Mortality and Immortality

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ABSTRACT

Mortality and Immortality[*]

It has been known for centuries that the rich and famous have longer lives than the poor and ordinary. Causality, however, remains trenchantly debated. The ideal experiment would be one in which status and money could somehow be dropped upon a sub-sample of individuals while those in a control group received neither. This paper attempts to formulate a test in that spirit. It collects 19th-century birth data on science Nobel Prize winners and nominees. Using a variety of corrections for potential biases, the paper concludes that winning the Nobel Prize, rather than merely being nominated, is associated with between 1 and 2 years of extra longevity. Greater wealth, as measured by the real value of the Prize, does not seem to affect lifespan.

JEL Classification: I12

Keywords: longevity, status, health, wealth, mortality

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1 Introduction

This paper provides evidence consistent with the view that an increase in status raises a person's lifespan. It uses data on the lives of 528 Nobel Prize winners and nominees. The paper uncovers no evidence that the money that comes with the Prize affects the longevity of recipients.

Work in the medical sciences has shown that measures of socio-economic status (SES) are associated with better health and longer life. A famous demonstration of these results comes from two Whitehall studies of British civil servants. The first (Reid et al., 1974) covered 18,403 non-industrial male civil servants aged 40-64 years. Analysis of mortality rates for coronary heart disease (CHD) at follow-up times of 7.5 years (Marmot, Rose & Hamilton, 1978), 10 years (Marmot, Shipley & Rose, 1984) and 25 years (Marmot & Shipley, 1996) all found occupational grade to be an important explanatory factor, with mortality rates successively decreasing with each occupational grade. In the 7.5 year follow-up, men in the lowest grade were 3.6 times more likely to die of CHD than were men in the highest grade, with only around 40% of this difference being explained by obvious risk factors that differ across grade, such as smoking, body mass index, blood pressure, and plasma cholesterol. Importantly, the finding pertained even within the uppermost grades, which exclusively contained well-educated professionals and top administrators, so it could not be explained solely by poverty.

Other studies have confirmed the independent association of civil service grade, after controlling for known risk factors, to mortality rates for a range of other common diseases (van Rossum et al., 2000; Marmot, Shipley & Rose, 1984) and in women civil servants as well as men (Marmot et al., 1991; Ferrie et al., 2002). The association has also been found in all other industrialized countries where the issue has been studied, including amongst populations other than civil servants (Adams et al., 2003; Adler & Ostrove, 1999; Adler et al., 1993).

The question of cause-and-effect, however, remains. Two influential accounts of the causal process based on perceived social status, and its psychobiological consequences, have been proposed by Marmot (2004) and Wilkinson (2000). Lower levels of SES are argued to raise levels of psychological stress, primarily through the association of low SES with low levels of job control (Marmot et al., 1991), and low levels of fall-back social support (Putnam, 2000; Marmot et al., 1991). A growing body of evidence indicates that lack of control in the workplace is associated with increased stress and depressive symptoms (Steptoe et al., 2003a; Stansfeld et al., 1999; Karasek & Theorell, 1990), while the degree to which an individual has social support is believed to determine the degree to which they can buffer job strain (Falk et al., 1992; Johnson & Hall, 1988). Wilkinson (2000) argues that perceived low social standing is linked with feelings of shame, humiliation, disrespect, and social anxiety. It is argued that increased levels of psychological stress can lead to the maintenance over time of high levels of stress hormones, and that this damages endocrine and immunological processes (Steptoe & Marmot, 2005). The result is a causal pathway from SES to mortality.

Important in this account is the idea that human societies are structured into hierarchies. As in other natural species, these occur whenever a species must ration access to scarce resources such as food or mates. Social status reflects position in the various societal hierarchies, and is therefore a fundamentally relative phenomenon. In principle, it can be measured distinct from economic measures of status, although in human societies the two routinely go hand-in-hand.

Experimental studies on rhesus macaques that control for dietary and living conditions, and in which rank can be manipulated, indicate that causation runs from hierarchical position to the presence of stress hormones (Shively, 2000). Sapolsky (1993) finds that levels of biological stress markers follow a hierarchical pattern in troops of wild baboons. However, studies of the cortisol stress marker in humans so far yield mixed results (Steptoe & Marmot, 2005; Kunz-Ebrecht et al., 2004, Steptoe et al., 2003b). When social and economic status go together, the pursuit of social status is reinforced by the greater entitlement to economic resources. Theoretical and experimental research into the

economic consequences of status-seeking behavior includes Becker, Murphy & Werning (2005), Ball et al. (2001), Clark and Oswald (1998), Fershtman, Murphy & Weiss (1996), Frank (1985), Oswald and Powdthavee (2007), and Rablen (2006)).

While medical scientists support the idea that causation runs from SES to health, many economists -- see for example the review by Cutler et al (2006) -- have taken seriously two other possibilities.   One is that causation may be from health to SES (social selection).   Another is that there may exist common factors, the most obvious being unobserved behavioral or genetic heterogeneity, that influence both susceptibility to disease and the determinants of SES. Of course, these routes are not mutually exclusive; it seems likely that all three combine in subtle ways to create the observed association between SES and health. Economists have questioned the psychological notion of perceived social status, or hierarchical position, and wondered whether it is unworkable for policy or testing (e.g. Deaton, 2003). The simplest social selection hypothesis argues that bouts of ill health lead to serious loss of income, so health has a causal link to SES. Economists have gone a step further to argue that individuals who expect a long duration of life may accumulate assets to finance their retirement, and that healthy workers may be paid higher wages due to their higher productivity (Luft, 1975). While social selection is widely thought nugatory by medical scientists, economists give it greater credence. Smith (1998, 1999, 2005) argues persuasively that there are significant pathways from health to economic measures of SES.

Measuring SES in terms of income or education, economists have uncovered mixed evidence for a causal association from SES to health. The majority of the evidence on economic measures of SES suggests that any causal effect is weak and small in magnitude. For instance, Meer, Miller & Rosen (2003) reject causation from wealth to health in US data. Also, holding constant initial health, Gardner & Oswald (2004) find no significant effect from income to later mortality risk using the British Household Panel Survey. Gravelle and Sutton (2006) and Miller and Paxson (2006) cannot find persuasive evidence of health effects from relative income. However, some innovative recent work

by Frijters, Haisken-DeNew & Shields (2005a,b) does suggest a role for income.[1]

An association between education and mortality has been independently confirmed by several authors since an early finding by Kitagawa & Hauser (1973). More recent research argues that a causal connection can be established from education to health (see e.g. Lleras-Muney, 2005; Smith, 2005). An important contribution by Adams et al. (2003) tests for the absence of direct causality from SES to health, using both economic and education measures of SES. The hypothesis of non-causality is, in general, only accepted if no direct causal link is present *and* there are no persistent common factors at work. The authors perform their tests on data from the Asset and Health Dynamics (AHEAD) Panel covering Americans aged 70 and older, and cannot reject the hypothesis of no causal link from SES to overall mortality after conditioning upon initial health conditions. However, for three of the top four causes of death amongst men the hypothesis of no direct causation can be rejected (Adda et al., 2003). The hypothesis of no direct causation is rejected for some mental, chronic, and degenerative conditions, which may be of importance as these are the types of conditions that can result from sustained levels of biological stress.

Testing for the role of social status has taken a different approach. Redelmeier & Singh (2001a) consider the longevity of Oscar winning actors and actresses relative to those who had been nominated for an award but had never won. Winning an Oscar, it is reasoned, should have a positive effect on feelings of perceived social standing. Indeed, as almost as many Americans watch the Oscar ceremony as vote in presidential elections, there are perhaps few greater public validations of self worth in the USA (Davey Smith, 2001). Additionally, film stars generally do not have a substantial amount of education, and are far from the poverty line irrespective of whether they win an Oscar. Finally, while being nominated for an Oscar may be down to ability, it is sometimes said that winning one is due partly to luck. The authors conclude that Oscar winners have a life expectancy 3.6 years longer than the control group of Oscar nominees, which is only slightly reduced

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[1] However, see Snyder & Evans (2006) for evidence that higher-income individuals die slightly younger -- not older -- than lower-income individuals.

by controlling for possible causality from longevity to winning an Oscar. Despite this remarkable finding, a follow-up study (Redelmeier & Singh, 2001b) that performed an identical analysis on Oscar winning and Oscar nominated screenwriters found that the unsuccessful Oscar nominees live on average 3.6 years longer than do winners. Thus the findings of the Redelmeier & Singh papers contradict each other.[2]

1.1 A Test

The paper contributes to this debate by using data on Nobel Prize winners and nominees in Physics and Chemistry between 1901-1950. Winners of a Nobel Prize are held in esteem, achieve international recognition of their work, and are often awarded titles or honors in their home countries. In addition to these markers of social distinction, the prize (which can be shared by up to three people) currently amounts to $1 - $1.5 million, although most winners donate a proportion of their winnings to academic and charitable causes. There is some consensus in the literature (see e.g. Frey, 2005; Layard, 1980), as well as among Nobel Prize winners themselves, that the intrinsic value of a Nobel Prize outweighs its accompanying or subsequent monetary rewards. Accordingly, we interpret winning the Prize as primarily a one-time innovation to social status.[3] The innovation to wealth cannot, though, be ignored. However, we show that there is no relationship over time between the real value of the Prize and the longevity of Prize winners. The idea that the demand for awards relies partly on individuals' desire for distinction, rather than from direct monetary concerns, is a plank of a nascent economic literature on awards (Frey, 2005). Without this, it is difficult to explain the large number of non-monetary awards that exist in many walks of life.

Thus we view winning the Nobel Prize as akin to an extraneous[4] or 'exogenous' boost to status. We attempt to measure the impact of winning a Nobel Prize on longevity by

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[2] After the first draft of our paper was written, our attention was also drawn to the note by Miskie et al. (2003), which discusses the lifespans of Nobel Prize winners but does not correct for the biases discussed in our paper. Their note argues that this Redelmeier-Singh paper is unpersuasive.

[3] In Economics, some believe the Prize is approximately a lifetime achievement award, with a timing that is influenced by the health status of the individual. In the sciences, however, the Prize is more generally viewed as a reward for a single (sometimes quite recent) key discovery.

[4] We thank Angus Deaton for helpful discussions about exogeneity and extraneity.

comparing winners to a control group, namely, those scientists nominated for a Nobel Prize who were never successful. These persons in themselves are top scientists who made important contributions in their individual fields, and are likely to be similar to Nobel winners in terms of SES characteristics and behavioral traits.

The raw data described in Section 2 and analyzed in Section 3 show that historically winners live just under 1.4 years longer than nominees. However, this does not establish a causal link from winning a Nobel Prize to longevity. As all scientists must be alive at the time they are nominated for a Nobel Prize, reverse causality from longevity to winning a Nobel Prize can arise in two ways. Even if scientists are thought to sequentially explore new ideas, some of which turn out to be good and others less so, the longer this process is allowed to continue the more chance any scientist has of stumbling upon a major discovery. Weinberg & Galenson (2005) suggest that this is especially the case for scientists who work inductively, accumulating knowledge from experience, for they tend to write their best works later in their careers than do scientists who work deductively from abstract principles. Second, there is a variable lag, in some cases large, between the award of a Nobel Prize and the completion of the work for which it is awarded. Low survival increases the likelihood of dying before receiving a Nobel Prize, even when the Prize would have been merited.

For these reasons, the remainder of the paper is devoted to modes of analysis that aim to overcome these difficulties. In Section 3 we present a simple non-parametric test of the null hypothesis that winners and nominees have identical survival. This proposes one way to overcome the issue of reverse causality. It compares the longevity of each given winner against the expected longevity of a nominee, *conditional on that nominee having lived to the age at which the winner won a Nobel Prize*. This is a simple matching test. The test relies upon the near absence of censoring in the lifespan data, which is a feature of our data, but not of the datasets used in the earlier research of Redelmeier & Singh (2001a,b).

In Section 4 we present a semi-parametric regression-based approach using the

proportional hazards model of Cox (1972). This allows us to control for heterogeneities between the two Prizes and across countries, as well as for possible sources of status other than winning a Nobel Prize. To mitigate reverse-causality bias we use the method of time-dependent covariates. Our final estimate here is that winners live 2.1 years longer than do nominees. Analysis of winners shows that there is no relationship over time between the real value of the Prize and the longevity of Prize winners. Hence, as well as being, in an important but controversial area, a test in the spirit of Redelmeier & Singh, this paper allows a test for the influence of an increase in wealth upon longevity. It also suggests a methodology not performed by Redelmeier & Singh. Section 5 concludes.

2   Data

The statutes of the Nobel Foundation mandate that the names of nominators and their nominees are to be kept secret for a period of 50 years. Because the Nobel Prizes in Chemistry, Literature, Peace, Physiology-or-Medicine, and Physics were first awarded in 1901, there are now more than 50 years of data on nominees for these Prizes. Our sample is drawn from a published census of Nobel Prize nominees in the Chemistry and Physics Prizes for the period 1901-1950 (Crawford, 2002). Of the 560 scientists nominated for one of these Prizes over this period, we were able to find the year of death for 532 scientists using the internet search engine Google, the `Recent Deaths' column of the academic journal Science, and a rolling scientific bibliographic dictionary initiated as Poggendorff (1863). Where possible, when information from the internet was conflicting, we used information linked to reputable published sources, such as the online entries from the Encyclopedia Britannica. Information on nationality, sex, year of birth, plus a full record of the years in which each scientist was nominated and/or won each Prize was collected from Crawford (2002). Three scientists are thought to be alive at time of writing. We were unable to find the year of death for the remaining 24 scientists assumed to be dead. They are therefore omitted.

Of the 532 scientists with known biographical details, we restrict attention to male scientists, of which there are 528, in order to escape the difference in life expectancy

across sexes. To avoid misplaced inference due to outliers, we drop a further four scientists who are known to have died prematurely from non-biological causes. These are Henry G. J. Moseley and Eduard Buchner, who both died in active combat in the First World-War; and Pierre Curie and Arnold J. W. Sommerfeld, who were both fatally wounded in traffic accidents. The final sample therefore contains some 524 scientists, of whom 135 won a Nobel Prize. For clarity we shall refer to these latter scientists as `winners'. Those scientists who never won a Nobel Prize we term `nominees'. The winners group contains twelve scientists who were nominated for, but did not win, either the Chemistry or Physics Prize, but instead won the Nobel Prize for Physiology or Medicine.[5] No scientist won either the Prize in Literature or the Prize in Physiology-or-Medicine in conjunction with the Chemistry or Physics Prizes.[6]

3   Non-Parametric Analysis

Average lifespan is just over 76 years, measured as the area under the survival function (Kaplan-Meier method). This is perhaps surprising for it is equivalent to male life expectancy for people born in the US in 2006, yet the sample was born on average in 1876, predominantly in rich countries with comparable levels of life expectancy. However, it can most probably be explained by the fact that all scientists in the sample survived infancy.

The baseline characteristics of winners and nominees are shown in Table 1 (standard deviations in parentheses).

Winners were born several years later in time than the sample average, but the difference is not statistically significant. Demographically, the two groups are similar. Two potentially important differences are that winners are first nominated for a Prize some ten

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[5] We count as a Physics winner a scientist who won the Prize in Physiology-or-Medicine and who was nominated for the Physics Prize, and similarly for chemists. This avoids (*i*) the necessity to somehow allow for `unsuccessful' nominees who won a Physiology-or-Medicine Prize and (*ii*) having to lose 12 winners.
[6] No individual has both won the Nobel Peace Prize and been nominated for (without winning) the Chemistry or Physics Prizes. However, Linus Pauling won both the Chemistry (1954) and Peace (1962) Prizes.

years earlier on average than are nominees, and are also nominated in more than 1.5 times as many distinct years.[7]

Second, we also decompose the data for each individual Prize. There appear to be more observations here due to the presence of 34 scientists who were nominated at various times for both Prizes. The statistics reveal some small heterogeneity between Prizes. Physicists lived almost a year longer than Chemists. Second, the Physics Prize has tended to draw from a smaller pool of scientists, with scientists first being nominated at a younger age and winning at a younger age also. Nevertheless, at least for the raw data, we cannot reject the hypothesis that the estimated survival functions for each Prize are the same using a log-rank test.

These raw data yield a first opportunity to assess the hypothesis that survival of winners differs from that of nominees.

In uncensored populations we calculate mean lifespan directly. In censored populations we predict mean lifespan as the area under the relevant survival function. Using the survival functions for winners and nominees (Figure 1), winners are estimated to live 1.4 years (77.2 v. 75.8) longer than nominees, which breaks down to an effect of 1.3 extra years in Chemistry (76.6 v. 75.3) and 1.2 extra years in Physics (77.4 v. 76.2).[8] However, the inferences we can make from these results are limited. Perhaps unsurprisingly, there is too much noise in the raw data to be able to show statistical significance at conventional levels for these differences: we cannot distinguish the survival functions for winners and nominees in Figure 1 using a log-rank test.

Second, these reported differences cannot be given a causal interpretation, for reasons

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[7] That winners were nominated 10 years earlier on average suggests that they arguably had more, and earlier, inherent status than did nominees. This does not affect the nature of the test described in this paper, but it could potentially lead to an underestimation of the true effect (if, hypothetically, those who went on to win already knew that they were particularly distinguished scientists, and thus gained less, psychologically, from the award of the Prize per se).
[8] The apparent discrepancy here is due to the effect of scientists nominated for both Prizes and the effects of rounding.

already discussed.
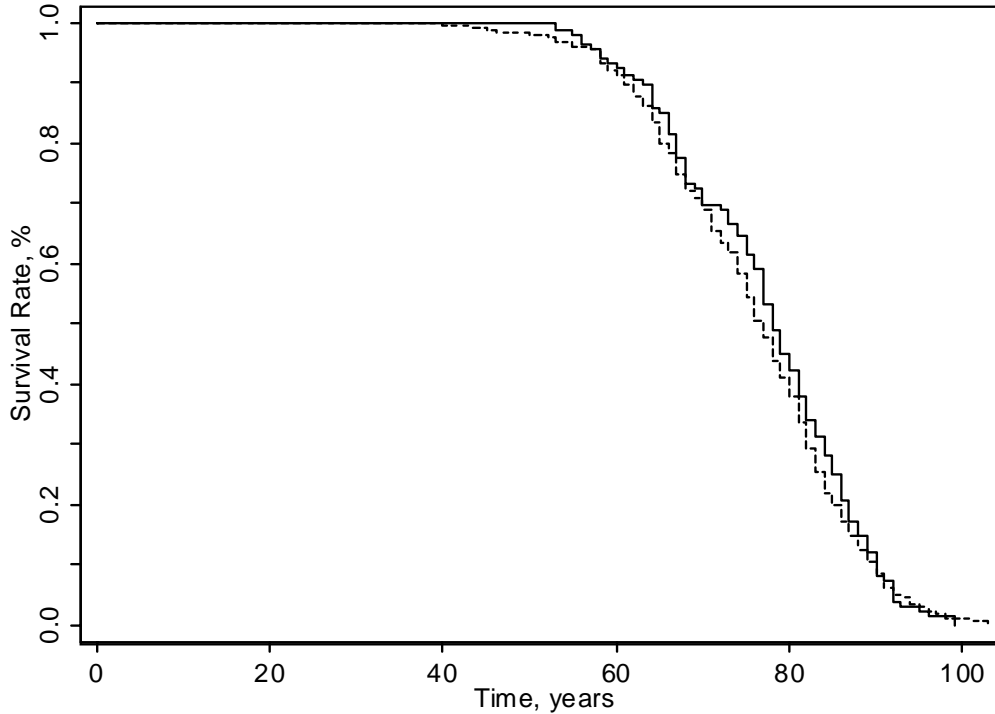
Figure 1: Survival Function for Winners (solid line) and Nominees (dashed line)

There are, however, simple non-parametric tests that can address the issue of reverse causality. If we take two identically aged scientists, one who will never win a Nobel Prize, and another who has already won a Nobel Prize, then later observed differences in lifespan cannot be explained in terms of a causal process from longevity to winning a Nobel Prize. We build on this idea.

Let the observed lifespan of each nominee j be denoted L_j^N and of each winner i, L_i^W. Denote the age at which each winner i won a Nobel Prize as A_i^W. A simple test is to compare for each winner the extra years of life lived after winning a Nobel Prize $\left(L_i^W - A_i^W\right)$ with $E_j\left[L_j^N - A_i^W \mid L_j^N \geq A_i^W\right]$, that is, the expected number of years lived by a nominee after age A_i^W, conditional on having reached age A_i^W. Under the null hypothesis

that winning a Nobel Prize has no effect on longevity, the two series of numbers should have the same mean. However, this test may have low power against the null hypothesis because it over-samples from the longest-lived nominees, who tend to be those born latest in the sample. To control for the growth of life expectancy over time, we limit the set of nominees over which we take expectations to those born within a small window of time of the winner. Looking ± 1 year around a winner's year of birth we term a window of three years (looking ± 2 years corresponds to a window of five years, and so on).

This test matches each winner with one or more nominees born in almost the same year as the winner. The algorithm used to group the data searches both forwards and backwards in time around each winner's birth year to find all nominees born within a window period of three years. If at least one nominee j is found such that $L_j^N \geq A_i^W$ then the winner is said to be matched. If not all winners are matched, the algorithm increases the window size by $+2$ and begins again. Therefore, the window size eventually employed is the smallest that enables us to match all the winners in the sample.

To obtain an estimate of the upward bias in the longevity of winners due to reverse causation, we can compare the difference in means from this test using the conditional expectation $E_j\left[L_j^N - A_i^W \mid L_j^N \geq A_i^W\right]$ with an otherwise identical test that instead employs the unconditional expectation $E_j\left[L_j^N - A_i^W\right]$.[9] A problem with implementing this test is that three scientists in our sample are still alive. In what follows, we assume for simplicity that each will die in the final time period (2006), which will under-estimate their true lifespans. We show later that our qualitative findings are robust to more extreme assumptions about their average lifespan.

The results of the conditional and unconditional matching test are reported in Table 2. These are respectively for the full sample; the USA; the European Union (EU); the most

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[9] Since we are computing an unconditional mean we need only check that there exists at least one nominee in the window, the condition that $L_j^N \geq A_i^W$ being redundant.

represented EU country (Germany); and the sample broken down by prize. `Conditional' here means that winners are matched only to nominees in the window who were alive at the age the winner won the Nobel Prize. The third and fourth columns report the difference in means between the lifespan of winners and nominees for the conditional and unconditional tests respectively. The unconditional differences are, by definition, uncorrected for bias, and we would therefore expect them to be positive. However, under the null hypothesis that the Nobel Prize has no effect on longevity, we would expect our corrected conditional estimates to lie around zero, with some estimates positive and others negative, due to the presence of noise in the data. Contrary to this prediction, in Table 2 we find positive differences in all the subsamples considered and for the sample as a whole. Winners outlive nominees.

It is noticeable that the estimated effect is larger in the single country sub-samples relative to those that mix scientists from different countries. This suggests that heterogeneities in the matches can obscure the longevity effect. In particular, we obtain estimates exceeding one and two years respectively for the Germany and the USA. For the matching test applied to the full sample, we find this issue so serious that we perform a further test that controls for unexplained variation in longevity across countries. In addition to restricting the expectation to nominees within the window, we further remove from the expectation nominees of a different nationality to the winner. Under this stricter matching rule it becomes impossible to match all winners without allowing the window size to converge towards the span of the full sample. In the row labelled `All (controls only of winner's nationality)' we present the results for a window-size of 13 years (the results at other window sizes being comparable). This procedure significantly increases our longevity estimates for the full sample by around two-thirds of a year.

Comparing the third and fourth columns yields initial estimates of the effect of conditioning on nominees living to at least the age their matched winner won a Nobel Prize. These estimates range from 2.56 years for the US to only 0.67 years for the EU. This is, then, an initial measure of the extent to which winners wrongly appear to live

longer due to causality from longevity to winning a Nobel Prize. A final thing to note from Table 2 is that the conditional test appears to exacerbate differences between the two Prizes, with no apparent winning effect visible for the Physics Prize. Again, however, it is worth treating this apparent finding with caution as the Physics and Chemistry sub-samples are heterogeneous in many respects.

The three scientists still alive have a current average age of 90.7 years. It is a simplification to assume that all die in 2006. Were we to assume that all three lived to 100, the conditional estimate for the full sample falls to 0.27. Were they to live to 110 it falls again to 0.19, but nevertheless remains positive. Since all three are chemists, the 0.04 years of the Physics test is unaffected. One of the scientists is German, but is not matched to any German winner by the grouping algorithm, so the German result of 1.30 years is unaffected. Another of the scientists is American. Were he to live even to 110, we still estimate a 1.87 year difference for the USA. We conclude that our qualitative conclusions are robust to reasonable assumptions concerning the lifespans of those scientists still alive.

Given the amount of noise in lifespan data, the size of sample, and the relatively small effect we are trying to isolate, we would not expect to find statistical significance at conventional levels from such a test. Nevertheless, the fact that the conditional matching test yields positive estimates from the full sample and all the main sub-samples, with the estimates in the most homogeneous sub-samples being the highest, is suggestive.

The `All' result of one-third of a year from winning a Nobel Prize is probably too low. When Americans are matched with fellow Americans, Germans with Germans, and EU scientists with others from the EU, the longevity effects of winning are between two-thirds of a year and 2 years. However, it is true to say that the non-parametric tests considered here are not capable of providing precise measures of the degree to which winners out-live nominees. In the next section we address this issue.

4 Semi-Parametric Analysis

We now estimate a hazard model to the data. The advantage of this approach over that of the previous section is that we can produce estimates of the effect of longevity from winning a Nobel Prize in an environment where we control for confounding influences, including the possibility that there might exist important sources of status other than winning a Nobel Prize.

The workhorses of much survival analysis in economics are the Mixed Proportional Hazards (MPH) model and the Proportional Hazards (PH) model. They represent a compromise between possibly over-specified fully parametric forms and the non-parametric approaches discussed in the previous section. The MPH model includes the PH as a special case, but has the advantage of allowing for unobserved heterogeneity at the cost of an additional parameter. We estimate both models and perform a Likelihood Ratio Test of the additional value of the extra heterogeneity parameter versus the PH model. For each regression equation, we find that the additional Likelihood between the MPH and PH models is insufficient to justify the additional parameter. We therefore present results for the more parsimonious PH model. The proportionality assumption maintained in the PH model cannot be rejected based on a global test using the unscaled Schoenfeld residuals, or from tests on the individual covariates. Analysis of the Cox-Snell residuals suggests that the PH model provides a good overall fit to the data.

In the first instance we measure time from the date of birth, thereby conforming the analysis to that of longevity. In choosing the covariates we control for systematic differences between winners and nominees on dimensions that are possible sources of status (other than from winning a Nobel Prize). It might be that being a successful scientist over a prolonged period is an important source of status independent of winning the Prize. This would produce confounding influences since winners are nominated over 1.8 times more years than nominees (Table 1). We therefore include as a covariate the number of distinct years each scientist was nominated for either of the two Prizes: the idea being that scientists were at the top of their respective fields during the period in

which they received nominations. The measure does not work well with respect to winners as they typically cease to be nominated after winning the Prize. Nevertheless, if time at or near the top is what is important, we would expect this to show up in an analysis of the nominees alone.

A second variable we include is an individual's age at first nomination, for this could influence our results in a number of ways. On the one hand, in essentially all professions, those who rise fastest are regarded highly, and therefore enjoy high social status. The ability to reach the top early in life may also be an indicator of underlying genetic qualities. Alternatively, it may be that there is adaptation over time to boosts to status. It might be better to boost status later in life, at a time when resistance to disease would be more valuable. Again, risk of confounding arises since winners are first nominated almost ten years younger than nominees (Table 1). In addition to these controls, we allow for systematic differences between the two Prizes by defining the dummy variable 'Chemistry' that takes a value of one for scientists nominated in the Chemistry Prize. We include dummies for country-specific effects.

Finally, within the regression context we must still deal with the issue of reverse causality. This is done by using time-dependent covariates within the PH model. Specifically, we estimate a model in which there is a step function for winning a Nobel Prize, such that a scientist is coded as a nominee until they win the Prize, and as a winner thereafter.

4.1 Results

Table 3 contains the results when time-zero is the year of birth. We term the analyses in Table 3 as `probability-of-death' equations, which, while not literally accurate, is to make clear that negative entries are associated with longer observed lifespans, while positive entries are associated with shorter observed lifespans. More precisely, the columns marked `% Mortality' show the percentage change in the relative mortality rate associated with a given covariate.

In regression I, winning a Nobel Prize is associated with a 32% decline in relative mortality. The comparable analysis of Oscar winners and nominees by Redelmeier & Singh (2001a) reported a 24-25% decline in relative mortality. Estimation of the baseline survival function allows us to convert these estimates into differences in expected lifespan. Doing this, the implied difference in years of life between winners and nominees is 3.6 years (78.7 v. 75.2). Consistent with expectation, we find evidence of increasing life expectancy across the period of the sample, equivalent to a decline in relative mortality of 1.6%. Paralleling the findings of Table 1, we also find an increased mortality risk associated with chemists relative to physicists, although we are unsure as to why. Age at first nomination is also found to be a strong predictor of longevity, with those older at first nomination living longer. However, interpreting this latter finding causally is dangerous.

In regression II we add the `years nominated (#)' covariate and additional controls. The `exposed to radiation' dummy is for three scientists (Henri Becquerel, Frederic Joliot & Wilhelm Rontgen) who are known to have been exposed to harmful amounts of radiation in the course of their research, at a time when the health effects of doing so were not fully understood. Two other prominent scientists (Marie and Pierre Curie) would also have entered this category were they not excluded on other grounds. We also know that at least two outliers in our data are scientists who committed suicide (Hans Fischer and Rudolf Schoenheimer). We decided that scientists who were exposed to radiation and who committed suicide should not be excluded from the analysis: in the case of suicide the cause of death cannot be assumed exogenous to the individual, while the extent to which radiation shortened the lives of particular scientists can only be guessed at, not proved. Nevertheless, it would be disconcerting if our results hinged on such factors, so we test for such a possibility. In Regression II, eliminating such scientists increases slightly our estimate of the effect of winning, though has little effect on the confidence intervals. Estimates for the other parameters are relatively unchanged. The number of years a scientist was nominated appears to be associated with small reductions in relative mortality risk, but the effect falls short of conventional significance levels.

Regressions III and IV are analogous to I and II, but now we adjust for the bias caused by reverse causality by specifying the winner covariate as a time-dependent step-function. The effect of this change is to reduce the estimated drop in relative mortality of winning from 32.4% to 25.3%. The co-efficient is sufficiently well estimated for this to be significant at the 5% level. Redelmeier & Singh (2001a) estimate a 20% relative reduction for Oscar winners and nominees with a time-varying step function. Converting our finding into life expectancy, we obtain a difference of 2.6 years (77.7 v. 75.1) between winners and nominees. There appear to be few differences in the estimation of the remaining variables relative to the analysis with fixed covariates. Regression IV shows that these qualitative conclusions continue to hold in the presence of further controls.

The positive co-efficient estimated on the `Age at first nomination' covariate contains a possible upward bias due to endogeneity effects. Furthermore, we know from Table 1 that winners are around 10 years younger than nominees at first nomination, so would be expected to have shorter lifespans than nominees. Since winners actually live longer than nominees in the data, this has to accounted for by an upwardly biased Winner co-efficient.

To check for this effect, we re-estimate the PH model, taking the year of first nomination as time-zero. Results for this test are shown in Table 4. Here, as well as allowing for age at first nomination, we also allow for the year of first nomination to capture any time trend due to life expectancy changes or changes in the average age of nominated scientists. Fixed covariate analysis (I and II) reveals a 28.7% reduction in relative mortality from winning the Prize. As anticipated, this is lower than the equivalent estimate taking time-zero as birth. The calculations, though still subject to bias from reverse causation, suggest that winners live 3.1 years longer than do nominees. Although a higher age at first nomination is associated with fewer extra years, the 9% increase in relative mortality is relatively modest, suggesting that years pre first-nomination are not perfect substitutes with years post first-nomination. Rather, those who are nominated later

in life appear to benefit the most in increased longevity.

Turning now to the time-varying covariate approach (regressions III and IV of Table 4), we estimate a reduction in relative mortality of 20.6% (regression III), which is here significant at the 5% level. As anticipated, this effect is again smaller than the estimate obtained taking time-zero as birth. In the final column IV of Table 4, the Winner co-efficient fractionally drops below significance at the 5% level. The point estimate is effectively the same as that in the previous equation, and the case for including the three extra controls is arguably weak as none approaches conventional significance levels. Calculating the implied longevity differences yields the result that winners live 2.15 years longer than do nominees. The analyses of Redelmeier & Singh (2001a,b) do not perform this test and therefore do not allow fully for possible reverse causality and endogeneity biases. Indeed Sylvestre et al. (2006) recalculate Redelmeier & Singh's (2001a) result on Oscar winners, allowing for such potential biases, and reach the finding that their 3.6 year estimate becomes closer to one year, and not significantly different from zero at the 95% confidence level.

4.2 Winners, Nominees, and Money

Breaking the sample down into winners and nominees allows a test of further hypotheses. First, one possible reason why the `years nominated (#)' covariate may appear insignificant in the pooled analysis is that winners cease being nominated after winning the Prize. Nevertheless, were a prolonged period of nominations an important source of status we would expect this to be evident when analyzing the nominees separately.

Second, it is interesting to examine whether the economic boost from winning a Nobel Prize may be a source of improved longevity (by the nature of their data Redelmeier & Singh were unable to explore the role of money). Although winners do donate a proportion of their prize-money, we can be reasonably sure that winning a Nobel Prize never harms the bank balance. Our approach is to test for a relationship between the real value of the Nobel prize-money and longevity of its recipients. The null hypothesis is that

the marginal wealth effect from differences in the real value of the Prize on longevity is zero. The test is aided by the fact that over the years there have been substantial fluctuations in the real value of the Prize (Figure 2). From the turn of the century until 1920 the Prize lost more than two-thirds of its real value. After something of a recovery in the 1930's it fell to new lows in the 1940's. It was not until the early 1990's that the prize-fund exceeded that of the inaugural Prize in 1901 in real terms. Third, we can examine whether there is any relationship between the fraction of a Nobel Prize won and longevity: do scientists awarded a full Nobel Prize live longer than scientists awarded only one-quarter of the Prize?[10]
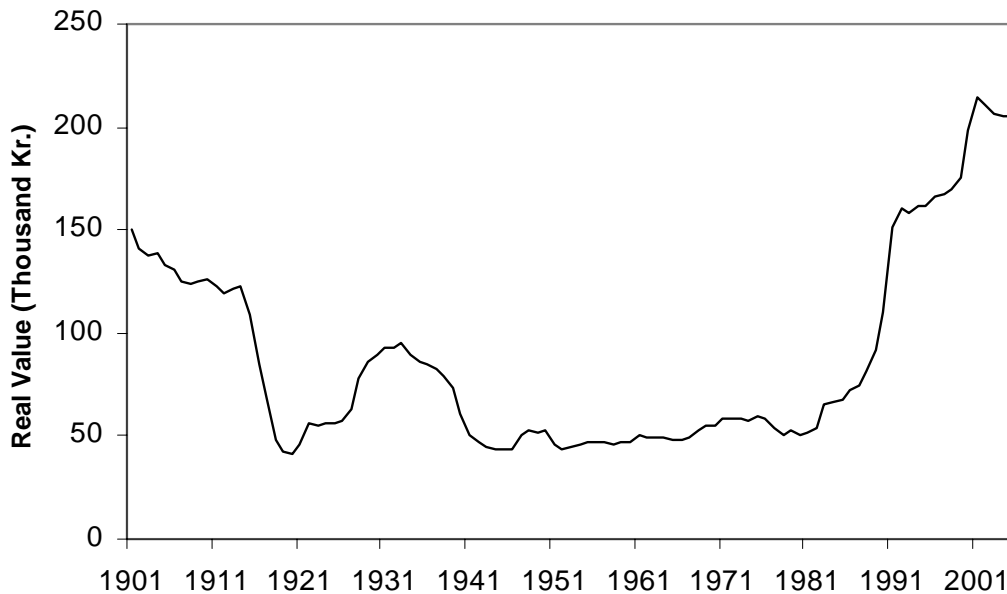
Figure 2: Real Value of the Nobel Prize

The results of this analysis are shown in Table 5. We collected data on the fraction of the Nobel Prize won by each scientist in the winner group, and the real value of the prize-fund (in thousand Swedish Kronor). The covariate `Prize Money' is the interaction between the fraction of the Prize won and the total prize fund (i.e. the amount the

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[10] Winning multiple Nobel Prizes may also be associated with additional status. However, as only two people have ever won more than one Nobel Prize, there are not the data to examine this issue.

scientist actually received). Regression I in Table 5 shows that, amongst the winners, age at first nomination is a statistically significant predictor of longevity, with an implied reduction in relative mortality of around 3%. However, this conclusion must again be treated with caution due to endogeneity bias. Nevertheless, this result, and those in previous sections, suggests to us that the weight of evidence favors the position that those who enter the fray later reap greater rewards in longevity. The fraction of the Prize won is poorly determined but, if anything, enters with an unexpected sign that implies that the higher the fraction, the higher the mortality risk. However, even were this effect statistically significant, it would not constitute decisive evidence against the importance of pure status, as arguably there is no important status difference for outright as opposed to shared winners: scientists who win any fraction of a Nobel Prize are in common parlance said to have won a Nobel Prize.

Importantly, there is no statistically discernible relationship between either the real value of the total prize fund or the real value of the amount received by the individual scientist. The finding is compatible with existing evidence, presented in the Introduction, on the apparently weak role of income and wealth in explaining health outcomes.

Turning to nominees (regressions III and IV of Table 5), we find no effect of the number of years of nominations on longevity. This is consistent with the result of Redelmeier & Singh (2001a), who show that controlling for exposures that cumulate over time, such as total films, and total nominations, has a negligible impact on their estimate of the longevity effect of winning an Oscar. The positive co-efficient implies that, if anything, more years of nominations are associated with shorter lifespans. Perhaps consistent with this, some research in social psychology suggests that the well-being of those who just miss out on a prize is reduced by the tendency to think about what might have been (Medvec, Gilovich & Madey, 1995).

This paper might be viewed as an attempt to move forward from the contradictory Oscar results of Redelmeier & Singh (2001a,b) and Miskie et al (2003). Redelmeier & Singh's (2001b) own account of their mixed findings rests upon the idea that behavioral factors

may be obscuring the underlying effect of social status on longevity: while actors are highly visible and must preserve their image, screenwriters live a life of anonymity, which permits a far wider range of behavior. There seems reason to be cautious of such an explanation, however, for the average estimated lifespan of all screenwriters (both winners and nominees) exceeds the equivalent estimate for all actors (76.9 v. 76.8), and the theory does not explain why Oscar-winning screenwriters supposedly engage in behavior more deleterious to health than do other screenwriters. Nevertheless, were behavioral influences the true culprit, we would argue that our study is less susceptible to such factors. So far as we know, academics are not renowned for the mixture of drugs, drink, sex, violence, monstrous egos, gangsterism, speed, and madness that Davey Smith (2001) argues characterizes the lives of Hollywood directors.

5   Conclusion

This paper finds that Nobel Prize winners go on to have longer lives than scientists who are merely nominated. Although it seems sensible to treat our results cautiously, they are consistent with some form of link between status and longevity. We considered a sample of 524 of the world's top scientists from the first half of the 20th century. These scientists made contributions of such significance to their disciplines that they were nominated at least once for the Nobel Prizes in either Physics or Chemistry. We set ourselves the seemingly demanding task of finding a systematic difference in the longevity between scientists who had actually won the Nobel Prize and those who had only been nominated.[11]

Two forms of empirical test are explored in the paper. First, the simplest evidence comes from within-country matched tests. These are done by pairing winners with nominees drawn solely from the same nation or group. Our matched nominees were born in approximately the same year as the winning scientist and were alive when their matched winner was awarded the Prize. For the USA, for example, Nobel Prize winners live 2.08

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[11] It is perhaps worth recording that when we began this exercise we were doubtful that, once corrections had been made, there would be a discernible effect in the data.

years longer than matched American nominees. For Germany, the figure is an extra 1.30 years of life. For Europeans, it is 0.69 years.

Second, semi-parametric Cox survival estimations are used. After controlling for other factors -- most significantly the possibility of reverse causation from longevity to winning a Nobel Prize -- the paper's best estimate is that winners live approximately two years longer than do nominees. Tests amongst the winners reveal no relationship between the real value of the Prize and longevity. Status, rather than money, appears to be responsible for our effect.

It might be argued that an approximately two-year gain in lifespan from winning a Nobel Prize is a small number of extra years. However, the controls here are extraordinarily successful scientists. By any usual standard, all are high-status individuals. If the idea that social status improves lifespan is truly correct, the size of the effect may in a practical sense be larger in a more normal population of people.

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Table 1: Means and Standard Deviations

Characteristics	Winners (n = 135)	Nominees (n = 389)	Chemistry (n = 296)	Physics (n = 262)
Mean year of birth	1881 (19.4)	1875 (21.3)	1876 (21.1)	1876 (21.1)
Mean lifespan	77.16	75.80	75.62	76.51
Mean age at first nomination	43.36 (9.12)	53.14 (12.4)	51.77 (12.7)	48.52 (12.0)
Mean age of winning	50.76 (10.2)	N/A	52.56 (9.47)	48.74 (10.4)
Mean number of years nominated	5.32 (3.95)	2.93 (3.27)	3.43 (3.68)	4.08 (3.90)
US nationality (%)	22.2	26.0	24.7	24.8
European nationality (%)	74.8	70.0	72.6	70.2

Table 2: Extra Years of Life from Winning (A Matching Test)

	Window (Years)	Matched Winners (#)	Conditional Test Diff. (Years)	Unconditional Test Diff. (Years)
USA*	3	30	2.08	4.64
Germany*	3	38	1.30	2.45
EU*	5	102	0.69	1.36
All	3	135	0.33	1.38
All * (controls are only of winner's nationality)	13	125	0.99	-
Physics*	3	77	0.04	0.83
Chemistry*	5	79	1.35	2.75

* Each winner is matched only with controls from the same nationality, continent or scientific discipline as specified.

Table 3: Probability-of-Death Equations (Time-Zero: Year of Birth)

	Fixed Covariates				Time-Varying Covariates			
	I		II		III		IV	
	% Mortality	95% Conf.	% Mortality	95% Conf	% Mortality	95% Conf	% Mortality	95% Conf
Winner	-32.4***	(-46;-15)	-33.6***	(-48;-16)	-25.3**	(-41;-5.9)	-25.7**	(-42;-5.6)
Age at 1st nomination	-3.4***	(-4.5;-2.3)	-3.5***	(-4.7;-2.4)	-3.2***	(-4.3;-2.1)	-3.4***	(-4.6;-2.2)
Birth year	-1.6***	(-2.3;1.0)	-1.7***	(-2.4;-1.0)	-1.6***	(-2.2;-0.9)	-1.6***	(-2.3;-0.9)
Chemistry	25.2**	(3.7;51)	25.8**	(4.1;52)	23.9**	(2.6;49)	24.5**	(3.1;50)
Years nominated (#)			-0.8	(-3.6;2.0)			-1.3	(-4.0;1.5)
Exposed to radiation			275.9**	(7.4;1,216)			236.1*	(-3.5;1,071)
Committed suicide			1,351***	(214;6,606)			1,235***	(190;6,041)
Country Dummies								
USA	-28.6***	(-43;-10)	-28.6***	(-43;-10)	-27.8***	(-43;-9.0)	-27.9***	(-43;-9.0)
France	-38.4***	(-54;-18)	-38.2***	(-54;-17)	-36.8***	(-53;-15)	-36.6***	(-53;-15)

Note: *** denotes significance at 1%, ** at 5%, and * at 10%.

Table 4: Probability-of-Death Equations (Time-Zero: Year of First Nomination)

	Fixed Covariates				Time-Varying Covariates			
	I		II		III		IV	
	% Mortality	95% Conf.	% Mortality	95% Conf	% Mortality	95% Conf	% Mortality	95% Conf
Winner	-28.7***	(-43;-11)	-28.9***	(-44;-9.9)	-20.6**	(-36;-0.2)	-20.4*	(-37;0.9)
Age at 1st nomination	9.3***	(8.2;10)	9.3***	(8.1;10)	9.5***	(8.4;11)	9.4***	(8.3;11)
Year of 1st nomination	-1.6***	(-2.2;-0.9)	-1.6***	(-2.3;-0.9)	-1.5***	(-2.2;-0.9)	-1.6***	(-2.3;-0.9)
Chemistry	29.1***	(6.7;56)	29.9***	(7.4;57)	27.8**	(5.7;55)	28.9***	(6.4;56)
Years nominated (#)			-1.0	(-3.7;1.8)			-1.5	(-4.2;1.3)
Exposed to radiation			193.0*	(-17;930)			166.2	(-24;835)
Committed suicide			407.0	(-35;3,850)			369.7	(-40;3,555)
Country Dummies								
USA	-28.6***	(-43;-10)	-28.8***	(-44;-10)	-27.8***	(-43;-8.8)	-28.1***	(-43;-9.2)
France	-38.1***	(-54;-17)	-37.9***	(-54;-17)	-36.5***	(-53;-15)	-36.4***	(-53;-14)
Belgium	186.8*	(-0.3;725)	187.6**	(0.0;727)	196.3**	(3.1;752)	196.5**	(3.1;753)

Note: *** denotes significance at 1%, ** at 5%, and * at 10%.

Table 5: Probability-of-Death Equations by Winner/Nominee

	Winners Only				Nominees Only			
	I		II		III		IV	
	% Mortality	95% Conf.	% Mortality	95% Conf	% Mortality	95% Conf	% Mortality	95% Conf
Years Nominated (#)	-2.7	(-7.8;2.8)	-3.4	(-8.6;2.1)	0.7	(-2.7;4.4)	0.8	(-2.7;4.4)
Age at 1st nomination	-3.3**	(-5.9;-0.6)	-4.3***	(-7.1;-1.4)	-3.2***	(-4.5;-1.9)	-3.2***	(-4.5;-1.9)
Birth Year	-1.9*	(-3.7;-0.1)	-2.4**	(-4.3;-0.4)	-1.4***	(-2.2;-0.6)	-1.4***	(-2.2;-0.6)
Fraction of Prize Won	35.3	(-69;482)	35.5	(-69;485)				
Total Prize Value	0.8	(-1.2;2.8)	0.7	(-1.3;2.7)				
Prize Money	-0.4	(-2.5;1.6)	-0.5	(-2.6;1.6)				
Chemistry			35.5	(-8.1;100)	24.6**	(0.2;55)	24.0*	(-0.3;54)
Exposed to radiation			188.9*	(-17;905)				
Committed suicide			773.0**	(14;6,595)			24,308***	(2,089;272,025)
Country Dummies								
USA					-34.4***	(-50;-14)	-35.2***	(-50;-15)
France					-42.8***	(-58;-21)	-43.0***	(-58;-22)
Italy	22,640***	(1,236;388,140)	23,197***	(1,256;400,019)				

Note: *** denotes significance at 1%, ** at 5%, and * at 10%.