Superstar Extinction

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Abstract

We estimate the magnitude of spillovers generated by 137 academic "superstars" in the life sciences onto their coauthors' research productivity. These researchers died while still being actively engaged in science, thus providing an exogenous source of variation in the structure of their collaborators' coauthorship networks. Following the death of a superstar, we find that coauthors suffer a lasting 8 to 18% decline in their quality-adjusted publication output. These findings are surprisingly homogenous across a wide range of coauthor and coauthor/superstar dyad characteristics. Together, they suggest that part of the scientific field embodied in the "invisible college" of coauthors working in that area dies along with the star — that the extinction of a star represents a genuine and irreplaceable loss of human capital.

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1 Introduction

Human capital, through its influence on the creation and adoption of improved technologies, has long been recognized as an important contributor to aggregate income (Nelson and Phelps, 1965; Schultz, 1967). Modern economic growth models have built upon this idea to argue that human capital externalities — knowledge spillovers across individuals — are the principal drivers of economic progress (Lucas, 1988; Romer, 1990). The skills and wisdom of some individuals, through interactions with others, increases these attributes in their peers. The increased stock of human capital in the economy generates more ideas and faster growth.

The empirical literature on human capital externalities is relatively small and principally focused on the estimation of peer effects among students (e.g., Rauch, 1993; Acemoglu and Angrist, 2001; Hoxby, 2000; Sacerdote, 2001). Yet, the endogenous growth literature typically envisions spillovers that occur in the workplace, where firms acquire ideas from their neighbors and employees learn from their co-workers. Indeed, the importance of learning through social interactions on the job can be traced back to turn of the century writings by Alfred Marshall (1890). Only a handful of empirical papers have examined peer effects in the employment setting.[1]

The paucity of literature is largely due to the difficulties involved in collecting the data necessary to measure these effects. In most employment settings, it is often impossible to identify peer groups at the individual level, and even harder to distinguish output produced jointly with the peer group from output produced independently of it. As a result, the existing research often relies on aggregate proxies — such as co-location — to estimate human capital externalities. Moretti (2004) exemplifies this approach. Using the share of college graduates in the workforce at the city level, he provides evidence for modest human capital externalities in the US manufacturing sector and also finds that spillovers are larger among

[1] A related literature examines the influence of peer effects on shirking behavior in the workplace (Ichino and Maggi, 2000; Costa and Khan, 2003; Bandiera et al., 2005; Mas and Moretti, 2006). Since shirking is easily observed by co-workers and "contagion" does not generally involve the transmission of knowledge or techniques, this type of spillover is conceptually distinct from human capital externalities.

firms that are economically similar. Since manufacturing is less human capital intensive than other sectors of the economy, the finding that spillovers are modest is perhaps not surprising.

In this paper, we attempt to relax the data constraint by focusing on a setting where human capital externalities are likely to be quite important — the academic life sciences.[2] This choice enables us to estimate the productivity benefits of knowledge spillovers at the individual level. It also allows us to trace these spillovers back to social interactions, independently of location decisions. The importance of social interactions is supported by recent evidence from Kim et al. (2006) who show that spillovers, at least among academic economists, have become less tied to physical location over time, perhaps due to improvements in information technologies. Moreover, our focus on connections between individuals of differing skill levels speaks directly to the types of human capital externalities envisaged by the early champions of modern economic growth theory, as exemplified by the following quote from Lucas (1988):

> *"Most of what we know we learn from other people. We pay tuition to a few of these teachers, either directly or indirectly by accepting lower pay so we can hang around them, but most of it we get for free, and often in ways that are mutual — without a distinction between student and teacher. Certainly in our own profession, the benefits from colleagues from whom we hope to learn are tangible enough to lead us to spend a considerable fraction of our time fighting over who they shall be, and another fraction traveling to talk with those we wish we could have as colleagues but cannot. We know that this kind of external effect is common to all the arts and sciences — the 'creative professions.' All intellectual history is the history of such effects."*

More specifically, we analyze the research productivity of coauthors for 137 eminent life scientists who die prematurely, while still being actively engaged in science. These scientists are drawn from a larger sample of superstar academics selected on the basis seven different criteria, including NIH funding, citations, patents, membership in the National Academy of Sciences, or appointment as Howard Hughes Medical Investigator. Using a matched faculty-university panel dataset, we measure how colleagues' scientific output, as measured by quality-adjusted publications, changes when their superstar collaborator passes away. To

[2]Weinberg (2007) pursues a similar strategy, focusing on physicists, and analyzing how geographic proximity to earlier Nobelists correlates with the start of the research that would eventually result in a Nobel Prize.

be clear, our focus is on faculty peers, not trainees, and thus our results should be viewed as capturing inter-laboratory spillovers rather than mentorship effects.

Economists have long recognized the difficulties involved in recovering causal effects in observational studies of peer influence (e.g., Manski 1993). Certainly, the formation of collaborative relationships should be understood as outcomes of a purposeful matching process (Fafchamps et al., 2006; Mairesse and Turner, 2005). Therefore, our approach is to condition inclusion in the sample on a coauthorship tie, and to focus instead on the deletion of this tie induced by the death of a prominent collaborator. Because we can identify 73 coauthors per superstar on average, we can exploit rich variation in the depth and length of interaction to examine potential heterogeneity in the treatment effect. Other economists have used the death of prominent individuals as a source of exogenous variation in leadership, whether in the context of business firms (Johnson et al., 1985; Bennedsen et al., 2007), or even entire countries (Jones and Olken, 2005). To our knowledge, however, we are the first to use this strategy to estimate the magnitude of knowledge spillovers.

Our results reveal an 8 to 18% decrease in the quality-adjusted publication output of coauthors in response to the sudden and unexpected loss of a superstar. When the superstar death is anticipated and thus less plausibly exogenous, our results are weaker but generally consistent with the effects due to unanticipated losses. More interestingly, the impact of star death is quite diffuse — output declines are similar irrespective of geographic distance between collaborators, the time since last collaboration, or whether the coauthor was formerly a trainee of the star. We also verify that the effects we measure are increasing in the superstar's intellectual accomplishments (measured by citation count at the time of death) and not their financial ones (measured by NIH grantsmanship at the time of death). This suggests that spillovers are indeed about knowledge and that the loss of a coauthor of lesser rank would not generate the long lasting effects of the type we find here. The only researchers that appear insulated from these negative effects of star death are those who are themselves among the highest achievers in their field, as measured by membership in the National Academy of Sciences, and those who are significantly more senior than the star. Together, these results paint a picture of an invisible college of coauthors bound together

by interests in a fairly narrow scientific area, which suffers a permanent and reverberating intellectual loss when it loses its star.

The rest of the paper proceeds as follows. In the next section, we provide a brief theoretical background for the study. Section 3 presents our sample of superstars and describes the matching process necessary for the identification of colleagues. Section 4 presents descriptive statistics, and reports econometric results. Section 5 concludes.

2 Theoretical Background

Knowledge can flow across time and space through three distinct mechanisms. The endogenous growth literature emphasizes the "standing on shoulders of giants" effect, whereby the current state of knowledge forms the basis for new knowledge-based innovation. This implies a role for education and training, which enable would-be innovators to be active at the frontier of knowledge (Jones, 2005). A second mechanism for knowledge diffusion is *involuntary* spillovers, whereby innovators obtain knowledge generated by others through observation and imitation. Finally, knowledge can be shared directly among members of innovating teams. Here we envision an "invisible college", where strong intellectual ties can create an informational network in which the main ideas of a new field are created (Crane, 1972). In this paper, we are interested in evaluating the extent to which such collaborations can foster the production of new knowledge.

Looking at the effect of collaborations is important because very few innovations result from the efforts of lone researchers engaged in otherworldly contemplation. The creative process is characterized by the search for useful recombinations of existing ideas (Weitzman, 1998), and the mixing of individuals with different backgrounds and education within the same team magnifies the number of combinations that can be evaluated. Moreover, members of these teams do not have to be colocated. In fact, they are increasingly distributed across

different locales, thanks to the use of information and communication technologies (Kim et al., 2006; Rosenblat and Mobius, 2004).[3]

When members of different skill level or experience match, less skilled agents might gain from their exposure to the ideas of the relatively more skilled, thus creating *voluntary* spillovers of knowledge. A skilled agent might match with a less skilled one by pure altruism, because of cultural norms that favor professional mentorship, or because the costs of collaboration with less-skilled agents are lower. Here, we will take the existence of such heterogeneous matches as given, and exploit the exogenous termination of these relations induced by death to study their impact on the output produced by the relatively less skilled. The impact of the skilled on the less skilled is especially interesting because the former might find it difficult to fully appropriate the benefits they confer on the latter. For example, it might be impossible to apportion credit between team members in ways that are verifiable by a court. Moreover, even when individual talent is perfectly observed in the market, asymmetric information over the mentorship abilities of skilled agents might both limit their mobility and ensure that they cannot extract their full marginal product (Lazear, 1986; Acemoglu and Pischke, 1999).

That knowledge spillovers are at least partially external means innovators will have too little incentive to innovate (Murphy et al., 1991). In such a world, the allocation of talent across firms and the technologies and policies that influence the flow of information between agents has important implications for the level and rate of technological innovation within the economy, and eventually for economic growth.

The setting chosen for our empirical work is the academic life sciences. This sector is an important one to study for several reasons. First, technological change has been enormously important in the growth of the health care economy, which accounts for roughly 15% of US GDP. Much biomedical innovation is science-based (Henderson et al., 1999), and interactions between academic researchers and their counterparts in industry appear

[3]In a related vein, Agrawal and Goldfarb (2006) provide persuasive evidence that the diffusion of BITNET, an early precursor to the internet, fostered collaboration between computer scientists in first- and second-tier research universities.

to be an important determinant of research productivity in the pharmaceutical industry (Cockburn and Henderson, 1998; Zucker et al., 1998; Powell et al., 2005). Second, and perhaps most importantly for our work, academic scientists are generally paid through soft money contracts. Salaries depend on the amount of grant revenue raised by faculty, thus providing researchers with high-powered incentives to remain productive even after they secure a tenured position. As such, academic life scientists can be viewed as entrepreneurs producing new knowledge through the management of small idiosyncratic firms, comprised of junior faculty, postdoctoral researchers, graduate students, and other research personnel. Lastly, there are large public subsidies for biomedical research in the United States. With an annual budget of $28 billion in 2004, support for the NIH dwarfs that of other national funding agencies in developed countries (Cech, 2005). Thus, estimating knowledge spillovers in this sector will allow us to better assess the return to these public investments.

Our focus on this setting — one with a very extensive paper trail of research output and collaboration histories — also offers practical benefits that better enable us to examine the magnitude of human capital externalities. By matching data on quality-adjusted publication output with an administrative dataset linking medical school scientists with their employers, we are able to create what is, to our knowledge, the first matched employee-employer dataset with individual-level measures of output.

The rich coauthorship tradition in the life sciences also allows us to identify peer groups more precisely than the standard approach in the literature, which uses colocation as a *de facto* measure of social interaction (Jaffe et al., 1993). While attending to geographic distance, we use collaborations between scientists to define actual interactions, thus drawing boundaries around the ethereal invisible college. Collaborations are important because research areas in the biomedical sciences are highly specialized and the set of individuals writing together on this subject is a better measure of the scientific field than school, department, or even discipline. To fix ideas, spheres of influence, and thus fields of inquiry, are not defined in broad categories such as genetics, but rather around ideas related to the regulation of vascular endothelial growth factor during nerve regeneration, perhaps only in mice and rats. As discussed earlier, we overcome concerns about the endogeneity of these

relations, by focusing on the impacts induced by the exogenous termination of collaborations due to the death of a superstar.

3 Data and Sample Characteristics

This section provides a detailed description of the process through which the matched coauthor/superstar data used in the econometric analysis was assembled. In order, we describe (1) the criteria used to select our sample of superstar life scientists; (2) the universe of potential colleagues for these superstars; and (3) the essence of the matching procedure implemented to identify actual colleagues from coauthorship records. We also present basic demographic characteristics for the superstars, as well as descriptive statistics for the individual coauthors and superstar/coauthor dyads.

3.1 Superstar Sample

Our basic approach is to rely on the death of "superstar" scientists to estimate the magnitude of knowledge spillovers onto colleagues. In particular, we limit our attention to those stars that were still active researchers when they died prematurely at an age less than or equal to 66 years. Our focus on superstars can be justified on both substantive and pragmatic grounds. Significant inequality in scientists' productivity has been widely documented. In a classic paper, Lotka (1926) showed that the most productive 6% of publishing physicists produced 50% of the papers in the journals he examined. An extensive literature in the sociology of science presents further evidence of the skewed distribution of productivity (e.g., Merton, 1973; de Solla Price, 1986). In a related vein, Zucker et al. (1998) established a robust correlation between the location of superstar life scientists and the number of new biotechnology firms spawned in a given locale. Thus, if one wants to find evidence of spillovers at the individual level, it seems logical to start with a sample of superstars, rather than with a random sample of scientists. From a practical standpoint, it is more feasible — though still surprisingly difficult — to trace back the careers of eminent scientists than to perform a similar exercise for less eminent ones. As we will see in Section 4, our motivation for focusing

on stars is also supported by our empirical work, which shows that the impact of star death is increasing in his/her accomplishments. The subset of stars used in our analysis is drawn from a larger pool of 7,276 eminent life scientists who are so classified if they satisfy at least one of the following seven criteria for scientific achievement.

- **Highly Funded Scientists.** Our first source is the Consolidated Grant/Applicant File (CGAF) from the U.S. National Institutes of Health (NIH). This dataset records information about grants awarded to extramural researchers funded by the NIH since 1938. Using the CGAF, and focusing only on research grants, we compute individual cumulative totals for the years 1977 to 2003, deflating the earlier years by the biomedical research producer price index. We also recompute these totals excluding large center grants that usually fund groups of investigators (M01 and P01 grants). Those scientists whose totals lie in the top ventile (i.e., above the 95^{th} percentile) of either of these two distributions constitute our first group of superstars. In this elite group, the least well-funded investigator had garnered $10.5 million in NIH career funding, and the most well-funded $462.6 million.

- **MERIT Awardees of the NIH.** In order to include a more explicitly quality-focused measure of grantsmanship, we also include MERIT awardees from the NIH. Initiated in the mid-1980s, the MERIT (Method to Extend Research in Time) Award program extends funding to experienced investigators with impressive records of scientific achievement in research areas of special importance or promise. Investigators cannot apply for a MERIT award and less than 1% of NIH-funded researchers are selected to receive them.

- **Highly Cited Scientists.** Despite the preeminent role of the NIH in the funding of public biomedical research, this indicator of "superstardom" biases the sample towards older scientists conducting relatively expensive research. We complement this first group with a second composed of highly cited scientists identified by the Institute for Scientific Information. A Highly Cited listing means that an individual was among the

250 most cited researchers for their published articles between 1981 and 1999, within a broad scientific field.[4]

- **Howard Hughes Medical Investigators.** We also drew on the population of current or former Howard Hughes Medical Investigators. Every three years, the Howard Hughes Medical Institute solicits nominations from research institution, with the aim of identifying researchers who have the potential to make significant contributions to science. Once selected, they continue to be based at their institutions, typically leading a research group of 10 to 25 students, postdoctoral associates and technicians. From our point of view, HHMIs are attractive in that they tend to be younger, "up-and-coming" scientists, rather than established investigators.

- **Pew and Searle Scholars.** We also included Pew and Searle scholars for the years 1981 through 2000. Every year, the Pew and Searle charitable trusts provide seed funding to 40 life scientists in the first two years of their careers as independent investigators. The Pew and Searle Scholarships are the most prestigious accolades that young researchers can receive at the start of their careers.

- **Top Patenters.** We add to these groups academic life scientists who belong in the top percentile of the patent distribution among academics — those who were granted 17 patents or more between 1976 and 2004.

- **National Academy of Sciences.** Finally, we add to these groups academic life scientists who were elected to the National Academy of Science between 1975 and 2007.

Tables 1A and 1B present individual-level details on the superstar sample, broken down by sudden and anticipated deaths, respectively. Heart attack is the most frequent cause of sudden death, while the vast majority of anticipated deaths are due to some form of cancer. Since most of the anticipated deaths are due to conditions with short life expectancies and

[4]The relevant scientific fields in the life sciences are microbiology; biochemistry; psychiatry/psychology; neuroscience; molecular biology & genetics; immunology; pharmacology; and clinical medicine.

those with longer ones are not necessarily viewed as terminal until the final stages, anticipated deaths should be thought of as those with no more than several years notice.

Many scientists achieve superstar status according to more than one metric. We trace back these scientists' careers with great care from the time they obtain their first position as independent investigators (typically after a postdoctoral fellowship) until 2006. We do so through *Who's Who* profiles, accolades/obituaries in medical journals, National Academy of Sciences biographical memoirs, and Google searches. As a result, we are able to trace back their path from humble beginnings to superstardom, and to examine whether spillovers vary over the life cycle, even though selection into the sample was based on *cumulative* achievement.

We record employment history, degree held, date of degree, gender, up to three departments, and whether the star held an administrative position, such as dean or hospital CEO. We cross-reference the superstar sample with other measures of scientific eminence. For example, our 137 superstars include one Nobel Prize winner and two Lasker awardees.

Table 2A provides descriptive statistics for the superstar sample. The average star received their degree in 1965, died at 58 years old, held 1.75 positions in their career, and worked with 73 coauthors in their lifetime. The youngest died at 35 and the oldest died at 66. The most gregarious worked with 225 coauthors over the course of their career. On the output side, the stars each received an average of roughly 22 million dollars in NIH grants, published 161 papers, garnered 10,240 citations, and held 2 patents. We also compute the h index due to Hirsch (2005), which is commonly used by bibliometricians: h is the highest integer such that an individual has h publications cited at least h times. In our sample, h is approximately 50. The most well-funded investigator received 203 million dollars in NIH funding and the most prolific wrote 533 papers. The subsample of superstars whose death was anticipated appears slightly more accomplished on average, but not dramatically so.

Table 2B provides additional information about the superstar sample. The sample is approximately 12% female and 84% US-born. 42% of our stars hold an MD degree, 47% a PhD, and the remainder hold dual MD/PhD degrees. Keeping in mind that our metrics of

superstardom are not mutually exclusive, roughly 7.3% are Howard Hughes Medical Investigators, 34.3% are MERIT awardees, and 21.9% are members of the National Academy of Sciences.

3.2 The Universe of Potential Colleagues

Information about superstars' colleagues stems from the Faculty Roster of the Association of American Medical Colleges, whose access we secured for the years 1977 through 2006 under a licensing agreement. The roster is an annual census of all U.S. medical school faculty, where each faculty is linked across yearly cross-sections by a unique identifier.[5] When all cross-sections are pooled, we obtain a matched employee/employer panel dataset. For each of the 222,478 faculty members that appear in the roster, we know the full name, the type of degrees received and the years they were awarded, gender, up to two departments, and medical school affiliation. Besides its comprehensiveness, an attractive feature of this data source is that it shares a common system of individual identifiers with the CGAF dataset.

Because the roster only lists medical school faculty, however, it is not a complete census of the academic life sciences. For instance, it does not list information for faculty at institutions such as MIT, University of California at Berkeley, Rockefeller University, the Salk Institute, or the Bethesda campus of the NIH; and it also ignores faculty members in Arts and Sciences departments — such as biology and chemistry — if they do not hold joint appointments at a local medical school.[6]

For the purposes of this paper, we define colleagues to be those faculty listed on the roster that are coauthors for each superstar. While this is certainly not the only basis

[5]Although AAMC does not collect data from each medical school with a fixed due date. Instead, it collects data on a rolling basis, with each medical school submitting on a time frame that best meets its reporting needs. Nearly all medical schools report once a year, while many medical schools update once semester. Other medical schools update once a month or even more often than that if the medical schools use the Faculty Roster as their primary database for maintaining faculty data.

[6]This limitation is less important than might appear at first glance. First, we have no reason to think that colleagues located in these institutions differ in substantive ways from those based in medical schools. Second, all our analyses focus on *changes* in research productivity over time for a given scientist. Therefore, the limited coverage is an issue solely for the small number of faculty who transition in and out of medical schools from (or to) other types of research employment. For these faculty, we were quite successful in filling out career gaps by combining the roster with the NIH data.

upon which colleagues could be defined, a coauthorship-based definition seems the most sensible.[7] The production of abstract knowledge depends on the extent to which ideas are shared among researchers, and an important mechanism for sharing knowledge is direct collaboration through coauthorship. Moreover, the coauthorship network is our best measure of scientific field in a universe with researchers who are highly specialized. It bears repeating that the typical social scientist's broad conceptualization of field is somewhat misguided here. A physiologist working on protein trafficking between intracellular organelles will not necessarily be influenced by developments in physiology writ large, but certainly follow closely the research of those working in his/her narrow area of inquiry. Indeed, the sphere of influence may even be limited to those engaged in research on protein trafficking for the Moloney murine leukemia virus.

3.3 Coauthor Matching

To identify coauthors, we have developed a custom software program, the Stars/Colleagues Generator, or S/CGen.[8] The source of the publication data is PubMed, an online resource from the National Library of Medicine that provides fast, free, and reliable access to the biomedical research literature. In a first step, the S/CGen downloads from the internet the

[7]Specifically, we could have labeled colleagues any faculty that was co-located with the superstar at any point during his or her career. This does not strike us as a meaningful definition of the term "peer," because medical schools are large research institutions (833 faculty on average in 2003). One could make use of department information to define narrower boundaries for peer groups, but this approach is too difficult to implement in practice. First, department affiliations are not fixed over time for most faculty — this is apparent in our sample of superstars, and many collaborations span departmental boundaries. Second, new departments were created during this period (e.g., Neuroscience, Genetics, or Biomedical Engineering), while others were phased out or dramatically shrunk (e.g., Anatomy). Third, the merging or survival of many departments is often a reflection of internal political struggles, rather than characteristics of the research conducted within them. For example, in some medical schools, orthopedic surgeons are in a separate department while in others, they are part of a large surgery department; In the basic sciences, many faculty would feel equally at home in cell biology, molecular biology, or biochemistry. Finally, three large departments (internal medicine, pediatrics, and surgery) tend to account for a large proportion of medical school employment, but their size masks enormous heterogeneity (e.g., neurosurgeons vs. cardio-thoracic surgeons; endocrinologists vs. infectious diseases specialists).

[8]The complete specifications are described in a technical working paper (Stellman et al., 2006), while the S/CGen software itself can be used by other researchers under a GNU license. Note that the S/CGen takes the faculty roster as an input; we are not authorized to share this data with third-parties. However, it can be licensed (for a fee) from AAMC, provided a local IRB gives its approval and a confidentiality agreement protects the anonymity of individual faculty members.

entire set of English-language articles for a superstar, provided they are not letters to the editor, comments, or other "atypical" articles. From this set of publications, the S/CGen strips out the list of coauthors, eliminates duplicate names, matches each coauthor with the Faculty Roster, and stores the identifier of every coauthor for whom a match is found. In a final step, the software queries PubMed for each validated coauthor, and generates publication counts as well as coauthorship variables for each superstar/colleague dyad, in each year.

S/CGen cannot generate a match for each coauthor. Some coauthors are postdocs, technicians, or graduate students who do not go on to faculty positions within our period of observation; other coauthors have positions in foreign institutions; others still publish under names that differ from the faculty roster listing (for instance by being inconsistent with the use of middle initials, suffixes, or hyphens). We are more worried about generating spurious matches, however.

An important limitation in the matching process is that PubMed does not record authors' full names, nor does it record their institutional affiliation; it only keeps track of authors by using a combination of last name, two initials, and a suffix (where the suffix and the second initial fields can be empty). There are no unique author identifiers, and no possibility to account for the different name variations that a given author uses throughout his/her career.

This absence of unique author identifiers could create a cascade of errors. For a superstar with a relatively common name (e.g., Thomas W. Smith), a simple query would return many publications that were, in fact, not his. As a consequence, a number of coauthors identified by our software would be spurious. This source of error would be compounded by the fact that when searching for the colleagues' publications, the output of those with relatively more frequent names will be imprecisely measured. The first source of error strikes us as potentially very serious, and to deal with it, we designed custom search queries that return a superstar's list of publications excluding those of any homonymous scientist.[9]

[9]These queries form part of the information that the software takes as an input. They can be up to 244 characters long (see Stellman et al. (2006) for more details).

After neutralizing this first source of error, it is still the case that S/CGen can generate more than one roster match for a given PubMed author name, and the quality of these matches will depend directly on the relative frequency of last names in the population. In order to ensure that publications are assigned to authors as precisely as possible, the analysis that follows is based only on those colleagues with unique PubMed names, *i.e.*, those combinations of last names, two initials, and suffix which correspond to one, and only one, faculty member in the roster.[10]

3.4 Control Coauthors

Our original research design calls can identify changes in output trends for coauthors after a superstar coauthor passes away, relative to before. With a single level of difference, we rely on the coauthors of stars who have not died yet as an implicit control group to pin down life cycle and calendar time effects. This will provide estimates that can be given a causal interpretation under fairly general assumptions regarding the exogeneity of the death event. However, the before/after contrast might be misleading if collaborations with superstars are subject to idiosyncratic dynamic patterns. For example, collaborations might grow stale over time, and our estimates might (in part) reflect this natural evolution rather than spillovers. Alternatively, happenstance might yield a sample of stars clustered in decaying scientific fields. Therefore, we adduce to the data a set of control coauthors so that we can analyze the impact of superstar death in a difference-in-differences set-up. In creating a suitable control group, we face two hurdles. First, we only have a small set of observable characteristics available to match coauthors. Second, the death of one of our 137 superstars should have no effect on the output of a suitable control coauthor. This is problematic because of indirect coauthorship ties: it is almost impossible to find scientists that are isolates in the network formed by life scientists and their collaborations.

Because of the first issue, we begin by finding controls for the 137 superstars who die among our set of 7,276 eminent scientists. We first eliminate from the set of potential controls

[10]To fix ideas, `Lechleiter JD` is an example of unique PubMed name. In contrast, `Weinstein SL` corresponds to two distinct faculty in the roster, `Miller MJ` to ten, and `Wang Y` to thirty six.

any scientist who coauthored with one of the treated superstar. We then run a logit, using a large number of covariates: publications, citations, funding, patents, number of coauthors, degree, gender, department, age, etc., to predict death in the set of 3,444 scientists who remain after applying this screen. Our favored specification has a pseudo-R^2 of .173, and we use the predicted value to select two "nearest neighbor" superstars for each of the superstars who dies. In other words, we identify 232 eminent scientists who appear similar (based on observable characteristics) to the 137 superstars that are the focus of the paper. The coauthors of those 232 superstars constitute our control group. Of course, it is sometimes the case that scientists who coauthor with the 232 control superstars also coauthor with one or more of the 137 focal superstars. In every specification that makes use of the controls, we eliminate from the estimation sample these "problematic" coauthors. This implies that the minimum path length between a control coauthor and a treated coauthor is 3 when we constrain the paths to pass through at least one of our 7,276 superstars. As a result, we are quite confident that our control sample is relatively "uncontaminated" by indirect coauthorship ties. Figure 1 presents a stylized schema of the coauthorship ties between control and treatment coauthors that are allowed by our data assembly process, and contrast them with the ties that we rule out.[11]

3.5 Descriptive Statistics

When applied to our sample of 232+137 = 369 superstars, S/CGen software identifies 15,715 scientists with unique PubMed names (11,033 controls and 4,682 treated coauthors). This translates into approximately 73 coauthors per superstar on average (the median is 68) — the distribution for the 137 superstars that die is displayed in Figure 2. Approximately 46% of all coauthors are MDs, 45% are PhDs, and the remainder are joint degree holders (see Table 3). It is appropriate to describe the data at two different levels, that of the individual

[11]Nothing precludes one of the 7,276 eminent scientists to play the role of coauthor in some dyads (as is the case for Europa, Orion's coauthor in Figure 1). In fact, such "star/star" dyads correspond to approximately 20% of the data. This reflects the importance of assortative matching in determining the structure of collaboration networks.

peer, and that of the superstar/coauthor dyad. The software generates 7,392 treatment dyads, for a total of 191,046 dyad-year observations between 1975 and 2006.

Colleague characteristics. The demographic characteristics for the coauthors are presented in Table 5. Approximately two-thirds are affiliated with a clinical, rather than basic science, department. Nearly half hold an MD degree and 19% of them are female. In terms of research productivity, they lag behind their superstar counterparts, but the difference is not dramatic (99 vs. 161, on average). This can be explained in two ways. First, these eminent academics' careers were cut short, and as a result, they have had less time to accumulate publications. Second, assortative matching implies that many of their coauthors will also be eminent scientists. The average coauthor has approximately 99 career publications, $5 million in career NIH funding, and one patent. Approximately 88.2% of colleagues publish with only one of our 369 superstar in their careers, with the remainder predominantly publishing with two superstars. Only 53 colleagues coauthor with 4 or more of our superstars in their career (see Table 4). Note that treated and control coauthors are not perfectly balanced on observables. The latter are more likely to be female, slightly less likely to receive NIH funding, and publish 5 papers less on average over their entire career. This lack of perfect balance is not surprising given the fact that the fit of the logit predicting death among superstars (on the basis of which the control coauthors were selected) is relatively low.

Dyad characteristics. Here we find it useful to distinguish between coauthors that have published papers with stars that died suddenly from those who collaborated with stars whose death was anticipated. Table 6 presents summary statistics for both groups, both of whom display strikingly similar levels of output. About 23% of the coauthors were closely located to their superstar coauthors at their time of death, whereas approximately 8% are former trainees of the star. The descriptive statistics also reveal that these coauthors are far from a random sample of scientists, as more than half were independently funded by the NIH at the time of their superstar coauthor's death. Finally, the distribution of coauthorship intensity is extremely skewed. As such, we define "casual" coauthors as those that have two or less coauthorships with the star, "regular" coauthors as those with three to ten coauthorships,

and "close" coauthors as those with ten or more coauthorships. Using these definitions, "regular" dyads correspond to those between the 75^{th} and the 95^{th} percentile of the dyad-level coauthorship distribution and "close" coauthors correspond to those above the 95^{th} percentile (displayed in Figure 3).

3.6 Econometric Modeling

Our estimating equation relates peer j's output in year t to characteristics of i and j:

$$E\left[y_{jt}|X_{ijt}\right] \;=\; exp\left[\beta_0 + \beta_1 AFTER\_DEATH_{it} + f(AGE_{jt}) + \delta_t + \gamma_{ij}\right] \qquad (1)$$

where y is a measure of publication output, $AFTER\_DEATH$ denotes an indicator variable that switches to one the year after the superstar dies, $f(AGE_{it})$ corresponds to a flexible function of the colleague's career age, the δ_t's stand for a full set of calendar year indicator variables, and the γ_{ij}'s correspond to dyad fixed effects, consistent with our approach to analyze *changes* in j's output following the passing of superstar i.

The dyad fixed effects control for many individual characteristics that could influence research output, such as gender, degree, and scientific field (although changes in department affiliations are quite frequent). Academic incentives depend on the career stage; given the shallow slope of post-tenure salary increases, Levin and Stephan (1991) suggest that levels of investment in research should vary over the career life cycle. To flexibly account for life cycle effects, we include seven indicator variables corresponding to different career age brackets, where career age measures the number of years since a scientist earned his/her highest doctoral degree (MD or PhD).[12]

Econometric considerations. The number of articles published, patents applied for, or NIH grants awarded are examples of count dependent variables — non-negative integers with many zeros and ones. For example, 20.22% of the dyad/year observations in the data correspond to years of no publication output; the figure climbs to 85.46% if one focuses on

[12]The omitted category corresponds to faculty members in the very early years of their careers (age 0 to 4). It is not possible to separately identify calendar year effects from age effects in the "within" dimension of a panel in a completely flexible fashion, because one cannot observe two individuals at the same point in time that have the same (career) age but earned their degrees in different years (Hall et al., 2005).

the count of successful grant applications, and to 96.92% for the count of patent applications that eventually issue. Following a long-standing tradition in the study of scientific and technical change, we present conditional maximum likelihood estimates of eqn. [1] based on the fixed-effect Poisson model developed by Hausman et al. (1984). Because the Poisson model is in the linear exponential family, the coefficient estimates remain consistent as long as the mean of the dependent variable is correctly specified (Gouriéroux et al., 1984). Further, "robust" standard errors are consistent even if the underlying data generating process is not Poisson. In fact the Hausman et al. estimator can be used for any non-negative dependent variables, whether integer or continuous (Wooldridge, 1997; Santos Silva and Tenreyro, 2006), as long as the variance/covariance matrix is computed using the outer product of the gradient vector.[13] We make our inference conservative by clustering all standard errors around superstar scientists.

Quality adjustment. To adjust publications for quality, we make use of the Journal Citation Reports, published yearly by the Institute for Scientific Information. ISI ranks journals by impact factor (JIF) in different scientific fields. The impact factor is a measure of the frequency with which the "average article" in a journal has been cited in a particular year. We weight each article published by the scientists in our sample by the corresponding journal's JIF, and compute quality-weighted publication counts in this way.[14]

Competing stories. Of course, the death of a superstar colleague can influence the productivity of their colleagues through channels other than knowledge spillovers. Coauthors may go through a period of bereavement or even question their commitments to work after losing a prominent coauthor. Alternatively, the death of a star may represent a loss of social connections rather than one directly related to knowledge. For example, stars may

[13] In other words, inference on the coefficient estimates presented below do not make use of the Poisson variance assumption. We assume only that the conditional mean of our dependent variables can be written $E[y|X] = exp(X\beta)$, a requirement we consider fairly innocuous, given the non-negative nature of the outcomes considered here.

[14] Basically a ratio between citations and recent citable items published, JIFs suffer from built-in biases: they tend to discount the advantage of large journals over small ones, of frequently-issued journals over less frequently-issued ones, and of older journals over newer ones. Nonetheless, they convey quite effectively the idea that the *New England Journal of Medicine* (Impact Factor = 23.223 in 1991) is a much more influential publication than the *Journal of General Internal Medicine* (Impact Factor = 1.056 in 1991).

have whispered in the ears of Deans and Project Officers to advocate on the behalf of their coauthors. Absent the star, these researchers may find it increasingly difficult to access the financial resources that enable them to conduct research. These competing hypotheses regarding the effects of superstar death imply a distinct signature of effects across different types of colleagues. In the case of bereavement, for example, we would expect the effects of star death to be immediate and more pronounced among closer colleagues. For internal social connections, co-located peers should experience larger effects, while impacts related to external connections should be more pronounced for those working with superstars that are more centrally located in the coauthorship network. In order to rule out these alternative explanations (or at least rule them out as the dominant phenomenon underlying our measured effects) we examine heterogeneity in the response to a break in exposure to superstar talent more closely. In particular, we interact the treatment effect with various fixed characteristics of the superstar, the colleague, or the dyad at the time the superstar passes away. These characteristics include geographic proximity, career stage, former trainee status, etc. Such interactions identify how various coauthor subgroups are differentially impacted by the death. These differential impacts directly inform our interpretation of the results, *i.e.*, they can help us distinguish between distinct mechanisms.

4 Results

We begin by validating our contention that our sample of superstars is composed of scientists still actively engaged in science at the time of their death. In Table 7, we present results for specifications in which the quality-adjusted publication output of our superstars is regressed on a series of indicator variables corresponding to the timing of death: 4 years before the year of death, 3 years before the year of death, and so on, up until two years after the year of death (a scientist can, and often does, publish after his death because his/her coauthors will typically push articles through the pipeline on his behalf). We stack the deck in favor of finding preexisting trends in the data by using solely the publications in which the star appears as last author (last author status is invariably reserved to the head of a

laboratory/research group in the life sciences). All models include superstar scientist fixed effects, and we use as a control group the set of control superstars described above. The inclusion of controls is important insofar as it enables us to pin down the effect of age and calendar time, which might be correlated with the death effect.

All estimates are presented in the form of incidence rate ratios; the formula $(e^{\beta}-1)\times 100\%$ (where β denotes an estimated coefficient) provides a number directly interpretable in terms of elasticity. Model (1), for instance, implies that output falls $1 - 0.61 = 39\%$ in the year following the year during which the superstar passed away. Column (1) uses all the data, and uncovers no evidence of decline in output prior to the superstar's death. In column (2), we drop the 80 colleagues whose death was anticipated, and obtain similar results. Conversely, column (3) excludes 57 superstars whose death was sudden and unexpected. The indicator variables are therefore identified solely off the superstars whose death was anticipated. In this case, we find modest evidence that scientists who died exhibited *higher* output in the year immediately preceding their death. This effect is only significant at the 10% level. In light of these results, we feel confident that our scientists were still actively engaged in science at the time of their deaths.

Table 8 presents our core results. The first two columns in Panel A examine the output of our superstars' coauthors, regardless of cause of death. We find a sizable and significant 7% decrease in the total number of quality-adjusted publications they produce after the star dies. The following two columns concentrate on the coauthors of stars that died suddenly. Here, we find rather similar results overall — roughly a 10% decrease in total publications, with a slightly larger effect for regular collaborators. When we turn our attention to anticipated deaths (columns 5 and 6), the overall impact of star death is less substantial, but remains negative. We also see virtually no net effect for regular coauthors. While nearly all star coauthors witness a drop in their total publication output, Panel B reveals that close and, to a lesser extent, regular coauthors do manage to find replacement collaborators to partially offset their loss. Close collaborators experience between a 17% and 26% increase in their quality-adjusted publications without the star (columns 2, 4, and 6).

Panel C in Table 8 presents results from estimating eqn. [1] for total publications with the inclusion of the control sample. Here we see the overall effect of star death is a remarkably consistent 18% across all specifications. The effect is still greater for regular coauthors of stars that died suddenly, with a net decline in excess of 20%, and somewhat smaller net decline 4% for anticipated deaths. These interaction terms are not estimated precisely, however. When we turn our attention to Panel D, we again find strong evidence of coauthor substitution for close collaborators along with weaker and more mixed evidence for regular coauthors.

We also explore the timing of the effects uncovered in Panel A of Table 8. We do so by estimating specifications in which the treatment effect is interacted with a set of indicator variables corresponding to a particular year before or after the superstar's death, and then graphing the effects and the confidence interval around them for expositional purposes (Figure 4). In the left panel, we focus on coauthors of stars whose death was anticipated. We find evidence of a slight preexisting positive output trend, but it is imprecisely estimated. In contrast, for the case of sudden death (right panel), we find no evidence of a direction in the output trend prior to the superstar's death, and a steady, linear decrease afterwards, becoming statistically significant in the third year after the superstar's death. This increasingly negative trend is consistent with two related observations. First, it may be that scientists continue to pursue the lines of superstar research inquiry, but that this becomes increasingly difficult over time since the star is no longer around to infuse the field with fresh ideas. Second, most NIH grants last for three to five years, so the impacts of a superstar's absence may not really be felt until it becomes time to apply for a new grant. It is also noteworthy that we find no evidence of recovery. It is certainly possible for coauthors to adjust to superstar extinction — for example, by changing the trajectory of their scientific investigations — but identifying these adjustments might be a more demanding task than our data can be reasonably expected to bear. Thus, there appears to be some tension between the importance of the star and his/her legacy.

Table 9 examines the relationship between knowledge spillovers and the accomplishments of the star. We find that the (negative) impact of star death is increasing in quartiles of the

superstars citations at the time of their death (columns 1a and 1b), a pattern that remains when citations are adjusted for career length (columns 2a and 2b). We see no such pattern for quartiles of career NIH funding for the star (columns 3a and 3b). Together, these results suggest that it is the quality of ideas emanating from stars, rather than the availability of research funding, that increases the productivity of colleagues. They also suggest that using the same empirical strategy, but applying it to a sample of "humdrum" coauthors who die, would not uncover effects similar in magnitude to those we observe here.

Tables 10, 11A, and 11B attempt to address the key competing hypotheses regarding the impacts of star death raised earlier. Table 10 employs three measures of network centrality and shows that spillovers are not increasing based on the centrality of the superstar. The first four columns in Table 11a show that the effect of losing a star does not differ for those co-located with the star at death. Being a former trainee of the star also has no differential effect (columns 4a and 4b). The effect of death is slightly reduced for recent coauthors (column 3a), but this advantage disappears once controls are added to the regression (column 3b). Table 11b shows that the effects of losing a superstar are experience equally by senior and junior colleagues as well as by those with or without an NIH grant at the time of death. These findings suggest that the negative effects of star death on their colleagues are not well explained by either a bereavement effect or a loss of either internal or external social connections.

Tables 11C and Table 12 provide additional tests of our intuition regarding the nature of the spillover effects. Consistent with the notion that colleagues with longer collaboration histories with the star have been more imprinted by their expertise and are better able to continue on in their absence, columns 1a and 1b of Table 11C show that the effect of star death is decreasing in the age of the colleague-star relationship. In a similar vein, columns 2a and 2b reveal that colleagues in later stages of their career when the star dies are less impacted by that death. The effect of peer maturity is also demonstrated in columns 1a and 1b of Table 12. Here we see that, when the coauthor is at least 15 years more senior than the star, death has no effect. Table 12 also suggests that, as one might expect, those coauthors that are very accomplished themselves are immune to the negative impacts of star death

(columns 2a and 2b). Reassuringly, columns 3a and 3b show no effect when we examine the impact of a star that dies beyond the highly creative stages of their career, *i.e.*, when the star is at least 75 years old at their time of death. Finally, we also find no effect when we generate placebo dates of death among our control superstars (where those dates are chosen to mimic the distribution of death events across years among our extinct superstars) and examine how output changes following these manufactured events solely among the control coauthors.

5 Conclusion

In this paper, we have presented an analysis of scientific collaborations in the academic life sciences, focusing more particularly on the impact of superstars on their collaborators. Using the premature death of still active superstars, we uncover strong evidence in support of spillover effects in this setting. When an eminent scientist dies, nearly all of his peers experience a sizable and permanent decrease in their total research output. The claim that this is a genuine star effect is supported by the evidence that effects are increasing in the accomplishments of stars. Moreover, the breadth of the impacts, which do not vary across a wide range of individual and individual-star dyad characteristics, make alternative explanations for this phenomenon less likely. If the effect were due to bereavement, we would expect more recent and closer colleagues to experience a more negative effect. We should also see an immediate impact that diminishes over time. If the effect were due to social connections, we would expect effects to be larger for closer colleagues as well as for those working at the same institution. Star centrality within the professional network should also matter. We see none of this. Rather, our results are consistent with the idea that part of the scientific field embodied in the invisible college of coauthors working in that area dies along with the star — that superstar extinction represents a genuine and irreplaceable loss of human capital.

At this stage, the interpretation of our estimates as genuine externalities must remain tempered. We observe output, not productivity; an interpretation of our results is that

superstars shift their peer's aspiration level, thus triggering more effort, even in collaborations that do not involve the star. While this corresponds to a real effect of the star, the term externality should not apply because the peer presumably bears the cost of increased effort. We must also resist calling these effects externalities because we document the benefits of collaborations, but scientific coauthorships also entail coordination costs. These could be borne by the peer in the form of lower wages, or by the star, who might divert some of his/her effort towards mentorship activities. Yet, we suspect that these spillovers are not fully internalized by the scientific labor market. In academia, pay exhibits much less dispersion than talent, and labor market frictions limit superstars' ability to extract their full marginal product.

The data presented here provide a platform to explore the "technology" of knowledge spillovers. Much more could be done to explore human capital externalities in this setting. To what extent are information technologies contributing to the diffuse impacts of star death? Are certain fields more susceptible to spillovers than others? Do we see similar effects when output is measured in terms of grants or patents? And, of course, how might these results be altered for relationship changes other than death? Answering these questions are the next steps of our research agenda.

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Table 1A: Superstar Sample, Sudden Deaths

Name		Degree/Year		Cause of Death	Institutional Affiliation	Career Patents	Career Pubs	Career Cites	Nb Coauthors
Alan P. Wolffe	(1959-2001)	PhD	1984	car accident	NIH	0	245	19,238	46
Stanley R. Kay	(1946-1990)	PhD	1980	heart attack	Albert Einstein College of Medicine	0	93	5,467	21
Joaquim Puig-Antich	(1944-1989)	MD	1967	asthma attack	University of Pittsburgh	0	83	4,849	42
Matthew L. Thomas	(1953-1999)	PhD	1981	died while travelling	Washington University in St. Louis	0	82	8,867	74
Mu-En Lee	(1954-2000)	MD/PhD	1984	complications from routine surgery	Harvard Medical School/MGH	17	83	6,289	74
Howard S. Tager	(1945-1994)	PhD	1971	heart attack	University of Chicago	0	99	5,638	47
John J. Wasmuth	(1946-1995)	PhD	1973	heart attack	University of California - Irvine	1	170	7,687	161
Richard E. Heikkila	(1942-1991)	PhD	1969	murder	UMDNJ Robert Wood Johnson Medical School	0	138	10,862	26
Harold A. Menkes	(1938-1987)	MD	1963	car accident	Johns Hopkins University	0	93	2,827	55
Roland L. Phillips	(1937-1987)	MD/PhD	1971	glider plane accident	Loma Linda University School of Medicine	0	35	3,323	29
Neil S. Jacobson	(1949-1999)	PhD	1977	heart attack	University of Washington	0	46	3,569	19
D. Michael Gill	(1940-1990)	PhD	1967	heart attack	Tufts University	0	75	8,019	26
Emil T. Kaiser	(1938-1988)	PhD	1959	complications from kidney transplant	Rockefeller University	7	143	6,253	70
Gary J. Miller	(1950-2001)	MD/PhD	1978	heart attack	University of Colorado HSC	0	98	3,297	98
Roland D. Ciaranello	(1943-1994)	MD	1970	heart attack	Stanford University	0	107	3,781	61
John B. Penney, Jr.	(1947-1999)	MD	1973	heart attack	Harvard Medical School/MGH	0	164	13,549	112
Mary Lou Clements	(1946-1998)	MD	1972	airplane crash	Johns Hopkins University	0	126	6,897	133
James R. Neely	(1936-1988)	PhD	1966	heart attack	Penn State University	0	91	8,732	26
Hymie L. Nossel	(1930-1983)	MD/PhD	1962	heart attack	Columbia University	0	80	5,000	45
Simon J. Pilkis	(1942-1995)	MD/PhD	1971	heart attack	University of Minnesota	0	166	8,970	68
Fredric S. Fay	(1943-1997)	PhD	1969	heart attack	UMASS	7	108	7,947	55
Jeffrey M. Isner	(1947-2001)	MD	1973	heart attack	Tufts University	9	373	29,075	213
Roy D. Schmickel	(1936-1990)	MD	1961	died tragically	University of Pennsylvania	0	63	3,531	60
Roger R. Williams	(1944-1998)	MD	1971	airplane crash	University of Utah	0	175	8,778	123
Julio V. Santiago	(1942-1997)	MD	1967	heart attack	Washington University in St. Louis	0	119	7,081	112
William L. McGuire	(1937-1992)	MD	1964	scuba-diving accident	University of Texas HSC at San Antonio	4	296	27,508	135
Walter F. Heiligenberg	(1938-1994)	PhD	1964	plane crash	UCSD	0	51	1,881	8
George J. Schroepfer, Jr.	(1932-1998)	MD/PhD	1961	heart attack	Rice University	2	183	5,230	44
George Streisinger	(1927-1984)	PhD	1953	scuba-diving accident	University of Oregon	0	38	3,765	11
D. Martin Carter	(1936-1993)	MD/PhD	1971	dissecting aortic aneurysm	Rockefeller University	0	87	2,678	61
Verne M. Chapman	(1938-1995)	PhD	1965	died suddenly while attending meeting	Roswell Park Cancer Institute/SUNY Buffalo	0	151	7,546	112
Dolph O. Adams	(1939-1996)	MD/PhD	1969	unexpected	Duke University	0	123	7,721	43
Don C. Wiley	(1944-2001)	PhD	1971	accidental fall	Harvard University	0	202	30,974	90
Edward V. Evarts	(1926-1985)	MD	1948	heart attack	NIH	0	79	5,254	16
G. Scott Giebink	(1944-2003)	MD	1969	heart attack	University of Minnesota	0	178	4,302	100
Ronald G. Thurman	(1941-2001)	PhD	1967	massive heart attack	University of North Carolina	3	444	15,289	103
Raymond R. Margherio	(1940-2000)	MD	1965	aneurysm	Wayne State University School of Medicine	0	26	697	15
Christopher A. Dawson	(1942-2003)	PhD	1969	suddenly	Medical College of Wisconsin	0	192	3,936	90
Donald C. Shreffler	(1933-1994)	PhD	1961	heart attack	Washington University in St. Louis	0	166	8,295	84
DeWitt S. Goodman	(1930-1991)	MD	1955	pulmonary embolism	Columbia University	0	216	15,586	75
John H. Walsh	(1938-2000)	MD	1963	heart attack	UCLA	1	370	16,854	186
Donald T. Witiak	(1935-1998)	PhD	1961	stroke	University of Wisconsin	29	120	2,028	63
Thomas F. Burks, II	(1938-2001)	PhD	1967	heart attack	University of Texas HSC at Houston	1	254	8,355	73
Norbert Freinkel	(1926-1989)	MD	1949	heart attack	Northwestern University	0	188	9,833	76
Robert M. Macnab	(1940-2003)	PhD	1969	accidental fall	Yale University	0	111	6,881	22
Philip J. Fialkow	(1933-1996)	MD	1960	trekking accident in Nepal	University of Washington	0	168	10,806	100
John J. Jeffrey, Jr.	(1937-2001)	PhD	1965	stroke	Albany Medical College	0	123	7,367	95
Demetrios Papahadjopoulos	(1934-1998)	PhD	1963	adverse drug reaction/multi-organ failure	UCSF	13	203	25,372	96
Gerald D. Aurbach	(1927-1991)	MD	1954	hit in a head by a stone	NIH	0	227	16,448	87
Takis S. Papas	(1935-1999)	PhD	1970	unexpected and sudden	Medical University of South Carolina	9	195	9,763	98
James N. Davis	(1939-2003)	MD	1965	airplane crash	SUNY HSC at Stony Brook	0	98	5,005	44
Sandy C. Marks, Jr.	(1937-2002)	DDS/PhD	1968	heart attack	UMASS	0	214	5,105	69
George B. Craig, Jr.	(1930-1995)	PhD	1956	heart attack	University of Notre Dame	0	74	1,710	18
Paul B. Sigler	(1934-2000)	MD/PhD	1967	heart attack	Yale University	0	132	18,527	95
Gerald P. Murphy	(1934-2000)	MD	1959	heart attack	Roswell Park Cancer Institute/SUNY Buffalo	2	404	14,667	130
Patricia S. Goldman-Rakic	(1937-2003)	PhD	1963	struck by a car	Yale University	2	285	29,271	85
Zanvil A. Cohn	(1926-1993)	MD	1953	aortic dissection	Rockefeller University	0	276	38,918	87

Table 1B: Superstar Sample, Anticipated Deaths

Name		Degree/Year		Cause of Death	Institutional Affiliation	Career Patents	Career Pubs	Career Cites	Nb Coauthors
Richard C. Parker	(1951-1986)	PhD	1979	lymphoblastic T-cell lymphoma	Columbia University	0	14	1,883	12
Ernest G. Peralta	(1959-1999)	PhD	1986	brain cancer	Harvard University	0	41	5,359	17
George Khoury	(1943-1987)	MD	1970	lymphoma	NIH	0	134	11,305	66
B. Frank Polk	(1942-1988)	MD	1967	brain cancer	Johns Hopkins University	0	107	8,226	121
Joel D. Meyers	(1944-1991)	MD	1970	colon cancer	University of Washington/FHCRC	0	171	14,717	120
Melvin L. Marcus	(1940-1989)	MD	1966	colon cancer	UMASS	0	225	15,034	96
Harold Weintraub	(1945-1995)	MD/PhD	1973	brain cancer	University of Washington/FHCRC	3	154	31,562	83
Richard K. Gershon	(1932-1983)	MD	1959	lung cancer	Yale University	0	182	12,007	73
Markku Linnoila	(1947-1998)	MD/PhD	1974	cancer	NIH	6	513	21,254	175
Theodore S. Zimmerman	(1937-1988)	MD	1963	lung cancer	Scripps Research Institute	20	132	12,264	75
Larry C. Clark	(1948-2000)	PhD	1981	prostate cancer	University of Arizona	1	33	2,387	33
Robert F. Spencer	(1949-2001)	PhD	1974	gastric carcinoma	Medical College of Virginia	0	59	2,136	25
Janis V. Giorgi	(1947-2000)	PhD	1977	uterine cancer	UCLA	1	130	10,212	137
Samuel W. Perry, 3rd	(1941-1994)	MD	1967	pancreatic cancer	Weill Medical College ¯ Cornell University	0	44	1,210	31
Edward C. Franklin	(1928-1982)	MD	1950	brain cancer	New York University	0	224	12,761	69
Gerald T. Babcock	(1946-2000)	PhD	1973	cancer	Michigan State University	0	123	8,511	49
Lois K. Miller	(1945-1999)	PhD	1972	melanoma	University of Georgia	9	120	8,261	20
Tai-Shun Lin	(1939-1994)	PhD	1970	non hodgkin's lymphoma	Yale University School of Medicine	19	91	3,102	42
Ora M. Rosen	(1935-1990)	MD	1960	breast cancer	Sloan Kettering Institute for Cancer Research	0	150	14,587	72
Edwin H. Beachey	(1934-1989)	MD	1962	cancer	University of Tennessee at Memphis	10	192	10,080	57
Elizabeth A. Bates	(1974-2003)	PhD	1974	pancreatic cancer	UCSD	0	81	1,985	18
Helene S. Smith	(1941-1997)	PhD	1967	breast cancer	UCSF	5	97	5,634	59
Murray Rabinowitz	(1927-1983)	MD	1950	muscular dystrophy	University of Chicago	0	146	8,229	51
C. Richard Taylor	(1939-1995)	PhD	1963	heart failure	Harvard University	0	100	6,377	26
Norton B. Gilula	(1944-2000)	PhD	1971	lymphoma	Scripps Research Institute	3	99	13,147	62
Ira Herskowitz	(1946-2003)	PhD	1971	pancreatic cancer	UCSF	4	159	17,457	85
Bernard N. Fields	(1938-1995)	MD	1962	pancreatic cancer	Harvard Medical School/Brigham & Women's Hospital	1	181	10,505	102
Wallace P. Rowe	(1926-1983)	MD	1948	colon cancer	NIH	0	232	21,432	79
Allan C. Wilson	(1934-1991)	PhD	1961	leukemia	UC Berkeley	0	164	25,915	48
Priscilla A. Campbell	(1940-1998)	PhD	1968	cervical cancer	University of Colorado HSC/Nat. Jewish center	0	77	3,914	31
Laird S. Cermak	(1942-1999)	PhD	1968	leukemia	Boston University	0	73	2,338	25
Peter A. Kollman	(1944-2001)	PhD	1970	cancer	UCSF	0	189	9,095	79
Elizabeth M. Smith	(1939-1997)	PhD	1978	cancer	Washington University School of Medicine	0	47	1,265	17
Nelson Butters	(1937-1995)	PhD	1964	Lou Gehrig's disease	UCSD	0	191	12,555	105
Aaron Janoff	(1930-1988)	PhD	1959	long illness	SUNY HSC at Stony Brook	0	131	8,590	58
David G. Marsh	(1940-1998)	PhD	1964	glioblastoma	Johns Hopkins University	1	142	6,395	58
G. Harrison Echols, Jr.	(1933-1993)	PhD	1959	lung cancer	UC Berkeley	0	112	9,190	56
George Némethy	(1934-1994)	PhD	1962	brain cancer	Mount Sinai School of Medicine	0	76	7,079	16
Mette Strand	(1937-1997)	PhD	1964	cancer	Johns Hopkins University	4	128	6,044	58
Lawrence H. Piette	(1932-1992)	PhD	1957	cancer	Utah State University	0	56	2,722	19
William L. Chick	(1938-1998)	MD	1963	diabetes complications	UMASS	0	90	5,108	70
Charles A. Janeway, Jr.	(1943-2003)	MD	1969	B-cell lymphoma	Yale University	0	313	31,455	141
Howard M. Temin	(1934-1994)	PhD	1959	lung cancer	University of Wisconsin	4	210	17,276	46
Joachim G. Liehr	(1942-2003)	PhD	1968	pancreatic cancer	University of Texas Medical Branch at Galveston	0	134	6,272	71
Harvey D. Preisler	(1941-2002)	MD	1965	lymphoma	Rush Medical College	0	304	7,069	107
Keith Green	(1940-2001)	PhD	1964	died after lengthy illness	Medical College of Georgia	0	206	2,572	44
Donald J. Cohen	(1940-2001)	MD	1966	ocular melanoma	Yale University	0	298	12,166	160
Gregory Mooser	(1942-2003)	DDS/PhD	1972	complications from alzheimer's disease	University of Southern California	0	23	962	9
Roy H. Steinberg	(1935-1997)	MD/PhD	1965	multiple myeloma	UCSF	3	121	6,707	25
Thomas W. Smith	(1936-1997)	MD	1965	mesothelioma	Harvard Medical School/Brigham & Women's Hospital	0	158	11,136	108
John C. Liebeskind	(1935-1997)	PhD	1962	cancer	UCLA	0	147	10,160	30
Marian W. Fischman	(1939-2001)	PhD	1972	colon cancer	Columbia University	0	157	5,910	42
Richard J. Wyatt	(1939-2002)	MD	1964	lung cancer	NIH	2	533	21,692	205
Sydney E. Salmon	(1936-1999)	MD	1962	pancreatic cancer	University of Arizona	8	286	20,024	204
Sidney H. Ingbar	(1925-1988)	MD	1947	lung cancer	Harvard Medical School/Beth Israel Medical Center	1	290	14,523	75
Eva J. Neer	(1937-2000)	MD	1963	breast cancer	Harvard Medical School/Brigham & Women's Hospital	0	104	10,652	60
Kiichi Sagawa	(1926-1989)	MD/PhD	1958	cancer	Johns Hopkins University	0	119	9,491	39
Charles D. Heidelberger	(1920-1983)	PhD	1946	carcinoma of nasal sinus	University of Southern California	0	242	19,302	59
Robert J. Fass	(1939-2002)	MD	1964	lung cancer	Ohio State University	0	132	3,703	77
Gerald L. Klerman	(1928-1992)	MD	1954	diabetes	Weill Medical College ¯ Cornell University	0	252	19,458	114
Eleanor M. Saffran	(1938-2002)	PhD	1968	amyotrophic lateral sclerosis	Temple University School of Medicine	0	41	2,811	14
Merton Bernfield	(1938-2002)	MD	1961	Parkinson's Disease	Harvard Medical School/Children's Hospital	8	137	11,962	87
Richard P. Bunge	(1932-1996)	MD	1960	esophageal cancer	University of Miami	0	157	10,828	70
Irving Kupfermann	(1938-2002)	PhD	1964	Creutzfeldt-Jacob's disease	Columbia University	0	120	7,635	23
Jiri Palek	(1934-1998)	MD	1958	2 year illness	Tufts University	0	127	5,487	51
Harold C. Neu	(1934-1998)	MD	1960	glioblastoma	Columbia University	1	530	18,498	102
Edwin L. Bierman	(1930-1995)	MD	1955	bone cancer	University of Washington	0	204	14,583	70
Edgar Haber	(1932-1997)	MD	1956	multiple myeloma	Harvard University School of Public Health	29	372	27,458	175
Joseph Stokes, 3rd	(1924-1989)	MD	1949	cancer	Boston University School of Medicine	0	62	2,815	46
J. Christian Gillin	(1938-2003)	MD	1966	esophageal cancer	UCSD	0	355	15,729	225
Leo J. Neuringer	(1928-1993)	PhD	1957	cancer	MIT	0	39	1,264	31
Frank Lilly	(1930-1995)	PhD	1965	prostate cancer	Albert Einstein College of Medicine	0	94	4,245	55
Jane Pitt	(1938-2003)	MD	1964	chronic lymphocytic leukemia	Columbia University College of Physicians and Surgeon	0	95	3,235	120
Barbara H. Bowman	(1930-1996)	PhD	1959	cancer	University of Texas Medical School at San Antonio	1	115	3,371	72
Charlotte Friend	(1921-1987)	PhD	1950	lymphoma	Mount Sinai School of Medicine	0	98	5,486	34
Albert Dorfman	(1916-1982)	MD/PhD	1944	kidney failure	University of Chicago	0	183	9,660	45
William H. Tooley	(1925-1992)	MD	1949	long illness	UCSF School of Medicine	0	77	5,890	62
John R. Williamson	(1934-2000)	PhD	1959	cancer	University of Pennsylvania School of Medicine	0	178	13,661	86
Henry S. Kaplan	(1918-1984)	MD	1940	lung cancer	Stanford University School of Medicine	3	395	27,480	132
Charles G. Moertel	(1927-1994)	MD	1953	Hodgkin's Disease	Mayo Medical School	0	290	21,372	166

Table 2A: Summary Statistics for Superstars

	Mean	Std. Dev	Min.	Max.
Anticipated (N=80)				
Age at Death	58.56	6.30	35	66
Year of Death	1995.19	6.06	1982	2003
Career Age at Death	31.96	7.05	7	44
Degree Year	1963.23	8.72	1940	1986
# Positions	1.79	0.92	1	6
# Coauthors	72.21	48.24	9	225
NIH funding (total)	$23,766,286	$30,722,321	$0	$202,505,312
NIH funding (excl. center grants)	$12,744,421	$10,820,568	$0	$57,208,996
# Papers (total)	164	109	14	533
# Citations	10,448	7,359	962	31,562
# Patents	1.84	4.77	0	29
h index	50.16	19.77	10	89
Sudden (N=57)				
Age at Death	56.46	6.57	42	66
Year of Death	1995.67	5.42	1983	2003
Career Age at Death	29.30	7.25	10	41
Degree Year	1966.37	7.76	1948	1984
# Positions	1.70	0.78	1	4
# Coauthors	73.81	43.44	8	213
NIH funding (total)	$18,935,842	$15,794,497	$0	$73,509,824
NIH funding (excl. center grants)	$11,060,302	$9,982,714	$0	$61,924,784
# Papers (total)	157	93	26	444
# Citations	9,949	8,333	697	38,918
# Patents	1.88	5.00	0	29
h index	48.49	19.77	14	108
Total (N=137)				
Age at Death	57.69	6.48	35	66
Year of Death	1995.39	5.79	1982	2003
Career Age at Death	30.85	7.23	7	44
Degree Year	1964.53	8.45	1940	1986
# Positions	1.75	0.86	1	6
# Coauthors	72.88	46.14	8	225
NIH funding (total)	$21,756,539	$25,626,239	$0	$202,505,312
NIH funding (excl. center grants)	$12,043,729	$10,475,725	$0	$61,924,784
# Papers (total)	161	103	14	533
# Citations	10,240	7,753	697	38,918
# Patents	1.85	4.85	0	29
h index	49.47	19.71	10	108

Table 2B: Summary Statistics for Superstars (Counts)

	N	MD	PhD	MD/ PhD	NAS	HHMI	MERIT	Female	US born
Anticipated	80	36	38	6	20	4	26	14	68
Sudden	57	21	26	10	10	6	21	2	47
Total	137	57	64	16	30	10	47	16	115

Table 3: Degree Breakdown for coauthors

Degree	**Frequency**	**Percent**
MD	7,267	46.24%
PhD	7,006	44.58%
MD/PhD	1,442	9.18%
Total	15,715	100%

Table 4: Number of Superstar Coauthors per Colleague

	Freq.	Proportion
1	13,863	88.22%
2	1,539	9.79%
3	260	1.65%
4	42	0.27%
5	11	0.07%
Total	15,715	100%

Table 5: Summary Statistics for Coauthors of Control and Extinct Stars

	Mean	Std. Dev.	Min.	Max.
Coauthors of Control Stars (N=11,033)				
Female	0.198	0.399	0	1
MD	0.465	0.499	0	1
PhD	0.447	0.497	0	1
MD/PhD	0.088	0.283	0	1
Basic Science Dept.	0.33	0.47	0	1
Ever received NIH grant	0.605	0.489	0	1
Member of the NAS	0.018	0.133	0	1
Degree Year	1976.604	11.572	1936	2002
# Patents (over the career)	0.641	3.199	0	157
# Papers (over the career)	0.198	0.399	0	1
NIH funding	$4,637,426	$12,073,809	$0	$462,601,248
Coauthors of Extinct Stars (N=4,682)				
Female	0.177	0.382	0	1
MD	0.476	0.499	0	1
PhD	0.421	0.494	0	1
MD/PhD	0.103	0.304	0	1
Basic Science Dept.	0.336	0.472	0	1
Ever received NIH grant	0.644	0.479	0	1
Member of the NAS	0.018	0.133	0	1
Degree Year	1974.59	9.773	1943	1998
# Patents (over the career)	0.752	3.166	0	70
# Papers (over the career)	104.071	99.879	2	935
NIH funding	$5,298,970	$12,792,579	$0	$359,540,992
All Coauthors (N=15,715)				
Female	0.192	0.394	0	1
MD	0.468	0.499	0	1
PhD	0.439	0.496	0	1
MD/PhD	0.092	0.289	0	1
Basic Science Dept.	0.332	0.471	0	1
Ever received NIH grant	0.617	0.486	0	1
Member of the NAS	0.018	0.133	0	1
Degree Year	1976.002	11.103	1936	2002
# Patents (over the career)	0.674	3.19	0	157
# Papers (over the career)	101.173	99.236	1	935
NIH funding	$4,834,521	$12,295,663	$0	$462,601,248

Table 6: Summary Statistics for Superstar/Colleague Dyads

	Mean	Std. Dev	Min.	Max.
Anticipated (N=4,303)				
JIF-weighted Papers (w/o star)	450.674	569.065	0.311	5705.146
JIF-weighted Papers (total)	542.481	645.322	1.246	6293.937
Non-weighted Papers (total)	140.198	131.307	2	1002
# Coauthorships	3.117	5.935	1	112
Trainee of star	0.083	0.276	0	1
Coauthor is senior to star	0.037	0.188	0	1
R01 grant at time of death	0.547	0.498	0	1
Career age at time of death	22.688	8.936	5	40
Same school at time of death	0.244	0.429	0	1
Within 10 miles at time of death	0.278	0.448	0	1
At least 1 coauthorship in 5 years prior to death	0.347	0.476	0	1
Sudden (N=3,089)				
JIF-weighted Papers (w/o star)	475.036	581.844	0.388	5067.218
JIF-weighted Papers (total)	556.654	651.196	1.378	6293.937
Non-weighted Papers (total)	142.299	134.428	2	1002
# Coauthorships	3.196	6.261	1	99
Trainee of star	0.073	0.26	0	1
Coauthor is senior to star	0.06	0.237	0	1
R01 grant at time of death	0.564	0.496	0	1
Career age at time of death	22.205	8.745	5	40
Same school at time of death	0.211	0.408	0	1
Within 10 miles at time of death	0.216	0.411	0	1
At least 1 coauthorship in 5 years prior to death	0.36	0.48	0	1
Total (N=7,392)				
JIF-weighted Papers (w/o star)	460.854	574.526	0.311	5705.146
JIF-weighted Papers (total)	548.404	647.777	1.246	6293.937
Non-weighted Papers (total)	141.076	132.615	2	1002
# Coauthorships	3.15	6.073	1	112
Trainee of star	0.079	0.269	0	1
Coauthor is senior to star	0.046	0.21	0	1
R01 grant at time of death	0.554	0.497	0	1
Career age at time of death	22.486	8.859	5	40
Same school at time of death	0.23	0.421	0	1
Within 10 miles at time of death	0.252	0.434	0	1
At least 1 coauthorship in 5 years prior to death	0.353	0.478	0	1

Table 7: Trends in Publication output in the years immediately preceding/following a superstar's death

	(1)	(2)	(3)	(4)
	137 Superstars, Age at death≤66	**Excluding 80 whose death was anticipated**	**Excluding 57 whose death was sudden**	**60 Superstars Age at death>66**
	232 Controls	**103 Controls**	**143 Controls**	**61 Controls**
2 years after year of death	0.268**	0.248**	0.284**	0.235**
	[7.09]	[4.24]	[5.75]	[4.58]
1 year after year of death	0.609**	0.693†	0.541**	0.612*
	[4.09]	[1.71]	[4.98]	[2.18]
year of death	1.079	0.989	1.134	0.800
	[0.64]	[0.05]	[0.92]	[1.21]
1 year before year of death	1.170	1.100	1.219†	0.775
	[1.58]	[0.55]	[1.79]	[1.51]
2 years before year of death	0.999	0.943	1.022	0.866
	[0.01]	[0.36]	[0.17]	[0.86]
3 years before year of death	1.046	1.138	0.969	0.844
	[0.43]	[0.84]	[0.24]	[0.82]
4 years before year of death	1.106	0.941	1.233†	0.906
	[1.03]	[0.34]	[1.91]	[0.87]
Log Quasi-Likelihood	-59,933	-26,823	-35,255	-18,470
Nb. of Observations	10,120	4,379	6,188	3,375
Nb. of Scientists	369	160	223	121

The estimates above are taken from a conditional fixed effects Poisson specification that also include 7 indicator variables corresponding to different age brackets and a full suite of calendar year effects (estimates not reported). The estimates are displayed as incidence rate ratios, e.g., the estimate in column (1) implies a statistically significant (1-0.276)=72.4% decrease in the rate of publication two years after a superstar scientist passes away (regardless of cause of death). Robust (QML) z-statistics are reported in brackets. The dependent variable is the weighted article count for the superstar, including only those publications in which the superstar appears in last position on the authorship list. The weights used to create these counts are Journal Impact Factors (JIF) published by the Institute for Scientific Information.

†significant at 10%; *significant at 5%; **significant at 1%

Table 8: Impact of Superstar Death on Coauthors' Publication Rates

Panel A: Treatment Dyads Only, JIF-weighted Total Publications

	(1) All	(2) All	(3) Sudden	(4) Sudden	(5) Anticip.	(6) Anticip.
After Death	0.929** [4.01]	0.932** [3.60]	0.895** [4.11]	0.914** [2.96]	0.959† [1.75]	0.947* [2.13]
After Death × Regular Collab.		1.008 [0.25]		0.907* [2.00]		1.084* [1.96]
After Death × Close Collab.		0.926 [1.52]		0.954 [0.85]		0.911 [1.22]
Log Quasi-Likelihood	-1,271,471	-1,271,339	-531,709	-531,503	-738,531	-738,173
Nb. of Obs.	191,046	191,046	79,232	79,232	111,814	111,814
Nb. of Dyads	7,392	7,392	3,089	3,089	4,303	4,303
Nb. of Superstars	137	137	57	57	80	80

Panel B: Treatment Dyads Only, JIF-weighted Publications written with others

	(1) All	(2) All	(3) Sudden	(4) Sudden	(5) Anticip.	(6) Anticip.
After Death	0.964* [2.06]	0.941** [3.25]	0.922** [3.17]	0.919** [2.88]	1.000 [0.01]	0.959† [1.72]
After Death × Regular Collab.		1.059† [1.76]		0.949 [1.09]		1.142** [3.12]
After Death × Close Collab.		1.201** [3.51]		1.256** [3.71]		1.168* [2.01]
Log Quasi-Likelihood	-1,246,076	-1,245,380	-520,674	-520,189	-724,236	-723,539
Nb. of Obs.	191,046	191,046	79,232	79,232	111,814	111,814
Nb. of Dyads	7,392	7,392	3,089	3,089	4,303	4,303
Nb. of Superstars	137	137	57	57	80	80

Estimates are displayed as incidence rate ratios (exponentiated coefficients). For example. the estimates in column (4) of Panel A imply that casual coauthors suffer a statistically significant (1-0.914)=8.6% decrease in the rate of publication after one's superstar coauthor passes away, but that regular coauthors (between 3 and 9 publications) incur an additional decrease of 1-0.910)=9%. All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. †significant at 10%; *significant at 5%; **significant at 1%.

Panel C: Treatment and Control Dyads, JIF-weighted Total Publications

	(1) All	(2) All	(3) Sudden	(4) Sudden	(5) Anticip.	(6) Anticip.
After Death	0.824**	0.824**	0.818**	0.828**	0.837**	0.829**
	[8.89]	[8.39]	[5.63]	[5.40]	[6.62]	[6.14]
After Death × Regular Collab.		1.026		0.967		1.070
		[0.64]		[0.49]		[1.33]
After Death × Close Collab.		0.922		0.940		0.911
		[1.21]		[0.85]		[0.89]
Log Quasi-Likelihood	-2,441,055	-2,440,952	-1,109,941	-1,109,914	-1,425,233	-1,425,087
Nb. of Obs.	426,306	426,306	191,151	191,151	252,310	252,310
Nb. of Dyads	17,944	17,944	8,111	8,111	10,585	10,585
Nb. of Superstars	369	369	160	160	223	223

Panel D: Treatment and Control Dyads, JIF-weighted Publications written with others

	(1) All	(2) All	(3) Sudden	(4) Sudden	(5) Anticip.	(6) Anticip.
After Death	0.866**	0.834**	0.855**	0.835**	0.882**	0.841**
	[6.91]	[8.00]	[4.45]	[5.20]	[5.05]	[5.83]
After Death × Regular Collab.		1.098*		1.031		1.146*
		[2.24]		[0.46]		[2.56]
After Death × Close Collab.		1.282**		1.315**		1.259*
		[3.53]		[3.25]		[2.17]
Log Quasi-Likelihood	-2,374,273	-2,373,559	-1,077,733	-1,077,415	-1,387,548	-1,387,064
Nb. of Obs.	426,306	426,306	191,151	191,151	252,310	252,310
Nb. of Dyads	17,944	17,944	8,111	8,111	10,585	10,585
Nb. of Superstars	369	369	160	160	223	223

Estimates are displayed as incidence rate ratios (exponentiated coefficients). For example. the estimates in column (6) of Panel D imply that casual coauthors suffer a statistically significant (1-0.88)=12% decrease in the rate of publication written with others after one's superstar coauthor passes away, but that close coauthors partly shift their effort towards other collaborations, resulting in a net increase of (1-1.169)-(1-11.6)=5.3%. All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. †significant at 10%; *significant at 5%; **significant at 1%.

Table 9: Ideas Spillovers Are Increasing in Superstar's Accomplishments

	Superstar's Total Cites at Time of Death		Superstar's Total Cites at Time of Death, normalized by career length		Superstar's Career NIH Funding	
	(1a) w/o Controls	(1b) with Controls	(2a) w/o Controls	(2b) with Controls	(3a) w/o Controls	(3b) with Controls
After Death × Star in 1st Quartile	0.962 [0.97]	0.880* [2.46]	0.966 [0.75]	0.888† [1.84]	0.910* [2.32]	0.824** [3.98]
After Death × Star in 2nd Quartile	0.946 [1.35]	0.846** [3.28]	0.962 [1.27]	0.855** [3.44]	0.936† [1.81]	0.834** [3.98]
After Death × Star in 3rd Quartile	0.923** [2.66]	0.803** [6.19]	0.937† [1.81]	0.839** [4.39]	0.908** [2.58]	0.823** [5.02]
After Death × Star in 4th Quartile	0.911** [3.37]	0.812** [5.84]	0.901** [4.00]	0.790** [7.11]	0.919** [3.31]	0.817** [5.68]
Log Quasi-Likelihood	-1,271,362	-2,440,916	-1,271,232	-2,440,808	-1,179,140	-2,387,544
Nb. of Obs.	191,046	426,306	191,046	426,306	177,379	417,551
Nb. of Dyads	7,392	17,944	7,392	17,944	6,874	17,604
Nb. of Superstars	137	369	137	369	130	362

Conditional dyad fixed effects quasi-MLE estimates for the determinants of JIF-weighted publications among coauthors of academic life sciences superstar academics. Estimates are displayed as incidence rate ratios (exponentiated coefficients). All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. We interact the treatment variable with 4 indicator variables corresponding to quartiles for four distinct metrics of achievement for the superstars at the time of their death: total citations, total citations normalized by years of career, and career NIH funding. In the latter case, we exclude 7 scientists who spend all their careers at NIH campus in Bethesda, MD, and are therefore not eligible to receive extramural NIH funding.

†significant at 10%; *significant at 5%; **significant at 1%.

Table 10: Spillovers Are Not Increasing in Superstar's Network Centrality

	Betweenness Centrality		Eigenvector Centrality		Bonacich Centrality	
	(1a) w/o Controls	(1b) with Controls	(2a) w/o Controls	(2b) with Controls	(3a) w/o Controls	(3b) with Controls
After Death × Star in 1st Quartile	0.842* [2.31]	0.815* [2.44]	0.842* [2.31]	0.815* [2.44]	0.842* [2.31]	0.815* [2.44]
After Death × Star in 2nd Quartile	0.971 [0.66]	0.867* [2.47]	0.971 [0.66]	0.867* [2.47]	0.971 [0.66]	0.867* [2.47]
After Death × Star in 3rd Quartile	0.973 [0.97]	0.873** [3.68]	0.973 [0.97]	0.873** [3.68]	0.973 [0.97]	0.873** [3.68]
After Death × Star in 4th Quartile	0.910** [4.11]	0.799** [7.95]	0.910** [4.11]	0.799** [7.95]	0.910** [4.11]	0.799** [7.95]
Log Quasi-Likelihood	-1,271,085	-2,440,786	-1,271,085	-2,440,786	-1,271,085	-2,440,786
Nb. of Obs.	191,046	426,306	191,046	426,306	191,046	426,306
Nb. of Dyads	7,392	17,944	7,392	17,944	7,392	17,944
Nb. of Superstars	137	369	137	369	137	369

Conditional dyad fixed effects quasi-MLE estimates for the determinants of JIF-weighted publications among coauthors of academic life sciences superstar academics. Estimates are displayed as incidence rate ratios (exponentiated coefficients). All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. We interact the treatment variable with 4 indicator variables corresponding to quartiles for four distinct measures of star centrality within the coauthorship network among 7,276 eminent life scientists. Because raw centrality measures are heavily correlated with publication output, the centrality measures on which the estimates are based are residuals from a simple regression of (raw) centrality on the stars' stock of publications and a constant.

†significant at 10%; *significant at 5%; **significant at 1%.

Table 11A: Interactions with Location, Coauthorship recency, and Former Trainee Status

	Star and Coauthor Co-located at Time of Death		Star and Coauthor Separated by Less than 10 Miles at Time of Death		Recent Coauthorship		Coauthor is Superstar's Former Trainee	
	w/o Controls	with Controls	w/o Controls	with Controls	w/o Controls	with Controls	w/o Controls	with Controls
	(1a)	(1b)	(2a)	(2b)	(3a)	(3c)	(4a)	(4c)
After death	0.898** [3.48]	0.824** [4.55]	0.904** [3.29]	0.830** [4.54]	0.851** [5.48]	0.796** [5.81]	0.896** [3.98]	0.819** [5.33]
After Death × Co-located at Time of Death	0.984 [0.33]	0.966 [0.53]						
After Death × Within 10 Miles at Time of Death			0.952 [1.05]	0.931 [1.30]				
After Death × At least one coauthorship in last 5 years					1.124* [2.31]	1.075 [1.23]		
After Death × Coauthor is Former Trainee							0.973 [0.47]	0.999 [0.01]
Log Quasi-Likelihood	-531,703	-1,109,928	-531,656	-1,109,885	-531255	-1,109,855	-531,705	-1,109,941
Nb. of Obs.	79,232	191,151	79,232	191,151	79232	191151	79,232	191,151
Nb. of Dyads	3,089	8,111	3,089	8,111	3089	8111	3,089	8,111
Nb. of Superstars	57	160	57	160	57	160	57	160

Conditional dyad fixed effects quasi-MLE estimates for the determinants of JIF-weighted publications among coauthors of academic life sciences superstar academics. Estimates are displayed as incidence rate ratios (exponentiated coefficients). All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. We interact the treatment variable with characteristics of the dyad or coauthor.

†significant at 10%; *significant at 5%; **significant at 1%.

Table 11B: Interactions with NIH Grantee Status and Career Age Difference

	NIH Grantee Satus		Career Age Difference		
	w/o Controls	with Controls	w/o Controls	with Controls	
	(1a)	(1b)	(2a)	(2b)	
After death	0.947 [1.21]	0.862** [2.70]	0.892** [3.77]	0.809** [4.88]	
After Death × Coauthor Holds R01 Grant at Time of Death	0.924† [1.85]	0.924 [1.41]			
After Death × Coauthor is a Junior to the Star			1.014 [0.27]	1.046 [0.77]	
After Death × Coauthor has no other superstar collaborator					
Log Quasi-Likelihood	-531,540	-1,109,843	-531,705	-1,109,917	
Nb. of Obs.	79,232	191,151	79,232	191,151	
Nb. of Dyads	3,089	8,111	3,089	8,111	
Nb. of Superstars	57	160	57	160	

Conditional dyad fixed effects quasi-MLE estimates for the determinants of JIF-weighted publications among coauthors of academic life sciences superstar academics. Estimates are displayed as incidence rate ratios (exponentiated coefficients). All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. We interact the treatment variable with characteristics of the dyad or coauthor.

†significant at 10%; *significant at 5%; **significant at 1%.

Table 11C: Interactions with Relationship Age and Collaborator Age at Time of Death

	old vs. new relationship		Coauthor Old vs. Young at Superstar's Time of Death	
	(1a) w/o Controls	(1b) with Controls	(2a) w/o Controls	(2b) with Controls
After Death × Relationship less than 5 years old	0.990 [0.19]	0.875* [1.96]		
After Death × Relationship b/w 5 and 10 years old	0.870** [3.66]	0.841** [3.54]		
After Death × Relationship b/w 10 and 20 years old	0.831** [5.57]	0.753** [6.75]		
After Death × Relationship more than 20 years old	0.836* [2.44]	0.827* [2.23]		
After Death × Coauthor less than 10 years of career age at TOD			0.761** [4.06]	0.768** [3.28]
After Death × Coauthor b/w 10 and 20 years of career age at TOD			0.864** [3.35]	0.797** [4.45]
After Death × Coauthor b/w 20 and 30 years of career age at TOD			0.921* [2.19]	0.841** [3.97]
After Death × Coauthor more than 30 years of career age at TOD			0.954 [1.07]	0.835** [2.69]
Log Quasi-Likelihood	-530,935	-1,109,671	-531,426	-1,109,885
Nb. of Obs.	79,232	191,151	79,232	191,151
Nb. of Dyads	3,089	8,111	3,089	8,111
Nb. of Superstars	57	160	57	160

Conditional dyad fixed effects quasi-MLE estimates for the determinants of JIF-weighted publications among coauthors of academic life sciences superstar academics. Estimates are displayed as incidence rate ratios (exponentiated coefficients). All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. We interact the treatment variable with characteristics of the dyad or coauthor.

†significant at 10%; *significant at 5%; **significant at 1%.

Table 12: Reality Checks

	Coauthor is At Least 15 Years the Star's Senior		Coauthor is member of the NAS at Time of Death		Superstar Dies Past Age 67	Placebo Death Dates for Control Superstars
	(1a) w/o Controls	(1b) with Controls	(2a) w/o Controls	(2b) with Controls	(3) w/o Controls	(4) Controls Only
After death	0.856 [1.16]	0.718 [1.33]	0.930 [1.17]	0.837 [1.20]	0.973 [0.67]	1.004 [0.32]
Log Quasi-Likelihood	-6,400	-28,752	-30,633	-6,572	-452,598	
Nb. of Obs.	937	4555	2,304	541	76,638	391,507
Nb. of Dyads	38	238	83	19	2,919	16,252
Nb. of Superstars	14	76	31	13	60	232

Conditional dyad fixed effects quasi-MLE estimates for the determinants of JIF-weighted publications among coauthors of academic life sciences superstar academics. Estimates are displayed as incidence rate ratios (exponentiated coefficients). All models incorporate year effects and seven age category indicator variables (career age less than 5 years is the omitted category). Absolute value of robust (QML) z-statistics in brackets, clustered at the level of the superstar. We interact the treatment variable with characteristics of the dyad or coauthor.

†significant at 10%; *significant at 5%; **significant at 1%.

Figure 1: Avoiding Contamination of the Control Sample

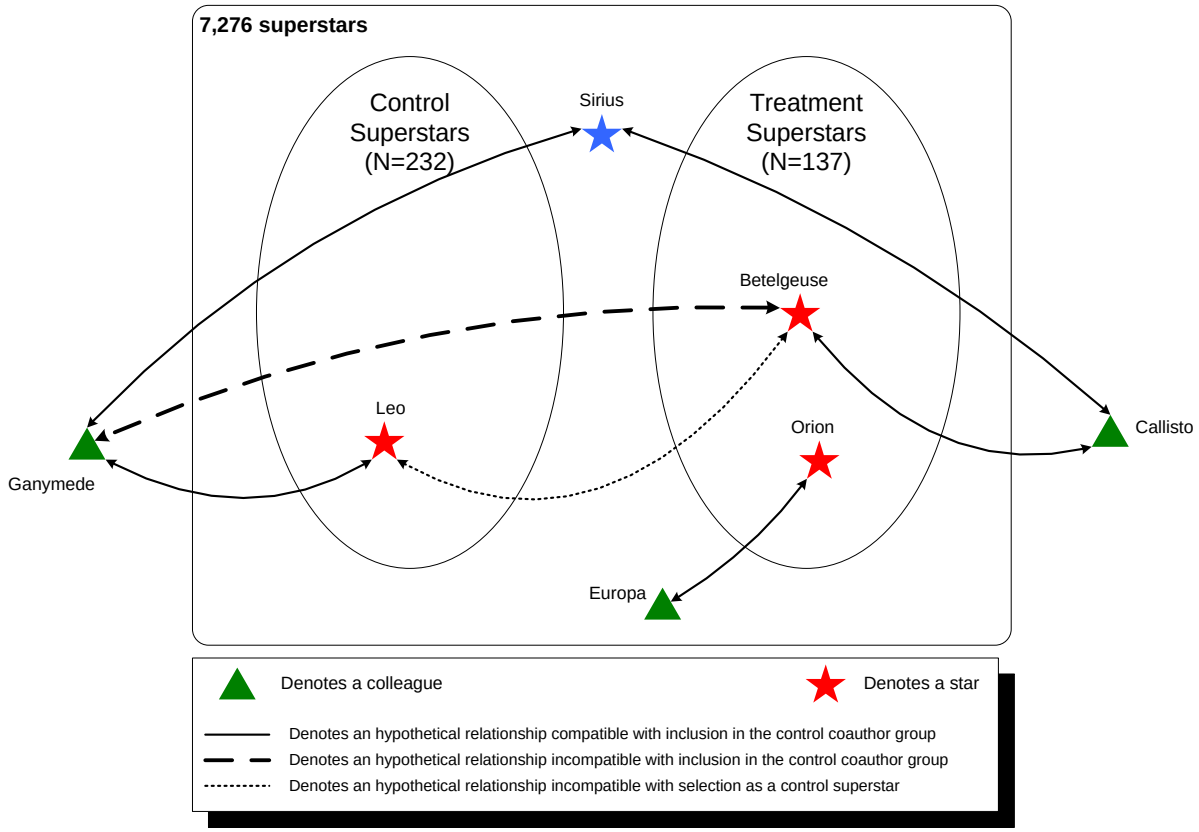


Figure 2: Number of Coauthors per Superstar

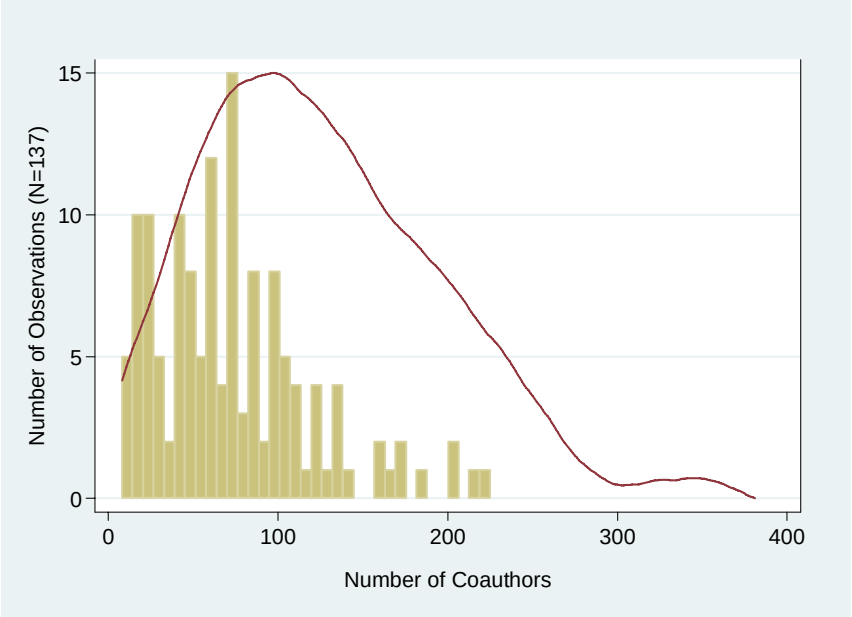


Figure 3: Distribution of Coauthorships at the Superstar/Colleague Level

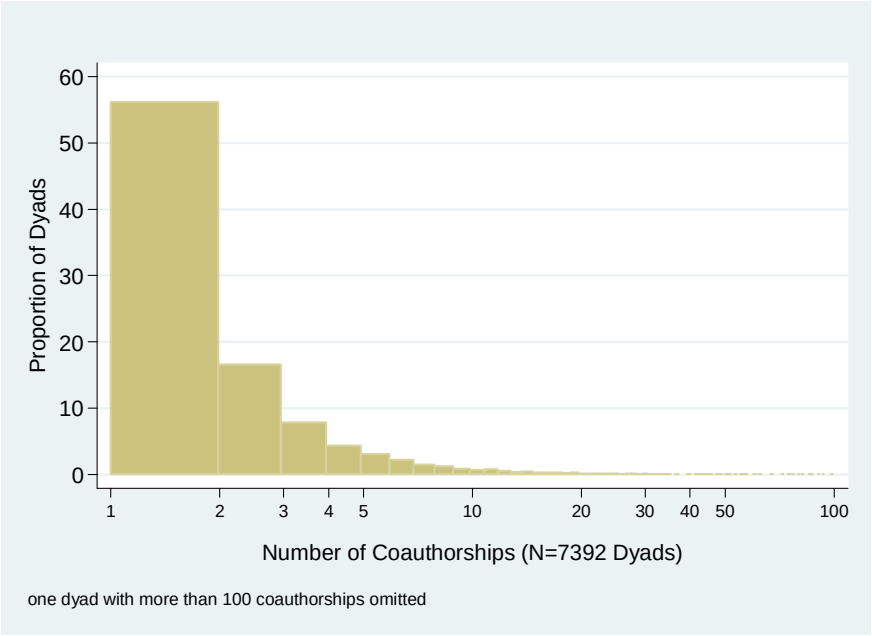
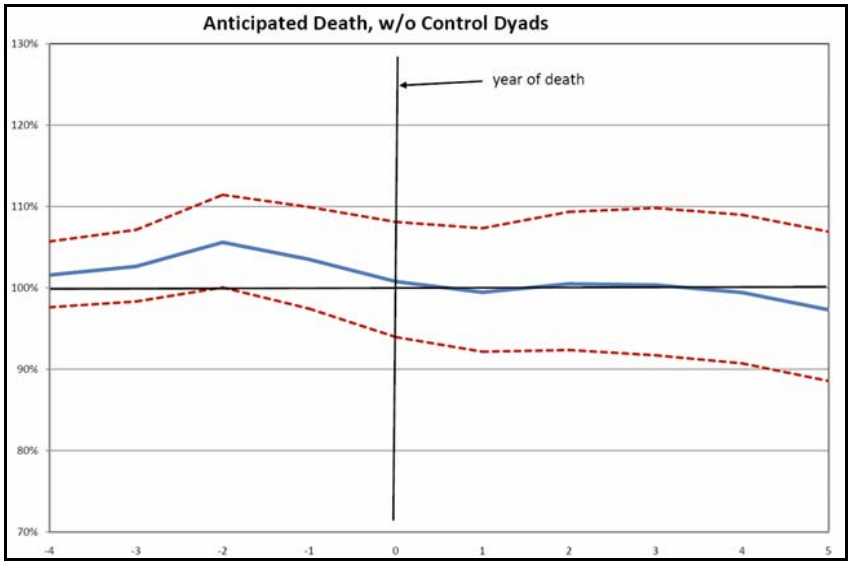
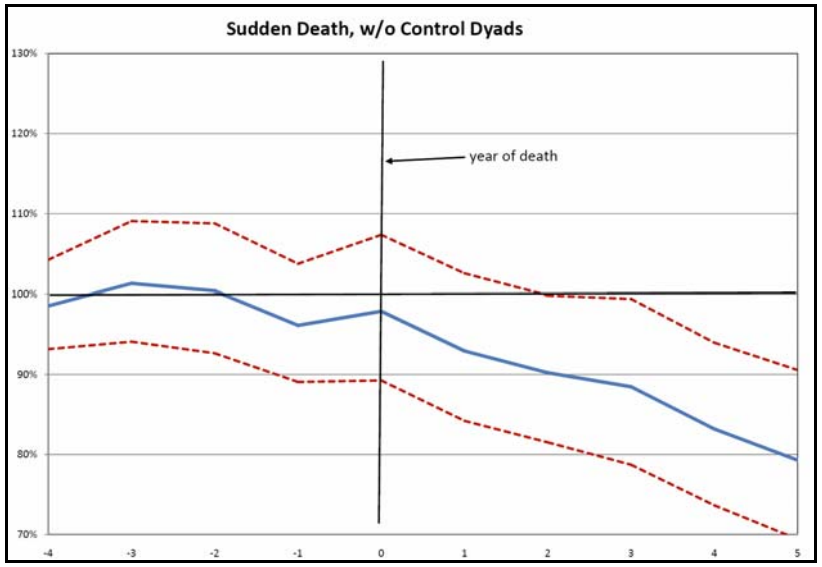


Figure 4: Time plot of coefficient estimates for the treatment effect interacted with years before and after superstar death.





The solid blue lines in the above plots correspond to the coefficient estimates for the incidence rate ratios of a Poisson regression in which the weighted publication output of a colleague with other faculty than the dead superstar is regressed onto year effects, 7 indicator variables corresponding to different age brackets, and interactions of the treatment effect with 11 dummy variables corresponding to 4 years before the year of death, 3 years before the year of death,..., 5 years after the year of death, and 6 years after the year of death and above (not plotted). The 95% confidence interval (corresponding to robust standard errors, clustered around supertsras) around these estimates is plotted with dashed red lines.