l}. \tag{4.21}$$

where $R_v(0)$, $R_z(0)$, and $R_w(0)$ are respectively the marginal effects of reproductive, growth, and survival investments on $R(0)$. Note that, with respect to survival investment, the change in resource surplus must be taken into account.

These equations show that MRS depends on various factors. Equation (4.19) illustrates that, in adulthood, MRS consists of three components; the mortality rate, the change in the reproductive efficiency, and the change in the marginal contribution of resources. This result is consistent with Sozou and Seymour (2003), Chu et al. (2010), and the model in Chapter 3.

In childhood, on the other hand, equation (4.20) shows that MRS consists of two components; the change in the growth efficiency and the change in the marginal contribution of resources. As in Chu et al. (2010), the mortality rate does not appear in MRS in childhood.

Furthermore, equation (4.21) shows that, throughout the life course, MRS can be calculated as the sum of three components; the change in the marginal contribution of resources, the change in the value of survival, and the mortality rate.

Despite these variations, however, the values of MRS derived in Equations (4.19)-(4.21) are all equal to the mortality rate when resources are optimally allocated. In equation (4.19), the second and the third terms cancel out on the optimal path as indicated in equation (4.7), and MRS is reduced to the mortality rate. Similarly, as implied in equation (4.8), the two terms in equation (4.20) sum up to the mortality rate. Turning to equation (4.21), it is also reduced to the mortality rate since, as indicated in equation (4.18), the first two terms cancel out.

This result is intuitive in the reproductive period. The change in the reproductive efficiency is neutralized by the change in reproductive investment on the optimal path. However, giving up a unit of current reproductive investment for future investment still accompanies the mortality risk. Thus, to keep $R(0)$ constant, the mortality risk needs to be compensated.

By contrast, the result that MRS is equal to the mortality rate during the growth period might be counter-intuitive. As children do not yet reproduce, the timing of growth investment seems irrelevant as long as they build up the same level of reproductive capacity at maturity.

The reason for this result is that the marginal productivity, $Bf_z(z)$, changes with age. As shown in Subsection 4.2.2, the marginal productivity decreases as the chance of reaching maturity increases, and its changing rate is exactly equal to the mortality rate. This is intuitive considering that the change in the marginal productivity is originated by the change in the survival probability to maturity. For example, if B is constant across ages, the marginal productivity decreases with age to satisfy $-\frac{f_{zx}(z)}{f_z(z)} = -\frac{l_x}{l}$. Therefore, to give up a unit of current growth investment for future investment, the reduction in the marginal productivity must be compensated, and this compensation rate is given by the mortality rate. For this reason, MRS in childhood is, as in adulthood, equal to the mortality rate.

4.3.2 Growth Process and Mortality

The result that MRS in childhood on the optimal path is equal to the mortality rate does not depend on the type of growth process. For example, consider as in Chu et al. (2010) that the amount of resources, $y(x)$, is determined by body size, which in turn depends on growth investment up to age x. I assume for simplicity that $y(x)$ is equal to body size at age x.

In this case, the growth process can be written as

$$y(x) = y(0) + \int_0^x g[s(\hat{x}), \hat{x}]d\hat{x} \tag{4.22}$$

where $s(x)$ is growth investment allocated for increasing body size and $g[s(x), x]$ is the contribution of the investment at each point in time. Here, I assume that $g[s(x), x]$ is sufficiently concave in $s(x)$ to have an interior solution.[9]

Incorporating this size effect into the lifetime budget constraint in equation (4.5), we obtain the first-order conditions:

$$g_s[s(x), x]\bar{L}(x) = 1 \tag{4.23}$$

for all x where $\bar{L}(x) = \int_x^\infty \frac{l(\hat{x})}{l(x)}d\hat{x}$, i.e., the remaining life expectancy at age x. This shows that the marginal return of investing in body size is equal

[9]We can also assume that $y(x)$ depends on knowledge learned in the past and interpret $g[s(x), x]$ as the increment of knowledge. See Ng (1991) for how knowledge affects fitness.

to its cost. The return is measured by the amount of resources that the individual is expected to accrue in the future, and the cost is equal to one, representing a unit of resource necessary for the investment.

Equation (4.23) further illustrates that the change in the marginal productivity, $g_s(s)$, depends on the change in the remaining life expectancy. While the remaining life expectancy is high, the marginal productivity is low so as to take advantage of the higher expected return. Therefore, if the growth efficiency is independent of age, $g_s(s)$ increases and s decreases with age.[10] This is consistent with Chu et al. (2010).

The next step is to calculate MRS. By applying the same method as before, it is given by

$$\frac{-\partial \log R_s(0)}{\partial x} = -\frac{g_{sx}(s)}{g_s(s)} - \frac{\bar{L}_x}{\bar{L}} - \frac{l_x}{l}. \tag{4.24}$$

The first term represents the change in the marginal productivity, the second term expresses the change in the remaining life expectancy, and the third term is the mortality rate. As equation (4.23) indicates, the first two terms cancel out on the optimal path. As a result, MRS in childhood is again given by the mortality rate.

4.3.3 Time Preference

While the intratemporal allocation of resources is involuntary, depending mostly on physiology, the intertemporal allocation is voluntary. It primarily depends on behavior since we can deliberately control the allocation by deciding how much to consume at present.

This implies that, to further analyze the intertemporal allocation, we need to move on to the economic framework that allows us to study voluntary behavior. We need to recall that humans do not intend to maximize fitness but rather behave in a utility-maximizing manner, and that those who had preferences that coincided with the fitness-maximizing behavior spread over and fixed in the population.

To do this, I employ a standard economic model in which the objective function is given by lifetime utility,

$$U = \int_0^\infty \beta(x) u[c(x), x] dx, \tag{4.25}$$

[10] The results that the two types of growth investment, s and z, change in the opposite direction suggests that the age-trajectory of whole growth investment can be either increasing or decreasing.

where $\beta(x)$ is the discount factor for future utility, $u[c(x), x]$ is a strictly concave instantaneous utility function, and $c(x)$ is consumption, which can be regarded as the sum of growth, reproductive, and survival investments. Note that the lifetime budget constraint presented in equation (4.5) is still relevant.

Here, the instantaneous utility depends on age. Given that preferences were shaped by natural selection, the instantaneous utility should reflect the effect of consumption on reproductive success at the corresponding age. To incorporate this aspect into the model, I assume that the marginal utility on the fitness-maximizing consumption path is constant across ages. This type of utility induces the individual to consume more while its marginal effect on fitness is high.

In this setting, MRS in utility is given by

$$-\frac{\beta_x(x)}{\beta(x)} - \frac{u_{cx}[c(x), x]}{u_c[c(x), x]}. \tag{4.26}$$

The individual applies this rate to evaluate future consumption and determines the intertemporal allocation. Thus, for the voluntary allocation to match the fitness-maximizing allocation, this rate must be consistent with MRS in fitness obtained in Subsection 4.3.1, and must be equal to the mortality rate when it is evaluated on the fitness-maximizing consumption path. In particular, when the instantaneous utility depends on age as described above, $u_{cx}[c(x), x]$ is equal to zero on the fitness-maximizing path, and thus $-\frac{\beta_x(x)}{\beta(x)}$, i.e., the *pure* rate of time preference, is equal to the mortality rate.[11]

These results further imply that, considering that time preference is psychologically embedded, the endowed rate of time preference is equal to the mortality rate in our evolutionary past when we existed as hunter-gatherers. Thus, presuming that the age-trajectory of mortality in our evolutionary past is similar to the one in modern hunter-gatherer populations, we can predict that the endowed rate of time preference is U-shaped in age, reaching its lowest level in early adulthood, as is the age-trajectory of the mortality rate in modern hunter-gatherer populations (e.g., Hill and Hurtado 1996; Hill et al. 2007).

This is consistent with empirical findings. As discussed in Section 4.1, the discount rate decreases in childhood. In adulthood, on the other hand,

[11] If we correctly perceive the survival probability and discount future utilities accordingly, the pure rate of time preference would be equal to zero. However, there is no biological basis to suppose that we are able to correctly perceive the survival probability. Given that discounting future consumption is not limited to humans, it makes more sense to consider that time preference is psychologically embedded.

while still empirically inconclusive, Trostel and Taylor (2001) and Read and Read (2004) found that the discount rate increases with senescence.[12] As the present study focuses on the biological discount rate and ignores the effect of social factors such as education, the discount rate in adulthood in the present study may be estimated higher than the actual rate in modern human populations. Nevertheless, the age-trajectory of time preference predicted in the present analysis is still in line with the empirical findings.

4.4 Concluding Remarks

This study aims to understand the mechanism that coordinates intertemporal choice, paying particular attention to the impatience of children. To do this, it examines human life-history strategies, incorporating the growth period to maturity. The results show that the endowed rate of time preference is equal to the mortality rate in the entire life course, and, thus, is U-shaped in age, indicating that it is higher for children than for young adults.

At the behavioral level, this provides a biological explanation as to why parents and children often have conflicts in the allocation of resources when they make collective decisions. Due to the difference in the discount rate, parents, who follow their own time preference, would allocate less to the present than their children would. Namely, the difference in time preference generates parent-offspring conflict over the intertemporal allocation of resources, affecting the decision-making process particularly within the household.[13]

One limitation of this study, however, is that it treats the growth period as one state and compresses the reproductive capacity into one parameter. This is equivalent to assuming that a unit of growth investment is exchangeable between any points in time within the growth period although such a transaction may incur additional costs.

In reality, it is often not the case. A deficiency in a particular type of growth investment at a certain age, for example, may not be compensated by

[12] These studies, however, differ in the age at which the discount rate reaches the lowest level. While Trostel and Taylor (2001) found that the discount rate is lowest among individuals in their twenties, Read and Read (2004) argued that it is lowest in their forties. Thus, the age at which time preference reaches its lowest level is still an open question.

[13] The parent-offspring conflict in the present study differs from the original parent-offspring conflict in Trivers (1974). While the ultimate cause of the original parent-offspring conflict is the difference in relatedness, the ultimate cause in the present study is the difference in life-history phase.

the same kind of investment at a later age, and may have a permanent effect. In such cases, intertemporal choice would not be as simple as the present analysis suggests, and the impatience of children, in particular, would be underestimated.

Nevertheless, as the first-order approximation, this study provides an explanation for the age-trajectory of time preference, connecting it to mortality in both childhood and adulthood. The future is discounted because survival is uncertain (Yaari 1965), and survival is uncertain because the future is discounted (Kirkwood 1977; Kirkwood and Rose 1991). Both propositions hold in the entire life course because time discounting and survival uncertainty are two sides of the same coin, reflecting the change in the value of survival. There are other factors, including the variability of the environment, sexual reproduction, and genetic relatedness, that possibly affect time preference, but mortality is the baseline for time preference through the entire course of life.

This study also indicates that the traditional view that time discounting is something unfavorable is not entirely accurate. Time discounting generally carries negative connotations and has been described as cognitive deficiency, impatience, shortsightedness, myopia, irrationality, and so forth. For example, Ramsey (1928) regarded time-discounting behavior as "a practice which is ethically indefensible and arises merely from the weakness of the imagination (p. 543)." However, as discussed in this study, being impatient is not a deficiency, but an optimal trait in the biological sense that was acquired in the course of evolution. Our surrounding environment has changed since our evolutionary past, and the endowed rate of time preference is no longer optimal in the economic sense. Nevertheless, being too patient is not necessarily favorable either as it would cause other problems such as dynamic inefficiency in macroeconomics and the postponement of reproduction that, together with the decline in reproductive efficiency, results in below-replacement fertility. Given our human nature, an appropriate level of impatience may in fact be beneficial to our well-being.

4.5 Appendix: The Value of Survival

The value of survival derived in this and previous chapters can be used for explaining other human-specific characteristics. The prominent example is the post-reproductive life in human females. Given that the value of survival includes both reproductive and productive contributions, we can argue that the post-reproductive life emerged as the result of specialization in pro-

duction at older ages. With resource transfers, specializing in production at older ages can contribute to their own fitness through supporting their children to reproduce in a shorter interval. Namely, post-reproductive life and a short birth interval, which is also unique to humans, are considered to have evolved together with the extensive use of resource transfers. Kaplan and Robson (2009) reach the same conclusion in a different setting.

Furthermore, the value of survival sheds light on the relationship between economics and evolutionary biology since the value of survival mathematically corresponds to the value of life in economics (see, e.g., Shepard and Zeckhauser 1984). The difference arises only from the measurement unit. While the value of survival is measured at the fitness level, the value of life in economics is measured at the utility level.

This raises a question of how we reconcile the value of life and the value of survival. As both economics and biology deal with behavior, predictions derived in the two disciplines need to be consistent with each other, and, thus, the two values ought to be somehow related to each other.

However, it is not necessarily the case under standard assumptions in economics. One major difference is that, in the standard time-separable framework, instantaneous utility function is generally assumed time-invariable while instantaneous reproduction function is time-variable. As a result, the age-trajectories of the two values differ from each other, potentially leading to different predictions in human behavior. One way to overcome such inconsistency is to examine how the shape of utility function changes with age and incorporate the results into economics.

Nonetheless, considering that the the value of life exactly matches the value of survival would be overly simplistic. Biological studies suggest that other factors, such as group selection and sexual reproduction, affect fitness. For example, Chu et al. (2008) show that the value of survival includes the compounding effect that arises from investing in somatic growth. Similarly, economic studies point to the complexity of the value of life. Kuhn et al. (2010), for example, incorporate the value of progeny and demonstrate that the value of life can include both biological and economic terms. The comparison of the two values requires further examination.

Chapter 5

Happiness and Sex Difference in Life Expectancy

5.1 Background

Happiness and health are correlated. Using micro data sets, a number of studies have reported both that healthier individuals are happier (see, e.g., Diener et al. 1999; Frey and Stutzer 2002; Helliwell 2003; Borooah 2006), and that happier individuals are healthier (see Pressman and Cohen 2005; Veenhoven 2008, for reviews).

This relationship has also been examined at the aggregate level, using national life expectancy as a proxy for the health of particular countries. Ovaska and Takashima (2006) and Deaton (2008), for example, both report that life expectancy is an important factor in explaining cross-country differences in life satisfaction, which correlates to happiness. By contrast, Bjørnskov (2008) uses a two-stage-least-square (2SLS) approach and finds that happiness has a *negative* impact on life expectancy.

That some studies model happiness as the dependent variable and life expectancy as the explanatory variable, while others model them the other way round, illustrates that the causality is not unidirectional. Happiness affects health and health affects happiness, and both are further correlated with third variables such as income, lifestyle, and education, leading to complex patterns of correlation that do not reflect simple patterns of causation. This not only renders the ordinary-least-square (OLS) estimator biased, but also leads to difficulty in finding appropriate instruments for 2SLS estimation.

To further complicate matters, cross-country studies often encounter small sample sizes and unbalanced panels. Large numbers of explanatory

variables reduce the efficiency of the regression models, and, especially, with high levels of multicollinearity, there may not be an analytical means to partition their separate effects. Taking these difficulties into account, it is not surprising that no studies have found the expected positive impact of happiness on life expectancy.

In light of these challenges, the present study takes on a different approach to find out whether the findings in micro studies that happier people live longer are still valid at the aggregate level. It employs the sex difference in, rather than the level of, life expectancy as the dependent variable.

The rationale for using the sex difference in life expectancy comes from the findings that physiological and behavioral responses to psychological stress differ between women and men. Weidner and Cain (2003), for example, show that the substantial increase in coronary heart disease observed in Eastern Europe after the fall of communism, which resulted in the region's dramatic health deterioration, was principally caused by psycho-social stress, and that this had a bigger impact on men because men cope less effectively with stress. Similarly, Möller-Leimkühler (2003) argues that men cope less effectively with psychological stress and adopt maladaptive strategies such as excessive alcohol consumption, risk-taking behavior, and violence.[1]

Furthermore, Helliwell (2007) shows that, while social factors affect women's and men's life satisfaction similarly, they influence the suicide rates quite differently, leading to much higher suicide rates among men. This result also points to the significant difference between sexes in physiological and behavioral responses to mental conditions and their underlying determinants.

From the biological perspective, these findings are not surprising. As a number of biological studies have pointed out, compared to their female counterparts, males are more vulnerable and more prone to death in various animal species, including humans, and this vulnerability is expected to make their mortality more sensitive to psychological conditions.

The ultimate, or evolutionary, cause of male vulnerability and high mortality is intense intrasexual competition that arises from their lower reproductive costs. The proximate causes, on the other hand, are their propensity to engage in risky behavior and their physiological fragility.

Males tend to engage in risky behavior because the reproductive return of such behavior is higher for males than for females (e.g., Wilson and

[1]This, however, does not necessarily mean that the level of psychological stress is higher for men. As found in Mirowsky and Ross (1995), women are generally at a higher risk of depression. The ways that women and men react to psychological stress are simply different. As described in Nathanson (1977), "women get sick and men die."

Daly 1985; Kruger and Nesse 2006; Kraus et al. 2008). Males that take risks and succeed can possibly reproduce at a much higher rate than their female counterparts. For example, if a male succeeds in monopolizing multiple females, his reproductive return would increase substantially, whereas a female monopolizing multiple males would not greatly enhance her reproductive return. This makes male-male competition fiercer, increasing their mortality.

Male physiological fragility is a by-product of physiological necessity to invest more resources in intrasexual competition. As male-male competition requires resources to make him competitive, it crowds out investments in the maintenance of the soma and makes him fragile. For example, male-female physical size dimorphism, which points to the significance of male-male competition, is found positively correlated with excess male morality across taxa (Promislow 1992; Moore and Wilson 2002; Clutton-Brock and Isvaren 2007).

These biological findings provide an explanation for why psychological stress is more influential on men's mortality than on women's. In bad conditions where male-male competition becomes more intense, greater psychological stress causes adverse physiological responses and promotes risky behavior, both of which increase men's mortality. Although women face similar pressure as surrounding factors affect women as well, men's propensity to engage in risky behavior and their physiological fragility increase the variability of their mortality.

Putting these arguments together, we can hypothesize that, as long as psychological stress and happiness are negatively correlated, happiness has a greater impact on men's life expectancy, and negatively affect the difference in life expectancy between women and men. This relationship may be easier to capture than the relationship between happiness and life expectancy itself. Considering that the sex difference in stress responsiveness is physiologically rooted, cross-country variations in the life expectancy gap should reflect cross-country differences in happiness.

For this reason, the present study focuses on the life expectancy gap. This provides a technical advantage as well. Since variables that similarly affect women's and men's mortality are eliminated altogether, the regression model is kept relatively simple.

However, the problem of reverse causality still exists. This is because the life expectancy gap influences the women's widowhood ratio. Since the widowed are, on average, less happy than those who are not, an increase in the life expectancy gap that raises the women's widowhood ratio is expected to lower women's average happiness. This effect must be properly controlled

to obtain unbiased estimates.

The remainder of this study is organized as follows. Section 5.2 addresses the regression strategy, including the problem of reverse causality. Section 5.3 presents the regression results. The details of the data, such as data sources and sample countries, are presented in the appendix. The main hypotheses to be tested are that (1) happiness negatively affects the life expectancy gap, and that (2) the happiness gap between women and men positively affects the life expectancy gap. The latter hypothesis is a simple reflection of the idea that happier individuals live longer. The results support the first hypothesis, but not the second hypothesis. Section 5.4 concludes.

5.2 Data and Regression Strategy

5.2.1 Life Expectancy Gap

The difference in life expectancy at birth between women and men, $LEGAP$, is used as the dependent variable. Although this is not a perfect measure to examine the relationship between happiness and population health because life expectancy at birth includes children while happiness data do not, the data on life expectancy of adult cohorts are limited to fewer countries. Thus, we employ the life expectancy gap at birth, as in previous studies, as an indicator of health across the different populations. The data are taken from U.N. (UN 2008). The cross-country average is about 5 years, ranging from -0.46 in Zimbabwe to 13.3 years in Russia (2000-2005 data).

Genetic and physiological features that possibly contribute to women's advantage in survival include the compensatory effects of the second X chromosome, longer telomeres, stronger immune systems, better protection against oxidative stress, and the protective effects of estrogen (Austad 2006; Eskes and Haanen 2007). These features lower women's mortality risks, especially those associated with cardiovascular disease.

Large cross-country variations, at the same time, suggest that behavioral and socio-economic factors influence $LEGAP$. Behavioral factors include lifestyle, smoking, drug and alcohol consumption, violence, and accidents (McKee and Shkolnikov 2001; Luy and Di Giulio 2006; Phillips 2006). Socio-economic factors include social and economic conditions, such as the availability of financial, medical, and technical resources that affect population health. As women tend to invest more resources in health, the availability of these resources may affect women and men differently. Therefore, when examining the explanatory power of happiness on the life expectancy gap, behavioral and socio-economic factors need to be taken into account.

5.2.2 Happiness

Happiness data are taken from the European and World Values Surveys, Waves 1 (1981-1984), 2 (1989-1993), 3 (1994-1999), and 4 (1999-2004). Among others, one question asks, "Taking all things together, would you say you are: very happy (4), quite happy (3), not very happy (2), or not at all happy (1)?"

The average level of happiness, HP, and the difference in happiness levels between women and men, $HPGAP$, are calculated for each country in each wave (country-wave). The average number of respondents with personal data covering age, sex, and marital status that can be separated into the married, the separated or divorced, the widowed, and the never married, is 1,380 (717 women and 663 men) per country-wave. The largest number is 4,599 (2,297 women and 2,302 men) in Turkey (Wave 4), and the least is 303 (164 women and 128 men) in Malta (Wave 1). The number of countries in each wave are, respectively, 19 (Wave 1), 43 (Wave 2), 54 (Wave 3), and 68 (Wave 4).

The cross-country-wave averages of HP and $HPGAP$ and the corresponding figures for each marital status are reported in Table 5.1. It shows that happiness varies with marital status. In particular, losing one's spouse has a significant negative impact on happiness. It also indicates that, while $HPGAP$ is almost negligible, the corresponding gaps in each marital-status category are significant. This points to the possibility that the sex gap in happiness has been underestimated. While the sex gap in happiness has been regularly ignored because its average is so small, it may not be as small as generally thought after controlling for marital status.

5.2.3 Reverse Causality

There is good reason to suspect that the causality runs in both directions. In the reverse direction from the life expectancy gap to national happiness, the intermediary is the women's widowhood ratio, WR. As the life expectancy gap reflects the sex difference in the survival probabilities at middle and old age, an increase in the life expectancy gap raises the women's widowhood ratio. Then, as the widowed are, on average, less happy, it is expected to lower women's average happiness, national average happiness, and the happiness gap.

To examine the significance of this effect, the relationships between $LEGAP$, WR, and $HPGAP$ are illustrated in Figures 5.1, 5.2 and 5.3.[2]

[2]The relationships for HP are not presented here because the expected effects are

Table 5.1: Happiness and happiness gap

	Average	Married	Separated or Divorced	Widowed	Never Married
HP					
Both Sexes	3.019	3.082	2.753	2.762	3.001
Women	3.020	3.097	2.769	2.778	3.022
Men	3.019	3.068	2.733	2.707	2.985
HPGAP	0.001	0.028	0.037	0.071	0.037
Ratio					
Women	1.00	0.62	0.07	0.11	0.20
Men	1.00	0.66	0.05	0.03	0.26

The number of observations is 144. Average *HP* can be regarded as the weighted average of marital-status-specific *HP*. The observations (country-waves) with fewer than five respondents in either sex in any of the marital statuses are excluded.

Figure 5.1 shows $LEGAP$ and WR are positively correlated, indicating that a larger life expectancy gap leads to a higher widowhood ratio for women. Figure 5.2 demonstrates that WR and $HPGAP$ are negatively correlated, suggesting that a higher widowhood ratio among women results in a smaller happiness gap. Connecting these two relationships, Figure 5.3 shows that $LEGAP$ is negatively correlated with $HPGAP$. Namely, a larger advantage in women's survival increases the likelihood of widowhood and reduces their edge in happiness. As previously noted, $LEGAP$ is not a perfect measure for the survival gap at middle and old age. Nonetheless, we can still observe the expected relationships by using $LEGAP$.

To further examine this reverse effect, Table 5.2 presents the correlation coefficients between $LEGAP$ and $HPGAP$ after decomposing $HPGAP$ by marital status. It shows that the correlation coefficients between $LEGAP$ and the happiness gaps in each marital-status category are, in absolute value, smaller than the coefficient between $LEGAP$ and $HPGAP$, i.e., their weighted average value. For example, while the correlation coefficient between $LEGAP$ and $HPGAP$ is -0.48, the corresponding figure for the widowed is -0.11. This points to the existence of the marital-status compositional effect. A large part of the correlation between $LEGAP$ and $HPGAP$ is generated by the compositional changes in marital status. The same thing can be said about the relationship between $LEGAP$ and HP. The correlation coefficients between $LEGAP$ and happiness in each marital-status category are smaller in absolute value. These results demonstrate that the

negative in both directions and the direction of the causality cannot be differentiated.

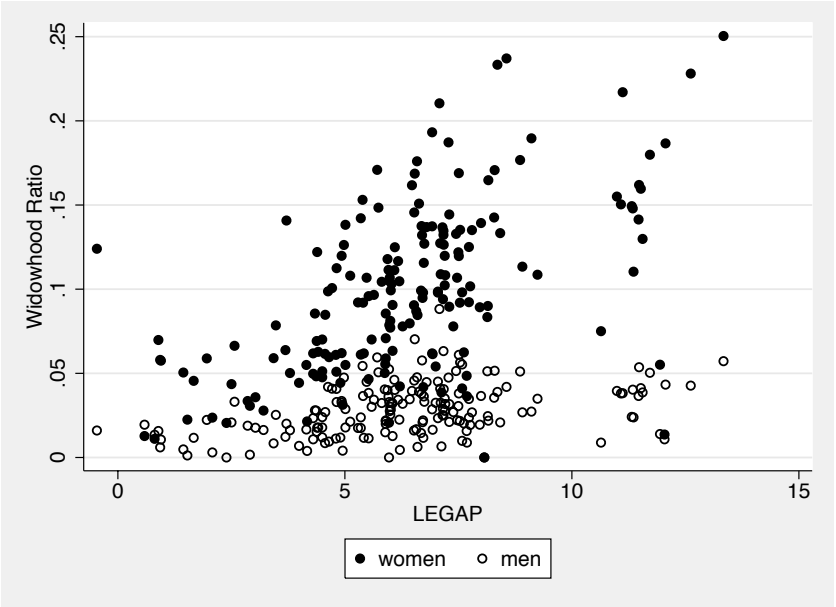


Figure 5.1: Correlation between widowhood ratio and life expectancy gap

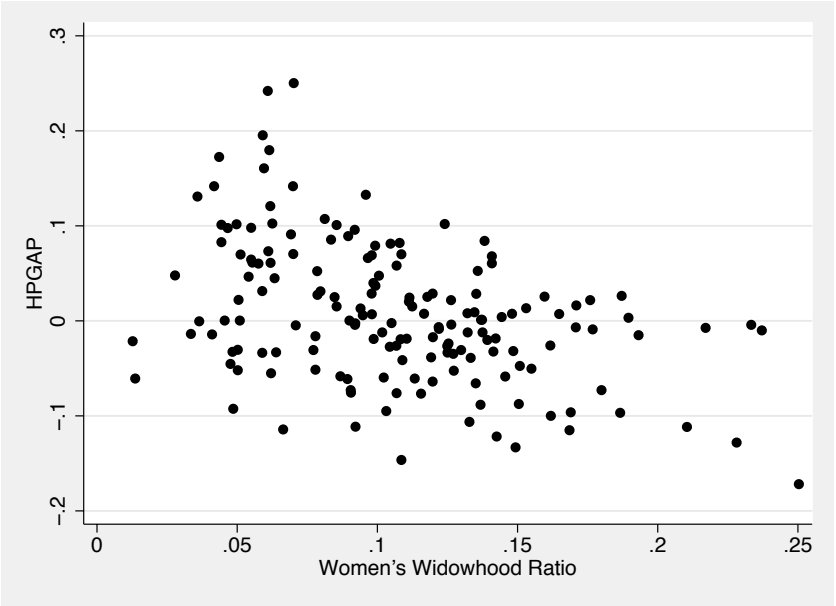


Figure 5.2: Correlation between happiness gap and widowhood ratio

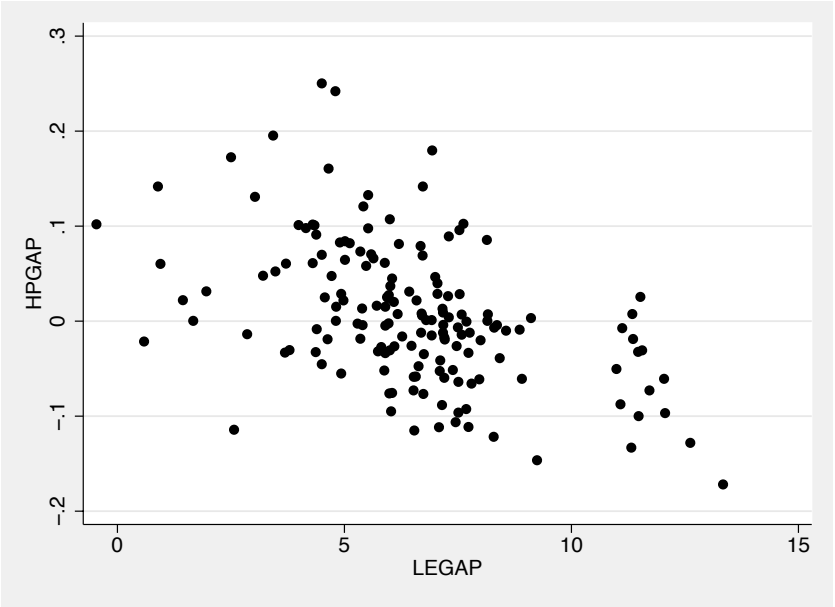


Figure 5.3: Correlation between happiness gap and life expectancy gap

reverse causality is significant. Therefore, it needs to be properly controlled in the regression analysis.

5.2.4 Explanatory Variables

The regression analysis requires various explanatory variables. As listed in Table 5.3, they are the women's labor force ratio, WLR, the log of purchasing-power-parity adjusted per-capita GDP, $LYPC$, hospital beds per 1,000 people, HB, physicians per 1,000 people, PH, fertility rate, FT, and the level of life expectancy at birth for both sexes, LE. The latter

Table 5.2: Correlation with $LEGAP$

	Average	Married	Separated or Divorced	Widowed	Never Married
HP	-0.51	-0.48	-0.44	-0.48	-0.49
HPGAP	-0.48	-0.29	-0.02	-0.11	-0.3

The number of observations is 139.

part of the analysis also includes the difference in the smoking rates between women and men, $SMGAP$, the difference in education (average years of schooling) between women and men, $EDGAP$, and the Gini coefficient, $GINI$. These last three variables are initially excluded so as to increase the number of observations. They cover only a fraction of the observations, and the sample size becomes only 33 if they simultaneously enter into the regression equation.

The reasons for employing these explanatory variables are as follows. The women's labor force ratio aims to capture the sex difference in lifestyle. The expected sign of the coefficient is, however, ambiguous. On one hand, a higher WLR may indicate greater autonomy in women, and, thus, increase $LEGAP$ by raising women's life expectancy. On the other hand, a higher WLR may imply that women are leading a less healthy lifestyle as their habits become similar to men's, and reduce $LEGAP$ by lowering the women's life expectancy.

The log of PPP per-capita GDP, hospital beds per 1,000 people, and physicians per 1,000 people aim to capture the effects of economic and medical resources. As women generally invest more resources in health, these variables are expected to be more influential on women's life expectancy than on men's life expectancy. Thus, they are expected to affect $LEGAP$ positively.

Fertility rate and the level of life expectancy represent the country's demographic characteristics. FT captures the risk of giving birth, and the expected sign of the coefficient is negative. LE, on the other hand, examines how the level and the gap of life expectancy are related. As found in various studies, $LEGAP$ has been shrinking since the 1970s in many industrialized countries (Trovato and Lalu 1996; Trovato 2005; Trovato and Heyen 2006; Glei and Horiuchi 2007).

This narrowing $LEGAP$ is often attributed to behavioral and medical factors such as the shrinking smoking gap and medical technological progress that benefits men more than women. If these factors thoroughly explain the correlation between $LEGAP$ and LE, LE would be insignificant after controlling for social and behavioral factors. However, at the same time, it is also attributed to the sex difference in the shape of the survival curves (Glei and Horiuchi 2007). As women's deaths are less dispersed across age, the same rate of mortality decline produces smaller gains in life expectancy for women than for men. This suggests that $LEGAP$ would be negatively correlated with LE even after controlling for behavioral and medical factors. Therefore, the coefficient of LE is expected to be either insignificant or negative.

Table 5.3: Definition of variables

Variable	Definition	Data Source
HP	National average happiness	EWVS 2008
HPGAP	The difference in average happiness between women and men	EWVS 2008
HPM, HPD, HPW, HPN	Average happiness for the married, the separated or divorced, the widowed, and the never married. The observations with fewer than five respondents in either sex of the marital status used in that particular analysis are excluded.†	EWVS 2008
HPGAPM, HPGAPD, HPGAPW, HPGAPN	The difference in average happiness between women and men for the married, the separated or divorced, the widowed, and the never married. The observations with fewer than five respondents in either sex of the marital status used in that particular analysis are excluded.	EWVS 2008
LEGAP	The difference in life expectancy at birth between women and men	UN 2008
LE	Life expectancy at birth for both sexes	UN 2008
LYPC	Log of GDP per capita (purchasing-power-parity adjusted)	PWT 2008
PI	Price level of investment	PWT 2008
FT	Fertility rate (births per woman)	World Bank 2008
WLR	Women's labor force ratio	World Bank 2008
HB	Hospital beds per 1,000 people	World Bank 2008
PH	Physicians per 1,000 people	World Bank 2008
GC	General government final consumption expenditure (percentage of GDP)	World Bank 2008
SMGAP	The difference in smoking rates between women and men	WHO Europe 2007
EDGAP	The difference in average years of schooling between women and men among those aged 25 and above	Barro & Lee 2008
GINI	GINI coefficient	LIS 2008
Regional Dummies	The division follows the subregions defined by UN. However, based on their cultural similarities and in an effort to make the number of observations in each region sufficiently large, the following subregions are integrated: Caribbean and Central and South America; North America, Australia, and New Zealand; Eastern, Southern, Middle, and Western Africa; Central and Western Asia and Northern Africa.	

† The averages are taken without controlling for the sex difference. Controlling for the sex difference does not change the results in any meaningful way.

Next, the sex difference in the smoking rates aims to capture the effect of smoking, and the expected sign of its coefficient is negative. As more women smoke, their advantage in life expectancy becomes smaller.

The sex difference in education is for assessing the effect of education, and its expected effect is positive. While women's education raises life expectancy for both women and men since women generally play more important roles in household activities, education is still expected to affect one's own health most. Incorporating $EDGAP$, however, gives rise to the possibility of reverse causality. Since the expectation of a longer life increases the demand for education, the causality runs in both directions. To cope with this problem, $EDGAP$ is included with and without additional instrument variables.

Finally, the Gini coefficient tests the effect of income inequality. Since life expectancy is more elastic to income when income level is low, an increase in income inequality is expected to widen the difference in life expectancy between the rich and the poor. As a result, if income affects women's survival more than men's, based on the speculation that economic resources are more important for women's life expectancy, $GINI$ would negatively affect $LEGAP$ by influencing women's life expectancy more significantly. But, if being relatively poor leads to greater stress and this effect is not captured by either HP or $HPGAP$, it would positively affect $LEGAP$ by lowering men's life expectancy more significantly.

On top of these variables, instrumental variables are required to control for the reverse causality. As mentioned earlier, the life expectancy gap affects both happiness and the happiness gap through the composition of marital status. The most prominent instruments are happiness and the happiness gap of a specific marital status. By controlling for marital status, the marital-status compositional effect should be removed and the reverse effect of $LEGAP$ should be substantially reduced.

Among the four types of marital statuses, the ones for the widowed, HPW and $HPGAPW$, are the best instruments because the individuals in this category have already gone through the hardship of losing a spouse and $LEGAP$ should not have any further impact on them. On the other hand, happiness and the happiness gap of the other marital statuses could be influenced by $LEGAP$ since $LEGAP$ affects the chance of being widowed.

However, there are two drawbacks in using happiness and the happiness gap of a specific marital status. First, the small number of respondents available for calculating these variables, particularly HPW and $HPGAPW$, may deem the results unreliable. Even though a survey contains, on average, 1,380 respondents in each country-wave, the widowhood ratio is only about

5%, and, consequently, the number of the widowed is, on average, only 70, or 35 for each sex. To minimize this problem, the observations with fewer than five widows or widowers are omitted in the analysis.

The second problem lies in the possibility that the effect of $LEGAP$ may remain in happiness and in the happiness gap even after controlling for marital status. For example, Barber (2009) argues that deviations from the standard sex difference in life expectancy reflect gender-specific disadvantage of particular countries and explain the cross-country variations in happiness. Should it be the case, the correlation between $LEGAP$ and HP would be the result of a third factor, and, thus, using happiness and the happiness gap of a specific marital status would not be an adequate solution. To cope with this problem, we also use instruments that are not directly related to happiness.

5.2.5 Methods

Using HPW and $HPGAPW$ as instruments, the data set is the four-period panel containing 142 observations (69 countries). For cross-country panels, a common method of estimation is to apply the fixed-effect model with country dummies. In this way, country dummies capture the unobservable country-specific effects. However, the present data set is heavily unbalanced. Only three countries (Spain, Sweden, and the USA) have full observations and 23 countries have only one observation. This implies that applying the fixed-effect model with country dummies is not realistic. Therefore, the data set is treated as a pooled data set, and, instead of country dummies, regional dummies are included. In other words, the regression model is regarded as the fixed-effect model with region-specific constants. Regions with only one observation are omitted.

5.3 Regression Results

5.3.1 Marital-Status Compositional Effect

Before regressing the life expectancy gap on happiness, we estimate the reverse effect of the life expectancy gap. For this purpose, we regress HP on HPW and $LEGAP$, and $HPGAP$ on $HPGAPW$ and $LEGAP$. Here, HPW and $HPGAPW$ are included to capture the country's basic levels that are independent of compositional changes in marital status.

The results are presented in Table 5.4. The top figures are the estimated coefficients, and the bottom figures are the heteroskedasticity-robust

t statistics. The results indicate that $LEGAP$ negatively affects both HP and $HPGAP$. The levels of significance are, respectively, 5 and 1%. Including regional dummies and period dummies does not change the results. These results support the existence of the marital-status compositional effect.[3]

5.3.2 Model Specification for Main Regression Analysis

The results of the main regression model are presented in Table 5.5. Here, we control for the reverse causality and test the explanatory powers of HP and $HPGAP$ on $LEGAP$. Equations (1) and (2) are for model specification. As HB captures the effect of medical resources better than PH, we omit PH and keep HB as a proxy for medical resources. Doing so increases the number of observations.

The period dummies, which are incorporated to capture time trend, are not significant at the 10% level. Equation (2) shows that regressing only with the samples in the forth period, i.e., the period with the most number of observations, yields similar results to those of the pooled data set. For

[3]There are two methods to calculate the marital-status compositional effect. In the first method, we estimate the effect of $LEGAP$ on WR, which corresponds to the slope of the fitted line for women's data in Figure 5.1, and calculate the impact of WR on women's average happiness using the data in Table 5.2. By multiplying these two effects, we can indirectly estimate the marital-status compositional effect. Using this method, a year increase in $LEGAP$ is estimated to raise WR by 1.06%, and one percent increase in WR is estimated to lower women's average happiness by 0.0032 point. Thus, a year increase in $LEGAP$ is expected to lower women's average happiness by 0.0034 point.

In the second method, we directly regress either $HPGAP$ or women's average happiness on $LEGAP$, controlling for the country's basic level of happiness. The current regression analysis corresponds to this method, and equations (3) and (4) in Table 5.4 show that a year increase in $LEGAP$ would lower women's average happiness by 0.015 point more than that of men. Similarly, by replacing HP and HPW in equations (1) and (2) with women's data, a year increase in $LEGAP$ is estimated to lower women's average happiness by, respectively, 0.023 and 0.019 points, both at the 1% level of significance.

These results indicate that the estimated effects of $LEGAP$ in the second method are about five to seven times larger than that of the first method. One possible cause for this difference is the weak explanatory power of the widowed data. After replacing happiness of the widowed with that of the married, a year increase in $LEGAP$ is estimated to lower women's average happiness by, respectively, 0.0072 and 0.010 points, both at the 1% level of significance. These figures are much closer to the estimated figure of the first method.

Alternatively, the difference could be due to the existence of other factors that connect the life expectancy gap to happiness. This would be an interesting topic to pursue. However, to proceed to the main regression analysis, it is suffice to show that $LEGAP$ is significant, controlling for the country's basic happiness level, and that the martial-status compositional effect exists.

Table 5.4: Regression results (Dependent variable: HP and $HPGAP$)

	HPW	*HPGAPW*	*LEGAP*	Dummies		*R-sq*	obs. #
				Region	Period		
HP							
(1)	0.681		-0.015	incl	incl	0.89	142
	13.38***		-2.31**				
(2)	0.726		-0.011	excl	excl	0.86	142
	23.60***		-2.42**				
HPGAP							
(3)		0.068	-0.015	incl	incl	0.49	142
		3.46***	-5.50***				
(4)		0.082	-0.015	excl	excl	0.31	142
		3.45***	-6.05***				

The top figures are the estimated coefficients. The bottom figures are heteroskedasticity-robust *t*-statistics. ***, **, and * respectively indicate the significance levels at $p<0.01$, $p<0.05$, and $p<0.10$.

these reasons, we omit the period dummies in the following analysis. This also helps to reduce the chances of having biased estimates. As countries with similar characteristics, such as newly independent countries in certain regions, may be omitted at some periods in a non-random manner, the inclusion of period dummies may generate a sample-selection bias.

5.3.3 Happiness Gap

Equation (3) shows the results without PH and period dummies. It indicates that $HPGAP$ is not significant although its coefficient takes the expected positive sign. One possible reason for this result is the weak explanatory power of instruments, in particular, of $HPGAPW$. Shea's Partial R2 (Shea 1997) for $HPGAP$ is 0.066 whereas it is 0.467 for HP. Equation (4) shows that excluding regional dummies does not improve the result.

It is worth noting, however, that the coefficient of $HPGAP$ becomes significantly negative with OLS estimation as in equation (5). This contradicts the hypothesis as well as the result of 2SLS estimation, pointing to the existence of reverse causality. The OLS estimate captures the reverse effect of $LEGAP$ on $HPGAP$.

Using happiness and the happiness gap of other marital statuses does not improve the results. As shown in equation (6), using those of the married yields the results similar to those of OLS estimation. This suggests that

Table 5.5: Regression results (Dependent variable: *LEGAP*

	Method & Instruments	*HP*	*HPGAP*	*LE*	*LYPC*	*FT*	*WLR*	*HB*	*PH*	UIDT	WIDT	*R-sq*	obs. #
(1)	2SLS	-3.216	2.722	-0.391	1.601	-1.004	0.0499	0.166	0.1959	8.08	3.80	0.81	135
	HPW / HPGAPW	-3.71***	0.39	-5.40***	4.77***	-2.33**	1.63	3.10***	0.79	0.00	7.03		
(2)	2SLS	-3.628	3.013	-0.394	1.455	-0.283	0.0960	0.364	0.0326	2.18	0.72	0.87	51
	HPW / HPGAPW	-2.96***	0.26	-2.07**	2.12**	-0.65	1.26	2.72***	0.07	0.14	7.03		
(3)	2SLS	-3.405	2.031	-0.359	1.632	-1.060	0.0611	0.147		8.56	4.41	0.80	142
	HPW / HPGAPW	-3.93***	0.30	-5.15***	5.01***	-2.55**	2.22**	3.61***		0.00	7.03		
(4)	2SLS	-4.664	8.667	-0.300	2.028	-0.619	0.0984	0.063		6.58	3.55	0.49	142
	HPW / HPGAPW	-2.69***	0.95	-3.94***	3.83***	-1.33	3.13***	1.09		0.01	7.03		
(5)	OLS	-2.774	-3.224	-0.325	1.461	-0.940	0.0475	0.150				0.82	142
		-4.89***	-2.01**	-6.46***	5.69***	-2.87***	2.21**	3.47***					
(6)	2SLS	-3.196	-2.933	-0.341	1.650	-1.058	0.0294	0.130		32.28	202.97	0.83	150
	HPM / HPGAPM	-5.62***	-1.71*	-6.74***	6.23***	-3.59***	1.40	3.55***		0.00	7.03		
(7)	2SLS	-1.942	6.222	-0.554	2.128	-1.925	0.0432	0.136		7.80	5.58	0.79	142
	HPD / HPGAPD	-1.62	0.81	-3.57***	5.13***	-3.64***	1.12	3.61***		0.01	7.03		
(8)	2SLS	-2.507	0.267	-0.402	1.686	-1.220	0.0410	0.133		23.44	18.64	0.83	156
	HPN / HPGAPN	-4.24***	0.09	-6.62***	6.17***	-3.85***	1.90*	3.60***		0.00	7.03		
(9)	2SLS	-3.259		-0.343	1.576	-1.004	0.0553	0.147		38.46	158.86	0.81	142
	HPW	-4.71***		-6.98***	5.97***	-3.03***	2.64***	3.57***		0.00	16.38		
(10)	2SLS	-4.398		-0.312	1.691	-0.888	0.0494	0.134		9.56	15.95	0.80	142
	PI	-3.10***		-5.50***	5.41***	-2.39**	2.27**	3.12***		0.00	16.38		
(11)	2SLS	-3.322		-0.341	1.582	-0.998	0.0550	0.147		38.74	84.25	0.81	142
	HPW / PI	-4.84***		-6.97***	6.00***	-3.01***	2.63***	3.56***		0.00	19.93		

	Method & Instruments	*HP*	*LE*	*LYPC*	*FT*	*WLR*	*HB*	*SMGAP*	*EDGAP*	*GINI*	UIDT	WIDT	*R-sq*	obs. #
(12)	2SLS	-2.521	-0.356	1.471	-0.680	0.0572	0.037	-0.0535			14.66	48.71	0.86	79
	HPW	-2.59**	-5.50***	3.90***	-1.55	2.14**	1.03	-3.21***			0.00	16.38		
(13)	2SLS	-1.531	-0.297	1.482	-0.613	0.0331	0.093		1.251		7.60	2.54	0.80	93
	HPW / GC / PH	-1.87*	-4.20***	3.06***	-1.77*	0.94	2.49**		3.24***		0.02	13.43		
(14)	2SLS	-1.334	-0.264	1.531	-0.403	0.0303	0.103		0.777		20.19	60.85	0.82	98
	HPW	-1.72*	-4.90***	3.39***	-1.37	1.11	2.96***		5.45***		0.00	16.38		
(15)	2SLS	-2.452	-0.307	0.724	-0.822	0.0466	0.114			3.32	12.43	71.62	0.80	57
	HPW	-2.69***	-3.37***	1.36	-1.51	1.55	1.83*			1.39	0.00	16.38		
(16)	2SLS	-3.346	-0.262	1.001	0.261	0.0263	0.018	-0.0956	0.917	-7.91	5.86	6.74	0.80	33
	HPW	-1.77*	-1.53	0.82	0.28	0.44	0.39	-2.14**	1.80*	-1.64	0.02	16.38		
(17)	2SLS	-2.787	-0.193					-0.0837	0.856	-11.00	9.50	16.67	0.79	33
	HPW	-2.30**	-4.00***					-3.24***	2.34**	-3.14***	0.00	16.38		

The top figures are the estimated coefficients, the bottom ones are heteroskedasticity-robust *t*-statistics. ***, **, and * respectively indicate the significance levels at $p<0.01$, $p<0.05$, and $p<0.10$. UIDT: Under-ID test. Kleibergen-Paap rk LM statistic at the top, and the corresponding *p*-value at the bottom (Kleibergen and Paap 2006). WIDT: Weak-ID test. Kleibergen-Paap rk Wald *F* statistic at the top, the Stock-Yogo weak ID test critical value for the Cragg-Donald i.i.d. case for a 10% bias at the bottom (Kleibergen and Paap 2006; Stock and Yogo 2005). Eq (1) includes period dummies, and eqs (1) to (3) and (5) to (14) include region dummies. Eqs (16) and (17) are estimated without additional instruments for controlling for the endogeneity of *EDGAP*. Over-identification test statistics are as follows (Hansen 1982): for eq (11), Hansen *J* statistic is 0.87 and the *p*-value is 0.35, for eq (13), they are respectively1.63 and 0.20.

reverse effect exists among the married. The risk of being widowed in the future seems to affect their happiness. This reverse effect is not detected among the separated or divorced or the never married as in equation (7) or (8), but either of them does not change the significance of $HPGAP$ in any meaningful way.

In sum, we cannot confirm the explanatory power of $HPGAP$ on $LEGAP$ at this point. Subsequently, we omit $HPGAP$ in the following analysis.

5.3.4 Happiness

Equation (9) shows the results without $HPGAP$. The coefficient of HP is significantly negative at the 1% level, and its estimated value is -3.26. The validity of the instrument is supported by both the under- and weak-identification tests (Stock and Yogo 2005; Kleibergen and Paap 2006).

Replacing the instrument by the price level of investment, PI, does not change the results.[4] Equation (10) shows that the coefficient of HP becomes -4.40, which is not significantly different from the estimate in equation (9). In equation (11) where both HPW and PI are included, the over-identification test (Hansen 1982) indicates that these instruments are uncorrelated with the residual. These results suggest that both HPW and PI are valid instruments.

Turning to the other explanatory variables, the results are also consistent with our expectation. The coefficients of LE and FT are significantly negative and the coefficients of $LYPC$ and HB are significantly positive. As for WLR, the positive effect of women's autonomy seems to be more significant.

To further examine these results, we add $SMGAP$, $EDGAP$, and $GINI$. Equation (12) presents the results with $SMGAP$. It indicates that, while $SMGAP$ is significant with the expected sign, HP is also still significant at the 5% level. However, FT and HB become insignificant. This may be due to the inclusion of $SMGAP$, or to the change in sample characteristics. Because the smoking data set covers only European and the former Soviet Asian countries, many developing countries with low levels of medical resources are excluded, resulting in a more homogeneous sample.

Equations (13) and (14) show the results with $EDGAP$. Equation (13) estimates the coefficients with additional instruments (the share of general government final consumption expenditure, GC, and physicians per 1,000

[4] Among a variety of variables not directly related to happiness, using PI alone yields the best results in the first-stage regression. Thus, we employ PI to test the validity of HPW. We refer to Bjørnskov (2008) to look for appropriate instruments.

people, PH), and equation (14) without additional instruments. In both cases, the explanatory power of $EDGAP$ on life expectancy is extracted at the 1% level of significance. The inclusion of $EDGAP$, on the other hand, lowers the significance level of HP, but it is still significant at the 10% level in either equation.

Equation (15) presents the results with $GINI$. As the sample size becomes much smaller and multicollinearity becomes severe, we drop the regional dummies. The results show that, while $GINI$ is not significant at the 10% level, HP is significant at the 1% level.

Finally, all of these three variables are included in equations (16) and (17). While the results must be interpreted with caution because the sample size is small, HP is still significant at the 5% level.

These results support the importance of happiness in explaining the life expectancy gap. A reduction in happiness by 0.1 point would widen the life expectancy gap by 0.25 to 0.35 year.

5.3.5 Regressing Life Expectancy

To further evaluate these results, we regress the level of life expectancy for both sexes and then separately for women and for men. As previously noted, this is not an easy task. The causality runs both directions between life expectancy and happiness, and both are further correlated with behavioral and socio-economic variables. Performing an elaborate regression analysis to explain life expectancy would require at least one independent study.

At the same time, however, comparing the regression results on the life expectancy gap to those on the level of life expectancy in the same framework may provide interesting insight. Thus, we perform regression analysis on life expectancy, aiming only to assess whether regressing life expectancy would yield compatible results to those on the life expectancy gap.

Therefore, we keep the same explanatory variables as those in the basic equations in the main regression model, i.e., happiness, $LYPC$, FT, WLR, and HB, as in equation (9) in Table 5.5. We also include the smoking rate and $GINI$. Although incorporating these two variables reduces the sample size, they cannot be eliminated given their importance on population health and on life expectancy (as for $GINI$, see, e.g., Wilkinson 2000; Deaton 2003). In addition, we employ period dummies in order to capture the technological progress. As for happiness and the smoking rate, we use either the sex-average values or sex-specific values relevant to the dependent variable. To control for the reverse causality between life expectancy and happiness, we continue to use PI as the instrument. Note that HPW is not

an appropriate instrument anymore.

The results are presented in Table 5.6. Equations (1) and (2) indicate that regressing life expectancy for both sexes yields ambiguous results. The coefficient of HP is positive as expected in either equation, but it is significant at the 5% level only if region dummies are omitted.

Equations (3) and (4) suggest that regressing women's life expectancy follows a similar pattern. While the coefficient for women's happiness is positive in either equation, its significance level depends on whether region dummies are included.

On the other hand, regressing men's life expectancy yields more solid results. As shown in equations (5) and (6), the coefficients of men's happiness are significant at the 5% level regardless of whether region dummies are included. These results are consistent with our expectation. The impact of happiness on life expectancy should be easier to capture for men since men's survival, more than women's survival, is strongly tied to happiness.

Furthermore, equations (4) and (6) indicate that the coefficient of happiness is larger for men than for women. This accords with the hypothesis as well as the regression results on the life expectancy gap. While the difference is somewhat larger, as a decrease in happiness by 0.1 point is estimated to raise the life expectancy gap by 0.5 year, it is still a good approximation, considering the experimental nature of the current regression model.

These results also provide an explanation for why it is difficult to capture the effect of happiness on the level of life expectancy. Its impact differs between women and men. Therefore, life expectancy for women and men should be treated differently to analyze the impact of happiness.

5.3.6 Comparison with Previous Empirical Findings

One major limitation of this study is that the data cannot be treated as panel data. Thus, the results might have picked up uncontrolled cross-country differences. This casts doubt on the credibility of the regression results.

To cope with this issue, we compare this study to previous studies that regress the life expectancy gap on behavioral and socio-economic factors with cross-country panel data. Specifically, we look at Pampel and Zimmer (1989) and Ram (1993), which examine how women's status influences their survival. As the present study shares the same dependent variable and most of the explanatory variables with these previous studies, the present study can, in one sense, be viewed as an extended model that incorporates happiness but with an unbalanced panel. Inconsistent results would indicate

Table 5.6: Regression results (Dependent variable: LE, LE (women), and LE (men))

	Happiness	*LYPC*	*FT*	*WLR*	*HB*	*Smoking*	*GINI*	Region Dummies	UIDT	WIDT	*R-sq*
LE											
(1)	8.701	-0.054	1.450	0.009	-0.008	-0.0756952	-14.00	incl	4.92	5.80	0.91
	1.68	-0.05	0.84	0.01	-0.05	-1.32	-2.30**		0.03	16.38	
(2)	8.142	0.945	-3.393	-0.184	-0.207	-0.1334461	-15.7	excl	10.35	21.35	0.85
	2.19**	0.56	-1.77*	-1.60	-1.32	-2.15**	-2.84***		0.00	16.38	
LE (women)											
(3)	5.943	0.161	1.556	0.065	0.002	-0.045305	-9.60	incl	4.35	4.75	0.88
	1.21	0.20	0.86	0.47	0.02	-0.91	-1.85*		0.04	16.38	
(4)	5.615	1.514	-2.648	-0.129	-0.176	-0.0876942	-13.02	excl	10.70	30.34	0.79
	1.85*	1.12	-1.44	-1.21	-1.30	-1.74*	-3.00***		0.00	16.38	
LE (men)											
(5)	12.649	-0.908	1.011	-0.027	0.054	-0.1203013	-14.97	incl	6.33	8.87	0.91
	2.42**	-0.55	0.49	-0.17	0.28	-2.06**	-2.19**		0.01	16.38	
(6)	10.586	-0.017	-4.213	-0.243	-0.168	-0.1631694	-16.07	excl	9.42	13.90	0.88
	2.05**	-0.01	-2.05**	-1.86*	-0.92	-3.38***	-2.51**		0.00	16.38	

The top figures are the estimated coefficients. The bottom figures are heteroskedasticity-robust t-statistics. ***, **, and * respectively indicate the significance levels at $p<0.01$, $p<0.05$, and $p<0.10$. The number of observations is 42. All equations include period dummies. To control for the endogeneity of happiness, *PI* is used as the instrument. As for happiness and smoking, eqs (1) & (2) employ the sex-average values, and the others use those of the relevant sex. With regard to under- and weak-ID tests, see Table 5.5.

possible errors in model specification in the present study.

We then focus on the explanatory power of each independent variable. Pampel and Zimmer (1989) find significant positive effects in the women's labor force ratio, income inequality (Gini), cigarette consumption per capita, and alcohol consumption per capita, and a significant negative effect in the sex difference in smoking prevalence (the ratio of lung cancer deaths that occur to women). Economic and health resources (GDP per capita and physicians per 1,000 people) and the sex gap in education (women's tertiary education enrollment ratio) are found insignificant. On the other hand, Ram (1993) reports significant positive effects in the women's labor force ratio, economic resources (energy consumption per capita), and the sex difference in education (women's secondary education enrollment ratio), and a significant negative effect in fertility. Health resources (population per physician) and income inequality (Gini) are found insignificant.

The findings in the present study do not contradict these results. While several variables are significant in some of the studies while insignificant in the others, the coefficients of the significant variables share the same sign. For example, all of the three studies have found significant positive effects in the women's labor force ratio. Similarly, Pampel and Zimmer (1989) and the present study have found significant negative effects in the sex difference in smoking prevalence. This consistency lends support to the present analysis.

5.4 Concluding Remarks

The aim of this study is to test the explanatory power of happiness on survival at the aggregate level. Based on the findings that psychological stress adversely affects survival and that its effect on survival is more severe for men, this study employs the sex difference in life expectancy as the dependent variable, and shows that happiness is significant in explaining the cross-country differences in the life expectancy gap. As national average happiness declines, the sex difference in life expectancy increases. This result suggests that happiness has a significant impact on survival even at the aggregate level.

This study also finds that the relationship between happiness and the life expectancy gap is not unidirectional. While happiness affects the life expectancy gap, the life expectancy gap influences happiness through the composition of marital status and, possibly, also through other unknown factors. This demonstrates the importance of controlling for endogeneity to study the relationship between happiness and survival at the aggregate

level. As found in the relationship between the happiness gap and the life expectancy gap, the OLS method may give misleading estimates.

Finally, evaluating the results from policy perspectives suggests that the returns of policies that enhance happiness are larger than are generally considered. Improving men's happiness would, *ceteris paribus*, increase their survival probability, lower the women's widowhood ratio, and raise women's happiness as well. For example, if the government succeeds in raising national happiness by 0.1 point, the life expectancy gap would shrink by 0.25-0.35 year by saving men's lives, and national happiness would further increase by 0.0004-0.0005 point by improving women's happiness.

This, however, should be understood merely as an example of the feedback effects that happiness generates. Happiness intertwines with a number of behavioral and socio-economic variables, making it difficult to quantify the returns of policies that are erected to happiness. Therefore, such policies must be evaluated in various perspectives in order to comprehend their extensive effects.

5.5 Data Appendix

5.5.1 Sample Periods

The sample periods consist of four periods: 1980-1984 (1), 1990-1994 (2), 1995-1999 (3), and 2000-2004 (4). This follows the sample periods of the dependent variable, $LEGAP$. Happiness data are attached to these periods according to wave number. For the variables taken from PWT, LIS, WHO Europe, and the World Bank, the averages are calculated within each period. For $EDGAP$, although the data are generally calculated every 5 years (e.g., 1980, 1990, and 1995), the newest data are of 1999. Thus, the 1999 data are used for the fourth period.

5.5.2 Sample Countries and Sample Periods in Table 5.5

Equations (3 *to* 5*, and* 9 *to* 11): Albania (3, 4), Algeria (4), Azerbaijan (3), Argentina (2, 3, 4), Australia (1, 3), Austria (2, 4), Bangladesh (3), Armenia (3), Belgium (1, 2, 4), Bosnia and Herzegovina (3, 4), Brazil (2, 3), Belarus (3, 4), Canada (1, 2, 4), Chile (2, 3, 4), China (2, 3, 4), Colombia (3), Croatia (3, 4), Czech Republic (2, 4), Denmark (1, 2, 4), El Salvador (3), Estonia (2, 3, 4), Finland (2, 3, 4), France (1, 2, 4), Georgia (3), Germany (2, 3, 4), Greece (4), Hungary (2, 3, 4), Iceland (1, 4), India (2, 4), Ireland (1, 2, 4), Italy (1, 2, 4), Japan

(1, 3, 4), Jordan (4), Republic of Korea (3), Kyrgyzstan (4), Latvia (3, 4), Lithuania (2, 3, 4), Luxembourg (4), Malta (1, 4), Mexico (2, 3, 4), Republic of Moldova (3, 4), Morocco (4), Netherlands (2, 4), New Zealand (3), Norway (2, 3), Pakistan (4), Peru (3), Philippines (4), Poland (2, 3, 4), Portugal (2, 4), Puerto Rico (3), Romania (2, 3, 4), Russia (2, 3, 4), Singapore (4), Slovakia (2, 3, 4), Vietnam (4), Slovenia (2, 3, 4), Spain (1, 2, 3, 4), Sweden (1, 2, 3, 4), Switzerland (2), Turkey (2, 3, 4), Ukraine (2, 3), Macedonia (3, 4), Egypt (4), UK (1, 2, 3), US (1, 2, 3, 4), Uruguay (3), Venezuela (3, 4)

Equation (12): Albania (4), Austria (2), Armenia (3), Belgium (1, 2, 4), Bosnia and Herzegovina (4), Belarus (3, 4), Croatia (3, 4), Czech Republic (2, 4), Denmark (2, 4), Estonia (2, 3, 4), Finland (2, 3, 4), France (1, 2, 4), Georgia (3), Germany (3, 4), Greece (4), Hungary (2, 3, 4), Iceland (4), Ireland (1, 2, 4), Italy (2, 4), Kyrgyzstan (4), Latvia (3, 4), Lithuania (2, 3, 4), Luxembourg (4), Malta (4), Republic of Moldova (1), Netherlands (2, 4), Norway (2, 3), Poland (2, 3, 4), Portugal (2), Romania (2, 4), Russia (2, 3, 4), Slovakia (2, 3), Slovenia (2, 3, 4), Spain (2, 3, 4), Sweden (1, 2, 3, 4), Switzerland (2), Turkey (4), Ukraine (3, 4), Macedonia (3), UK (1, 2, 3)

Equation (14): Algeria (4), Argentina (2, 3, 4), Australia (1, 3), Austria (2, 4), Bangladesh (3), Belgium (1, 2, 4), Brazil (2, 3), Canada (1, 2, 4), Chile (2, 3, 4), China (2, 3, 4), Colombia (3), Denmark (1, 2, 4), El Salvador (3), Finland (2, 3, 4), France (1, 2, 4), Germany (2, 3, 4), Greece (4), Hungary (2, 3, 4), Iceland (1, 4), India (2, 4), Ireland (1, 2, 4), Italy (1, 2, 4), Japan (1, 3, 4), Jordan (4), Republic of Korea (3), Malta (1, 4), Mexico (2, 3, 4), Netherlands (2, 4), New Zealand (3), Norway (2, 3), Pakistan (4), Peru (3), Philippines (4), Poland (2, 3, 4), Portugal (2, 4), Singapore (4), Spain (1, 2, 3, 4), Sweden (1, 2, 3, 4), Switzerland (2), Turkey (2, 3, 4), Egypt (4), UK (1, 2, 3), US (1, 2, 3, 4), Uruguay (3), Venezuela (3, 4)

Equation (15): Australia (1, 3), Austria (2, 4), Belgium (2, 4), Canada (1, 2, 4), Czech Republic (2), Denmark (2, 4), Estonia (4), Finland (2, 3, 4), France (1, 2, 4), Germany (2, 4), Greece (4), Hungary (2, 3), Ireland (2, 4), Italy (2, 4), Luxembourg (4), Mexico (2, 3, 4), Netherlands (2), Norway (2, 3), Poland (2, 3), Romania (3), Russia (2, 3, 4), Slovakia (2, 3), Slovenia (3), Spain (1, 2, 3, 4), Sweden (1, 2, 3, 4), UK (2, 3), US (2, 3, 4)

Equations (16, 17): Austria (2), Belgium (2, 4), Denmark (2, 4), Finland (2, 3, 4), France (1, 2, 4), Germany (4), Greece (4), Hungary (2, 3), Ireland (2, 4), Italy (2, 4), Netherlands (2), Norway (2, 3), Poland (2,

3), Spain (2, 3, 4), Sweden (1, 2, 3, 4), UK (2, 3)

5.5.3 Data Sources

Barro, R.J., & Lee, J.W. (2000). International data on educational attainment: Updates and implications. *CID Working Paper*, 42. Center for International Development, Harvard University.

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Chapter 6

Exploring the Myth of Unhappiness in Former Communist Countries: The Roles of the Sex Gap in Life Expectancy and the Marital Status Composition

6.1 Introduction

Disparities between former communist European countries and the other European countries, often referred to as "East-West divides," have been observed in various disciplines. An example comes from happiness studies, indicating that national average happiness is generally lower in former communist countries. Between the scale of 1 (not at all happy) to 4 (very happy), their cross-country average is 2.71, as compared to 3.20 for other European countries (European and World Values Surveys, Wave 4).

Similarly, the difference in happiness between women and men points to the existence of an East-West divide. The cross-country average for former communist countries is -0.025, as compared to -0.004 for other European countries.

Figure 6.1 presents the correlation between national average happiness, HPN, and the difference in happiness between women and men, $HPGAP$

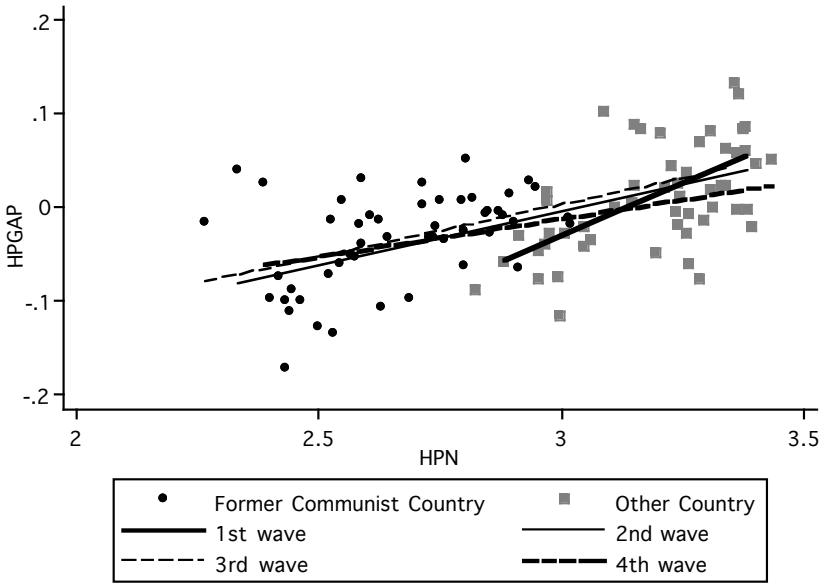

Figure 6.1: Correlation between HPN and $HPGAP$

(EWVS, Waves 1-4). The solid lines illustrate simple linear relationships in each wave. As expected from the findings that both HPN and $HPGAP$ are lower in former communist countries, they are positively correlated. This suggests that women in former communist countries are the group with lowest level of happiness. They are on average less happy than men in the same countries and than women in other European countries.

To explain this positive correlation, one might hypothesize that women's happiness rises more than men's happiness in a favorable environment. This seems consistent with the idea that men are emotionally poor, or that men's happiness is less elastic to external stimuli. In fact, happiness data show that the ratio of respondents who choose "very happy", the top category of happiness, and the ratio of those who choose "not at all happy", the bottom category, are both higher for women. According to the European cross-country data, 20.5% of women choose the top category and 38.7% the bottom category, while the corresponding figures for men are respectively 19.9% and 34.6% (EWVS, Wave 4).

Similarly, one might consider that the positive correlation results from gender-specific social norms that pressure women to be feminine and men to be masculine. While strict gender-specific social norms make both women

and men less happy, their impacts might be more significant for women. Tesch-Römer et al. (2008), for example, argue that societal gender inequality explains varying gender differences in subjective well-being in countries, favoring a culture of gender equality.

The commonality in these arguments is that they connect social factors and happiness at the individual level. By assuming implicitly that the population of each sex is sufficiently homogeneous, they focus on the happiness of the representative woman and the representative man, and consider that the difference in their psychological responses to different social environments gives an explanation for the correlation between happiness and the happiness gap at the cross-country level.

On the other hand, it is also possible to hypothesize that the correlation results from compositional differences in the population that consists of heterogeneous individuals. A major factor generating the heterogeneity is the difference in survival probabilities between women and men. It influences the composition of marital status through the widowhood ratio among women, and, as the widowed are on average less happy, it negatively affects women's average happiness. This implies that HPN and $HPGAP$, both of which reflect women's average happiness, also respond negatively to the difference in survival probabilities, making HPN and $HPGAP$ positively correlated. Chapter 5 in this thesis shows that the sex difference in survival probabilities influences both HPN and $HPGAP$, employing the difference in life expectancy at birth between women and men, $LEGAP$, as its proxy.

At the same time, Chapter 5 also demonstrates that the relationships between HPN and $LEGAP$ and between $HPGAP$ and $LEGAP$ are not uni-directional in that not only does $LEGAP$ affect HPN and $HPGAP$, but HPN, and potentially $HPGAP$, also influence $LEGAP$.

The reasons are as follows. With respect to the effect of $HPGAP$ on $LEGAP$, as found in studies using micro data set, it is simply because happier individuals are healthier and live longer (see Pressman and Cohen 2005; Veenhoven 2008, for reviews). This suggests that a rise in women's relative happiness increases their advantage in health status. As a result, although no previous study has confirmed this effect with aggregate data, $HPGAP$ is expected to positively influence $LEGAP$.

Turning to the effect of HPN on $LEGAP$, the rationale comes from the findings that physiological and behavioral responses to psychological stress (unhappiness) differ between women and men, and that men's mortality responds more elastically to psychological stress (see e.g. Weidner and Cain

2003; Möller-Leimkühler 2003; Helliwell 2007).[1] This indicates that a decline in HPN raises men's mortality more than women's, and results in a larger $LEGAP$. As discussed in Chapter 5, this is consistent with a bio-evolutionary perspective that men are more fragile because of their fiercer intrasexual competition.

These arguments point to the possibility that, in the correlation between HPN and $HPGAP$, $LEGAP$ plays a more important role than exogenously providing an explanation. A decline in HPN widens $LEGAP$ by increasing men's excess mortality, and an increase in $LEGAP$ lowers $HPGAP$ by raising the women's widowhood ratio. Putting these relationships together suggests that $LEGAP$ is possibly the cause that connects $HPGAP$ to HPN.

Taking these hypotheses into consideration, this study aims to assess the following relationships. First, it tests whether, after controlling for socio-economic factors, the cross-sectional correlation between HPN and $HPGAP$ is attributed to their direct relationship, or, alternatively, explained by $LEGAP$. If, for example, variables influencing the correlation are unobservable and cannot be differentiated from HPN even after controlling for socio-economic factors, HPN itself should become the proxy for the unobservable variables and yield strong explanatory power on $HPGAP$. On the other hand, if the sex difference in survival probabilities significantly influences HPN and $HPGAP$, $LEGAP$ should be significant for explaining both HPN and $HPGAP$, given that $LEGAP$ is a good approximation for quantifying the survival probability of adults.

Second, it tests whether $LEGAP$ endogenously generates the correlation between HPN and $HPGAP$. By estimating the impacts of HPN and $HPGAP$ on $LEGAP$ and vice versa, we calculate the significance of $LEGAP$ in the correlation between HPN and $HPGAP$.

The remainder of this study is organized as follows. The next section discusses data and regression strategy, and Section 6.3 presents the results. The details of data, such as data sources and sample countries, are presented in the Appendix. The results show that, while HPN and $HPGAP$ are insignificant in explaining each other, $LEGAP$ is significant in explaining $HPGAP$ and HPN. The reverse effects of HPN and $HPGAP$ on $LEGAP$ are also found significant. These results support the hypothesis that an exogenous decrease in HPN, *ceteris paribus*, widens $LEGAP$ and

[1] This, however, does not necessarily mean that the level of psychological stress is higher for men. As found in Mirowsky and Ross (1995), women are generally at a higher risk of depression. The ways that women and men react to psychological stress are simply different. As described in Nathanson (1977), "women get sick and men die."

this in turn lowers $HPGAP$. This effect accounts for about one-third of the correlation between HPN and $HPGAP$. Section 6.4 concludes.

6.2 Data and Regression Models

6.2.1 Happiness, Happiness Gap, and Life Expectancy Gap

Happiness data are taken from the European and World Values Surveys, Waves 1 (1981-84), 2 (1989-93), 3 (1994-99), and 4 (1999-2004). Among other questions, one question asks, "Taking all things together, would you say you are: very happy (4), quite happy (3), not very happy (2), or not at all happy (1)?"

HPN and $HPGAP$ are calculated for each country in each wave (country-wave). In our sample that contains 82 country-waves (36 countries), the average number of respondents with personal data covering age, sex, and marital status that can be separated into the married, the separated or divorced, the widowed, and the never married, is 1,282 (687 women and 595 men) per country-wave. The largest number is 4,072 (2,180 women and 1,892 men) in Spain (Wave 2), and the least is 359 (191 women and 168 men) in Malta (Wave 2). The number of countries in each wave is, respectively, 7 (Wave 1), 26 (Wave 2), 20 (Wave 3), and 29 (Wave 4).

Table 6.1 presents the data on happiness and the happiness gap averaged separately for former communist countries and for other European countries. To see the effect of the marital status composition on happiness, it presents not only the national averages, i.e., HPN and $HPGAP$, but also marital status-specific figures and the composition of each marital status. Note that, apart from rounding errors, the national averages correspond to the weighted averages of all marital statuses.

The data on happiness indicate that the marital status composition alone cannot fully explain the East-West divide in happiness. Even after decomposing happiness by marital status, happiness is lower in former communist countries.

By contrast, the data on the happiness gap point to the significance of the marital status composition. Compared to the national average, marital status-specific figures are much greater. This is especially apparent in former communist countries. While the national average is negative, marital status-specific figures are all positive.[2] This demonstrates that the marital status

[2]This result also casts a doubt on the general idea that women are less happy than men in European countries.

Table 6.1: Happiness and happiness gap

	National Average	Married	Separated or Divorced	Widowed	Never Married
Former Communist Countries					
Happiness					
Both Sexes	2.720	2.781	2.450	2.441	2.803
Women	2.710	2.792	2.467	2.449	2.832
Men	2.736	2.771	2.417	2.378	2.780
Happiness Gap	-0.027	0.022	0.050	0.071	0.052
Composition					
Women	1.00	0.56	0.10	0.17	0.17
Men	1.00	0.67	0.06	0.04	0.23
Other European Countries					
Happiness					
Both Sexes	3.209	3.300	3.012	2.961	3.147
Women	3.212	3.313	3.026	2.974	3.154
Men	3.207	3.288	2.986	2.890	3.140
Happiness Gap	0.005	0.024	0.040	0.083	0.014
Composition					
Women	1.00	0.55	0.09	0.10	0.26
Men	1.00	0.58	0.07	0.03	0.32

The numbers of observations available in Wave 4 are 14 for both former communist countries and other European countries. The observations (country-waves) with fewer than five respondents in either sex in any of the marital statuses are excluded. This makes *HPN* and *HPGAP* slightly different from the figures in Section 6.1.

composition significantly influences $HPGAP$. The sex difference in the marital status composition, especially, the larger widowhood ratio among women, is a major factor lowering women's average happiness relative to men's.

To further examine the significance of the marital status composition, Table 6.2 presents the correlation coefficients between $LEGAP$ and HPN, between $LEGAP$ and $HPGAP$, and those for each marital status category. As the causality runs in the both directions, we must be cautious about the interpretation. Nonetheless, it clearly shows that the correlation coefficients between $LEGAP$ and the happiness gap for each marital status category are, in absolute value, much smaller than the coefficient between $LEGAP$ and $HPGAP$. For example, while the correlation coefficient between $LEGAP$ and $HPGAP$ is -0.55, the corresponding figure for the widowed is 0.02. This points to the significance of the marital status composition. A large part of the correlation between $LEGAP$ and $HPGAP$ is generated by composi-

Table 6.2: Correlation with $LEGAP$

	Average	Married	Separated or Divorced	Widowed	Never Married
HPN	-0.75	-0.73	-0.66	-0.74	-0.71
HPGAP	-0.55	-0.24	-0.06	0.02	-0.18

The number of observations is 28 in Wave 4.

tional differences in marital status.

The same thing can be said about the relationship between $LEGAP$ and HPN. The correlation coefficients between $LEGAP$ and happiness for each marital status category are smaller in absolute value, although the differences are not as obvious. This supports the existence of the marital status composition effect in HPN.

6.2.2 Regression Models

We first regress $HPGAP$ on HPN and vice versa to test whether HPN and $HPGAP$ explain one another at the cross-country level. As the number of countries in each wave is small, we use all four waves with period dummies.[3] We also include $LEGAP$ and socio-economic variables to control for cross-country differences.

For regressing $HPGAP$, socio-economic variables include per-capita real GDP (thousands of international dollars), YPC, price level, PL, the women's labor force ratio, WLR, fertility rate, FTR, openness, $OPEN$, government share of GDP, GS, the growth rate of per-capita real income, $GYPC$, the average age of the respondents, AGE, and the former-communist dummy, FCD. As no general theory is established for explaining cross-sectional differences in $HPGAP$, the choice of these variables is admittedly arbitrary. They are included on the basis of their potential importance on $HPGAP$.

In addition, we employ an instrumental variable to control for the reverse effect of $HPGAP$ on $LEGAP$. The reverse causality makes the ordinary-least-square (OLS) estimator biased, and thus calls for a two-stage-least-square (2SLS) approach. For this purpose, we employ the difference in smoking rate between women and men, $SMGAP$, as the instrument. The analysis in Chapter 5 shows that $SMGAP$ is significantly correlated with

[3] As the data set is heavily unbalanced and eight countries have only one observation, we treat it as pooled data set. Thus, the present analysis intends to test the cross-sectional correlation.

$LEGAP$, and is an appropriate instrument to control for the reverse causality.

For estimating HPN, the regression model takes the same approach as that of $HPGAP$. To control for the reverse effect of HPN on $LEGAP$, it applies the 2SLS method and employs $SMGAP$ as the instrument. The differences are the inclusion of life expectancy at birth for both sexes, LE, and the replacement of YPC by the log of the per-capita real income, $LYPC$. While YPC is more significant on $HPGAP$, $LYPC$ is more significant on HPN.

It is worth noting that the present analysis does not mean to provide a thorough explanation for cross-country differences in HPN. The current data set is not suitable for this purpose as the sample size is small. Regressing $HPGAP$, on the other hand, is less complex as variables that equally affect women's and men's happiness can be omitted. Thus, the results obtained from regressing HPN should be interpreted with caution.

Next, we regress $LEGAP$ to test the reverse effects of HPN and $HPGAP$ on $LEGAP$. It is again estimated with 2SLS to control for the simultaneous effects. To control for cross-country differences, the regression model includes LE, $LYPC$, $SMGAP$, and WLR, which are found significant in Chapter 5 with a larger data set, and FCD.

As for instrumental variables, we employ happiness of the widowed and the happiness gap of the married. By controlling for marital status, the marital status composition effect should be removed, and the effects of $LEGAP$ on HPN and on $HPGAP$ should be substantially reduced.

Among the four types of marital statuses, the ones for the widowed are expected to be the most suitable instruments. This is because the individuals in this category have already gone through the hardship of losing a spouse, and $LEGAP$ should not have any further composition effect on their happiness.

At the same time, the problem is that the number of widows and widowers are very small. Even though a survey contains on average 1,282 respondents in each country-wave, the numbers of widows and widowers are on average only 88 and 20 respectively. This may deem the results unreliable. To minimize this problem, the observations with fewer than five widows or widowers are omitted in the analysis. However, even after this manipulation, the explanatory power of the happiness gap of the widowed on $HPGAP$ is still too weak. Thus, we use the happiness gap of the married as the instrument for $HPGAP$.

6.3 Regression Results

6.3.1 Correlation between Happiness and Happiness Gap

Table 6.3 presents the regression results for $HPGAP$. Equations (1-1) and (1-2) regress $HPGAP$ on HPN without socio-economic variables. While equation (1-1) includes HPN alone, equation (1-2) incorporates FCD and period dummies. The coefficients of HPN are, with no surprise, significantly positive in both equations.

Equations (1-3) and (1-4) additionally include $LEGAP$. Both equations indicate that HPN loses its explanatory power with the inclusion of $LEGAP$ while the coefficients of $LEGAP$ are significantly negative at the 1% level. The test scores for under- and weak-identification show no sign of identification problems (Stock and Yogo 2005; Kleibergen and Paap 2006), indicating that $SMGAP$ is an appropriate instrument. These results suggest that the correlation between HPN and $HPGAP$ is spurious. HPN picks up the explanatory power of $LEGAP$ when $LEGAP$ is excluded.

Equation (1-5) includes socio-economic variables. After omitting insignificant variables, equation (1-6) shows that the coefficient of $LEGAP$ is still significantly negative. Furthermore, equation (1-7), where HPN is included instead of $LEGAP$, indicates that HPN is significant at the 10% level. This is consistent with the results above that HPN picks up the explanatory power of $LEGAP$.

Turning to other variables, the coefficient of YPC is negative, suggesting that an increase in average income contributes to men's happiness more than women's. Intuitively, this is reasonable, considering that men are more keen on income. On the other hand, the rationale for the positive coefficient of PL is unresolved. It probably captures the effects of unobserved factors. As for FTR and GS, their coefficients are positive, indicating that they are more elastically correlated with women's happiness. Finally, the coefficient of FCD is found positive, suggesting that $HPGAP$ in former communist countries is not necessarily smaller after controlling for socio-economic variables.[4] These results show that $LEGAP$, as well as socio-economic factors, explains cross-country variations in $HPGAP$.[5]

[4]Since the outcome that the coefficient of FCD is significantly positive could potentially be caused by the non-linear relationship between $HPGAP$ and HPN, we also test if adding the square of HPN in equations (1-1) and (1-6) changes the regression results. However, the results do not change in any meaningful way. Both HPN and HPN squared are insignificant in both equations.

[5]WLR and FCD compete for explanatory power. If WLR is included instead of FCD in equation (1-6), the coefficient of WLR becomes significantly positive at the 5%

Table 6.3: Regression results (Dependent variable: $HPGAP$)

	(1-1)	(1-2)	(1-3)	(1-4)	(1-5)	(1-6)	(1-7)
HPN	0.116	0.154	-0.006	0.053	-0.014		0.067
	6.24***	4.64***	-0.14	1.13	-0.30		1.90*
LEGAP			-0.020	-0.019	-0.012	-0.012	
			-3.50***	-3.25***	-2.08**	-3.12***	
YPC					-0.0043	-0.0033	-0.0041
					-3.22***	-2.69***	-3.34***
PL					0.0018	0.0016	0.0016
					4.98	5.64***	4.94***
WLR					0.0017		
					1.01		
FTR					0.038	0.030	0.033
					2.39***	2.47**	2.27**
OPEN					0.0003	0.0002	0.0003
					1.75*	1.66	1.94*
GS					0.0024	0.0025	0.0014
					2.29**	2.77***	1.45
GYPC					0.0006		
					0.31		
AGE					0.002		
					0.89		
FCD		0.030		0.043	0.038	0.046	0.041
		1.68*		1.99**	1.68*	2.25**	2.12**
Period D	excl.	incl.	excl.	incl.	incl.	excl.	excl.
Under-ID			12.14	12.00	20.08	25.45	
Test			0.00	0.00	0.00	0.00	
Weak-ID			23.53	23.42	41.26	63.56	
Test			16.38	16.38	16.38	16.38	
R-sq	0.38	0.40	0.23	0.30	0.62	0.58	0.59

The number of observation is 82. The top figures are the estimated coefficients, and the bottom figures are heteroskedasticity-robust *t*-statistics. ***, **, and * respectively indicate the significance level at $p<0.01$, $p<0.05$, and $p<0.10$. Under-ID test: Kleibergen-Paap rk LM statistic at the top, and the corresponding *p*-value at the bottom (Kleibergen & Paap, 2006). Weak-ID test: Kleibergen-Paap rk Wald *F* statistic at the top, the Stock-Yogo weak ID test critical value for the Cragg-Donald i.i.d. case for a 10% bias at the bottom (Kleibergen & Paap, 2006; Stock & Yogo, 2005). The instrumental variable is *SMGAP*.

Table 6.4 presents the regression results for HPN. With respect to the direct relationship between HPN and $HPGAP$, the results are essentially the same as the ones for $HPGAP$. Equations (2-1) to (2-4) show that the inclusion of $LEGAP$ makes $HPGAP$ insignificant while $LEGAP$ becomes significant. This supports the finding above that the correlation between HPN and $HPGAP$ is spurious. In addition, equations (2-5) and (2-6) show that $LEGAP$ does not lose its significance with the inclusion of socio-economic variables. Again, the test scores for under- and weak-identification show no sign of identification problems.

As for other variables, the results are consistent with our expectation. The coefficients of PL, FTR, $OPEN$, and GS are significantly positive at least at the 10% level. FCD, on the contrary, loses the explanatory power when socio-economic variables are included.

Comparing the results in these two regression models, however, we obtain one unexpected result. The coefficient of $LEGAP$ on HPN is, in absolute value, larger than the one on $HPGAP$. The estimated coefficients are respectively -0.041 and -0.012 in equations (2-6) and (1-6). This indicates that the coefficient of $LEGAP$ on HPN contains something more than the marital status composition effect. As the marital status composition effect works on women's average happiness, its impact on HPN is expected to be smaller than that on $HPGAP$.

Although inconclusive, one potential reason for this result is that $LEGAP$ picks up the uncertainty of survival. A larger $LEGAP$ mirrors less certainty in survival, and therefore indicates a higher chance of unexpected death. This should significantly lower happiness of those left behind, including those who are not the spouse of the deceased.

6.3.2 Life Expectancy Gap

Table 6.5 presents the regression results for the reverse effects of HPN and $HPGAP$ on $LEGAP$. After omitting period dummies as they are insignificant in equation (3-1), equations (3-2) and (3-3) show that both HPN and $HPGAP$ are significant at least at the 10% level with the expected signs. The test scores for under- and weak-identification show no sign of identification problems.

Here, the result that the coefficient of $HPGAP$ is significantly positive is noteworthy. This is the first result supporting the micro findings that

level while other results remain the same. Assuming that a higher WLR indicates greater autonomy in women and more gender equality, this result suggests that women's autonomy and gender equality contribute to women's happiness.

Table 6.4: Regression results (Dependent variable: HPN)

	(2-1)	(2-2)	(2-3)	(2-4)	(2-5)	(2-6)
HPGAP	3.260	1.503	0.776	0.528	0.041	
	9.69***	4.98***	1.23	1.16	0.11	
LEGAP			-0.110	-0.078	-0.040	-0.041
			-5.16***	-3.82***	-1.89*	-3.43***
LE					0.0003	
					0.02	
LYPC					0.0931	0.084
					1.11	1.36
PL					0.0041	0.0042
					2.91***	4.32***
WLR					-0.004	
					-0.99	
FTR					0.103	0.102
					1.45	1.77*
OPEN					0.0006	0.0008
					2.10**	3.04***
GS					0.0058	0.0047
					1.46	1.83*
GYPC					0.0036	
					0.80	
AGE					0.009	0.009
					1.50	1.55
FCD		-0.445		-0.228	-0.0005	
		-10.70***		-3.48***	-0.01	
Period D	excl.	incl.	excl.	incl.	incl.	excl.
Under-ID			30.93	18.35	18.26	22.15
Test			0.00	0.00	0.00	0.00
Weak-ID			39.60	23.42	36.61	54.73
Test			16.38	16.38	16.38	16.38
R-sq	0.38	0.79	0.61	0.77	0.89	0.89

See Table 6.3.

happier people live longer at the aggregate level using the date on the sex difference. However, as the sample size is small, we must be cautious about the interpretation. We need to examine this issue with a large panel data set.

To test whether happiness of the widowed is a valid instrument for HPN, equation (3-4) employs PL instead of happiness of the widowed, and equation (3-5) employs both of them.[6] As noted earlier, the correlation between HPN and $LEGAP$ possibly contains something more than the marital sta-

[6]We use PL because it is most significant in explaining HPN in the previous regression model.

Table 6.5: Regression results (Dependent variable: $LEGAP$)

	(3-1)	(3-2)	(3-3)	(3-4)	(3-5)	(3-6)
HPN	-2.28	-2.61	-3.49	-4.07	-3.61	
	-1.58	-1.87*	-3.07***	-2.89***	-3.34***	
HPGAP	6.28	6.82	7.43	7.82	7.58	
	1.80*	1.93*	2.09**	2.20**	2.16**	
LE	-0.28	-0.33	-0.35	-0.34	-0.35	-0.31
	-3.75***	-5.02***	-5.53***	-5.10***	-5.46***	-5.31***
LYPC	2.30	2.32	2.14	2.26	2.17	1.91
	5.32***	5.24	4.72***	4.28***	4.72***	4.79***
SMGAP	-0.092	-0.084	-0.077	-0.073	-0.076	-0.090
	-4.44***	-4.26***	-4.24***	-4.04***	-4.27***	-6.55***
WLR	0.066	0.058	0.069	0.062	0.068	0.081
	2.59**	2.14**	2.61**	2.34**	2.64***	2.79***
FCD	1.078	0.782				1.289
	1.72*	1.44				3.17***
Period D	incl.	excl.	excl.	excl.	excl.	excl.
Under-ID	16.25	15.77	16.95	16.68	19.64	
Test	0.00	0.00	0.00	0.00	0.00	
Weak-ID	18.78	21.29	27.74	13.69	19.00	
Test	7.03	7.03	7.03	7.03	13.43	
R-sq	0.87	0.86	0.85	0.84	0.85	0.86

The number of observations is 78. In equations (3-1) to (3-3), the instrumental variables are happiness of the widowed and the happiness gap of the married. In equation (3-4), *PL* is employed instead of happiness of the widowed, and, in equation (3-5), both happiness of the widowed and *PL* are used. Over-identification test statistics (Hansen 1982) in equation (3-5) show that Hansen *J* statistic is 0.22 and that the corresponding *p*-value is 0.64. For other information, refer to Table 6.3.

tus composition effect, and the reverse effect of $LEGAP$ on HPN may remain in happiness of the widowed. If it were severe, happiness of the widowed would not be an appropriate instrument.

The results nevertheless suggest that happiness of the widowed is a valid instrument. In both equations, the regression results do not change in any meaningful way. The over-identification test (Hansen 1982) in equation (3-5) also indicates that the instruments are uncorrelated with the residual, supporting the use of both instruments.

With respect to FCD, equations (3-2) and (3-6) show that the inclusion of HPN and $HPGAP$ substantially reduces the explanatory power of FCD. While FCD is significant without HPN and $HPGAP$, it becomes insignificant at the 10% level with the inclusion of HPN and $HPGAP$. This suggests that HPN plays an important role in explaining large $LEGAP$ in former communist countries.

Turning to other variables, the results are consistent with those in Chapter 5. The coefficient of LE is positive, supporting the result in Glei and

Horiuchi (2007). The coefficient of $LYPC$ is also positive, indicating that economic resources are more influential on women's life expectancy. Consistent with the generally accepted assumption that smoking is bad for health, the coefficient of $SMGAP$ is negative. As for WLR, it indicates that the positive effect of women's autonomy is significant. These results also accord with Pampel and Zimmer (1989) and Ram (1993), both of which employ panel data sets. This consistency lends support to the present analysis.

6.3.3 Life Expectancy Gap in the HPN-HPGAP Relation

Using these results, we can numerically calculate the effect of HPN on $HPGAP$ endogenously generated by $LEGAP$. A decline in HPN accompanies a decline in $HPGAP$ since, as found in the regression models, a decrease in HPN widens $LEGAP$ and this in turn lowers $HPGAP$. Therefore, even without any direct relationship between HPN and $HPGAP$, HPN influences $HPGAP$.

To do this, we use the estimated coefficients in equations (1-6), (2-6), and (3-3). Equations (1-6) and (2-6) provide the coefficients of $LEGAP$ on $HPGAP$ and HPN, and equation (3-3) provides the ones of HPN and $HPGAP$ on $LEGAP$. Here, equations (1-6) and (2-6) are estimated again with the same sample as equation (3-3) that includes 78 country-waves.[7]

Taking recursive effects into account, the results show that a decline in HPN by 0.1 point would, *ceteris paribus*, widen $LEGAP$ by 0.37 year, reduce $HPGAP$ by 0.004 point, and further lower HPN by 0.015 point. This indicates that a point decline in HPN would result in 0.036 point reduction in $HPGAP$. As the slope of $HPGAP$ on HPN in Figure 6.1 is 0.112 with the 78 country-wave sample, it accounts for 32% of the correlation between HPN and $HPGAP$. Namely, about one-third of the correlation is endogenously generated by the sex difference in life expectancy.

6.4 Concluding Remarks

This paper examines aggregate measures of happiness from a demographic perspective. The results show that a significant portion of the cross-sectional correlation between national average happiness and its sex gap is attributed to the heterogeneity in marital status generated by the sex difference in

[7]The coefficients of $LEGAP$ on $HPGAP$ and HPN are respectively -0.011 and -0.041, both at the 1% level of significant. These results are virtually equivalent to the results of the previous sample.

survival probabilities. After controlling for the life expectancy gap as well as socio-economic factors, a typical woman in former communist countries per se is no less happy than a typical woman in other European countries.

These results point to the importance of the demographic composition of the population when dealing with aggregate happiness measures. For example, if we had not paid attention to the marital status composition, we could have concluded that happiness directly influences the happiness gap. Similarly, by looking at the happiness gap averaged for former communist countries in Table 6.1, we could have agreed that a typical woman in these countries is less happy than a typical man in the same countries. However, we now know that these results are artifacts of the marital status composition.

Thus, we must keep in mind that aggregate happiness measures contain composition effects. In fact, the marital status composition effect might be only one of many. To correctly interpret aggregate happiness measures and use them as national happiness indicators, we must continue to investigate how the demographic composition of the population influences aggregate happiness measures.

6.5 Data Appendix

6.5.1 Data sources

HPN, $HPGAP$, AGE, and other happiness-related variables: European and World Values Surveys (2006). *European and World Values Surveys four-wave integrated data file*, 1981-2004, v.20060423. Surveys designed and executed by the European Values Study Group and World Values Survey Association. File Producers: ASEP/JDS, Madrid, Spain and Tilburg University, Tilburg, the Netherlands. File Distributors: ASEP/JDS and GESIS, Cologne, Germany.

YPC, $LYPC$, PL, $OPEN$, GS and $GYPC$: Heston, A., Summers, R., & Aten, B. (2006). *Penn world table version* 6.2. Center for International Comparisons of Production, Income and Prices, University of Pennsylvania.

$SMGAP$: WHO Regional Office for Europe (2007). *Health for all database* (http:// www.euro.who.int/hfadb).

WLR and FTR: World Bank (2008). *World development indicators* 2008. Washington DC.

$LEGAP$ and LE: United Nations Population Division (2007). *World population prospects: The* 2006 *revision* (http://data.un.org/).

6.5.2 Sample Periods

The sample periods consist of four periods: 1980-1984 (1), 1990-1994 (2), 1995-1999 (3), and 2000-2004 (4), following the data in UN. Happiness data are attached to these periods according to wave number. For the variables taken from PWT, WHO Europe, and the World Bank, the averages are calculated within each period.

6.5.3 Sample Countries and Sample Periods

Albania (4), Austria (2), Belgium (1, 2, 4), Bosnia and Herzegovina (4), Belarus (3, 4), Croatia (3, 4), Czech Republic (2, 3, 4), Denmark (2, 4), Estonia (2, 3, 4), Finland (2, 3, 4), France (1, 2, 4), Germany (3, 4), Greece (4), Hungary (2, 3, 4), Iceland (2, 4), Ireland (1, 2, 4), Italy (2, 4), Latvia (2, 3, 4), Lithuania (2, 3, 4), Luxembourg (4), Malta (2, 4), Republic of Moldova (4), Netherlands (1, 2, 4), Norway (1, 2, 3), Poland (2, 3, 4), Portugal (2), Romania (2, 4), Russia (2, 3, 4), Slovakia (2, 3), Slovenia (2, 3, 4), Spain (2, 3, 4), Sweden (1, 2, 3, 4), Switzerland (2, 3), Ukraine (3, 4), Macedonia (3), UK (1, 2, 3). Note that, for regressing $LEGAP$, Iceland (2), Latvia (2), Malta (2), and Netherlands (1) are excluded.

Chapter 7

Concluding Remarks

In this thesis, I have demonstrated how bio-demography can approach economics. As presented here, there are various routes for this approach, and any of the routes taken can enrich economics by providing bases of human nature.

To avoid unnecessary confusion, it is worth noting here that this approach is complementary to the conventional approach in economics and will not replace existing economics. What bio-demography can provide for social science is the foundations of human nature that was embedded in humans in the evolutionary time scale. Thus, bio-demography does not necessarily offer direct and relevant explanations for human behavior in today's world. For example, the rate of time discounting discussed in Chapters 3 and 4 is no longer optimal today at both the fitness and utility levels due to the drastic changes in our surrounding environment in the last two million years. Besides, various functions developed in the course of human history, such as culture and learning, affect time discounting behavior as well as other behaviors in today's world.

Nevertheless, the bio-demographic approach is necessary to sort out the assumptions that are biologically valid from those that are not. This operation is indispensable for connecting economics to natural science. Furthermore, it refines economic assumptions and results in improving the accuracy of economic predictions. Using the terms in behavioral economics (Kahneman 2003), this approach provides explanations of behavior at the System 1 level, but not necessarily at the System 2 level although it can account for the evolutionary origin of System 2.

Last but not least, I need to acknowledge that this approach is not new. It is as old as the discipline of economics. As evidence of this, let me quote

an argument that Adam Smith made in the Theory of Moral Sentiments (Smith 1790, first published in 1759).

> Thus self-preservation, and the propagation of the species, are the great ends which Nature seems to have proposed in the formation of all animals. Mankind are endowed with a desire of those ends, and an aversion to the contrary; with a love of life, and a dread of dissolution; with a desire of the continuance and perpetuity of the species, and with an aversion to the thoughts of its intire extinction. But though we are in this manner endowed with a very strong desire of those ends, it has not been intrusted to the slow and uncertain determinations of our reason, to find out the proper means of bringing them about. Nature has directed us to the greater part of these by original and immediate instincts (II.1.27, note 2).

In this argument, Smith placed humans within the animal kingdom, and then explained the biological roots of human instincts. Whereas this argument is no longer exactly accurate, it is worth being credited, given that this book was first published a century before the publication of the Origin of Species (Darwin 1859), which marked the birth of modern biology. In Smith's books, this type of biological views appear in various places, ranging from the association between the value of life and how we sympathize others, the relationship between the kin link and affection, to the biological root of marriage.

It is on this bio-demographic consideration about human nature that economics was established. In the Wealth of Nations, Smith derived "the propensity to truck, barter, and exchange one thing for another (I.2.1)" by comparing behaviors between men and dogs, and it, together with self-love, provided a basis of invisible hand (Smith 1904, first published in 1776).

These bio-demographic perspectives disappeared in the course of scientific progress in economics. While such abstraction is necessary and beneficial for focusing on particular aspects of human behavior, it should not be considered as the one and only way that economics can make advancements. As demonstrated in this thesis, we can import scientific findings of other disciplines into economics. I argue that this is a promising direction to which economics can head in order to establish the science of man.

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