Mortality and Immortality: The Nobel Prize as an Experiment into the Effect of Status upon Longevity

Matthew D. Rablen
matthew.rablen@hmrc.gsi.gov.uk

Andrew J. Oswald
andrew.oswald@warwick.ac.uk

University of Warwick and Cornell University

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Abstract

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 extra status could somehow be dropped upon a sub-sample of individuals while those in a control group of comparable individuals received none. This paper attempts to formulate a test in that spirit. It collects 19th-century birth data on science Nobel Prize winners. Correcting for potential biases, we estimate that winning the Prize, compared to merely being nominated, is associated with between 1 and 2 years of extra longevity.

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1. <u>Introduction</u>

High socio-economic status (SES) is associated with good health and long life. A famous demonstration comes from the Whitehall studies of British civil servants. The first such study (Reid et al., 1974) covered a large sample of male civil servants. Analysis of mortality rates for coronary heart disease (CHD) at follow-up times of 7.5 years (Marmot, Rose and Hamilton, 1978), 10 years (Marmot, Shipley and Rose, 1984), and beyond, found occupational grade to be an important explanatory factor. Mortality risk decreased with each extra increment in job seniority. Other research went on to confirm the independent association of grade, after controlling for known risk factors, with common diseases (Marmot, Shipley and Rose, 1984) and in women as well as men (Marmot et al., 1991; Ferrie et al., 2002). This statistical association has now been found in many populations (Adams et al., 2003; Adler and Ostrove, 1999; Adler et al., 1993).

Nevertheless, important questions remain about cause-and-effect: see Pearl (2000) for one of the clearest methodological discussions. At least three pathways are possible: (i) a direct causal effect from status to health; (ii) a reverse causal path from health to status; (iii) unmeasured common causes, which produce a kind of unobserved heterogeneity. A fourth possibility (sample selection) is that statistical limitations in the data incorrectly suggest an underlying causal mechanism where there is none.

Accounts of the beneficial effects of perceived social status, and its psychobiological consequences, have been proposed by Marmot (2004) and Wilkinson (2000). It is argued that lower levels of SES raise levels of psychological stress, and that this happens 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). Empirically, lack of control in the workplace is associated with stress and depressive symptoms (Steptoe et al., 2003a; Stansfeld et al., 1999; Karasek and 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 and Hall, 1988). Wilkinson (2000) proposes that perceived low social standing is linked with feelings of shame, humiliation, disrespect, and social anxiety. It is argued that strain can lead to high levels of stress hormones, and that this in turn damages immunological processes (Steptoe and Marmot, 2005). The result is a potential pathway from SES to longevity.

Studies on rhesus macaques, in which rank can be manipulated, seem to indicate that causation runs from hierarchical position to the presence of stress hormones (Shively, 2000). Using data on troops of wild baboons, Sapolsky (1993) shows that levels of biological stress markers follow a hierarchical pattern. However, studies of cortisol in human beings yield mixed results (Steptoe and Marmot, 2005; Kunz-Ebrecht et al., 2004, Steptoe et al., 2003b). When social and economic status go together, as often in human life, the pursuit of social status is reinforced by the greater entitlement to economic resources. Theoretical and experimental research into the consequences of status-seeking includes Ball et al. (2001), Brown et al. (2008), Clark and Oswald (1998), Frank (1985), Oswald and Powdthavee (2007), and Rablen (2008).

Many economists -- for example Cutler et al. (2006) -- have pointed out that causation may run from health to SES (so-called selection). There may also exist common factors, such as genetic heterogeneity, that influence SES and susceptibility to disease. Investigators have questioned the psychological notion of perceived social status, and wondered whether it is unworkable for policy or testing (e.g. Deaton, 2003). Ill health leads to loss of income, so health can have a causal link to SES. Someone who expects a long life may accumulate assets to finance their retirement, and healthy workers may be paid more because of their higher productivity (Luft, 1975). While selection is widely thought nugatory by medical scientists, economists give it greater credence. Smith (1998, 1999, 2005) argues persuasively that there are pathways from health to SES. Meer, Miller and Rosen (2003) reject causation from wealth to health in US data. Holding constant initial health, and using the British Household Panel Survey, Gardner and Oswald (2004) find no significant effect from income on to later mortality risk. Gravelle and Sutton (2006) and Miller and Paxson (2006) do not uncover persuasive evidence of health effects from relative income. However, innovative work by Frijters, Haisken-DeNew and Shields (2005a,b) does suggest a role for income.[1]

A causal connection can be established from education to health (see e.g. Lleras-Muney, 2005; Smith, 2005). Adams et al. (2003) tests for the absence of direct causality from SES to health, using both economic and education measures of SES. The authors draw upon data from the Asset and Health Dynamics (AHEAD) Panel, on Americans

aged 70 and older, and after conditioning upon initial health conditions cannot reject the hypothesis of no causal link from SES to overall mortality. 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).

Tests for the effects of social status have taken a different approach. Redelmeier and 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 – particularly because as many Americans watch the Oscar ceremony as vote in presidential elections (Davey Smith, 2001). The authors conclude that winners have a life expectancy 3.6 years longer than the control group of nominees. Despite this remarkable finding, a follow-up study (Redelmeier and Singh, 2001b) on Oscar winning and Oscar nominated screenwriters concluded that the unsuccessful Oscar nominees live on average longer than do winners. Thus the two papers seem to contradict each other.[2]

2. The Empirical Approach

We try to contribute to this debate. We use data on Nobel Prize winners and nominees in Physics and Chemistry between 1901 and 1950. In addition to social distinction, the prize (which can be shared by up to three people) carries an amount of $1 - $1.5 million. It is often thought (see e.g. Frey, 2005; Layard, 1980) that the intrinsic value of a Nobel Prize outweighs its monetary rewards. Accordingly, we interpret winning the Prize as primarily a one-time innovation to social status.[3] The idea that the demand for awards relies partly on individuals' desire for distinction is a plank of a nascent economic literature on awards (Frey, 2005).

This study views 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 comparing winners to a control group, namely, scientists nominated for the Prize who

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

[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 the Redelmeier-Singh study of Academy Award winners 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.

were unsuccessful.

Our raw data reveal that winners live just under 1.4 years longer than nominees. However, this does not establish a causal link from winning 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 at least two ways. First, the longer a scientist lives, the greater the chance of stumbling upon a major discovery. Weinberg and Galenson (2005) suggest that this is especially the case for scientists who work inductively. Second, the Prize is not given posthumously. Low survival rates increase the likelihood of dying before receiving the Prize, even when it would have been merited.

Two different approaches are implemented here:

(i)   a matching test

(ii)   a time-varying covariates regression test.

Initially we provide a non-parametric test of the null hypothesis that winners and nominees have identical survival. This proposes a particularly simple way to overcome problems of reverse causality. It compares the longevity of each given winner against the expected longevity of a nominee, *conditional on that nominee being alive when the winner won a Nobel Prize*. In other words, we measure how long both nominees and winners subsequently manage to stay alive. As there might be concerns about some remaining 'immortal time bias', we also present a semi-parametric regression-based approach. It uses 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 help mitigate reverse-causality bias, we use the method of time-dependent covariates. Our final estimate here is that winners live 1.6 years longer than nominees.

Our analysis uncovers no relationship between the real value of the Prize and the longevity of Prize winners.

The sample is drawn from a published census of Nobel Prize nominees[5] 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

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[5] 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.

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). A further three scientists are thought to be alive at time of writing. We were unable to find the year of death for the remaining 28 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.[6] No scientist won either the Prize in Literature or the Prize in Physiology-or-Medicine in conjunction with the Chemistry or Physics Prizes.[7]

Mean lifespan in the data is 76 years, measured as the area under the Kaplan-Meier survival function. This seems remarkable: it is equivalent to male life expectancy for people born in the US in 2006, yet the sample was born on average in 1876.

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,

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[6] We count as a Physics winner any scientist who both won the Prize in Physiology-or-Medicine and who was nominated for the Physics Prize-- and equivalently 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.

[7] Nobody 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.

winners and nominees are similar. Two potentially important differences are that winners are first nominated for a Prize some ten years earlier on average than are nominees, and are also nominated in more than 1.5 times as many distinct years.[8]

Second, we also decompose the data for each individual Prize. The Physics Prize has tended to draw from a smaller pool of scientists, with scientists first being nominated at a younger age and winning earlier also. Nevertheless, at least for the raw data, using a log-rank test we cannot reject the hypothesis that the estimated survival functions for each Prize are the same.

In uncensored populations we calculate 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).[9] However, unsurprisingly, there is too much noise in the data to be able to show statistical significance at conventional levels: we cannot distinguish the survival functions for winners and nominees in Figure 1 using a log-rank test. Second, these differences cannot be given a causal interpretation, for reasons already discussed.

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

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[8] 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).
[9] The apparent discrepancy here is due to the effect of scientists nominated for both Prizes and the effects of rounding.

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. 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).

Our test deliberately matches each winner with one or more nominees born in approximately the same year as the winner. The algorithm searches both forwards and backwards in time around each winner's birth year to find all nominees born within the window period of a winner's year of birth. 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 winners in our sample.

To estimate any 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 $E_j \left[L_j^N - A_i^W \right]$.[10] A problem with this test is that three scientists in our sample are still alive. Therefore we assume 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 other assumptions.

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

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[10] 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.

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, our corrected conditional estimates should lie around zero, with some positive and others negative. Interestingly, contrary to this, in Table 2 we see positive differences in all the subsamples considered and for the sample as a whole. Winners go on to outlive nominees.

We obtain estimates exceeding one and two years respectively for Germany and the USA. 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 (results at other window sizes are comparable). This procedure increases our longevity estimates for the full sample by around two-thirds of a year.

Comparing the third and fourth columns in Table 2 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 live to 110 it falls to 0.19, but nevertheless remains positive. 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 to 110, we still estimate a 1.87 year life gain for the USA.

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. This issue is addressed later.

We now estimate a hazard model to the data. The advantage of this approach over that of the previous section is that it produces 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[11]. 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, the additional Likelihood between the MPH -- our MPH models take the moment of mixing to be the moment of first nomination -- 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 unscaled Schoenfeld residuals, or from tests on the individual covariates. Analysis of the

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[11] Another reason to estimate a hazard model, with time-varying influences, is that it might be argued that even our matching test might fail to purge all immortal-time bias if one of the reasons the matched nominee never subsequently won the prize is because they died too early to win it. A referee has suggested matching instead on age of nomination itself, but that also is open to objections.

Cox-Snell residuals suggests that the PH model provides a reasonable fit to the data.

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.

A second variable we include is a scientist's age at first nomination. Winners are younger when first nominated (Table 1). We allow for differences between Prizes with a dummy variable 'Chemistry'. Dummies are included for country-specific effects.

Finally, we must deal with the issue of reverse causality. This is done 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.

Table 3 treats time-zero as the year of nomination. 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 column I of Table 3, it can be seen that those who win a Nobel Prize exhibit lower mortality than the control group (that is, those nominated for the Nobel Prize but who did not win). The reduction is estimated at 21%. Column II leads to a fractionally larger estimate; it allows for a further range of dummy variables.

The comparable analysis of Oscar winners and nominees by Redelmeier and Singh (2001a) reported a 24-25% decline in relative mortality. It should be emphasized that here we allow for time co-varying variables. 'Winner' enters as a step function, but age is also included as a time-varying influence. For each scientist, the age variable changes by an increment of +1 each year up until the age of death. Age is allowed to

enter flexibly into the model as a linear spline function with knots at the 33rd and 66th percentiles. This gives three variables, as shown in the Table. Two dummy variables in Table 3 deserve mention. 'World-War 1' is for scientists who died in the years 1914-1918. 'World-War 2' is for scientists who died during 1939-1945.

Our key finding here is that winners live 1.62 years longer than nominees. Paralleling Table 1, we also find an increased mortality risk associated with chemists relative to physicists, although we are unsure as to why.[12] Regression II shows that these qualitative conclusions continue to hold in the presence of further controls.

The analyses of Redelmeier and Singh (2001a,b) do not perform this kind of test and therefore do not allow fully for possible reverse causality and endogeneity biases. Indeed, Sylvestre et al. (2006) recalculate Redelmeier and 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.

Breaking the sample down into winners and nominees allows a test of further hypotheses. 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 and 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

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[12] The 'exposed to radiation' dummy is for three scientists (Henri Becquerel, Frederic Joliot and 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.

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?[13]

The results of this analysis are shown in columns III and IV of Table 3. 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 scientist actually received). Regression III in Table 3 shows that, amongst the winners, age is a statistically significant predictor of longevity. However, this conclusion must again be treated with caution due to endogeneity bias. Nevertheless, this result suggests to us that the weight of evidence favors the position that those who enter the fray later reap greater rewards in longevity.

Importantly, there is no statistically discernible relationship between mortality and either the real value of the total prize fund, the prize money received by the individual scientist, or the fraction of the Prize won. The finding is compatible with existing evidence, presented in the Introduction, on the apparently weak role of income and wealth in explaining health outcomes. If anything, although poorly determined, the real value of the prize money received by the individual scientist enters with an unexpected sign that implies that the higher the amount received, the higher the mortality risk.

Rablen and Oswald (2007) reports some additional tests performed solely on the nominees. A 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. However, we find no statistically discernible relationship among nominees between mortality and years of nomination. 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

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[13] Winning multiple Nobel Prizes may bring additional status. As only two people have done so, there are not the data to examine this.

tendency to think about what might have been (Medvec, Gilovich and Madey, 1995).

3. Conclusion

This study finds that Nobel Prize winners go on to have longer lives than scientists who are merely nominated. Although it is important to treat these results cautiously, they appear to be consistent with a causal link between status and later lifespan.[14]

Two complementary kinds of test are explored in the paper. First, simple evidence comes from within-country matched samples. We pair winners with nominees drawn from the same nation or group. By design, our matched nominees are born in approximately the same year as the winning scientist and, crucially, are alive when their matched winner is awarded the Prize. The results of this exercise seem of interest. For the USA, Nobel Prize winners go on to live 2.08 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, we use semi-parametric Cox survival estimation. After controlling for other factors -- most significantly the possibility of reverse causation from longevity to winning a Nobel Prize -- our paper's estimate is that winners live approximately 1.6 years longer than nominees. Tests amongst the winners reveal no relationship between the value of the Prize and longevity. The money *per se* is apparently not creating an effect.

This paper could be viewed as an attempt to move forward from the contradictory results of Redelmeier and Singh (2001a,b) and Miskie et al. (2003). Redelmeier and Singh's (2001b) own account of their mixed findings rests upon the idea that behavioral factors may obscure 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, because 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 engage in behavior more deleterious to health than do other screenwriters. Nevertheless, were behavioral

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[14] It is may be worth recording that when we began this exercise we were sceptical and thought that once proper corrections were done there might well be no discernible longevity effect left. We remain concerned about the chance of false positives (see the useful discussion in Sterne and Davey Smith, 2001, and Ioannidis, 2005) in statistical research of this kind. We are also conscious that our test depends on the assumption that winners and nominees are, ex ante, intrinsically as healthy as one another.

factors the true culprit, we would argue that our study may be 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.

References

Adams P, Hurd MD, McFadden D, Merrill A, Ribeiro T. Healthy, wealthy, and wise? Tests for direct causal paths between health and socioeconomic status. Journal of Econometrics 2003; 112; 3-56.

Adda J, Chandola T, Marmot MG. Socio-economic status and health: Causality and pathways. Journal of Econometrics 2003; 112; 57-63.

Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, Syme SL. Socioeconomic status and health: The challenge of the gradient. American Psychologist 1993; 49; 15-24.

Adler NE, Ostrove JM. Socioeconomic status and health: What we know and what we don't. Annals of the New York Academy of Sciences 1999; 896; 3-15.

Ball S, Eckel C, Grossman PJ, Zame W. Status in markets. Quarterly Journal of Economics 2001; 116(1); 161-188.

Brown GDA, Gardner J, Oswald AJ, Qian J. Does wage rank affect workers' well-being? Industrial Relations 2008, forthcoming.

Clark AE, Oswald AJ. Comparison-concave utility and following behaviour in social and economic Settings. Journal of Public Economics 1998; 70;133-150.

Cox DR. Regression models and life tables. Journal of the Royal Statistical Society Series B 1972; 34; 187-220.

Crawford E. The Nobel population 1901-1950: A census of the nominators and nominees for the prizes in physics and chemistry. Universal Academy Press: Tokyo; 2002.

Cutler D, Deaton A, and Lleras-Muney A. The determinants of mortality. Journal of Economic Perspectives 2006; 20(3); 97-120.

Davey Smith G. Death in Hollywood: Any relation between self worth and mortality is uncertain. British Medical Journal 2001; 323; 1441-1442.

Deaton A. Health, inequality, and economic development. Journal of Economic Literature 2003; 41(1); 113-158.

Falk A, Hanson BS, Isacsson SO, Ostergren PO. Job strain and mortality in elderly men: Social network, support, and influence as buffers. American Journal of Public Health 1992; 82(8);1136-1139.

Ferrie JE, Shipley MJ, Davey Smith G, Stansfeld SA, Marmot MG. Change in health inequalities among British civil servants: the Whitehall II study. Journal of Epidemiology and Community Health 2002; 56; 922-926.

Frank RH. The demand for unobservable and other nonpositional goods. American Economic Review 1985; 75; 101-116.

Frey BS. Knight fever: Towards an economics of awards. CESifo working paper 1468; 2005.

Frijters P, Haisken-DeNew JP, Shields MA. Socio-economic status, health shocks, life satisfaction and mortality: Evidence from an increasing mixed proportional hazards model. Discussion paper 496, Centre for economic policy research, Australian National University; 2005a.

Frijters P, Haisken-DeNew JP, Shields MA. The causal effect of income on health: Evidence from German reunification. Journal of Health Economics 2005b; 24; 997-1017.

Gardner J, Oswald AJ. How is mortality affected by money, marriage, and stress? Journal of Health Economics 2004; 23; 1181-1207.

Gravell H, Sutton M. Income, relative income, and self-reported health in Britain 1979-2000. University of York, CHE research paper 10; 2006.

Ioannidis JPA. Why most published findings are false. PLoS Medicine 2005; 2; 696-701.

Johnson JV, Hall EM. Job strain, work place, social support and cardiovascular disease: A cross-sectional study of a random sample of the Swedish working population. American Journal of Public Health 1988; 78(10); 1336-1342.

Karasek R, Theorell T. Healthy work: Stress, productivity and the reconstruction of working life. Basic Books: New York; 1990.

Kunz-Ebrecht SR, Kirschbaum C, Steptoe A. Work stress, socioeconomic status and neuroendocrine activation over the working day, Social Science and Medicine 2004; 58; 1523-1530.

Layard R. Human satisfactions and public policy. Economic Journal 1980; 90; 737-750.

Lleras-Muney A. The relationship between education and adult mortality in the United States. Review of Economic Studies 2005; 72(1); 189-221.

Luft HS. The impact of poor health on earnings. Review of Economics and Statistics 1975; 57(1); 43-57.

Marmot, MG. The status syndrome: How social standing affects our health and longevity. Owl Books: New York; 2004.

Marmot, MG, Rose G, Hamilton PJS. Employment grade and coronary heart disease in British civil servants. Journal of Epidemiology and Community Health 1978; 32(4); 244-249.

Marmot MG, Shipley MJ, Rose G. Inequalities in death -- Specific explanations of a general pattern? Lancet 1984; 323; 1003-1006.

Marmot MG, Davey Smith G, Stansfeld S, Patel C, North F, Head J, White I, Brunner E, and Feeney A. Health inequalities among British civil servants: the Whitehall II study. Lancet 1991; 337; 1387-1393.

Medvec VH, Gilovich T, Madey SF. When less is more - Counterfactual thinking and satisfaction among Olympic medallists. Journal of Personality and Social Psychology 1995; 69(4); 603-610.

Meer J, Miller D, Rosen H. Exploring the health-wealth nexus. Journal of Health Economics 2003; 22; 713-730.

Miskie B, Near S, Hegele R. Survival in Academy Award-winning actors and actresses. Annals of Internal Medicine 2003; 138(1); 77-78.

Miller DL, Paxson C. Relative income, race, and mortality. Journal of Health Economics 2006; 25(5); 979-1003.

Oswald AJ, Powdthavee N. Obesity, unhappiness, and the challenge of affluence: Theory and evidence. Economic Journal 2007; 117(521); F441-454.

Pearl J. Causality: models, reasoning and inference. Cambridge University Press: Cambridge; 2000.

Poggendorff JC. Biographisch-literarisches handwörterbuch der exakten naturwissenschaften. 1863 Johann Ambrosius Barth, Leipzig.

Putnam RD. Bowling alone: The collapse and revival of American community. Simon and Schuster: New York; 2000.

Rablen MD. Relativity, rank and the utility of income. Economic Journal 2008; 118; 801-821.

Rablen MD, Oswald AJ. Mortality and immortality. 2007 IZA Discussion Paper 2560.

Redelmeier DA, Singh SM. Survival in Academy Award-winning actors and actresses. Annals of Internal Medicine 2001a; 134; 955-962.

Redelmeier DA, Singh SM. Longevity of screenwriters who win an Academy Award: Longitudinal study. British Medical Journal 2001b; 323; 1491-1496.

Reid DD, Brett GZ, Hamilton PJS, Jarrett RJ, Keen H, Rose G. Cardio-respiratory disease and diabetes among middle-aged male civil servants: A study of screening and intervention. Lancet 1974; 303; 469-473.

Sapolsky R. Endocrinology alfresco: Psychoendocrine studies of wild baboons. Recent Progress in Hormone Research 1993; 48; 437-468.

Shively CA. Social status, stress and health in female monkeys. In: Tarlov AR, St Peter RF (Eds.), The society and population health reader - A state and community perspective. New Press: New York; 2000.

Smith JP. Socioeconomic status and health. American Economic Review 1998; 88(2); 192-196.

Smith JP. Healthy bodies and thick wallets: The dual relation between health and economic status. Journal of Economic Perspectives 1999; 13(2); 145-166.

Smith JP. Unraveling the SES-health connection. Population and Development Review 2005; 30; 108-132.

Snyder SE, Evans WN. The effect of income on mortality: Evidence from the social security notch. Review of Economics and Statistics 2006; 88(3); 482-495.

Stansfeld SA, Head J, Fuhrer R, Shipley MJ, Marmot MG. Work characteristics predict psychiatric disorder: Prospective results from the Whitehall II study. Occupational and Environmental Medicine 1999; 15; 302-307.

Steptoe A, Kunz-Ebrecht S, Owen N, Feldman PJ, Rumley A, Lowe GDO, Marmot MG. Influence of socioeconomic status and job control on plasma fibrogen responses to acute mental stress. Psychosomatic Medicine 20003a; 65(1); 137-144.

Steptoe A, Kunz-Ebrecht S, Owen N, Feldman PJ, Willemsen G, Kirschbaum C, Marmot MG. Socioeconomic status and stress-related biological responses over the working day. Psychosomatic Medicine 2003b; 65(3); 461-470.

Steptoe A, Marmot MG. Socioeconomic status and coronary heart disease: A psychobiological perspective. In: White LJ (Ed.), Aging, health and public policy. Population Council: New York; 2005.

Sterne JA, Davey Smith G. Sifting the evidence—What's wrong with significance tests. British Medical Journal 2001; 322; 226–231.

Sylvestre MP, Huszti E, Hanley JA. Do oscar winners live longer than less successful peers? A re-analysis of the evidence. Annals of Internal Medicine 2006; 145(5); 361-363.

Weinberg BA, Galenson DW. Creative careers: The life cycles of Nobel laureates in economics. 2005 NBER Working Paper 11799, Cambridge Mass.

Wilkinson R. Mind the gap: Hierarchies, health and human evolution. Weidenfeld and Nicolson: London; 2000.

Figure 1: Survival Function for Winners (solid line) and Nominees (dashed line)
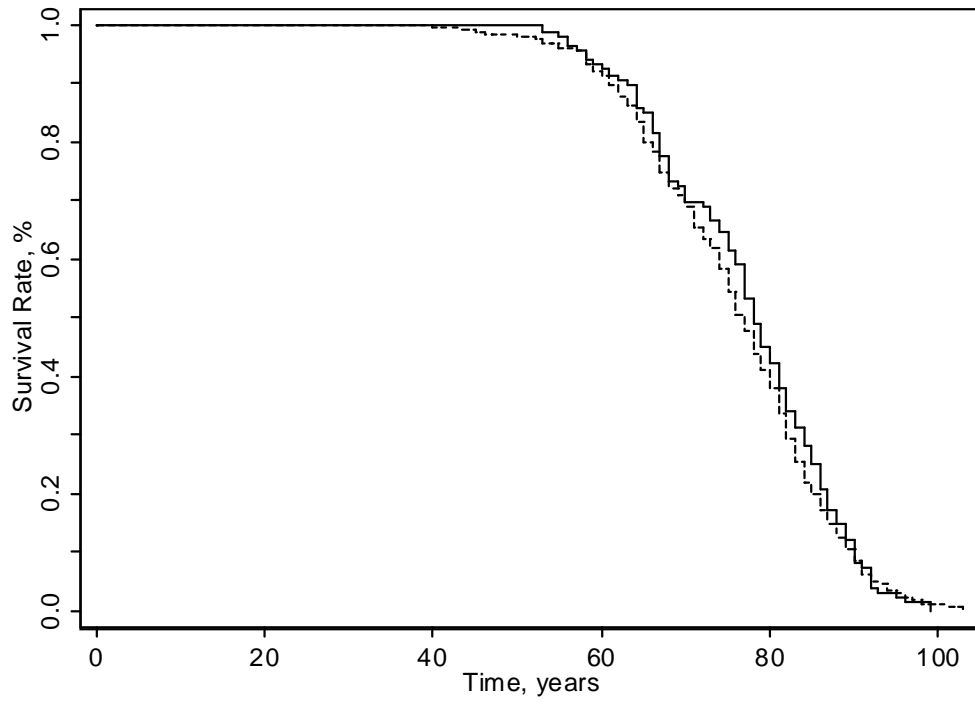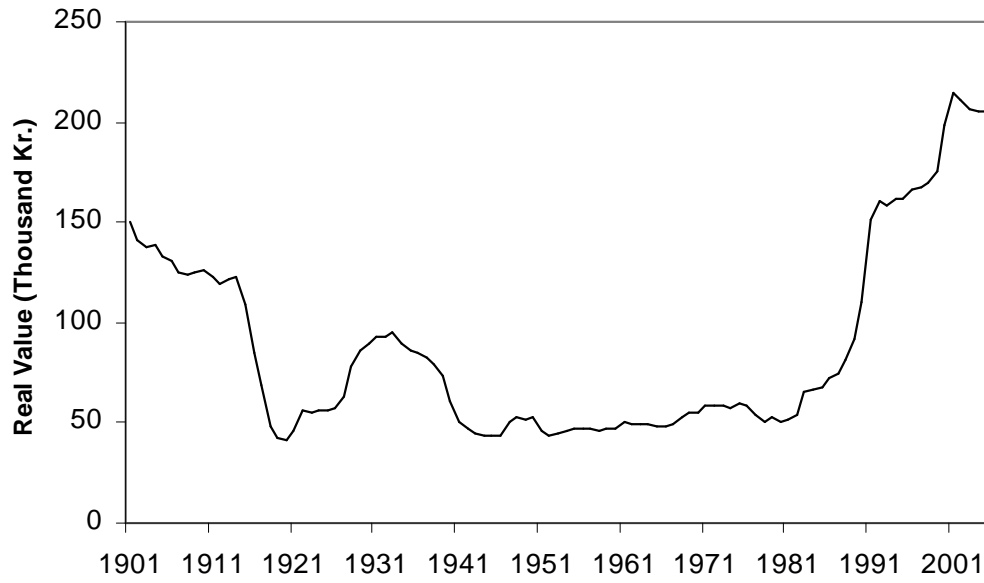
Figure 2: Real Value of the Nobel Prize

Note: In the regression equations of Table 3, this data series is used only up to the early 1950s. Recent winners of the Nobel Prize cannot be included in the sample.

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 First Nomination) with Time-Varying Covariates

| | Winners and Nominees | | | | Winners only | | | |
| | I | | II | | III | | IV | |
	% Mortality	95% Conf.	% Mortality	95% Conf	% Mortality	95% Conf	% Mortality	95% Conf
Winner	-21.0**	(-37;-0.7)	-22.3**	(-39;-1.6)				
Age (lower third)	13.9***	(5.8;23)	13.6***	(5.4;22)	75.5	(-24;307)	68.9	(-27;292)
Age (middle third)	6.1***	(2.7;9.6)	6.1***	(2.7;9.7)	4.5	(-3.6;13.3)	3.5	(-4.8;13)
Age (upper third)	8.0***	(6.1;9.9)	7.8***	(5.9;9.7)	9.0***	(5.0;13.2)	7.8***	(3.5;12)
Year born	-1.5***	(-2.2;-0.9)	-1.3***	(-2.1;-0.6)	-1.4	(-3.2;0.3)	-2.0*	(-4.0;0.2)
Chemistry	27.9**	(5.8;55)	28.3**	(6.0;55)	32.5	(-10;96)	35.3	(-10;104)
Fraction of prize won					14.8	(-76;443)	11.9	(-78;458)
Total real prize value					1.0	(-1.1;3.1)	0.4	(-1.7;2.7)
Prize money					-0.2	(-2.3;2.0)	-0.1	(-2.4;2.2)
Years nominated (#)			-1.1	(-3.8;1.8)			-3.3	(-8.7;2.5)
Exposed to radiation			217*	(-10;1,017)			226*	(-20;1,229)
Committed suicide			267	(-54;2,821)			727*	(-11;7,620)
World-War 1			66.0	(-11;211)			85.9	(-59;741)
World-War 2			31.8*	(-1.7;77)			40.1	(-32;188)
Country Dummies								
USA	-27.7***	(-43;-8.8)	-26.4**	(-42;-6.8)				
France	-36.5***	(-53;-15)	-34.7***	(-51;-12)				
Belgium	194**	(2.3;746)	183*	(-2.0;716)				

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