Death of the Salesman but Not the Sales Force: How Interested Promotion Skews Scientific Valuation[1]

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Whereas research has demonstrated how social cues appearing as disinterested social validation can skew valuation processes, interested promotion may be at least as important. This factor is examined here via the premature death of 720 elite life scientists. Especially when scientists are young and their articles have received little attention, their deaths stimulate a long-lasting, positive increase in citation rates, relative to trajectories for equivalent articles authored by counterfactual (i.e., still-living) scientists. These patterns seem largely explained by a spike in posthumous recognition efforts by the deceased scientists' associates. The upshot is clear evidence of informational inefficiency, which derives from the challenges of absorbing the massive volume of research produced by the scientific community and from its ambivalence about the norm of disinterestedness.

INTRODUCTION

While it may seem obvious that buyers should not "judge a book by its cover," sellers' promotional efforts continue apace, in the apparently reasonable

[1] All authors contributed equally. Pierre Azoulay is a research associate at the National Bureau of Economic Research and acknowledges the financial support of the National Science Foundation through its SciSIP Program (award SBE-1735413). The results presented in the article leverage data provided by the Association of American Medical Colleges (AAMC). The views expressed herein are those of the authors and do not necessarily reflect the position or policy of the AAMC. The National Institutes of Health partially supports the AAMC Faculty Roster under contract 75N94019C00007, and the authors

0002-9602/2019/12503-0004$10.00

expectation that buyers will struggle and often fail to discount the seller's biases. This struggle is especially salient in meritocratic domains, those governed and justified by strong norms enjoining participants to ignore social and physical cues and instead to assess products and producers purely on the basis of underlying quality. Consider science as the quintessential meritocratic domain, marked by widespread deference to the norms of "universalism" and "disinterestedness" (Merton 1979). Consider too that an important way of judging the health of a scientific field is whether it is informationally efficient: when scientific advances are made (according to the criteria of the field's dominant paradigm, however imperfect it may be), are they recognized as such and does this recognition diffuse quickly? If some scientific papers owe their recognition not to the underlying quality of the work but to the fact that they benefited from more effective promotion, this would defy meritocratic norms and hinder informational efficiency. At the limit, if science were just about who had access to the biggest promotional platform or used it most cleverly, public confidence in science would be misplaced.

The question of whether such *interested promotion* of science limits the efficiency of scientific valuation can be better appreciated in the context of recent research on *disinterested validation* in meritocratic domains (see esp. Salganik, Dodds, and Watts 2006; Simcoe and Waguespack 2011; Azoulay, Stuart, and Wang 2014; van de Rijt 2019). Common to research on this type of social cue are three insights. First, given the widespread challenge of distinguishing higher-quality products and producers as well as the common need to coordinate on the basis of quality (Correll et al. 2017), third parties naturally emerge in meritocratic domains to aggregate and publicize informed assessments of quality (Zuckerman 1999; Espeland and Sauder 2007). Second, even when these assessments are produced in a disinterested manner—for example, by expert panels (Simcoe and Waguespack 2011; Azoulay et al. 2014) or by anonymous peers (Salganik et al. 2006; van de Rijt 2019)—they can skew valuations to produce informational inefficiency. In particular, when recognition is bestowed on one product/producer before it is bestowed on an equivalent one, the former may benefit from a "Matthew effect," whereby the initial validation skews subsequent sampling, evaluation, and investment patterns. Finally, such advantages can be empirically

express their appreciation to Hershel Alexander (AAMC Director of Data Operations and Services) for his support in providing access to the AAMC Faculty Roster data. We thank Angela Lu, Freda Lynn, Cat Turco, and Arnout van de Rijt, as well as seminar audiences at MIT, Columbia University, University of Pennsylvania, and USC Marshall for useful discussions. A replication archive is available online in a zip file. Direct correspondence to Pierre Azoulay, Massachusetts Institute of Technology, Sloan School of Management, 100 Main Street, E62-487, Cambridge, Massachusetts 02142. E-mail: pazoulay@mit.edu

identified via counterfactuals derived from situations in which the same evaluative standards are used but disinterested validation of some products is higher for reasons that are unrelated to quality. This can occur either because (i) an experimenter has subdivided a population into subpopulations, and public quality assessments of the very same products happen in a different sequence in each subpopulation (Salganik et al. 2006; van de Rijt 2019), or (ii) an agent can only validate the quality of a limited number of products, thus entailing that a subset of equivalent products will have the bad fortune of not being validated (Azoulay et al. 2014; Bol, de Vaan, and van de Rijt 2018). Overall, these studies have produced clear evidence of informational inefficiency, although it is hardly overwhelming in its magnitude.

But insofar as disinterested validation of these varieties is distinct from the types of efforts at interested promotion mentioned above, it is unclear whether the latter type of social cue might also skew valuation and produce unfair advantage. The norm of "disinterestedness" enjoins scientists and scientific institutions to sanction scientists for attempting to boost the value of their work for personal gain (Merton 1942, p. 124). Accordingly, self-citations are often eliminated when assessing scientific contributions, as they are thought to be biased. Yet given the overwhelming volume of scientific research that is produced and the career stakes involved in gaining recognition, it is hardly surprising that scientists may be seen promoting their work in a wide variety of ways—on their vitae, on their websites, at academic conferences, in the introductions to their papers, and so on.

Moreover, there is reason to think that such efforts at interested promotion can influence the reception of science despite widespread fealty to the norm of disinterestedness and efforts to enforce it. In short, it is often difficult and even undesirable for scientists to treat interested promotion as biased. In particular, those with the most interest in a given line of work are often regarded as the most knowledgeable and as having the greatest incentive to accurately assess its quality (Li 2017; Teplitskiy et al. 2018). After all, it is generally a worrying sign if producers are not willing to stand by their work. A related consideration is that scientific movements often require a critical mass of contributors to make progress; as such, it is quite natural for scientists to promote work as a way of enlisting additional hands on deck (Botelho 2018). Finally, even if it is reasonable to dismiss a scientist's efforts at self-promotion as irremediably biased, it is more questionable whether one should dismiss efforts by the scientist's colleagues on his behalf. The upshot is that interested promotion generally falls into a normative gray area, making it difficult and often inadvisable to discount for its influence.

One implication of these considerations is to provide another basis for the Matthew effect (Merton 1968), whereby effectiveness in the promotion of

scientific work is increasing in a scientist's status. But it also suggests that we can gain distinctive insight into the efficiency of the scientific valuation process by identifying contingencies that affect the manner and degree to which scientific work is actively promoted. In particular, research on reputational entrepreneurship in political and cultural contexts suggests that the death of a "producer" (i.e., an artist, politician, or scientist) provides a unique window into how shifts in the opportunity structure for interested promotion can have a significant impact on how the producer's work is valued.

This literature identifies two countervailing effects of a producer's death on such opportunities: on the one hand, death prevents the producer from playing the role of "salesman" in publicizing and promoting himself and his products, but on the other hand, the producer's death can influence how other parties play the role of a "sales force" in publicizing and promoting the producer's work (Bromberg and Fine 2002, p. 1139). In some cases, the death of the producer appears to have a negative effect on his legacy by eliminating the salesman. For example, in accounting for why U.S. President Warren Harding is the "worst president of all time" (Holmes and Elder 1989), Fine (1996) notes that Harding was a reasonably popular and effective president during his lifetime; however, his early death in 1923 prevented him from defending his reputation in the wake of the Teapot Dome scandal, while his erstwhile supporters had every incentive to let him take the blame. Yet while the death of the producer can have a negative impact on his legacy, it can paradoxically have a positive effect insofar as it mobilizes a sales force composed of people who were positively influenced by the producer during her lifetime. Thus, Lang and Lang (1988) document how the sudden death of young etchers mobilized friends and family to commemorate the oeuvre of the deceased, thereby making it less likely that the artist would be forgotten by the next generation. Fine (1996) too contrasts Harding's death with John F. Kennedy's, showing that Kennedy's supporters commemorated his life and work to such an extent that he became one of America's most popular presidents after his death, despite a rather brief and controversial term as president.

What then is the impact of scientists' deaths, especially the premature deaths of young scientists, on the valuation of those scientists' work? If the scientific valuation process is highly efficient (in discounting any bias in efforts to promote science), then the death of a scientist should have no impact on the valuation of her work. But if indeed a given scientific community is hard-pressed to absorb the work produced by its members and to discount any bias in promotional activities, contingent shifts in promotional opportunities can make a difference by either reducing recognition for the scientist's work (if what matters most is the scientist's self-promotion efforts) or increasing recognition for her work (if promotional efforts by supporters make the bigger difference).

To preview our findings, our analysis of elite academic life scientists shows that a scientist's death tends to provide a boost to her papers' citation trajectories, and it does so by mobilizing scholars seeking to memorialize the deceased, thereby promoting her work and reputation posthumously. As a result, these scholars' research enjoys greater recognition than that of still-living scientists. We also find that these effects appear to be long-lasting; for up to 10 years after their deaths (a relatively long time relative to the citation half-life of articles in this field), the authors' work continues to be cited more than comparable work by scientists who had not yet died. The effect is not uniformly distributed. It is more pronounced for those who are most memorialized, and consistent with findings by Lang and Lang (1988), such memorialization is disproportionate when the death occurs at a relatively young age. Additionally, it is the scientist's least-cited papers at the time of death that see the largest boost in posthumous citations. Taken together, these findings suggest that the promotional efforts of the sales force are effective in shifting valuations and that the effect occurs because of an attention shift in the context of limited capacity for attending to the massive amount of scientific output.

THEORY

> You have no control:
> Who lives
> Who dies
> Who tells your story?
> —Lin-Manuel Miranda, *Hamilton*

Our article examines the impact of contingent shifts in the opportunity structure for promotion on the informational efficiency of scientific valuation. To clarify the theoretical issues at stake, it is useful to consider what has been accomplished by recent research that examines the effect of contingent shifts in social cues on meritocratic valuation. In short, this research, which has largely been described as testing the Matthew effect (Simcoe and Waguespack 2011; Azoulay et al. 2014) or cumulative advantage (Salganik et al. 2006; Salganik and Watts 2008), has demonstrated that disinterested validation can shape which products/producers are more highly valued (as measured by citations or downloads, in the cases above). However, it is unclear whether interested promotion can have a substantial impact and what specific mechanisms might be responsible. To the extent that the Mertonian norms of universalism and disinterestedness govern science, one would expect scientists and scientific institutions to discount such efforts (Merton 1942). Yet scientific communities may find it difficult and even inadvisable to completely dismiss such promotional efforts, given that they may be reliable signals of quality. This ambivalence may make

interested promotion an effective means of boosting valuations, both by the focal scientist and by his supporters.

Disinterested Validation

A key contribution of recent research is methodological, in that it has shown that the clearest way to demonstrate that social signals shape valuation is through the use of counterfactuals that are identical or observationally equivalent to the focal products/services but do not enjoy the same degree of social validation. For example, the Columbia MusicLab experiment induces alternative popularity trajectories for the very same song, depending on whether it is evaluated in one of several different "social" worlds (in which popularity information is visible, such that songs' initial popularity influences their later popularity) or in an "asocial" world in which popularity information is not given (Salganik et al. 2006; Salganik and Watts 2008). Similarly, Azoulay and colleagues' (2014) study of how the conferral of status on life scientists by a prestigious foundation (the Howard Hughes Medical Institute, or HHMI) affects the citation trajectories of the scientists' previously published papers is based on the premise that near-equivalent scientists (not anointed by HHMI) and papers (as discussed below) may serve as counterfactuals.

It is important to appreciate what this literature has demonstrated to date and what its limitations are. First, this research is focused on informational efficiency rather than allocative efficiency (Stout 1995; Sethi 2010; Zuckerman 2012*b*). Put differently, this research focuses on whether a particular community assigns valuations in a consistent manner as specified by its dominant paradigm but does not address whether the dominant paradigm is in an objective sense "correct." This is most obvious in the case of the MusicLab, as the key question is the extent to which exposure to popularity information alters users' perceptions of what would meet their personal taste (Salganik et al. 2006, p. 854). The same question is also implicitly operative in Simcoe and Waguespack (2011) and Azoulay et al. (2014): although it is possible that the work of both the award winners and the counterfactual (i.e., still-living) scientists will eventually be dismissed as having little value (thus implying allocative inefficiency), this is a separate matter from the informational inefficiency implied when the work of the award winner is valued more highly than equivalent work by lower-status peers. Note that this focus on informational efficiency is consistent with the thrust of science studies since the 1970s (Bloor 1973; Latour and Woolgar 1979; Shapin 1982; Ziman 1983), which have assailed the epistemological premise that scientific valuations can achieve objectivity. Scientific valuation necessarily reflects contingent communal standards, and insofar as those standards are necessarily limited, allocative efficiency is unattainable. But

this begs the question of whether a community applies its standards (however limited) in a consistent way. That is the question of informational efficiency.

Second, each of these studies focuses on disinterested validation. In the case of the MusicLab, the implicit premise is that music fans are limited in their ability to sample the vast universe of songs, so they look to their peers—who are presumed to have similar tastes—to guide them.[2] This guidance is disinterested because it comes as a by-product of these peers' consumption behavior and because the anonymity of the setting ensures that no one has an interest in promoting one song or another. Note also that this guidance is meritocratic in that it is ostensibly based on "the satisfaction of quality standards that can be articulated independently of the options available" (Correll et al. 2017, p. 299). Research on the Matthew effect in science is similar in both these respects. For example, the procedures that govern appointments to coveted Howard Hughes Medical investigatorships are presumed to be both disinterested and meritocratic because of HHMI's institutional mandate to support high-quality research and from the review process's adherence to the norm of universalism.

Third, it is important to consider two notable differences in the mechanisms affecting the informational efficiency of science from those in cultural domains, especially as examined in experiments in which the participants are anonymous and thus indifferent to how their valuations appear to others (see Zuckerman 2012*a*): (i) the prospect of tangible rewards for scientific advances that are independent of the valuation of the academic community and (ii) career-based social pressures in science that make scientists sensitive to their colleagues' opinions. The first point derives from the premise that science is not purely a matter of taste; as such, there are significant rewards available to the scientist who challenges the dominant paradigm and successfully develops or inspires a piece of technology whose value becomes undeniable even to initial skeptics (e.g., polymerase chain reaction, CRISPR gene editing, or angiogenesis inhibitors). The second point derives from the premise that scientists' career outcomes are determined by their fellow scientists, and this can induce significant pressure to conform to the dominant paradigm (it can also induce pressure to differentiate from their colleagues as competitors; Zuckerman 2012*a*). Given these two countervailing effects, one that rewards scientists for challenging convention and the other for adhering to convention, it is unclear ex ante whether the effect of social signals on valuation should be stronger or weaker in science relative to cultural markets. It is instructive then that while the results of recent studies demonstrate that the Matthew effect is real, its magnitude seems relatively

[2] Notably, if they discover that their peers have very different tastes than they do, they tend to reject their guidance, and the social influence effect wanes (Salganik and Watts 2008; van de Rijt 2019).

small (Azoulay et al. 2014), thus implying a relatively low level of informational inefficiency.

Interested Promotion

Yet while this research has made important progress in assessing how social signals affect valuation, its focus on disinterested validation is necessarily limiting. After all, many social signals are conveyed by interested parties, and they too may have a significant impact on valuation. In cultural markets, such efforts are so commonplace as to be obvious: although Billboard may rank songs by market share (the equivalent of the disinterested validation provided in the MusicLab), this in no way deters artists and music labels from promoting their work through the use of advertisements, radio and playlist spots, television appearances, and so on.[3] The prevalence of such promotional efforts is important for present purposes because it implies that market participants do not think that the market is informationally efficient (see Zuckerman 1999, pp. 1430–31). Rather, given the vast number of options available and the search costs associated with sampling them, efforts to gain the attention of consumers seem necessary.[4] And as documented by marketing scholars (Van den Bulte and Lilien 2001), these efforts can pay off, by raising consumer awareness of the focal product or producer along with consumers' perceptions of quality. Although consumers are typically aware that such efforts are biased attempts to sway their consumption behavior, they may be quite effective nonetheless.

But it is an open question whether and to what extent interested promotion may shape social valuation in science, affecting the informational efficiency of a given domain and thus potentially allocative efficiency as well. Insofar as scientific communities are governed by the norm of disinterestedness (Merton 1942), we might expect promotional efforts to be limited. Yet the same conditions that provide an impetus for promotional efforts in other settings—very large number of options and significant search costs—apply in science as well. As such, and given competition for scarce jobs and resources, scientists have good reason to fear that their work will not be noticed, thereby leading them to act as "salesmen" in promoting their work. Such promotion does not stop with the focal scientist herself; scientists often promote the work of others they know and respect. Although such promotional efforts are often presented as being disinterested and they may be less

[3] It is possible—if unlikely—that some of MusicLab's participants had an interest in promoting the bands they favored. To the extent that this was the case, the social cues would be a mix of disinterested validation and interested promotion. The specific contribution of interested promotion efforts would remain unknown, however.

[4] Tucker and Zhang (2011) show that disinterested validation is more influential when there is less information available ex ante about consumption options.

self-interested than those of the salesmen, efforts by friends and colleagues—whom we term "the sales force"—to promote another's work are not disinterested to the same degree as an anonymous ranking system (such as the MusicLab) or a third-party award (such as the HHMI). In particular, there is no comparable mandate or commitment by the promoter to assess a range of potentially meritorious candidates. In addition, the promoter may benefit either from reciprocal arrangements or from the increased status of a shared field (Reschke, Azoulay, and Stuart 2018).

But does (interested) promotion of scientific work significantly shape scientific valuation, and if so, how? Note in this regard Merton's claim regarding the norm of disinterestedness was not that scientists are more moral and therefore less likely to attempt to boost scientific efforts for personal gain; rather, he argued that the institutions of science would be able to check such actions and prevent them from being effective (Merton 1942). Thus, one reason to doubt that interested promotion has a substantial impact is that scientific communities employ various practices—from removing self-citations from citation counts to avoiding advisors and coauthors when requesting journal referees and tenure letters—that are meant to counteract bias.

Yet as noted above, this is just one side of the coin. As with conflicts of interest in other domains, scientists' investment in a subfield or a particular line of work (their own or that of a colleague) actually has ambiguous implications.[5] In particular, someone who is interested in a particular domain may favor that domain, but she may also be more knowledgeable about it and more concerned about vetting the quality in it. Thus, as Li (2017) shows in her study of scientists assessing grants at the National Institutes of Health (NIH), while scientists may be biased in their valuations of quality in a manner that disproportionately benefits themselves and their colleagues, these (interested) scientists are also most accurate in their assessments, as they know more about their own domain and are most concerned about its trajectory. Moreover, given that scientific movements often require the mobilization of many colleagues to embark on complementary research, a natural consequence is that scientists will advertise their work so as to facilitate such mobilization. Indeed, the failure to promote one's work in this fashion could even be interpreted as a negative signal.

The larger implication is that it is ultimately unclear whether and how scientists should discount one another's promotional efforts, as they may be unsure whether such efforts are poor signals of quality due to bias or strong signals of quality due to aligned incentives. As such, there is good reason to expect that interested promotion has a substantial impact on the informational

[5] This debate is common in many other domains outside of science. For instance, there is a long-standing legal precedent for the common law requirement of "legal standing," meaning that parties must have been adversely affected themselves before they can bring a lawsuit forward (see, e.g., *Lujan v. Defenders of Wildlife*, 504 U.S. 555 [1992]).

efficiency of scientific communities. In particular, the general implication is that *scientific work that benefits from more effective promotional efforts is more highly valued than equivalent work that does not benefit from the same level and type of promotion*. A further implication is that *if for whatever reason, a work of science benefits from extra promotion that is ostensibly unbiased, it should have an even greater impact than work that receives the same level of promotion but is perceived as biased*.

Scientist's Death as a Window into the Importance of Promotion

In order to assess these implications, we examine contingent shifts in opportunities for promoting science occasioned by the premature death of scientists. Past research has demonstrated that a scientist's death can be effectively used to study a given scientist's impact on the production of science (Azoulay, Graff Zivin, and Wang 2010; Oettl 2012; Azoulay, Fons-Rosen, and Graff Zivin 2019). And as discussed above, research on reputational entrepreneurship in cultural and political domains suggests that we can make progress on the larger question of the impact of interested promotion on the efficiency of scientific valuation by examining how appreciation for a scientist's published work changes as a result of his death. Since the quality of such work (which was published in the past) is obviously unaffected by the death of its author, it should have no impact on how it is valued, as measured by the trajectory of citations to that paper.[6] More specifically, to the extent that promotional efforts are biased and the scientific community successfully discounts for such biases, any effect of changes in promotional efforts due to the death should be negligible.

We have noted, however, why it is unlikely that such biases are fully discounted. And the literature on reputational entrepreneurship in cultural and political domains implies two pathways by which the death of a producer can affect how his work is valued based on how the death affects promotional activity. One possibility, as reflected in Fine's (1996) study of Warren Harding discussed above, is that the valuation of scientific works will fall after the author's death. Scientists who believe their research is undervalued by the community may seek to raise awareness of it through press releases, teaching graduate courses, presenting at conferences, and so on. This implies that at any given point in time, the level of citations a paper

[6] Citations are necessarily a measure of attention (Merton 1988) but an imperfect measure of communal valuation given that some citations are negative. However, recent research (Catalini, Lacetera, and Oettl 2015) on a subfield (immunology) within the larger domain studied here finds that only 2.4% of the total citations have a negative valence. A more subtle issue is that citations may not reflect the citer's personal assessment of quality but rather the assessment of quality she thinks coordinates well with journal referees and readers (see Correll et al. 2017). We will return to this issue in the discussion.

receives is a function of the quality of the paper (according to the dominant paradigm) and the amount of "salesmanship" it has received. Thus, since the death of the scientist eliminates the latter factor, the number of citations should decline.

Second, as in the case of John F. Kennedy above, insofar as the death of a scientist leads scientists' supporters to "memorialize" their deaths, it may generate an increase in the valuation of her work. Lang and Lang's study of etchers provides intriguing evidence for how death can spur supporters to initiate celebrations of the artists' life and work via "recognition events"—biographies, news articles, and exhibits of their life and oeuvre (1988, p. 94). To be sure, recognitions of a producer's entire oeuvre often occur while she is still alive—a Festschrift is a common form of such recognition for scholars—but recognition events seem more common in the aftermath of the producer's death. In Lang and Lang's research, such events directed the etching field's attention to the work of the deceased, thereby raising its perceived value to such an extent that memorialized etchers were remembered vastly beyond their living counterparts, even those who did superior work (pp. 93–94).

Importantly, Lang and Lang report that such memorialization was most impactful when the artist died at a young age. Lang and Lang's (1988, pp. 93–94) example of Elizabeth Fyfe is emblematic:

> Fyfe, who died in Switzerland in 1933, just after her thirty-fourth birthday after a long bout with tuberculosis, had been hailed by British critics as "one of the most original and accomplished young etchers." That her name and her work, which amounted to just over 1,600 impressions, somehow survive, whereas those of others once equally or better known do not, has much to do with her premature death. Her teachers, her friends, her collectors, and other etchers rallied, while she was in the hospital, to organize an exhibition of her work, complete with catalog, and then used the proceeds from sales to help pay for the care she needed. Her dealer saw to it that her plates were printed when she could no longer do so herself and gave a full set of her prints to Fyfe's sister. In this way, the many persons mobilized by the tragedy helped to preserve the work and, thereby, to sustain the memory of the artist.

An important factor noted here—the preservation of the artist's otherwise perishable work—seems to apply to art but not to science. At the same time, science seems comparable to art and politics in that recognition events will be relatively rare for the young if they remain alive. Note further that young producers in a given domain tend to have more living supporters than those who die at an advanced age. Moreover, the deaths of those in the prime of their career are surprising and more likely to be experienced as tragic; as such, they may be more likely to mobilize a community that is keen to ensure that the scientist's work not be forgotten. However well intentioned, such collective efforts at interested promotion have the potential to provide an ironic benefit to the dead scientist's work via a boost in positive attention

as compared with equivalent scientists who have the good fortune to remain alive.

Empirical Implications

Thus, the death of a scientist implies a contingent shift in opportunities for interested promotion. As such, it provides a lens through which we can examine how the informational efficiency of a scientific field is affected by interested promotion. If a given scientific field quickly and fully incorporates new advances (according to the criteria of its dominant paradigm), this would imply that the timing of the deaths of authors should not matter for how their research is valued, as measured by citation trajectories. But if such incorporation is incomplete and the field is not able to discount for any bias produced by interested promotion, such promotion—as elicited by scientists—can shift the level of appreciation for their work in one or both of two ways.

In particular, there are four possible ways that the valuation of a scientist's work may be affected by her death. One possibility is that any shift in interested promotion has no impact, and scientific valuation is informationally efficient in this respect. The three other scenarios reflect some degree of informational inefficiency, whereby efforts at interested promotion are not fully discounted. Thus, a second possibility is that scientific valuations are significantly sustained by the efforts of the scientist himself; this would imply that the death of the salesman causes a decrease in citations to the scientist's papers. A third possibility is that scientific valuations are significantly sustained by the efforts of a scientist's supporters, and if the death of a scientist catalyzes the mobilization of this sales force, a boost in citations will ensue. Finally, it is possible that both channels have significant impact but cancel each other out. As long as either the underlying salesman or sales force effect can be identified, such an indeterminate outcome might still imply a significant degree of informational inefficiency in the field.

There is no strong theoretical basis for predicting which of these scenarios is most likely. At the same time, the first possibility seems unlikely. In general, informational efficiency in a domain requires effective tools for arbitrage (or "valuation opportunism"; see Zuckerman 2012*b*) whereby someone who recognizes a gap between quality and social valuation can profit from this gap even when others do not recognize it. But while such mechanisms do exist in various scientific fields (e.g., scientific contributions can be turned into technologies whose value is so apparent they cannot be denied), they tend to be relatively weak. More specifically, and as reviewed above, it seems unlikely that scientific fields are able to fully discount for biases that might be incorporated in efforts at interested promotion.

At the same time, it is not clear whether the mobilization of the sales force should overcome the absence of the salesman. On the one hand, the salesman has the most incentive to promote his own work and, therefore, is likely to do the most promoting. On the other hand, the efficacy of such efforts may be limited by the fact that the scientist is only one person, and his motives are transparently self-interested. As noted above, a key implication of our theoretical framework is that interested promotion should be more effective when those interests do not connote bias. Moreover, conditional on mobilization, the number of individuals in the sales force can potentially be much larger than a single scientist, and, as noted above, their efforts are unlikely to be viewed as entirely self-interested. These factors may be responsible for the evidence of the importance of posthumous "sales force" activity documented in the literature on reputational entrepreneurship (Lang and Lang 1988; Fine 2003). And yet we have noted that an important factor in such studies but absent from science is the role of the sales force in preserving a producer's work. As such, we make no prediction as to which of the three other scenarios is most likely. Rather, our goal is to leverage our analysis to make progress in understanding whether interested promotion skews valuations and which channel is most important in doing so.

Our goal of learning about the relative importance of different channels for interested promotion is furthered by two more specific goals: (i) to assess the importance of key contingency factors that might alter the balance of the salesman and sales force effects and (ii) to examine whether the sales force effect indeed works via a spike in recognition events for dead versus still-living scientists. With respect to the first of these goals, four contextual factors seem especially important. First, as reviewed above, there is reason to think that the sales force effect will be especially strong for the young, with the key reason being that these scientists would have received much less recognition had they remained alive. Second, variation in the "engagement style" of the scientist may have an important impact on either the salesman or sales force effects. For example, scientists who tend to work with large research teams (coauthors, trainees) may be expected to have larger (posthumous) sales forces. Also of interest is whether scientists were highly self-promotional while they were alive. On the one hand, they may be dynamic personalities whose death catalyzes their colleagues to promote their deceased cohorts' work in their stead. On the other hand, such scientists may be regarded as self-serving and be relatively ineffective at eliciting a posthumous sales force. Third, it will be instructive to examine how the shift in interested promotion affects scientists' papers on the basis of their baseline citation level before their death. If a scientist's most-cited papers earn the biggest citation boost from a scientist's death, this will imply that a version of the Matthew effect is at work whereby interested promotion is most effective in combination with other forms of validation. But if a scientist's

least-cited papers gain the most, this will imply a more narrow form of inefficiency, whereby a deceased scientist's papers compete with another's for scarce attention, with some losing out simply because of such scarcity. A spotlight on a scientist's work will then increase the likelihood that overlooked work will now get its due (Tucker and Zhang 2011). Finally, we will examine not only changes in the number of citations to papers of the deceased versus the still living but also changes in who the citers are (collaborators vs. noncollaborators, in the same field vs. outsiders, working for the same institution or not, etc.).

With respect to the second goal, it will be important to examine not only the effect of death on citations to a scientist's work but also the causes and effects of recognition events. Insofar as a scientist's death indeed elicits a positive boost in the valuation of his papers, it may not be due to promotion by the sales force. For instance, it is possible that competitors of the scientist who were stingy in their citations before now become more generous.[7] As such, it will be important to examine (i) whether indeed the death of a scientist elicits more recognition events than if he had remained alive and (ii) whether such recognition activity is responsible for any observed sales force effect on citations.

DATA AND EMPIRICAL DESIGN

The design of our empirical analysis unfolds in three separate steps. The first step is a causal analysis: we examine how the premature death of an eminent biomedical academic researcher changes the rate of citations to her work, compared to the work of other eminent researchers who do not die prematurely. The level of analysis for this step is an article/scientist pair, and the main challenge to be overcome is the building of a control group of articles that plausibly pin down the citation trajectories of the deceased scientists' articles had they remained alive. In the second step, we examine whether recognition activity is greater for deceased scientists, controlling for a host of important correlates of individual recognition; the level of analysis is the individual scientist, and the key challenge is the measurement of the recognition process, which is highly variegated and would, at first blush, appear to defy efforts at quantitative reduction. The third and final step ties the earlier analyses together. We ask whether the recognition process is a plausible mechanism through which scientific work gets remembered in the long run. The main challenge is one of prediction: for each article, we must be able to forecast the citation trajectory that would have been observed if the scientist had remained alive, so as to isolate a net citation

[7] Conversely, it is possible that evidence for the salesman effect is in fact evidence for diminished motivation to engage in strategic citation of the now-deceased scientist.

premium (or deficit) for this article. With these forecasts in hand, we can then examine whether variation in recognition intensity mediates the relationship between death and posthumous citations.

Below, we provide a detailed description of the process through which we assembled the data set used in the statistical analysis. We begin by describing the criteria used to select the sample of elite academics, with a particular focus on the timing and the manner of their deaths. The focus then shifts to the publications that deceased and still-living scientists authored during their lifetimes and how one might build a matched sample of publication/scientist pairs in which the citations received by articles authored by still-living scientists offer a plausible counterfactual to the citations that articles authored by deceased scientists would have received had they not died prematurely. Finally, we document how we measured the recognition process for each individual scientist. Throughout this description of the data, we outline how the construction of the sample addresses the empirical design challenges enumerated above.

Institutional Context

Our empirical setting is the academic life sciences. We focus on this domain for three reasons. The first is its sheer size: U.S. medical schools employ over 150,000 faculty members, and this figure underestimates the size of the labor market since it does not take into account scientists and engineers working at NIH, in nonprofit research organizations (such as the Salk Institute), for independent hospitals (such as the Cleveland Clinic), or within schools of arts and sciences (such as MIT; University of California, Berkeley; or Rockefeller University). Academic biomedical research also garners over 70% of all nondefense federal research and development dollars. The large size of the labor market is important for reasons of statistical power: our key source of variation is generated by the premature death of eminent scientists, and these events are relatively rare. Importantly, the members of this labor market share broadly similar norms, career goals, and incentives and operate within comparable institutional structures.

Second, scientific discoveries over the past half century have greatly expanded the knowledge frontier in the life sciences, and these advances have resulted in more specialization, as well as an increase in the size of collaborative teams (Wuchty, Jones, and Uzzi 2007). These trends help ensure that career shocks affect only relatively narrow swathes of the intellectual landscape. Were our research domain less balkanized across narrow subfields, it would be challenging for us to identify control articles or control scientists (Azoulay et al. 2019).

Third, and perhaps more pragmatically, our setting is blessed by an abundance of data sources. The careers of eminent, still-living life scientists are

extensively described in curriculum vitae, Who's Who profiles, or laboratory websites. We combine these data with the free and publicly available bibliographic database PubMed, citation information from the Web of Science, and administrative records from the Faculty Roster of the AAMC and NIH's Compound Grant Applicant File (CGAF). Together, these sources of information allow us to create an accurate longitudinal record of publications, citations, and funding for each scientist in the sample.

Our focus on the scientific elite is substantively justified in light of our goals. One would expect the articles of eminent scientists to be identified and evaluated immediately after their publication, relative to the articles authored by scientists of lesser repute. This should in turn lessen the relevance of interested promotion in influencing how science is valued. To some extent, this is testable since our metrics of eminence exhibit substantial heterogeneity even within our sample of eminent scientists. That said, this approach has limitations as well, which we discuss after presenting our findings.

Sample of Elite Academic Life Scientists

Following Azoulay et al. (2010, 2019), we begin by demarcating a set of 12,426 "elite" life scientists (roughly 5% of the entire relevant labor market) who are so classified if they satisfy at least one of the following criteria for cumulative scientific achievement: (i) highly funded scientists, (ii) highly cited scientists, (iii) top patenters, or (iv) members of the National Academy of Sciences (NAS) and the National Academy of Medicine. Because these four measures rely on achievements over the course of a scientist's career, they will tend to select older scientists. To create more demographic balance, we add three additional measures that capture individuals with promise at the early and middle stages of their scientific careers (regardless of whether that success endures): (v) NIH Method to Extend Research in Time awardees, (vi) Howard Hughes Medical Investigators, and (vii) early career prize winners. Online appendix A (apps. A–F are available online) provides additional details regarding these seven metrics of "stardom."

We trace back these scientists' careers from the time they obtained their first position as independent investigators (typically after a postdoctoral fellowship) until 2006. We do so through a combination of curriculum vitae, NIH biosketches, Who's Who profiles, accolades/obituaries in medical journals, NAS biographical memoirs, and Google searches. For each one of these individuals, we record employment history, degree held, date of degree, gender, and department affiliations, as well as a complete list of publications, patents, and NIH funding obtained in each year.[8]

[8] Online app. B details the steps taken to ensure that the list of publications is complete and accurate, even in the case of stars with common last names.

The next step in the sample construction process is to select a subset of scientists from this overall pool whose premature death will "treat" their past output. First, we select scientists whose death occurs between 1969 and 2003.[9] Second, we need to ensure that these scientists had not entered a preretirement phase of their career. This is trickier, because the timing of retirement is endogenous, and scientists who do not wish to retire can show great initiative in subverting rules surrounding mandatory retirement (which was legal in the United States until 1986). To overcome this challenge, we make full use of the narrative data contained in the dossiers we compiled for each scientist (deceased or not); we also examine publication output as well as funding received to remove from the sample those who either "meaningfully" retired or whose output shows sign of abating before their death or the end of the observation period.[10]

As a result of these steps, we identify 720 "treated" scientists (see table 1). The mean and median age at death is approximately 64, with the youngest scientist dying at age 33 and the oldest dying at age 91.[11] We then investigate the cause of death in this sample to classify their deaths as being either "sudden" or "anticipated." The main motivation here is to better identify when the sales force of those motivated to memorialize the scientist and his work would have become mobilized; insofar as the death is anticipated, this mobilization could begin before death. Distinguishing anticipated from sudden deaths is less difficult than it appears, since most obituaries typically are quite specific in this respect.[12] To distinguish sudden from anticipated deaths, we use an arbitrary distinction between deaths that likely occurred with six months' notice or less versus those that likely occurred with more than six months' notice. In practice, this "sudden" category mostly comprises fatalities due to heart attacks, car accidents, and sudden-onset illnesses. Conversely, most "anticipated" deaths are from various forms of cancer or other long-term illnesses. In the deceased scientist sample, 330 (46%) scientists died suddenly, while 352 (49%) died from an anticipated illness.

[9] An implication of this design choice is that even for the scientists who die "late" (e.g., in 2003), we will have at least three years of citation data to pin down how their passing changes the recognition of their work.

[10] In previous work, one of us has verified that it is essentially impossible to predict death in a related sample using measures of lagged publication output (Azoulay et al. 2010).

[11] How can one die at a very advanced age yet one's passing still be deemed "premature"? Easily, as it turns out. Aubrey Gorbman (1914–2003), described in academic obituaries as the "father" of the field of comparative endocrinology, succumbed to Parkinson's disease but still published two first-authored articles in the last year of his life.

[12] In some instances, when the cause of death could not be ascertained from the obituaries, we contacted former collaborators individually to clarify the circumstances of the superstar's passing. We were unable to ascertain the cause of death for 38 (5.28%) of the 720 deceased scientists. Some of these cases may have been suicides given the cultural taboo on publicizing suicide over much of this period.

TABLE 1
Summary Statistics of Deceased Scientists

	Mean	Median	SD	Min	Max
Year of birth	1926.48	1927	12.27	1893	1960
Degree year	1953.55	1954	12.94	1920	1988
Death year	1990.15	1991	9.30	1969	2003
Age at death	63.66	64	10.56	33	91
Female	.09	0	.28	0	1
MD degree	.46	0	.50	0	1
PhD degree	.45	0	.50	0	1
MD/PhD degree	.09	0	.29	0	1
Death was sudden	.46	0	.50	0	1
Death was anticipated	.49	0	.50	0	1
Unknown cause of death	.05	0	.22	0	1
Cumulative no. of publications	126	102	105	10	1,380
Cumulative no. of citations	7,228	4,624	8,088	77	76,231
Cumulative amount of NIH funding	16,601,680	10,742,377	25,919,386	0	329,968,960
Cumulative no. of trainees at death	5	3	6	0	44
Cumulative no. of coauthors at death	73	54	70	0	714
Cumulative no. of posthumous predicted citations	606	284	841	2	7,646
Cumulative no. of posthumous "excess" citations	−34	−34	443	−2,459	3,582
Memorialization events:					
Total no. memory events	4.38	3	4.84	0	65
Total no. academic memory events	2.33	1	3.20	0	30
New York Times obituary	.33	0	.48	0	3
Wikipedia page	.25	0	.44	0	1
Named award	.22	0	.42	0	1
Festschrift	.08	0	.28	0	1

Note.—Sample consists of 720 elite academic life scientists who died while still actively engaged in research. See online app. A for more details on the sample construction. National Institutes of Health (NIH) funding amounts have been deflated by the biomedical R&D Producer Price Index (base year = 2007). Forty-five (6.25%) of the deceased scientists are NIH intramural scientists and therefore not eligible for extramural NIH funding. The funding totals are computed for the 720 − 45 = 675 scientists eligible to receive NIH awards.

Table 1 provides descriptive statistics for this sample (see online app. F for a complete list of these individuals, along with basic demographic information, institutional affiliation, and a brief description of their scientific domain). The overwhelming majority (91%) are men.[13] Of note is the fact that even within this sample, substantial variation in status exists: whether one

[13] Per our tabulations of AAMC Faculty Roster data, the patterns of entry into this labor market have only recently equalized across genders, and our sample reflects the extreme gender imbalance that prevailed for most of the time period we study.

measures eminence through publications, NIH funding, or citations (excluding those citations that accrue after the scientist has passed), the mean is always much higher than the median.

Difference-in-Differences Estimation Framework

The death shock that provides the essential lever for our research design occurs at the level of the individual scientist. Similarly, recognition and memorialization efforts typically focus not on particular articles but on the overall body of work of a scientist. And yet, our research design focuses on studying changes in citations to discrete academic publications in the wake of their authors' passing, rather than changes in the flow of citations aggregated up to the scientist level of analysis.

We justify this crucial design choice as follows. Substantively, the norm of universalism emphasized by Merton as a hallmark of the scientific incentive system assumes that the identity of a scientific producer can be unbundled from her published works, at least in principle. The level of analysis in the first part of our study takes this distinction seriously. From an empirical standpoint, the scientist level of analysis is not well suited to the challenge of identifying the causal effect of death on the reception of an academic's work. The content of every scientific contribution remains fixed after it is published, and only the way it is understood, celebrated, or denigrated can change over time. In contrast, the dynamics of the flow of citations received by an individual scientist reflects both increments of recognition accruing to past work as well as additional recognition enabled by new resources (e.g., funding, disciples) secured as a by-product of the reception of past work. The article level of analysis enables us to filter out the effect of the second source of variation, by anchoring the design around a very natural datum that determines unambiguously a "before" and an "after" period for each article: the timing of its author's death.[14]

However, a simple difference between citations that accrue to a paper after, rather than before, the time of its author's death is not enough to yield estimates with a plausibly causal interpretation of the effect of a scientist's passing. This is because the memory of any article (or scientist) must eventually fade. Examine (in fig. 1*A*) the mean number of annual citations received by the 720 deceased scientists, both before and after the death. The

[14] This approach is not new (Farys and Wolbring 2017). For instance, Murray and Stern (2007) ask how citations to articles shift once the underlying results appear in a patent; Azoulay et al. (2014) ask how the receipt of an accolade changes the citation trajectories of articles that appeared before the accolade was received; Azoulay, Graff Zivin, and Sampat (2012) investigate how the mix of local to nonlocal citations changes after a scientist moves to a geographically distant institution.

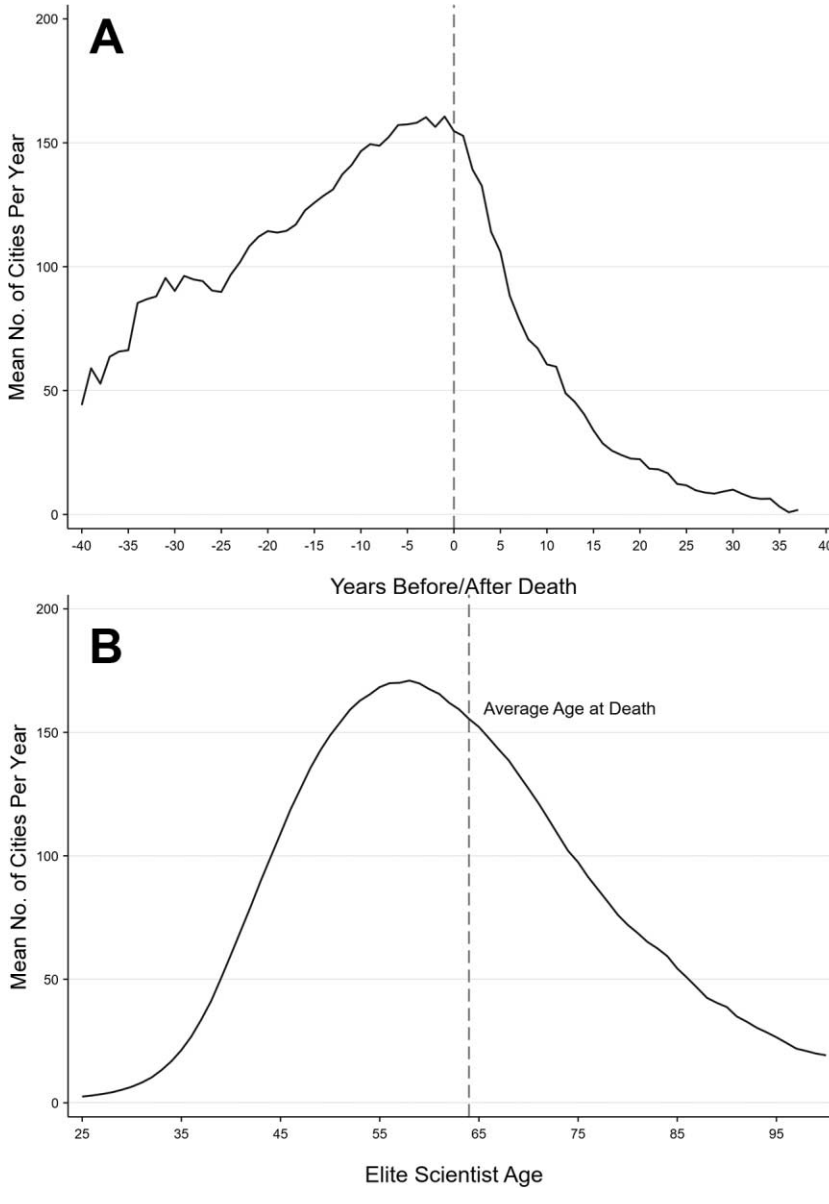


FIG. 1.—Citation life cycle for elite scientists. *A*, Total number of citations accrued per year by each of the 720 deceased scientists in the sample, within a window of ±40 years around their death. *B*, Number of citations accrued by each of the 8,326 still-living scientists who contribute at least one publication to the sample of control articles. Dashed vertical line indicates the average age at death for the treated sample, approximately 64 years old.

curve has an inverted U shape with a peak in the year before death, followed by an inexorable and steep decline, although it will take close to 40 years for the memory of any work by a deceased scientist to disappear from the scientific literature. Figure 1*B* produces a similar graph for the subset of still-living scientists who contribute articles to our control group (in a manner made precise below). In this case, we use their calendar birth age to display graphically the citation life cycle. The vertical dashed line age at 64 corresponds to the mean age at death in the deceased sample. There too, the flow of citations declines inexorably starting in a scientist's late 50s, but that decline is much more gradual than what is observed for scientists who died prematurely. Therefore, the question for our study is not whether the recognition given to the work of deceased scientists will decrease after they die, as it surely will. Rather, the challenge is to assess this decline relative to the citation trajectory of articles whose recognition potential was similar at the time of the scientist's passing. To do so, we need to construct a control group of articles that can plausibly capture this counterfactual.

Matched Sample of Articles

As in Azoulay et al. (2019), our approach is to identify control articles from the vast set of articles authored by elite scientists who did not die prematurely. For each article by a deceased scientist, we attempt to find at least one article by still-living scientists to pair it with. Although this step necessarily entails some degree of judgment, in order to yield valid comparisons, the matching procedure must meet a number of requirements. Notably, to contrast citation flows after the death shock, relative to before, we must be able to assign a counterfactual date of death to each control article as well as a counterfactual eminent scientist who could have died but did not. Pairing treated and control articles appropriately is therefore essential, since the control article will inherit certain characteristics from its matched treated article.

In particular, we require that each control article (i) be published contemporaneously with (and have a similar number of authors as) the article by a deceased scientist with which it is paired, (ii) be unrelated (in both an intellectual and a social sense) to the treated article with which it is paired, and (iii) have an author in last-authorship position who is a still-living elite scientist of approximately the same age as that of the deceased scientist on the article with which it is paired. The focus on the last-authorship position is a solution to the problem that modern science is a team sport, with steadily increasing rates of coauthorship over the past 40 years (Wuchty et al. 2007). Here, we are helped by a strong norm in biomedical research that invariably puts the principal investigator (PI) on a research project in last-authorship

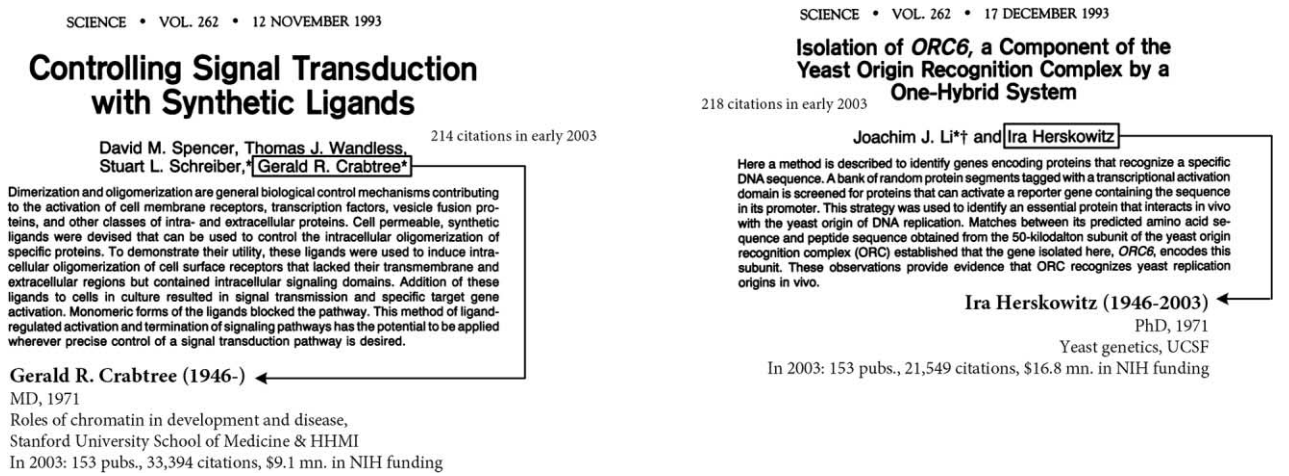
position on any paper that results from the funding she or he was able to mobilize (Nagaoka and Owan 2014).[15]

In addition, it is important that the control group of articles as a whole be broadly similar to the treated group of articles, where similarity should be understood as reflecting average balance across key covariates at baseline. Although it is impossible to identify for each treated article a "fraternal twin" that matches it exactly on an exhaustive list of author and article characteristics, it is possible to select article controls in a way that will make the control group as a whole similar to the treated group in terms of expected impact and scientific "fruitfulness" at the time of the scientist's death. Pragmatically, we specify a handful of covariates along which matched treated/control articles must resemble each other, and we implement a blocking procedure—described in detail in online appendix C—to identify all the articles among those published by still-living scientists that satisfy these criteria (so that each treated article can and typically does have more than one associated control article). Since judgment is required to choose the list of "blocking" covariates, online appendix C also provides two alternative matching schemes and probes the robustness of the core results when selecting one of these alternatives. Reassuringly, the main conclusions are robust to these variations. Our chosen approach yields a higher proportion of articles by deceased scientists with at least one match within the set of articles by still-living scientists. This has two benefits. First, the external validity of our findings is enhanced. Second, the larger sample size gives us more statistical power to detect heterogeneous effects by type of scientist or article.

Treated/Control Article Pair: An Example

Consider the paper "Isolation of *ORC6*" published in the journal *Science* in 1993 originating from the laboratory of Ira Herskowitz, an eminent University of California, San Francisco, geneticist who died in 2003 from pancreatic cancer. We match 34 publications to this article, also published in *Science* in 1993, on which a still-living star scientist occupies the last-authorship position. Figure 2 illustrates the matching with one of these articles, "Controlling Signal Transduction with Synthetic Ligands," which came out of the laboratory of Gerald Crabtree, a Stanford pathologist who studied the role of chromatin in development and disease. By the end of 2002, the Crabtree

[15] To be sure, a scientist can have a deep imprint on a research project and yet occupy authorship position other than last. In the case of interlab collaboration, for instance, it is not unusual to observe one of the PIs occupy the first-authorship position or the next-to-last position. What is important for our purposes is that it is difficult to imagine circumstances where an author does occupy the last author position and she or he is not closely identified with the work.



SCIENCE • VOL. 262 • 12 NOVEMBER 1993

Controlling Signal Transduction with Synthetic Ligands

214 citations in early 2003

David M. Spencer, Thomas J. Wandless, Stuart L. Schreiber,* Gerald R. Crabtree*

Dimerization and oligomerization are general biological control mechanisms contributing to the activation of cell membrane receptors, transcription factors, vesicle fusion proteins, and other classes of intra- and extracellular proteins. Cell permeable, synthetic ligands were devised that can be used to control the intracellular oligomerization of specific proteins. To demonstrate their utility, these ligands were used to induce intracellular oligomerization of cell surface receptors that lacked their transmembrane and extracellular regions but contained intracellular signaling domains. Addition of these ligands to cells in culture resulted in signal transmission and specific target gene activation. Monomeric forms of the ligands blocked the pathway. This method of ligand-regulated activation and termination of signaling pathways has the potential to be applied wherever precise control of a signal transduction pathway is desired.

Gerald R. Crabtree (1946-)
MD, 1971
Roles of chromatin in development and disease,
Stanford University School of Medicine & HHMI
In 2003: 153 pubs., 33,394 citations, $9.1 mn. in NIH funding

SCIENCE • VOL. 262 • 17 DECEMBER 1993

Isolation of *ORC6*, a Component of the Yeast Origin Recognition Complex by a One-Hybrid System

218 citations in early 2003

Joachim J. Li*† and Ira Herskowitz

Here a method is described to identify genes encoding proteins that recognize a specific DNA sequence. A bank of random protein segments tagged with a transcriptional activation domain is screened for proteins that can activate a reporter gene containing the sequence in its promoter. This strategy was used to identify an essential protein that interacts in vivo with the yeast origin of DNA replication. Matches between its predicted amino acid sequence and peptide sequence obtained from the 50-kilodalton subunit of the yeast origin recognition complex (ORC) established that the gene isolated here, *ORC6*, encodes this subunit. These observations provide evidence that ORC recognizes yeast replication origins in vivo.

Ira Herskowitz (1946-2003)
PhD, 1971
Yeast genetics, UCSF
In 2003: 153 pubs., 21,549 citations, $16.8 mn. in NIH funding

FIG. 2.—Matching procedure to identify treatment and control articles. The two articles, which appeared in the journal *Science* in 1993, help illustrate the matching procedure (online app. C provides more details). Note that Ira Herskowitz and Gerald Crabtree are both in the last-authorship position. They obtained their highest degree in the same year. This procedure led the Li and Herskowitz article to be matched with 34 other articles in addition to the Spencer et al. article. Note that the articles are in very different subfields of the life sciences. Formally, the Li and Herskowitz article is not in the list of PubMed Related Citation Algorithm neighbors for the Spencer et al. article (and vice versa).

paper had garnered 214 citations, relatively close to the 218 citations that had accrued to the Li and Herskowitz paper—both articles belong to the top percentile of the 2002 citation distribution for the universe of papers published in 1993. Notice as well that Crabtree and Herskowitz were born in the same year (1946) and received their highest degree in the same year (1971). This is not happenstance, as the matching procedure requires that the career age (years since the highest degree was earned) of the treated and control elite scientists be no more than two years apart.

Yet there are still observable differences between this pair of articles and their authors. The two PIs do not match particularly closely on all metrics of cumulative achievement, for example. This is less of a concern than might appear at first blush, since as will be described below, we have found that imposing balance on article-level characteristics yields, as a fortunate by-product, approximate balance on scientist-level characteristics as well.

Two additional facts about this pair of articles are worth mentioning since they hold true more generally in the sample. Crabtree and Herskowitz never collaborated. Furthermore, these two papers belong to very different subfields of the life sciences.[16] This is important insofar as a desirable feature of the control group is to be unaffected by the treatment event. By eliminating articles by collaborators as well as topically related articles from the list of eligible controls, we bolster the claim that the control articles can pin down a credible counterfactual citation trajectory.

Descriptive Statistics

The procedure described above yields a total of 454,599 papers authored by 8,326 control scientists, as well as 27,147 treated papers authored by the 720 deceased scientists.[17] On average, there are 16.7 control articles for each treated article, highlighting the one-to-many feature of the matching procedure. Table 2 presents descriptive statistics for control and treated publications in the baseline year, that is, the year that immediately precedes the year of death for the deceased scientist. A number of the covariates are balanced between treated and control publications solely by virtue of the matching procedure—for instance, the year the article was written and the number of authors. However, covariate balance in the level of eminence at the time of (actual or counterfactual) death for treated and control scientists (measured

[16] Formally, the PubMed Related Citation Algorithm (PMRA), which will be described in more detail below, does not list one as being topically related to the other.

[17] These 27,147 articles represent approximately 60% of the set of articles by treated scientists for which we attempted to find matches (i.e., original articles in journals indexed by PubMed and the Web of Science, in which the prematurely departed scientist occupies the last position on the authorship roster and published no later than the year before the year of death).

TABLE 2
Summary Statistics of Control and Treated Articles at Baseline

	Control Publications					Treated Publications				
	Mean	Median	SD	Min	Max	Mean	Median	SD	Min	Max
Article:										
Age in year of death	4.48	5	2.57	0	9	4.48	5	2.57	0	9
Year of publication	1976.64	1977	11.24	1950	2002	1976.64	1977	11.24	1950	2002
No. of authors	3.19	3	1.58	1	15	3.20	3	1.56	1	13
Citations at baseline	35.46	16	98.94	0	18,055	33.31	15	65.27	0	3,129
Citations by noncollaborators at baseline	33.25	15	95.57	0	17,797	31.25	14	61.97	0	3,082
Citations by collaborators at baseline	2.20	0	5.78	0	358	2.06	0	5.26	0	191
Citations outside of field at baseline	31.55	13	96.49	0	18,048	29.52	13	62.19	0	3,102
Citations within field at baseline	3.91	2	6.22	0	162	3.79	2	6.11	0	138
Citations from distant authors at baseline	34.57	15	96.03	0	17,646	32.46	15	63.47	0	3,085
Citations from colocated authors at baseline	.89	0	4.00	0	410	.85	0	2.91	0	122
Investigator:										
Year of birth	1928.30	1928	10.44	1895	1966	1927.53	1927	10.52	1893	1960
Degree year	1954.59	1954	10.76	1921	1989	1954.32	1954	10.83	1920	1988

Death year	1992.35	1994	8.32	1969	2003	1992.35	1994	8.32	1969	2003
No. of publications in matched sample	51.74	39	48.58	1	355	86.64	69	72.28	1	414
Cumulative no. of publications	199	160	145	1	1,124	219	169	188	10	1,380
Cumulative no. of citations	13,586	9,359	14,318	17	188,430	13,799	9,895	12,264	77	76,231
Cumulative amount of funding (×$1,000)	24,196	15,678	28,322	0	408,427	27,435	14,949	47,940	0	329,969
No. of trainees	9	7	10	0	87	10	7	9	0	44
No. of collaborators	112	85	93	0	1,052	117	95	103	0	714
N			454,599					27,147		

NOTE.—Sample consists of all of the publications for treated and control scientists that the matching procedure has culled from the universe of last-authored original publications by deceased and still-living scientists. The matching procedure is "one to many": each treated article is matched with zero, one, or more control articles. The procedure matches 62% of eligible treated articles. The average number of control articles per treated article in the matched sample is 16.75 (median = 6, SD = 28.6, min = 1, max = 281). The descriptive statistics are weighted by the inverse number of controls in a matching strata. All time-varying covariates are measured in the year of the scientist's death (or counterfactual year of death for the control scientist). The article-level citation counts correspond to the accumulated stock of citations up to the year of death. NIH funding amounts have been deflated by the biomedical R&D Producer Price Index (base year = 2007).

through NIH funding, number of articles published, or cumulative number of citations) was not guaranteed by the matching procedure.

Figure 3 examines differences in the shape of the distribution for citations received by treated and control articles, respectively, up to the baseline year. The two distributions exhibit very similar shape, including the far-left and the far-right tails. As highlighted below, balance in the stock of citations at baseline in the cross-sectional dimension of the data is not required for the validity of the empirical exercise. More important is the absence of differential trends in the flow of citations up until the time of treatment between the treated and control groups. An important step of the empirical analysis will be to verify, ex post, the absence of such trends before the death event.

Statistical Considerations

Our estimating equation relates the effect of a scientist's death on citations in the following way:

$$\begin{aligned} E(\text{cites}_{it}|X_{it}) = \exp[&\beta_0 + \beta_1 \text{AFTER\_DEATH}_{it} \\ &+ \beta_2 \text{AFTER\_DEATH}_{it} \times \text{TREAT}_i \\ &+ f(\text{AGE}_{it}) + \delta_t + \gamma_i], \end{aligned} \tag{1}$$

where cites_{it} is the number of citations paper i receives in year t (purged of self-citations), AFTER_DEATH denotes an indicator variable that switches to 1 in the year after the star scientist (treated or control) associated with i passes away, TREAT is an indicator variable set to 1 if the scientist dies during the period, $f(\text{AGE}_{it})$ corresponds to a set of indicator variables for the age of article i at time t (measured as the number of years since the year of publication), the δ_t's stand for a full set of calendar year indicator variables, and the γ_i's correspond to article fixed effects, consistent with our approach to analyze changes in the flow of citations within each article following the passing of an elite scientist.[18]

We follow Jaravel, Petkova, and Bell (2018) in including in our specification an indicator for the timing of death that is common to treated and control articles (whose effect will be identified by the coefficient β_1) in addition to the effect of interest, an interaction between AFTER_DEATH and TREAT (whose effect will be identified by the coefficient β_2). The effects of these two variables are separately identified because (i) deaths are staggered across our observation period and (ii) control publications inherit a

[18] To avoid confusion, we have suppressed any subscript for the scientist. This is without loss of generality, since each article is uniquely associated with a single scientist (i.e., there can only be one individual in last-authorship position for each article).

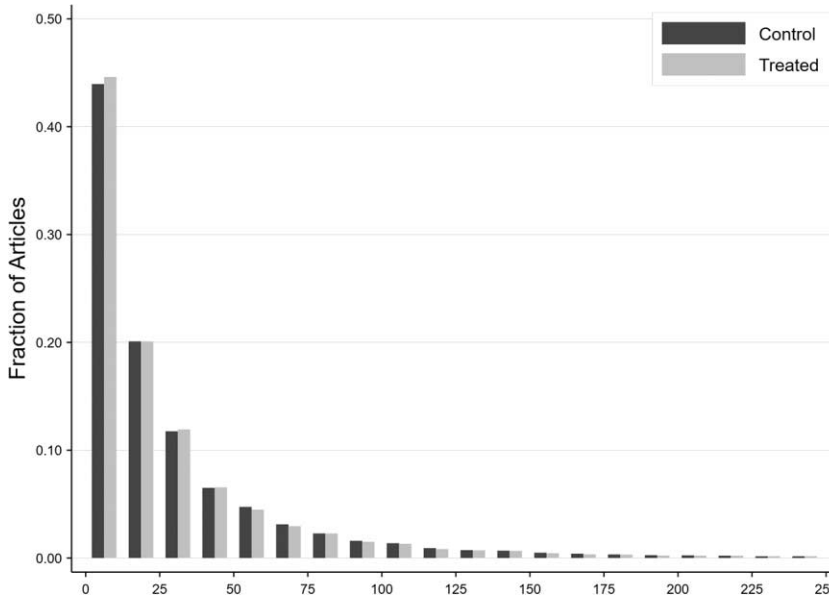


FIG. 3.—Baseline stock of citations. We compute the cumulative number of citations up to the baseline year, that is, the year that immediately precedes the year of death (or the counterfactual year of death) for the 27,147 publications by treated scientists and the 454,599 publications by control scientists. The histogram excludes articles with 250 or more accumulated citations in the year of death (approximately 0.5% of the sample).

counterfactual date of death since they are uniquely associated with a treated publication through the matching procedure described earlier. The inclusion of the common term addresses the concern that age and calendar year fixed effects may not fully account for shifts in citation activity around the time of the scientist's passing. If this is the case, AFTER_DEATH will capture the corresponding transitory dynamics, while AFTER_DEATH × TREAT will isolate the causal effect of interest. Empirically, we find that in some specifications, the common term has substantial explanatory power, although its inclusion does not radically alter the magnitude of the treatment effect.

Estimation.—The dependent variable of interest, citations accrued in each year (net of self-citations), is skewed and nonnegative. Specifically, 49.20% of the articles receive no citations in a given year, while 0.04% accumulate over 100. Following a long-standing tradition in the study of scientific and technical change, we present conditional quasi–maximum likelihood estimates based on the fixed effects Poisson model developed by Hausman, Hall, and Griliches (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, Monfort, and

Trognon 1984). We cluster the standard errors at the scientist level in the results presented below.

As discussed above, we pursue two empirical goals beyond testing for the overall effect of death on citation levels. One goal involves exploring four contextual factors. In addition to examining how the net effect varies depending on the relative youth of the scientist, we examine variables associated with three broad factors: (i) a paper's impact history, (ii) a scientist's engagement style, and (iii) the identity of citers.

Paper impact history.—The key consideration here is that papers may vary in their susceptibility to interested promotion on the basis of how much impact they have made up to the time of death (or counterfactual death). To get at this, we assign each article the percentile of the citation distribution to which it belongs, given its vintage. When computing these empirical distributions, we take into account both the year of death (citations that accrue after the year of death or counterfactual death are excluded) and the year of publication. This allows us to compare the citation impact of each article in the sample, regardless of the year in which it appeared and regardless of the time of treatment, relative to the article's age.[19] Using this information, for each scientist we create five distinct article subsamples: (1) the set of articles in the top 10% of impact at time of death, (2) the set of articles in the bottom 10% of impact at time of death, (3) the set of articles in the second and third quartile of the impact distribution at time of death, (4) the set of articles in the top 1% of impact at time of death in the PubMed universe, and (5) the set of articles published in a narrow window of three years before the time of death. Note that subsamples 1–3 use a relative benchmark to delineate a set of articles (e.g., every scientist in the data has a top 10% and a bottom 10%). The fourth subsample uses a universal benchmark, and it is possible for scientists in the data to contribute no articles to this subsample.

Engagement style.—The basic premise here is that the manner by which scientists engage with the scientific community (before death) may shape how their work is recognized (posthumously). We measure two aspects of such engagement style. The first reflects the "gregariousness" of the scientist, as reflected in the number of coauthors or trainees with whom he has worked. Arguably, we may expect such scientists to experience a more pronounced posthumous citation upsurge. The second reflects the scientist's predilection for self-promotion. One view might be that self-promotional activities while alive "prime the pump" for the posthumous mobilization of his supporters. Conversely, it might be that the activities of the salesman and the sales force

[19] For example, revisiting the example presented on fig. 2, Ira Herskowitz's *Science* publication belongs to the top percentile of the cumulative citation distribution for all articles published in 1993 and indexed jointly by PubMed and the Web of Science (only citations up to 2003, the year of death, are included in the computation). It is also ranked 10th among the 117 original articles he published before his death.

are substitutes, for example, because self-promoters are deemed unworthy of further glorification upon passing. Our proxy for self-promotion is a scientist's rate of "gratuitous" self-citation, which we define as the proportion of all citations that are self-citations for which the cited paper is in a subfield different from that of the citing paper (with subfields corresponding to those defined by PMRA), in the entire portfolio of publications for a scientist in the predeath period.[20]

Citer identity.—In order to better understand the activities of the sales force, characterizing the relationship between the citing authors and the cited is of interest. Specifically, are posthumous citations more likely to come from former collaborators or trainees? Are they more likely to originate from within the narrow subfield of the cited article or from outside that narrow subfield? Or are they more likely to be circumscribed in geographic space, for example, emerging from authors employed by the same institution as that of the deceased scientist? We parse all the citing-to-cited article pairs to distinguish between such relationships in social space, intellectual space, and geographic space.[21] We then aggregate these data up to the article-year level to compute citation counts from related versus unrelated authors.

Measuring Individual Recognition in Science

Recall that the second step of our empirical analysis involves comparing recognition activity for deceased versus still-living scientists. Our approach leverages the existence of institutionalized occasions over the course of a scientist's career in which her body of work is positively recognized. Perhaps most prominent among these include memorial events and obituaries written after death and Festschrifts or career awards (such as induction into the NAS or receipt of the Nobel Prize or Lasker Award) before death. In addition, professional journals routinely interview scholars to provide a perspective on the evolution of their fields or publish retrospective articles. The common thread across these "recognition events" is that they celebrate

[20] We experimented with several variants of this measure, including defining self-promotion as the proportion of gratuitous self-cites, as opposed to the proportion of all cites. The results presented below were qualitatively unchanged.

[21] Matching each author on citing and cited articles with the Faculty Roster of the AAMC allows us to distinguish between publications with and without former collaborators or trainees and with or without authors colocated with the focal elite scientist. Similarly, the use of the PMRA helps us distinguish between citations coming from within the same subfield, as opposed to outside the subfield. Importantly, this parsing can be implemented for the articles authored by both the treated and the placebo scientists, in a rigorously symmetric fashion. Finally, we distinguish between geographically proximate vs. distant citers using authors' institutional affiliation obtained from the AAMC Faculty Roster and NIH's CGAF database.

the scholar as an individual producer rather than narrowly shine a light on individual articles.[22] Importantly, the rate of arrival of these events (if they occur at all) is not exogenous but rather reflects an investment on the part of fellow scientists. A cynic could be forgiven for thinking (probably not out loud) that many such events would go unrecorded unless they served the memorializers' interest, in addition to the lofty and well-intentioned goal of enhancing or preserving the legacy or career of the individual being recognized.

Accordingly, we undertake a large-scale effort to collect articles recorded in academic journals that celebrate, recognize, or memorialize the scientists in our sample, whether they are deceased or still living. The challenge is to do so in a manner that is consistent over time and does not entail a built-in bias in favor of the deceased. To do so, we rely on PubMed, a publicly available bibliometric database curated by the Library of Medicine, which contains, as of the end of 2018, 29 million records for the biomedical research literature, life science journals, and online books. Helpfully, every publication indexed by PubMed is tagged by one or more of 80 distinct publication types, 10 among which could potentially denote a personal recognition event. Sifting through these articles in a systematic way, we build a data set of 5,850 distinct articles that pertain to one of the scientists in our database, deceased or still-living control.[23] While there are more events overall in the control sample, the average number of events per scientist is much higher for the deceased than for the still-living scientists (1.74 vs. 0.52 on average).

In order to compare the intensity of recognition between prematurely deceased and still-living scientists, we leverage our research design. Recall that a by-product of the matching procedure at the article level is to generate a counterfactual year of death for each elite scientist whose articles match those of treated scientists. This counterfactual year of death provides a temporal anchor to compare recognition for the deceased as well as the living. A slight complication arises since the same scientist can serve as a control multiple times, for different treated scientists who passed away in different years between 1969 and 2003. As a result, there is typically more than one counterfactual year of death for each control scientist. To get around this problem, we simply select at random one of the possible counterfactual years of death for each living scientist. We then use a window of one year before until four years after the year of death (or counterfactual death) symmetrically for

[22] Consider, e.g., Goldberg (2007). While the article highlights the impact of two articles in a specific subfield, it does so with a clear focus on the context that leads the investigator to develop a novel experimental paradigm to study visual perception in primates and features his picture prominently.

[23] Online app. E provides additional detail on the identification of these events that involved a manual hand-coding effort to weed out false positives due to homonyms.

deceased and control scientists and sum the number of recognition events for each scientist within that window.

Figure 4*A* displays the histogram for the distribution of events, broken down by treatment status. The distribution of recognition is extremely skewed for deceased and still-living scientists, but recognition is a relatively rare event for the 8,326 control scientists: only 6% are recognized at least once, whereas 49% of the deceased are the subject of a recognition event. This simple comparison provides important validation for a key premise of our argument, which is that death is an exogenous shock that shifts opportunities for interested promotion.[24]

Predicting Long-Run Posthumous Citations

The third and last step of our analysis examines whether recognition efforts plausibly lie on the causal pathway linking the premature death of a scientist with his citation "afterlife." To do so, we face the challenge that posthumous citations could be mechanically related to memorialization activities (e.g., the publication of a special volume dedicated to the work of the deceased, which necessarily includes citations to his work) and more broadly to the activities of the deceased's sales force. To avoid the reflection problem entailed by correlating two variables driven by the same underlying process—the mobilization of the sales force—we must predict posthumous citation using information available before the death event exclusively.

A natural starting point might be to use the estimates from the causal model to generate predictions. However, the difference-in-differences modeling strategy, while well suited to the challenge of estimating the causal effect of premature death on citation trajectories, is not adapted to the task of predicting, at the article level, the future time path of citations.[25] To generate article-level predictions, we begin by collapsing the data in the longitudinal dimension, such that for each article (treated or control) there are

[24] Given the sparsity of the recognition data—a vanishingly small number of still-living controls receive more than one event during the window—our empirical analysis at the scientist level will focus on the probability of being recognized (modeled with a logit specification), rather than the intensity of recognition. In online app. E, we present additional analyses of memorialization specifically for the sample of 720 scientists. In this smaller sample, there are enough events (especially after bringing in additional types of memorial events beyond those appearing in professional journals) to model the intensity of memorialization using count data models.

[25] In fact, the conditional fixed effects Poisson estimator only allows us to characterize how a scientist's death shifts the conditional mean of the flow of citation over time. It would be invalid to use the resulting estimates to compute a prediction for each article in the sample. Yet, it is the appropriate estimator for the causal analysis because it will generate consistent estimates under mild regularity assumptions (Wooldridge 1997).

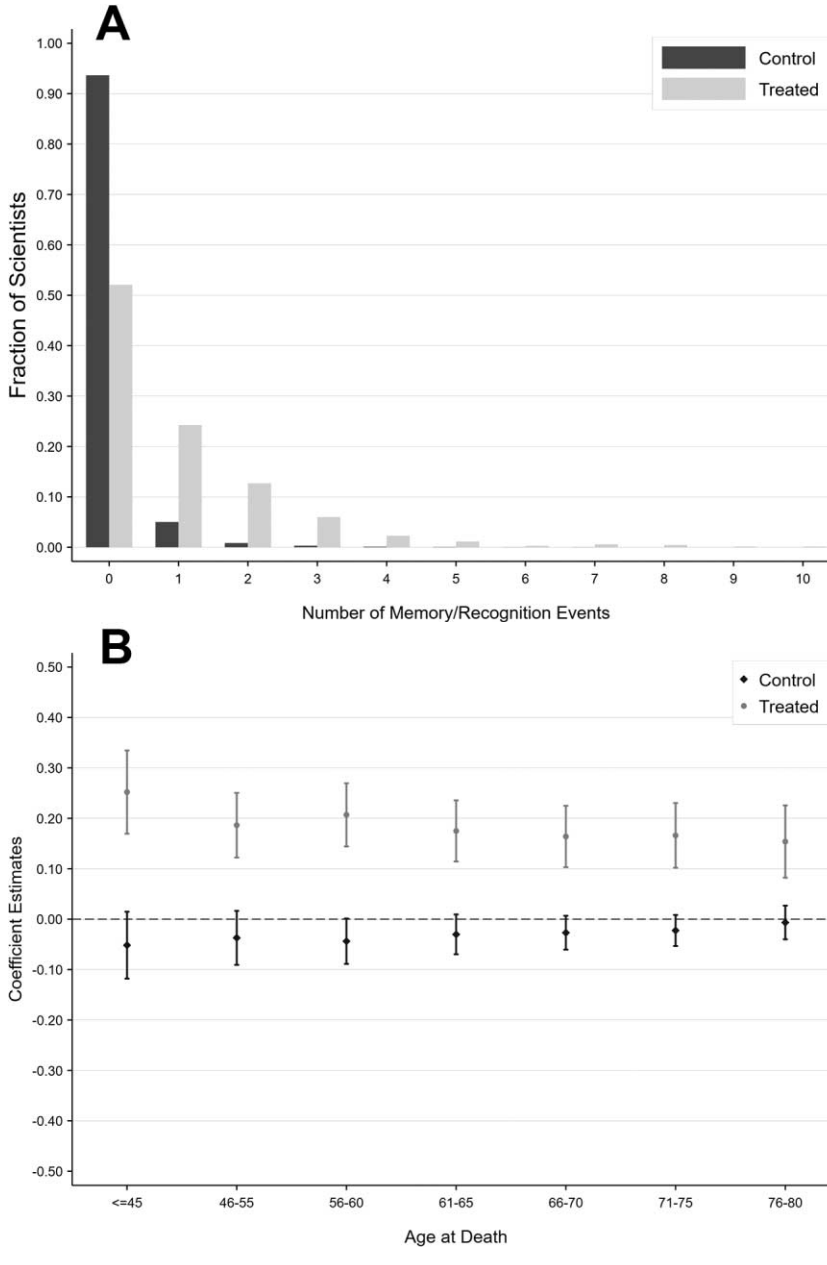


FIG. 4.—Academic memory and recognition events. *A*, Histogram of the number of academic memory events for the sample of 720 deceased scientists and 8,326 still-living scientists, within a window of one year before/five years after the year of death (for deceased scientists) or counterfactual year of death (for deceased scientists). *B*, Dots correspond to coefficient estimates for the marginal effects in logit specifications modeling the probability of an academic memory/recognition event for a scientist, as in table 7. In addition to the list of covariates in column 7, we include age-by-treatment interaction effects whose coefficients are depicted. Bars correspond to 95% confidence intervals (calculated using robust SEs).

exactly two observations, one before the year of death or counterfactual death and one from the year of death onward.

We then construct a list of 728 predictive features, including the number of citations that accrued to the article in the predeath (or pre–counterfactual death) period (log transformed), a female scientist indicator variable, year of publication effects, type of degree effects, a full suite of indicator variables for the scientists' year of (possibly counterfactual) death, a series of indicator variables for scientists' highest degree graduation years, and 472 indicator variables for each journal in which each article appeared. Using these features, we perform a penalized Poisson regression with Lasso regularization to generate predicted postdeath citation rates without overfitting the data.[26]

For each article, we compute the number of "excess" citations, that is, the difference between actual posthumous citations received and the predicted score. Panel *B* of figure D1 in the online appendix displays the histogram for the distribution of this measure, which is skewed and takes on negative values (the median of the distribution is −4.6). In the article-level sample of extinct scientists, we can run simple ordinary least squares (OLS) specifications in which excess citations are regressed on an indicator variable for having a deceased last author, a nonlinear function of the intensity of recognition activities for each scientist, as well as a large vector X of control variables (such as year of publication effects for each article, gender, highest degree, cause of death, age at death, and year of death indicators):

$$\begin{aligned}\text{NegLog}(\text{excess\_cites}_i) = \beta_0 + \beta_1 \text{DECEASED}_i + \sum_{k=1}^{3} \gamma_k 1_{\#\text{events}_i = k} \\ + \beta' X_i + \varepsilon_i,\end{aligned} \tag{2}$$

where $\text{NegLog}(x) = \log(x)$ if $x > 0$ and $-\log(-x)$ if $x < 0$ (Yeo and Johnson 2000). We are primarily interested in examining whether the correlation between death and posthumous citations is mediated by recognition efforts. If this were the case, the coefficient β_1 should decrease in magnitude or even vanish once the intensity of recognition is controlled for in the specification (by including the series of indicator variables, corresponding to three different levels of recognition intensity, as right-hand-side covariates).

[26] Online app. D provides more details, as well as sensitivity analyses, using a much more parsimonious negative binomial model estimated by maximum likelihood, as well as a high-dimensional fixed effects Poisson estimation routine (Correia, Guimarães, and Zylkin 2019). The variant we selected, based on the plug-in formula for the Lasso (Belloni, Chernozhukov, and Wei 2016), generates by far the best out-of-sample predictions (as ascertained by the deviance residuals) but interestingly exhibits the smallest correlation with actual posthumous citations.

RESULTS

The Effect of Premature Death on Citation Rates

Table 3 presents the main results for the first step of our analysis, and figure 5 provides corresponding event study graphs. These are created by estimating a specification in which the treatment effect is interacted with a set of indicator variables corresponding to a particular year relative to the scientist's death and then graphing the effects and the 95% confidence interval around them (e.g., figs. 5*A*, 5*B*, and 5*C* correspond to table 3 cols. 1, 2, and 3, respectively).[27] The estimate in table 3, column 1, implies that the papers by deceased scientists receive a boost in citations after the scientist passes away, relative to the papers of still-living scientists, with an estimated magnitude of 7.4%. Figure 5*A* shows that this effect is long-lasting. After a pronounced upsurge in citation rates in the three to four years that immediately follow the death event, the magnitude of the effect tends to attenuate and is less precisely estimated, although only in figure 5*C* (corresponding to older stars) is there clear evidence of reversion to the preevent mean of zero effect. Overall, it seems that however important a scientist is as the salesman for promoting his work, this pales in comparison to the promotional effect of the third-party sales force. More generally, we have clear evidence of a distortion in the informational efficiency of the scientific valuation process, whereby the death of a scientist seems to raise the valuation of a scientific paper by dint of the contingency of its lead author's untimely demise.

The additional results in table 3 and figure 5 shed light on two issues discussed above: (i) whether the death was anticipated and how such anticipation might alter the strength of our conclusions and (ii) whether the effect is more pronounced when the death occurred at a young age. With regard to the first issue, it seems clear from figures 5*A*, 5*B*, 5*C*, and 5*E* that there is no discernible evidence of an effect in the years leading up to the death. The absence of differential citation trends between treated and control articles provides an important ex post validation of our identification strategy. In figure 5*E* (corresponding to the subsample of articles by treated scientists who died from an anticipated illness and their associated control articles), one can observe a positive and marginally significant increase in citations in the year before death. It seems that for anticipated deaths, news of the scientists' terminal illness increases attention to the scientists' work before

[27] In these specifications, the AFTER_DEATH term that is common to treated and control publications is also interacted with a complete series of lags and leads relative to the year of death or counterfactual death.

TABLE 3
EFFECT OF SCIENTIST'S DEATH ON CITATION RATES, BY AGE AND CAUSE OF DEATH

	ALL CAUSES OF DEATH			SUDDEN DEATHS		ANTICIPATED DEATHS	
	All Ages (1)	≤65 at Death (2)	>65 at Death (3)	≤65 at Death (4)	>65 at Death (5)	≤65 at Death (6)	>65 at Death (7)
After death	.07*	.08*	.07	.10*	.03	.06	.13+
	(.03)	(.03)	(.06)	(.04)	(.08)	(.04)	(.08)
No. of investigators	9,038	8,567	4,500	7,524	3,533	6,749	3,568
No. of source articles	481,337	309,154	172,183	138,545	70,012	161,651	93,625
No. of source article-year observations	10,947,398	6,243,544	4,703,854	2,696,929	1,857,319	3,361,745	2,611,750
Log likelihood	−17,010,037	−10,262,936	−6,741,759	−4,421,808	−2,684,950	−5,563,598	−3,754,028

NOTE.—Estimates stem from fixed effects Poisson specifications. For each article, one observation per year is included in the sample in the window between the year of publication and 10 years after the (possibly counterfactual) event or 2006, whichever comes earlier. The dependent variable is the total number of citations accrued to a publication in a particular year. All models incorporate a full suite of year effects and nine article age effects, as well as a term common to both treated and control articles that switches from 0 to 1 after the death of the scientist, to address the concern that age, year, and individual fixed effects may not fully account for transitory citation trends after death. Exponentiating the coefficients and differencing from 1 yields numbers interpretable as elasticities. For example, the estimate in col. 1 implies that the papers of deceased scientists posthumously experience a $100 \times (\exp[0.07] - 1) = 7.25\%$ statistically significant increase in the number of citations relative to papers whose author remained alive. The number of observations varies slightly across columns because the conditional fixed effects specification drops observations corresponding to articles for which there is no variation in activity across the entire observation period. This is also true for the results reported in tables 4–6. Robust (quasi–maximum likelihood) SEs are in parentheses, clustered at the level of the star scientist.

+ $P < .10$.

* $P < .05$.

** $P < .01$.

their passing, especially if the news of the eminent scientist's illness spreads in the "invisible college" in which she or he participates.[28]

With regard to the second issue, we find that the citation boost that a scientist receives as a result of premature death is greater when the death occurs at a relatively young age. The overall difference can be seen most clearly from the comparison of figures 5*B* and 5*C*, given the stronger tendency toward reversion in later years among scientists who were older at the time of death. The overall difference is relatively slight, however, and is only statistically significant when the comparison is within scientists who die suddenly (whereas the difference is reversed when the comparison is within scientists whose deaths were anticipated). This could be because such deaths are experienced by the community as especially tragic (with particular sensitivity to the work the scientist might have produced had she lived), thereby triggering an especially strong mobilization on the part of the sales force.

Table 4 splits the sample over article-level characteristics that should correlate with the salience of discrete academic works within a larger portfolio of published articles for each scientist. The average effect (reproduced in col. 1) conceals striking heterogeneity in the magnitudes that apply to articles of different initial impact, assessed by cumulative citations received up to the year of death (or counterfactual death). For the articles that had already attracted the most notice at the time of death, either in a local (own top 10%) or global (universe top 1%) sense, the posthumous increase in citations is more than 17% (cols. 4 and 5), while for the least well-cited articles at the time of death (own bottom 10%), the boost is an even more remarkable 91% (col. 2). The papers that lie between the 25th and 75th percentile of citation impact at the time of death (col. 3) do not experience a posthumous citation boost. Finally, recently published articles, which are presumably salient in citers' minds, experience a somewhat greater increase (10.1%) than articles published earlier. These analyses imply that the increased attention received by the articles of deceased scientists after their passing is not uniformly distributed across their portfolio: the broad middle of the impact distribution receives no citation boost, while articles in the tail, and particularly the bottom tail, experience an upward shift in their citation trajectory. These contributions would have remained relatively obscure had their last author not prematurely died.

Table 5 reports the results with regard to engagement style. Columns 1 and 2 report estimates for the sample split across the median of the size of the sales force distribution and show that the postdeath effect is driven by the sample of stars who cultivated a larger number of coauthors while alive.

[28] Note, however, that anticipated deaths do not exhibit elevated rates of "recognition events" in the year of a scientist's death—or the two years that precede it—relative to scientists whose death was likely sudden.

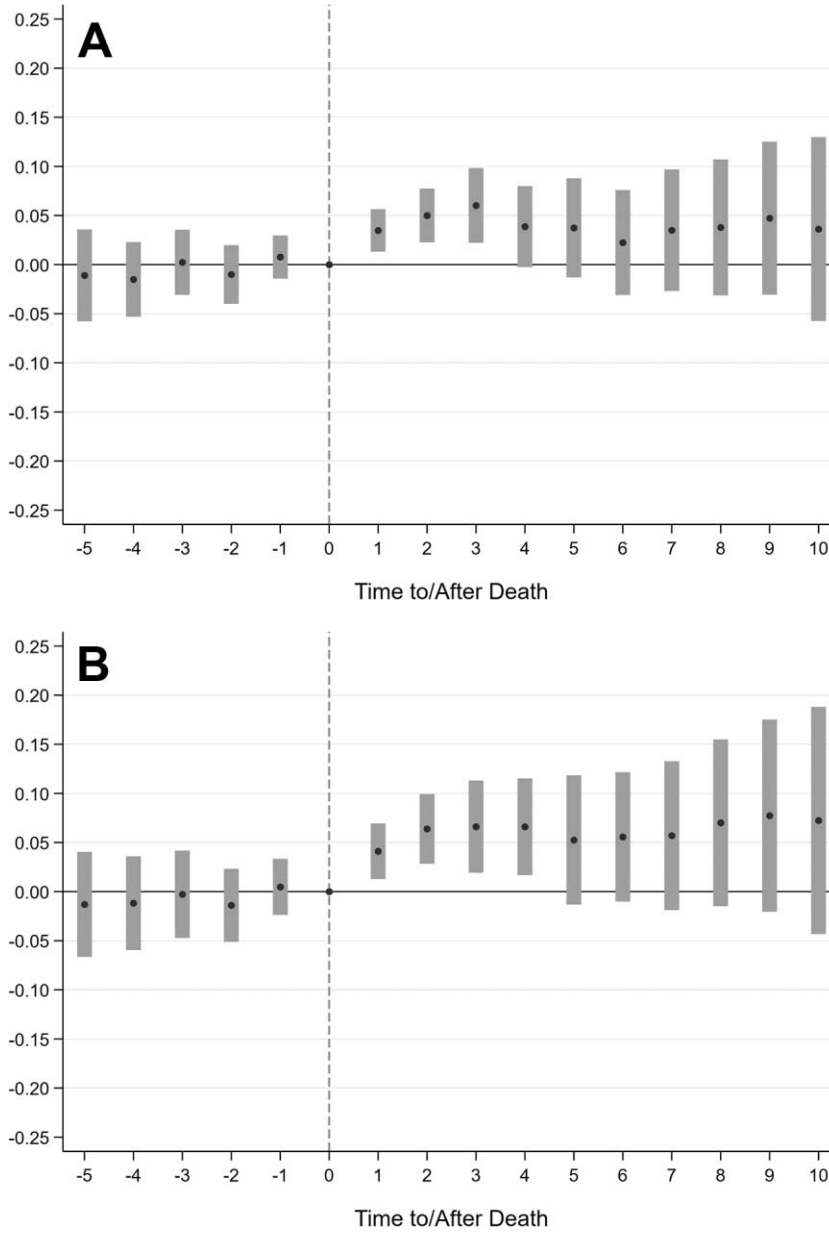


FIG. 5.—Effect of scientists' deaths on reception of their work—event study graphs: *A*, all ages, all causes of death; *B*, all causes of death, ≤65 at time of death; *C*, all causes of death, >65 at time of death; *D*, all ages, sudden deaths; *E*, all ages, anticipated deaths. Dots correspond to coefficient estimates stemming from conditional (scientist) fixed effects Poisson specifications in which citation flows are regressed onto year effects, article age effects, as well as 15 interaction terms between treatment status and the number of years before/after the death of the author (the indicator variable for treatment status interacted with the year of death is omitted). The specifications also include a full set of lead

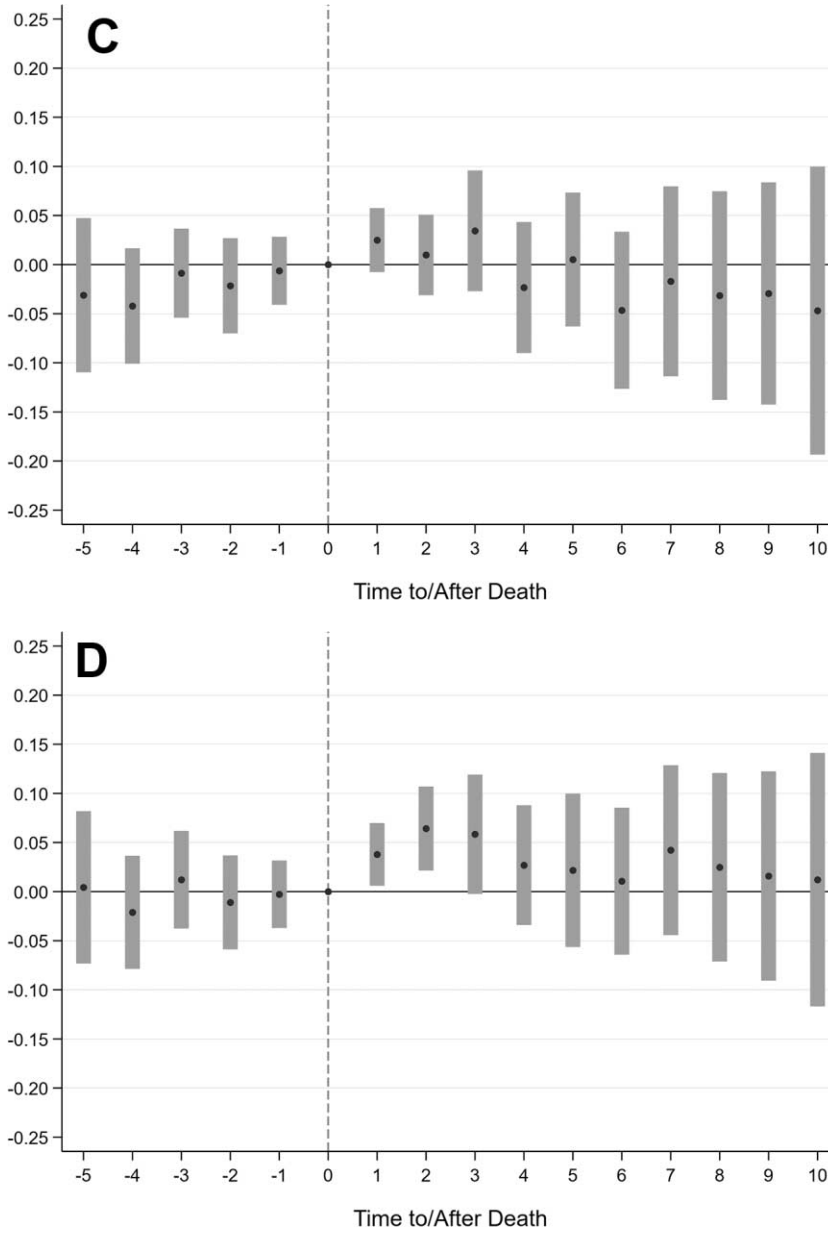


FIG. 5.—(*Continued*)

and lag terms common to both the treated and control articles, to fully account for transitory trends around the time of the event. The 95% confidence interval (corresponding to [quasi-maximum likelihood] robust SEs, clustered at the level of the scientist) around these estimates is plotted with bars. *A*, *B*, and *C* correspond to dynamic versions of the specifications in table 3 columns 1, 2, and 3, respectively.

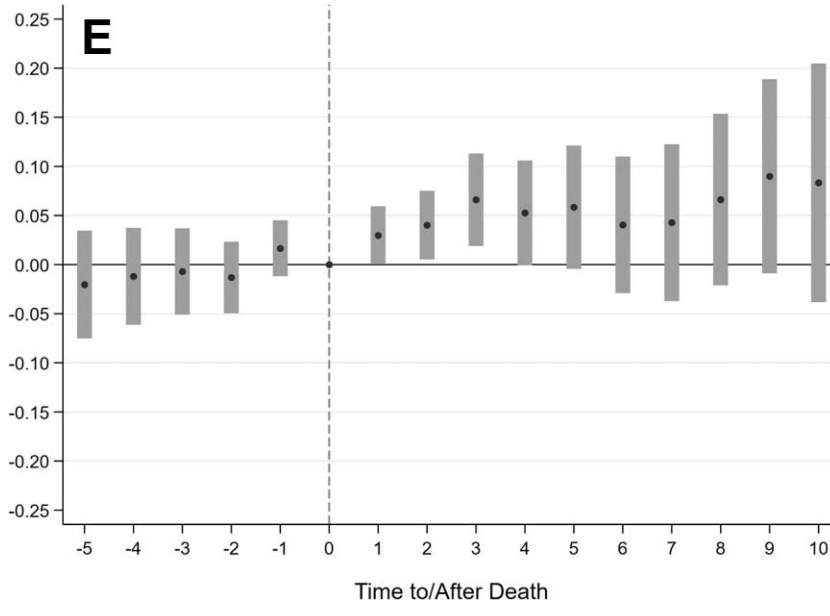


FIG. 5.—(*Continued*)

Columns 3 and 4 correspond to a sample split across the median of our proxy for self-promotional behavior. We find that the postdeath citation boost is twice as large in magnitude for the subsample of articles of more "humble" stars, consistent with the view that the salesman's promotional activities are discounted by the audience. However, we caution that the standard errors around the estimates are sufficiently large that we cannot reject the hypothesis that the two coefficients are in fact equal.

The results with regard to citer identity are presented in table 6. Note that the different columns do not correspond to splits of the sample; rather, it is only the dependent variable that changes across specifications. For instance, column 1 models the effect of the scientist's passing on the number of citations solely coming from articles that do not include a former collaborator of the deceased (or of the still-living control scientist). Overall, there is only modest evidence that postdeath citations are bestowed on the work of deceased scientists disproportionately by more proximate citers. While the magnitudes are higher for proximate citations (especially in the intellectual and spatial dimensions), the difference between the effect on proximate versus nonproximate citations is not itself statistically significant. We tentatively conclude that the citation boost documented in tables 3 and 4 (as well as fig. 5) reflects a diffuse and increased interest in the deceased's contributions.

TABLE 4

EFFECT OF SCIENTIST'S DEATH ON CITATION RATES, BY ARTICLE IMPACT AT BASELINE

	All Publications (1)	Own Bottom 10% (2)	Own 25%–75% (3)	Own Top 10% (4)	Universe Top 1% (5)	3 Years before Death (6)
After death	.07*	.65**	.00	.17**	.16**	.10*
	(.03)	(.08)	(.03)	(.04)	(.05)	(.04)
No. of investigators	9,038	4,333	8,422	6,035	4,346	6,244
No. of source articles	481,337	17,747	264,631	55,558	25,197	51,930
No. of source article-year observations	10,947,398	368,091	5,945,660	1,351,435	580,912	610,367
Log likelihood	−17,010,037	−483,644	−9,030,902	−2,427,679	−1,182,256	−1,132,866

NOTE.—Estimates stem from conditional fixed effects Poisson specifications. For each article, one observation per year is included in the sample in the window between the year of publication and 10 years after the (possibly counterfactual) event or 2006, whichever comes earlier. The dependent variable is the total number of citations accrued to a publication in a particular year. All models incorporate a full suite of year effects and nine article age effects, as well as a term common to both treated and control articles that switches from 0 to 1 after the death of the scientist, to address the concern that age, year, and individual fixed effects may not fully account for transitory citation trends after death. Column 4 (col. 2) corresponds to an estimation sample comprising solely the top 10% (bottom 10%) of each scientist's publications, ranked in terms of cumulative citations at the time of death (or counterfactual time of death for control scientists). Column 3 limits the estimation sample to publications in the middle two quartiles of the citation distribution at the time of death. Column 5 limits the estimation sample to articles that fall above the 99th percentile of the vintage-specific citation distribution at the time of death in the universe of publications indexed by PubMed and the Web of Science. Exponentiating the coefficients and differencing from 1 yields numbers interpretable as elasticities. For example, the estimate in col. 1 implies that the papers of deceased scientists posthumously experience a $100 \times (\exp[0.07] - 1) = 7.25\%$ increase in the number of citations relative to papers whose author remained alive. The number of observations varies slightly across columns because the conditional fixed effects specification drops observations corresponding to articles for which there is no variation in activity over the entire observation period. This is also true for the results reported in tables 3, 5, and 6. Robust (quasi–maximum likelihood) SEs are in parentheses, clustered at the level of the star scientist.

$^{+}$ $P < .10$.

* $P < .05$.

** $P < .01$.

TABLE 5
HETEROGENEITY IN THE EFFECT OF SCIENTIST'S DEATH ON CITATION RATES, BY SCIENTIST ENGAGEMENT STYLE

	No. of Coauthors		Self-Promotion	
	Below Median (1)	Above Median (2)	Below Median (3)	Above Median (4)
After death	.02	.12**	.10**	.05
	(.04)	(.04)	(.04)	(.05)
No. of investigators	7,248	4,298	7,291	2,848
No. of source articles	244,293	237,044	240,139	241,198
No. of source article-year observations	5,544,118	5,403,280	5,171,366	5,776,032
Log likelihood	−8,105,971	−8,883,461	−8,046,445	−8,958,661

NOTE.—Estimates stem from conditional fixed effects Poisson specifications. For each article, one observation per year is included in the sample in the window between the year of publication and 10 years after the (possibly counterfactual) event or 2006, whichever comes earlier. The dependent variable is the total number of citations accrued to a publication in a particular year. The estimation samples in each column correspond to sample splits across the median of two individual characteristics of the scientists in the sample, assessed in the year of death: accumulated number of distinct collaborators and self-promotion behavior. All models incorporate a full suite of year effects and nine article age effects, as well as a term common to both treated and control articles that switches from 0 to 1 after the death of the scientist, to address the concern that age, year, and individual fixed effects may not fully account for transitory citation trends after death. Exponentiating the coefficients and differencing from 1 yields numbers interpretable as elasticities. For example, the estimate in col. 2 implies that the papers of deceased scientists with above the median number of coauthors at the time of their death posthumously experience a $100 \times (\exp[0.12] - 1) = 12.75\%$ increase in the number of citations relative to papers whose author remained alive (and are also below the median number of coauthors at the time of their counterfactual death). The number of observations varies slightly across columns because the conditional fixed effects specification drops observations corresponding to articles for which there is no variation in activity over the entire observation period. This is also true for the results reported in tables 3, 4, and 6. Robust (quasi–maximum likelihood) SEs are in parentheses, clustered at the level of the star scientist.

$^{+}$ $P < .10$.
* $P < .05$.
** $P < .01$.

The Determinants of Individual Academic Recognition

In clear violation of informational inefficiency, the results above demonstrate that the reception of scientists' work does change after their death, with articles by the deceased being shifted to a steeper citation trajectory relative to the articles of the living. This posthumous boost is particularly large for articles that had not attracted wide recognition and for young scientists who die suddenly. As a whole, the evidence suggests that death elicits a surge in interest in the deceased scientist's work relative to comparable work by still-living scientists.

TABLE 6
EFFECT OF SCIENTIST'S DEATH ON CITATION RATES, BY CITER IDENTITY

	SOCIAL SPACE		INTELLECTUAL SPACE		GEOGRAPHIC SPACE	
	Noncoauthored Cites (1)	Coauthored Cites (2)	Out-of-Field Cites (3)	In-Field Cites (4)	Noncolocated Cites (5)	Colocated Cites (6)
After death	.06	.07	.07*	.17**	.06	.18**
	(.04)	(.06)	(.03)	(.04)	(.04)	(.06)
No. of investigators	7,916	7,916	8,709	8,709	7,175	7,175
No. of source articles	300,821	300,821	408,730	408,730	189,881	189,881
No. of source article-year observations	6,701,406	6,701,406	9,101,153	9,101,153	4,246,421	4,246,421
Log likelihood	−12,247,248	−3,030,973	−14,364,645	−4,802,682	−8,940,616	−1,322,822

NOTE.—Estimates stem from fixed effects Poisson specifications. For each article, one observation per year is included in the sample in the window between the year of publication and 10 years after the (possibly counterfactual) event or 2006, whichever comes earlier. The dependent variable is the total number of citations of a particular type accrued to a publication in a particular year. The types of citations considered include (i) citations from coauthors vs. noncoauthors, (ii) citations from the same narrow subfield vs. those from other subfields (the PubMed Related Citation Algorithm is used to distinguish between in-field vs. out-of-field citations), and (iii) citations from colocated authors vs. distant authors. All models incorporate a full suite of year effects and nine article age effects, as well as a term common to both treated and control articles that switches from 0 to 1 after the death of the scientist, to address the concern that age, year, and individual fixed effects may not fully account for transitory citation trends after death. Exponentiating the coefficients and differencing from 1 yields numbers interpretable as elasticities. For example, the estimate in col. 6 implies that the papers of deceased scientists experience a posthumous $100 \times (\exp[0.18] - 1) = 19.72\%$ increase in the number of citations from colocated scientists. The number of observations varies slightly across columns because the conditional fixed effects specification drops observations corresponding to articles for which there is no variation in activity over the entire observation period. This is also true for the results reported in tables 3–5. Robust (quasi–maximum likelihood) SEs are in parentheses, clustered at the level of the star scientist.

$^{+}$ $P < .10$.

* $P < .05$.

** $P < .01$.

What these results do not explain, however, is why this mobilization occurred. In the second and third steps of our analysis, we examine the possibility that supporters of the deceased scientist promote her work via recognition events, whereas such interested promotion does not occur, or occurs with less intensity, for still-living scientists.

To measure the determinants of academic recognition at the scientist level, we model the probability of being memorialized (for the deceased) or recognized (for the still-living controls) at least once in an academic journal within a window of one year before to four years after the year of death (or counterfactual death). In the sample of 9,046 scientists, our minimal list of covariates to explain recognition includes an indicator variable for the deceased, the scientist's gender, her age in the year of death (captured with six indicator variables corresponding to different brackets, e.g., less than 45 years old, between 45 and 55 years old, etc.), indicator variables for the cause of death (anticipated death is the omitted category), and a full suite of indicator variables for the calendar year of death.[29]

Table 7 reports marginal effects from logit models. Consistent with figure 4*A*, the estimates in column 1 demonstrate that the deceased are more than 18% more likely to be recognized (at the means of the other covariates). Conversely, there does not seem to be much difference between the likelihood of recognition for scientists of different genders (with the caveat that the gender composition of the sample skews heavily male). We do not report the coefficients for the included age effects, but the age gradient is relatively flat, except at very old ages—the "forces of nature" who die past age 75 while still leading an active scientific career get memorialized more intensely than scientists whose death can more legitimately be deemed "premature."

Columns 2–4 of table 7 examine the role of eminence in shaping the intensity of recognition. All columns include an indicator variable for members of the NAS, which can be thought of as an "elite within the elite." The effect of NAS membership is always large and precisely estimated. Column 2 uses cumulative citations at death as an additional measure of eminence. Column 3 (respectively, col. 4) uses cumulative publications instead (respectively, cumulative NIH funding). The results indicate that eminence is, perhaps unsurprisingly, correlated positively with recognition. Column 5 includes all three measures in the specification, but the high correlation between them makes it difficult to interpret the results (although the cumulative citation measure is the one that appears to keep its sign and magnitude).

Columns 6 and 7 of table 7 retain eminence as a covariate (using NAS membership and citations at death) but also add two measures that aim to capture the size of the cohort of scientists who are probably most affected

[29] Recall that still-living scientists inherit both the year of death and the cause of death of the deceased scientist with whom they are matched in our research design.

TABLE 7
CORRELATES OF ACADEMIC RECOGNITION/MEMORIALIZATION

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Deceased	.19**	.18**	.18**	.19**	.18**	.18**	.18**	.21**
	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)
ln(cumulative citations)		.04**			.04**	.04**	.04**	.03**
		(.00)			(.01)	(.00)	(.00)	(.00)
ln(cumulative publications)			.05**		.01			
			(.00)		(.01)			
ln(cumulative funding)				.01**	.00			
				(.00)	(.00)			
Member of the NAS		.07**	.09**	.10**	.07**	.07**	.07**	.09**
		(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)
ln(no. of trainees)						−.00		−.00
						(.00)		(.00)
ln(no. of coauthors)							−.00	.00
							(.01)	(.00)
Self-promoter								.03**
								(.01)
Death is sudden	.01	.00	.00	.00	.00	.00	.00	.00
	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)
Female	.01	.02*	.02*	.01	.03**	.02*	.02*	.00
	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)	(.01)
Pseudo-R^2	.18	.24	.23	.21	.24	.24	.24	.20

NOTE.—Estimates are marginal effects from logit specifications. The unrounded coefficients/standard errors for the rounded coefficients that read .00 in the table are .001. The sample consists of a cross-section of the 720 deceased scientists and 8,326 still-living scientists. The response variable is the existence of at least one academic memory/recognition event created for a scientist in a window of one year before/five years after the death for the deceased (or counterfactual death for the still-living scientists). All models include—but do not report—controls for degree type (PhD and MD/PhD; MD is the omitted category), a suite of indicator variables for each death year, six indicator variables for age categories at the time of death (less than 45 years old, between 45 and 55 years old, between 55 and 60 years old, between 60 and 65 years old, between 65 and 70 years old, between 70 and 75 years old, and between 75 and 80 years old, above 80 years old is the omitted category), and an indicator variable for unknown cause of death. The number of coauthors measure excludes the number of trainees, so that there is no double counting of coauthors (trainees are only identified conditional on coauthorship). Self-promoter is an indicator variable equal to 1 for investigators above the median in terms of self-promotional behavior (as assessed by the number of unrelated—i.e., out-of-subfield—self-citations as a proportion of all citations in a deceased scientist's entire body of work). Number of scientists = 9,046. Robust SEs are in parentheses.

+ $P < .10$.
* $P < .05$.
** $P < .01$.

by the premature death of a scientist: former trainees and collaborators of the deceased, respectively.[30] The results in columns 6 and 7 do not seem

[30] Trainees are identified as the subset of coauthors who appear in first-authorship position when the star is in last-authorship position, in a window of five years around the time they earned their highest degree.

to indicate that the sheer quantity of trainees and former collaborators (which we might think of as constituting the deceased's "visible college") correlates strongly with memorialization activities. Column 8 presents the results for the most saturated model, which adds our index of self-promotional behavior as a covariate. We find that self-promotion is positively correlated with recognition. At the very least, it does not appear that humility makes it easier for the sales force to coalesce around the memory of the deceased.

Since the postdeath citation boost was especially startling for younger scientists, we also explore whether the age-recognition gradient differs for deceased and still-living scientists. We do so by including age at death × treatment status interactions in the memorialization regression model and displaying the marginal effects in figure 4*B*. Older scientists may be more memorialized than younger ones on average, but at every age, and especially younger ones, scientists who die get memorialized more than those who remain alive. Thus, consistent with Lang and Lang (1988), and consistent with the findings of the difference-in-differences model, the type of recognition events that accrue to very senior scientists in the twilight of their careers (or in retirement) are bestowed onto younger scientists only if they die prematurely.

Table E3 in the online appendix examines the authors of academic memory events in the subsample of 720 deceased scientists and demonstrates that the memorializers are either socially connected (coauthor or former trainee), intellectually connected (same subfield), or spatially connected (same institution) with the individual they recognize. The evidence is therefore consistent with a particular sequence unfolding after the death event, whereby close associates take on the burden of memorializing the deceased, and in certain conditions this triggers a much wider and diffuse response that expresses itself in the form of an elevated propensity to cite the work of the deceased. The next section attempts to substantiate empirically the last step of this sequence.

Long-Run Citation Afterlife and Its Relationship to Recognition Efforts

For the final step of our analysis, we test whether recognition events in a limited window around the time of death (or counterfactual death) mediate the effect of a scientist's passing on the rate of long-run posthumous citations for articles that were published before the death occurred. To do so, we regress "excess" cumulative citations on an indicator variable for the deceased, the intensity of recognition activity, along with the following covariates as controls: year of publication effects, gender, degree type, an indicator variable for sudden deaths and unknown causes of death, as well as a full set of indicator variables for the scientist's age at the time of death and for his or her calendar year of death. Because recognition efforts might have a nonlinear relationship with long-run citations, we break out the overall count of

academic recognition events: zero events (89.2% of the articles, the omitted category), exactly one recognition event (6.9% of the articles), exactly two recognition events (2.1% of the articles), and three or more recognition events (1.8% of the articles).

Table 8 reports OLS estimates. Because the distribution of excess citations is skewed and takes on negative values, we model it using a NegLog transformation (Yeo and Johnson 2000). In columns 1–3, we use all possible citations to build a predicted count for the "surprise" in citations for each article published by a scientist in the postdeath period. In columns 4–6, we use the same predictive model but omit citations that accrue in the five years that immediately follow the death (as well as citations from articles

TABLE 8
Long-Run Citation Afterlife and Its Relationship to Recognition Efforts

	All Citations			Excluding Citations in a Window of 5 Years Postdeath and Citations from Memorializers/Coauthors		
	(1)	(2)	(3)	(4)	(5)	(6)
Deceased	.11**		.06	.10**		.08*
	(.04)		(.04)	(.03)		(.03)
Scientists with 1 academic memory/recognition		.18**	.17**		.11**	.10**
		(.04)	(.04)		(.03)	(.03)
Scientists with 2 academic memories/recognitions		.21**	.20**		.13**	.12**
		(.05)	(.05)		(.04)	(.04)
Scientists with 3+ academic memories/recognitions		.21**	.20**		.12**	.11*
		(.05)	(.06)		(.05)	(.05)
Mean of dependent variable	−.88	−.88	−.88	−.44	−.44	−.44
Adjusted R^2	.05	.05	.05	.16	.16	.16

Note.—Estimates stem from ordinary least squares specifications. The dependent variable is the number of "excess" citations, which is simply the number of actual posthumous citations minus the number of predicted posthumous citations, based on the prediction model presented in online app. D. In cols. 1–3, all postdeath/post–counterfactual death citations are used to compute the prediction, whereas in cols. 4–6, citations that accrue in the first five years after death are excluded, as well as citations given by collaborators and memorializers of the deceased. Because the distribution of excess citations is both skewed and takes on negative values, a NegLog transformation of the dependent variable (Yeo and Johnson 2000) is performed before estimation. All models include (but do not report coefficients for) a full suite of indicator variables for age at death, year of death, year of the article's publication, degree type, and cause of death. Number of source articles = 481,746; number of investigators = 9,046. Robust SEs, clustered at the level of the scientist, are in parentheses.

+ $P < .10$.

* $P < .05$.

** $P < .01$.

written by coauthors or memorializers) to compute the prediction. The reason to exclude citations that accrue to the scientists' articles in the immediate aftermath of their deaths (or counterfactual deaths) is that these citations could reflect, at least in part, recognition efforts (e.g., it is not uncommon for obituaries and reminiscences published in scientific journals to have a list of references). By excluding from the count of excess citations those that accrue in the period of bereavement (or counterfactual bereavement), we can be more confident that our measure of excess citations does not reflect the mechanical impact of memorialization efforts.

In table 8, column 1, we confirm the effect found in the difference-in-differences analysis: the articles of deceased scientists receive 11.2% more posthumous citations on average, relative to those of still-living scientists. Results in column 2 are consistent with our argument that assigns a key role to recognition events: recognized scientists exhibit elevated rates of posthumous citations, relative to unrecognized ones. Column 3 simultaneously enters the deceased effect and the effects for the recognition events in the model. The magnitude of the deceased effect is halved and becomes imprecisely estimated. In contrast, the magnitudes of the recognition effects remain largely unchanged. From this analysis, it would appear that recognition processes largely mediate the effect of death on the allocation of the scientific community's attention toward scientific works that appeared before the death.[31]

The models in table 8, columns 4–6, paint a similar qualitative picture, with the caveat that the attenuation of the coefficient estimate for the effect of death itself is less stark in these models, which omit the short-run citation response.[32] In spite of this, the correlation between recognition intensity and posthumous citations does not appear to merely reflect awareness by the "visible college" during the turbulent years that immediately follow the passing of these scientists.

A necessary caveat is that the validity of a mediation analysis of this type requires (i) the absence of unmeasured treatment-outcome confounders, conditional on control covariates, and (ii) the absence of unmeasured mediator-outcome confounders, also conditional on covariates (Shaver 2005). The first assumption might be valid in our application, if we assume death to be an exogenous event.[33] The second assumption strikes us as being less tenable,

[31] Using a more parsimonious model with a single dichotomous mediator (recognized at least once vs. not), we perform a Sobel (1982) test and find that 41.1% of the effect of death is mediated by the recognition effect.

[32] The Sobel test implies that only 37.3% of the treatment effect of death is mediated by the recognition effect in this case.

[33] But even the exogenous character of death is open to challenge in our setting: in the case of anticipated events, elite scientists might have the opportunity to actively shape their legacy, including the identity of their future memorializers.

since more recognized scientists might differ from less recognized ones in myriad other ways that also correlate with unobserved determinants of posthumous citation rates. In the absence of exogenous variation in memorialization intensity, the evidence of partial mediation presented in table 8 must be considered as merely suggestive: individual recognition plausibly contributes to the triggering of a vibrant "citation afterlife" for deceased scientists.

When considered in the context of the results presented in tables 3–7, the evidence points to the following chain of events: the death of eminent scientists activates a narrow vanguard of colleagues who were proximate to the deceased.[34] It is this vanguard who engages in memorialization efforts, and these efforts in turn bring to the attention of the scientific community at large the work of the deceased—in particular, work that may have been overlooked while he was alive.

CONCLUSION AND DISCUSSION

Limitations

Before concluding, it is useful to consider our findings in light of the two principal limitations of our study: that our sample is limited to elite academic life scientists and that our method for identifying the effect of interested promotion focuses on the shock of a scientist's premature death. Recall that the main advantage of our sample is that the wealth of information on elite life scientists allows us to create precise and meaningful counterfactuals. And the main advantage of focusing on the effects of death is that the death of a scientist occasions a shift in promotional activity without any change in the underlying quality of what was produced. But to what extent do our findings generalize beyond what we can observe with this sample and method?

Regarding the limitations of focusing on elite scientists, some light may be shed by examining the variation in status within our sample. To see if higher status scientists receive a larger boost in citations after their death, we reprise the difference-in-differences empirical framework and split the sample at the median by cumulative publications, citations, and funding at the time of death. No clear pattern emerges from these analyses—displayed in table 9—except that the effect of death remains positive across all sample splits. The articles of more eminent scientists may experience a larger boost than those of the less eminent (when eminence is measured by cumulative publications at death) or a smaller boost (when eminence is measure by cumulative citations or funding at death). Moreover, in all cases the

[34] Proximity is multidimensional, corresponding to relationships that unfolded in geographic space (such as the case of department or university colleagues), in social space (such as between mentor and trainee or between coauthors), and in intellectual space (such as shared topics, research questions, and methodologies).

TABLE 9

HETEROGENEITY IN THE EFFECT OF SCIENTIST'S DEATH ON CITATION RATES, BY SCIENTIST STATUS

	PUBLICATIONS		CITATIONS		FUNDING	
	Below Median (1)	Above Median (2)	Below Median (3)	Above Median (4)	Below Median (5)	Above Median (6)
After death	.04	.11*	.09**	.06	.09+	.05
	(.03)	(.05)	(.03)	(.05)	(.05)	(.05)
No. of investigators	8,253	2,859	7,759	3,052	7,703	3,277
No. of source articles	241,403	239,934	239,165	242,172	240,863	221,903
No. of source article-year observations	5,253,983	5,693,415	5,393,368	5,554,030	5,176,767	5,358,076
Log likelihood	−7,982,519	−9,020,775	−7,062,688	−9,934,474	−7,966,382	−8,359,278

NOTE.—Estimates stem from conditional fixed effects Poisson specifications. For each article, one observation per year is included in the sample in the window between the year of publication and 10 years after the (possibly counterfactual) event or 2006, whichever comes earlier. The dependent variable is the total number of citations accrued to a publication in a particular year. The estimation samples in each column correspond to sample splits across the median of three individual scientist characteristics assessed in the year of death: cumulative publications, cumulative citations, and cumulative National Institutes of Health (NIH) funding. All models incorporate a full suite of year effects and nine article age effects, as well as a term common to both treated and control articles that switches from 0 to 1 after the death of the scientist, to address the concern that age, year, and individual fixed effects may not fully account for transitory citation trends after death. Exponentiating the coefficients and differencing from 1 yields numbers interpretable as elasticities. For example, the estimate in col. 2 implies that the papers of deceased scientists with above the median number of publications at the time of their death posthumously experience a $100 \times (\exp[0.11] - 1) = 11.63\%$ increase in the number of citations relative to papers whose author remained alive (and are also below the median number of publications at the time of their counterfactual death). The number of observations varies slightly across columns because the conditional fixed effects specification drops observations corresponding to articles for which there is no variation in activity over the entire observation period. Columns 5 and 6 drop from the sample articles written by 318 scientists (273 treated scientists and 45 control scientists) who are "intramural employees" of the NIH and therefore not eligible to receive extramural NIH funds. Robust (quasi–maximum likelihood) SEs are in parentheses, clustered at the level of the star scientist.

+ $P < .10$.

* $P < .05$.

** $P < .01$.

difference between the above-median and below-median coefficients is not itself statistically significant. Therefore, the data at our disposal do not support the idea that the efficacy of interested promotion varies with a scientist's status.

Yet there remain reasons to doubt that we can generalize from an elite sample to the general population of scientists. It is possible that interested promotion is more efficacious for lower-status scientists. This possibility is foreshadowed by the literature on the Matthew effect in that it highlights how the work of high-status scientists is more widely read (Merton 1968; Cole 1970; Allison, Long, and Krauze 1982; Simcoe and Waguespack 2011; Azoulay et al. 2014). Insofar as this is the case, it may be that the work of elite scientists is relatively insensitive to promotional efforts in general and posthumous memorialization in particular. Put differently, while we find that even the highest-status scientists have some work that has been overlooked by the community and is thus sensitive to interested promotion, this should a fortiori be true for low-status scientists. But while the efficacy of equivalent promotional activity may be greater for lower-status scientists, it may be more difficult to mobilize (posthumous) activity for such scientists. Our results regarding the correlates of individual academic recognition (e.g., table 7) demonstrate significant responsiveness to status differences. Since such efforts partly mediate the effect of death on posthumous citations (table 8), it follows that one might expect the death of lower-status scientists to be less effective in mobilizing a sales force and for this smaller sales force to be less effective in activating the community at large to pay homage to the work of the deceased.[35] Finally, it is also possible that interested promotion would be less valuable for lower-status scientists because audiences will find efforts to promote their work less credible.

Putting aside how the rate and effect of interested promotion might vary with the status of the scientist, promotional activities may vary with other contextual factors that are held constant in our study. In particular, it may be that the death of a scientist is an unusually good context for promoting her work because the norm of disinterestedness is suspended. The occasion of a death may also lend unusual credibility to assessments of a scientist's work because they occur sometime after publication and thus are not a snap judgment but can be made in light of subsequent work. Finally, since we identified the salesman effect indirectly, via the absence of a drop in citations due to death, there is reason to wonder whether scientists can sometimes be more effective as salesmen than our study suggests.

[35] The literature on the Matthew effect would also suggest that lower-status scientists attract smaller numbers of coauthors, research assistants, doctoral students, and admirers (see Zuckerman 1967; Allison and Stewart 1974; Goldstone 1979; Stewart 1983; Rossiter 1993; Dey, Milem, and Berger 1997)—in other words, a less vibrant sales force.

The upshot is that the current study is hardly the last word on how interested promotion skews scientific valuation or social valuation more generally. Our results provide evidence for informational inefficiency in a highly developed and broad scientific domain, but they are particular to that domain and a particular select group within it, a particular social cue, and a particular opportunity for viewing the effects of that social cue. Our discussion here provides some guidance for how our results may generalize along those dimensions, but we must await future research before drawing firmer conclusions.

Implications

The foregoing caveats notwithstanding, our study has significant implications for understanding the informational (in)efficiency of meritocratic systems, how science as a vocation shapes recognition and the allocation of credit, and reputational entrepreneurship more generally. We conclude by discussing each of these implications in turn.

The informational efficiency of meritocratic systems.—An important contribution of our article is to open up a new direction for the study of how social cues affect the informational efficiency of meritocratic systems. As reviewed above, recent research has made significant strides on this question. However, this literature is also limited because of the narrow range of social cues and situations it has examined. In short, it is potentially quite problematic to reduce all social cues to disinterested validation. One important limitation of this restricted focus has been stressed by some scholars (see Zuckerman 2012*a*, pp. 227–30; Turco and Zuckerman 2017, p. 1287) but not fully appreciated in the literature, that is, that anonymous evaluators (as in Salganik et al. [2006] or van de Rijt [2019]) are unusually impervious to social influence. In many social settings, actors are highly sensitive to the popularity of a practice or product, sometimes conforming and sometimes differentiating from others based purely on the prevalence and identity of others who have adopted it (e.g., Lieberson and Lynn 2003; Obukhova, Zuckerman, and Zhang 2014; Catalini and Tucker 2017). As such, whereas some scholars have concluded from studies of anonymous evaluators that social cues have limited impact in skewing valuations in meritocratic settings (see Salganik and Watts 2008; van de Rijt 2019), this conclusion is premature.

To be sure, some studies have indeed examined disinterested validation in settings where valuations are not anonymous. For example, studies based on natural experiments in scientific domains are focused on environments where the evaluators may be quite sensitive to the impressions their

evaluations make on others. In particular, scientists may often be reluctant to cite work that is rarely cited by others (or perhaps by lower-status scientists). Given that, it is notable progress to find that disinterested validation is responsible for a significant if modest degree of informational inefficiency (Simcoe and Waguespack 2011; Azoulay et al. 2014).[36]

Yet without broadening the social cues examined, from disinterested validation to interested promotion, our knowledge of how social cues affect informational efficiency is quite limited. It is unclear why (with the exception of the literature on reputational entrepreneurship) scholars have focused on disinterested validation rather than interested promotion.[37] One possibility is that it is challenging to study promotional activity in the laboratory, at least in a manner that would be generalizable. A second possibility is that scholars tend to assume that scientific fields, and meritocratic systems more generally, are governed by the Mertonian norms of disinterestedness and universalism. We have given ample reason not to rely on such an assumption, however. In light of the information frictions documented here and prior research, scientific communities may find it difficult and undesirable to dismiss promotional efforts, as they may have useful information in them. This ambiguity may make interested promotion an effective means of boosting valuations, both by the focal scientist and by her supporters.

If either the avowed norms of science were fully operative or the mechanisms underlying the scientific marketplace worked to distinguish better from worse work (given established paradigms), it would not matter whether the author of a scientific paper is dead or alive. But we find that it does matter, thus indicating the weakness of such norms and the limits to informational efficiency. In particular, the random event of an untimely death elicits commemoration activity, and such activity seems to raise the valuation of elite scientists' lesser-known work. As noted, these findings are hardly definitive. But insofar as we have identified a class of important social cues that shape valuations in meritocratic systems, future work will help flesh out our understanding of such effects given a wider range of social cues and social contexts.

[36] Note, however, that when evaluators are highly sensitive to making unusual valuations, this provides another reason why a system can be allocationally inefficient even while achieving informational efficiency. At the limit, if everyone conforms to established views, reactions to new work will be consistent but progress will never be recognized.

[37] As Arnout van de Rijt helpfully pointed out to us, an additional dimension along which social cues vary is the extent to which they occur via relationships. Thus, the mode of social influence found in book clubs (Rawlings and Childress 2019) is distinct from either that which occurs via the canonical studies of disinterested validation or the kind of interested promotion we have studied in this article—both of which operate largely outside direct relationships. A full account of how social cues skew valuations should consider this additional dimension.

How science as a vocation shapes recognition and the allocation of credit.—A second contribution of our article is to shed light on how careers within science shape the allocation of credit. Misvaluations arise in part because science struggles to divorce research from the identity of its author. The norms of disinterestedness and universalism belie the fact that science is a profession through which many individuals seek employment, status, and remuneration (Polanyi 1966; Merton 1968; Gieryn 1983). While scientific communities may seek to evaluate contributions in a manner that is blind to the identity of contributors, members can hardly be blind to identity when they recruit individuals to teach and to manage laboratories. Similarly, while citations and various awards may be conferred on papers, grants and other awards are given to individuals for broad research agendas. The paradox is that the scientific community is committed to assessing work independently of its producers even while evaluating producers on the basis of their work. This tension between universalism and science as an employment system is most observable in the debate over the "blinding" of the review process; although double-blinded reviews are most common in science, there is significant controversy over the practice precisely because some explicitly wish to use the author's identity as a signal of quality (Ceci and Peters 1984; Blank 1991). While the salience of identity to the valuation of scientific work is not new in the context of this debate, we demonstrate that even outside of it (or more specifically after it), the identity of the author materially affects the valuation of scientific work.

This struggle shows science to be nearer to art in its evaluation of work than it would at first appear to be. There is little debate that the value of a work of art is greatly affected by the identity of the artist. The salience of the artist's identity arises from the fact that art is assessed through the lens of the artist's style (Sgourev and Althuizen 2014; Wohl 2019). For this reason, art exhibitions are typically organized by artist (within genre) and reviews are most often done by well-known critics when the identity of both parties is plainly visible. Science at first blush seems to be organized in stark contrast, all in accordance to the norm of universalism (Merton 1979). But our findings demonstrate that these institutional arrangements are insufficient to overcome the incentives created by the employment and status system of science. Just as in the case of Lang and Lang's (1988) etchers, the valuation of scientific works is affected by the identity of the author via efforts at interested promotion. Discussions of a scientist's oeuvre at a retirement Festschrift or a memorial event bear many of the hallmarks of parallel events in the art world.

The logic of interested promotion.—Finally, our analysis advances our understanding of how interested promotion shapes producer legacies. Past

research in this area (labeled "reputational entrepreneurship") has focused on politics and art (Lang and Lang 1988; Fine 1996; Bromberg and Fine 2002; Jansen 2007; Kahl, Kim, and Philips 2010; McCormick 2015). This study used the context of science to analyze how a scientist's death affects the amount of interested promotion that her work receives, thus boosting positive recognition for her papers. This represents an advance both because this is a setting with especially strong meritocratic norms and because it allows for more careful identification. A key challenge in verifying any causal claim is to measure the impact relative to a counterfactual situation in which the event had not occurred (Lewis 1973). In politics, this is daunting because the number of observations is quite small and events are historically and contextually dependent. And identifying counterfactuals in art is challenging because of the absence of consensual criteria for judging pieces of art to be equivalent. In science, however, over 2.5 million articles are published annually after having completed peer review based on relatively consensual evaluation guidelines. As a result, we have been able to synthesize counterfactual cases in which death or interested promotion did not occur, by comparing articles with similar characteristics.

This approach yields striking results: interested promotion can permanently shift the valuation of prior work by up to 7.3% on average and upward of 90% in some cases. This research design also allows us to shed light on which actors are the most effective in promoting legacy. Prior work tended to focus on either the sales force (Lang and Lang 1988) or the salesman (e.g., Fine 1996) but tended not to directly compare the two. Our research design allows for this through the juxtaposition of living scientists and the memorializers of deceased ones. This comparison reveals the memorializers (sales force) to be more effective in changing valuations than is the scientist herself. This may reflect a very general pattern. It is intriguing to note how major religious movements (e.g., Christianity, Mormonism, Hasidism, Islam, and Buddhism) seem to get a boost from the founder's death, as it mobilizes efforts by disciples to ensure that the founder's life and vision are remembered and institutionalized.[38] But why might the sales force be more effective than the salesman? Two likely (but as yet untested) reasons are size and credibility. Individuals promoting their own work may be limited in that they can only be in one place at a time; by contrast, the sales force can have a much larger presence. Additionally, while communities may discount the efforts of the salesman as being self-interested, the motives of the sales force may be more difficult to impugn. As such, the larger community may be more receptive to their message and, therefore, likely to pay more attention.

[38] We are grateful to Angela Lu for pointing this out to us.

The impact of superstar scientist deaths.—Our article also sheds some initial light on how interested promotion works. Prior research on reputational entrepreneurship does not distinguish between shifts in attention and in valuation. By contrast, our results—in particular, that it is the least-cited papers that are most sensitive to reputational entrepreneurship—suggest that attentional processes may be especially important. Our study is not definitive in this regard, nor is it clear to what extent our results would generalize to domains beyond science, but they call into question a tendency to assume that reputational entrepreneurship operates by changing the valuations of existing audiences. In bringing overlooked work to the fore, the sales force is able to increase its valuation by changing the sample of work with which the community engages (Denrell and Le Mens 2016). That this mechanism is so effective in science, and especially in the work of elite scientists, is testimony to the extent to which search costs inhibit the scientific community's ability to digest new work.

We close by reflecting on how our findings at once resonate and are in tension with broader observations concerning the role of a prominent scientist's death in shaping her legacy. On the one hand, we have seen evidence of a general pattern by which death mobilizes (a large, credible, cadre) of supporters to promote the scientist's legacy. On the other hand, recent research (Azoulay et al. 2019) provides systematic evidence for Max Planck's quip that "science advances one funeral at a time." The idea is that prominent scientists are often conservative forces because of their control of resources and opportunities, such that their removal from the scene gives innovative outsiders (identified as scientists who did not collaborate with the dead scientist) the space they need to flourish. But then is a scientist's death a positive or a negative force from the standpoint of preserving a scientist's legacy?

A tentative answer is that there are two countervailing effects. On the one hand, the death of a scientist gives her supporters a temporary platform for calling attention to her work, thus helping her work gain recognition relative to other work. But on the other hand, unless these supporters have effective control of their field, their temporary platform does not block the arrival of outsiders who might wish to challenge the existing paradigm with new contributions—work that soon becomes more impactful as it facilitates a paradigm shift. A possible paradox then is that while the death of elite scientists provides a glimpse into the informational inefficiency of science, it also increases the allocational efficiency of science in the long run.

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Supplementary Online Material

Appendix A: Criteria for Delineating the Set of 13,426 Elite Scientists

Scientists enter the elite sample if they meet at least one of the following seven criteria:

Highly Funded Scientists. Our first data 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 direct costs associated with research grants, we compute individual cumulative totals for the decades 1977-1986, 1987-1996, and 1997-2006, deflating the corresponding amounts with 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). Scientists whose totals lie above the 95^{th} percentile of either distribution constitute our first group of superstars. In this group, the least well-funded investigator garnered $10.5 million in career NIH funding and the most well-funded $462.6 million.[i]

Highly Cited Scientists. Despite the preeminent role of the NIH in the funding of public biomedical research, the above indicator of "superstardom" biases the sample towards 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.[ii]

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.

Members of the National Academy of Science and of the Institute of Medicine. We add to these groups academic life scientists who were elected to the National Academy of Science or the Institute of Medicine between 1970 and 2013.

MERIT Awardees of the NIH. Initiated in the mid-1980s, the MERIT Award program extends funding for up to 5 years (but typically 3 years) to a select number of NIH-funded investigators *"who have demonstrated superior competence, outstanding productivity during their previous research endeavors and are leaders in their field with paradigm-shifting ideas."* The specific details governing selection vary across the component institutes of the NIH, but the essential feature of the program is that only researchers holding an R01 grant in its second or later cycle are eligible. Further, the application must be scored in the top percentile in a given funding cycle.

Former and current Howard Hughes Medical Investigators (HHMIs). Every three years, the Howard Hughes Medical Institute selects a small cohort of mid-career biomedical scientists with the potential to revolutionize their respective subfields. Once selected, HHMIs continue to be based at their institutions, typically leading a research group of 10 to 25 students, postdoctoral associates and technicians. Their appointment is reviewed every five years, based solely on their most important contributions during the cycle.[iii]

Early career prize winners. We also included winners of the Pew, Searle, Beckman, Rita Allen, and Packard scholarships for the years 1981 through 2000. Every year, these charitable foundations provide seed

[i] We perform a similar exercise for scientists employed by the intramural campus of the NIH. These scientists are not eligible to receive extramural funds, but the NIH keeps records of the number of "internal projects" each intramural scientist leads. We include in the elite sample the top five percentiles of intramural scientists according to this metric.

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

[iii] See Azoulay et al. (2011) for more details and an evaluation of this program.

funding to between 20 and 40 young academic life scientists. These scholarships are the most prestigious accolades that young researchers can receive in the first two years of their careers as independent investigators.

Appendix B: Linking Scientists with their Journal Articles

The source of our publication data is *PubMed*, a bibliographic database maintained by the U.S. National Library of Medicine that is searchable on the web at no cost.[iv] *PubMed* contains over 29 million citations from 4,800 journals published in the United States and more than 70 other countries from 1950 to the present. The subject scope of this database is biomedicine and health, broadly defined to encompass those areas of the life sciences, behavioral sciences, chemical sciences, and bioengineering that inform research in health-related fields. In order to effectively mine this publicly-available data source, we designed PubHarvester, an open-source software tool that automates the process of gathering publication information for individual life scientists (see Azoulay et al. 2006 for a complete description of the software). PubHarvester is fast, simple to use, and reliable. Its output consists of a series of reports that can be easily imported by statistical software packages.

This software tool does not obviate the two challenges faced by empirical researchers when attempting to accurately link individual scientists with their published output. The first relates to what one might term "Type I Error," whereby we mistakenly attribute to a scientist a journal article actually authored by a namesake; The second relates to "Type II error," whereby we conservatively exclude from a scientist's publication roster legitimate articles:

Namesakes and popular names. *PubMed* does not assign unique identifiers to the authors of the publications they index. They identify authors simply by their last name, up to two initials, and an optional suffix. This makes it difficult to unambiguously assign publication output to individual scientists, especially when their last name is relatively common.

Inconsistent publication names. The opposite danger, that of recording too few publications, also looms large, since scientists are often inconsistent in the choice of names they choose to publish under. By far the most common source of error is the haphazard use of a middle initial. Other errors stem from inconsistent use of suffixes (Jr., Sr., 2nd, etc.), or from multiple patronyms due to changes in spousal status.

To deal with these serious measurement problems, we opted for a labor-intensive approach: the design of individual search queries that relies on relevant scientific keywords, the names of frequent collaborators, journal names, as well as institutional affiliations. We are aided in the time-consuming process of query design by the availability of a reliable archival data source, namely, these scientists' CVs and biosketches. PubHarvester provides the option to use such custom queries in lieu of a completely generic query (e.g, `"azoulay p"[au]` or `"graff zivin js"[au]`). As an example, one can examine the publications of Scott A. Waldman, an eminent pharmacologist located in Philadelphia, PA at Thomas Jefferson University. Waldman is a relatively frequent name in the United States (with 208 researchers with an identical patronym in the AAMC faculty roster); the combination `"waldman s"` is common to 3 researchers in the same database. A simple search query for `"waldman sa"[au] OR "waldman s"[au]` returns 377 publications at the time of this writing. However, a more refined query, based on Professor Waldman's biosketch returns only 256 publications.[v]

[iv] `http://www.pubmed.gov/`

[v] `((((("waldman sa"[au] NOT (ether OR anesthesia)) OR ("waldman s"[au] AND (murad OR philadelphia[ad] OR west point[ad] OR wong p[au] OR lasseter kc[au] OR colorectal))) AND 1980:2013[dp])`

The above example also makes clear how we deal with the issue of inconsistent publication names. PubHarvester gives the end-user the option to choose up to four *PubMed*-formatted names under which publications can be found for a given researcher. For example, Louis J. Tobian, Jr. publishes under `"tobian l"`, `"tobian l jr"`, and `"tobian lj"`, and all three names need to be provided as inputs to generate a complete publication listing. Furthermore, even though Tobian is a relatively rare name, the search query needs to be modified to account for these name variations, as in `("tobian l"[au] OR "tobian lj"[au])`.

Appendix C: Construction of the Control Group

We detail the procedure implemented to identify the control publications that help pin down the life-cycle and secular time effects in our difference-in-differences (DD) specification. Happenstance might yield a sample of publications from aging scientists, or in out-of-fashion fields. More plausibly, article-level citation trends might be subject to idiosyncratic life-cycle patterns, reflecting the article's vintage, the age of its lead author, the vitality of its subfield, or the recency of its methods. Relying solely on publications treated earlier or later as an implicit control group raises the worry that these time-varying omitted variables will not be fully captured by publication age controls.

To address this concern, we create an additional level of difference by selecting control publications. Recall that we can accurately identify the complete publication history of all the elite scientists in the superstar sample (deceased, retired, or still living, cf. Appendix B). Therefore, we can potentially identify articles written by still-alive scientists who are "similar" to those written by the deceased scientists. But what are the characteristics of a satisfactory article control, and what are the characteristics of satisfactory control group? The distinction between the two is subtle and important. Our difference-in-differences empirical analysis relies on a counterfactual date of death for a control article, and a counterfactual eminent scientist who could have died, but did not, to produce various sample splits. It is therefore important that a control article might be paired appropriately, since it will inherit certain characteristics from its associated treated article.

Judgement is required to decide the list of covariates for which balance between control and treated units is required to generate internally valid comparisons. The analyst faces a sharp trade-off between internal and external validity, since an exhaustive list of covariates on which to guarantee balance *ex ante* would result in very few (and maybe even no) matches. Therefore, the principle that guides the selection of control articles is to choose the least restrictive set of covariates that results, *ex post*, in a control group that we can regard as comparable enough to not jeopardize the internal validity of the empirical analysis. Below, we make the modeling choices explicit in the spirit of giving the reader a view on the process through which our understanding of the setting translated into very practical considerations regarding matching.

In practice, we would like each control article to:

1. be published contemporaneously with, and to have a similar number of authors as, the article by a treated (i.e., deceased) scientist with which it is paired;
2. be unrelated (in both an intellectual and a social sense) to the article by a treated scientist with which it is paired;
3. have an author in last-author position who is a still-living elite scientist of approximately the same age as that of the deceased scientist on the article with which it is paired.[vi]

[vi] "Still living" means not only that the scientist is alive at the time of the counterfactual death, but that s/he will remain alive over the five years that follow.

We think of these "pair-level" requirements as a necessary baseline. Clearly, if control and treated articles are of vastly different vintage, or with a vastly different number of authors, it makes little sense to compare their citation trajectories. If they are related intellectually or socially (e.g., the elite scientist on one is a collaborator of the elite scientist on the other, or the *PubMed* Related Citation Algorithm lists one as an intellectual neighbor for the other), then the "control" could well be treated by the event as well. The last requirement is sensible once it is understood that there is a publication and citation life cycle for scientists in general (see Figure 1B), and that there is wide variation in the age at death in the sample of 720 deceased scientists.

In addition, we would like the control group of articles as a whole to be broadly similar to the treated group of articles, where similarity should be understood as reflecting average balance across key covariates at baseline—shortly before the death event.[vii]

As a result, we impose the following additional requirement to select control articles:

4. that they be of similar expected impact, relative to the article from the treated scientist;

To match on expected impact at baseline, we experimented with the following covariates: (i) the journal in which the treated article was published (so that the control will be recruited from the set of articles published in the same journal); (ii) the journal impact factor of the journal in which the treated article was published (when not imposing same-journal match); and (iii) the number of citations that cumulatively accrued to the treated article up to the baseline year (i.e. the year of death).

Because we found that the balance between the treated and control groups was compromised when we did not impose same-journal match, below we provide descriptive evidence with three alternative approaches. The least restrictive imposes same-journal match with no additional restriction on the number of citations received by treated and control articles at baseline; the intermediate version imposes same-journal match with relatively coarse restrictions on the number of citations received by treated and control articles at baseline; and the most restrictive imposes same-journal match with relatively fine restrictions on the number of citations received by treated and control articles at baseline.

Finally, since the combined treated and control article dataset will be analyzed in a difference-in-differences framework, rather than in the cross-sectional dimension of the data, the appropriateness of the control group must eventually be assessed on its ability to exhibit parallel citation trends (our outcome of interest) before the event of interest with those of the treated group of articles. In this respect, the work presented here is similar in flavor to recent studies that also rely on blocking techniques as a device to select a control group in the cross-sectional dimension of the data, before combining treated and control units in a panel dataset (e.g., Jaravel et al. 2018; Azoulay et al. 2019).

One can also ask how the step of selecting a control group might impact statistical inference in the second step of the analysis. Recent work by Abadie and Spiess (2019) suggests that clustering the standard errors at the level of the strata used to pair control and treated units results in conservative inference. Since we cluster our standard errors at the level of the scientist, a level that nests the matching strata, we can ignore the influence of the matching step in the difference-in-differences results we present.

Article-level or scientist-level covariates? An important design choice is whether to privilege balance on article-level characteristics or scientist-level characteristics. A case can be made that both are important: our difference-in-differences specification uses the article as the level of analysis, but the treatment corresponds to a scientist-level event, namely death. In practice, we found that imposing balance on article-level characteristics yielded approximate balance on scientist-level characteristics as well, as a fortunate byproduct, whereas the reverse was not true. As a consequence, we will focus on variants where we tweak the list of article-level characteristics that must match between treated and control articles.

Blocking on covariates. To meet these goals, we implement a blocking procedure in the spirit of coarsened exact matching (Blackwell et al. 2009). The first step is to select a relatively small set of covariates on which

[vii] Of course, balance on other moments of the distribution of these covariates would be desirable as well.

we need to guarantee balance *ex ante*, guided by the set of criteria listed above. The second step is to create a large number of strata to cover the entire support of the joint distribution of the covariates selected in the previous step. In a third step, each observation is allocated to a unique stratum, and for each observation in the treated group, control observations are selected from the same stratum. This procedure is "coarse" in the sense that we do not attempt to precisely match on covariate values; rather, we coarsen the support of the joint distribution of the covariates into a finite number of strata, and we match a treated observation if and only if a control observation can be recruited from this strata. The procedure is also "exact" in the sense that one either finds one control or more in a stratum, or one finds none, in which case the treated article is eliminated from the analytic sample. As a result, the more fine-grained the partition of the support for the joint distribution (i.e., the higher the number of strata), the larger the number of unmatched treated observations.[viii]

Implementation. We identify controls based on the following set of covariates: scientist career age; number of authors; position of the star author on the authorship roster (only last authorship position is considered); journal; and year of publication. The first two covariates only need to match within relatively coarse bins. For instance, we require that the career ages (years since the highest degree was earned) of the treated and control elite scientists be no more than two years apart. We coarsen the distribution of the number of authors by collapsing it onto four separate bins: solo-authored publications; publications with two, three, or four authors; publications with between five and eight authors; and publications with nine authors or more (the maximum is fifteen authors). In contrast, we impose an exact match on journal, publication year, and the star's authorship position.

To explore the sensitivity of our results to the choice of covariate blocking scheme, we propose three variants that make use of an additional covariate, the distribution of accumulated citations up to the baseline year (i.e., $t-1$ if t denotes the year of death). Under the most restrictive scheme, we coarsen the distribution of citations into the following bins: bottom ten percentiles; between the 10^{th} and the 25^{th} percentiles; second quartile; third quartile; between the 75^{th} and the 95^{th} percentiles; between the 95^{th} and the 99^{th} percentiles; and above the 99^{th} percentile.[ix]

The second variant coarsens the distribution of accumulated citations in $t-1$ slightly: bottom quartile; middle two quartiles; between the 75^{th} and the 95^{th} percentile; between the 95^{th} and the 99^{th} percentiles; and above the 99^{th} percentile. The third and final variant ignores the number of citations received at baseline altogether when selecting controls. It is therefore the least restrictive.

Regardless of the variant, we drop from the data any control article whose last author collaborated with the deceased scientist, as well as any control article who is a PMRA "intellectual neighbor" with its associated treated article. After these tweaks (which drop only a very small number of articles), we further drop from the sample any "orphan" article (i.e., a treated unpaired with any control, or a set of controls that has lost its treated source).

Figure D1 displays the distribution of the number of control articles per treated article under each scheme. Unsurprisingly, the size of the samples corresponding to each variant differ. The most restrictive and intermediate versions are quite similar in size, matching approximately 50% of the eligible treated articles. In contrast, the least restrictive variant matches 60% of the eligible treated articles, and can recruit many more controls in each strata (eighteen on average, versus approximately seven under the more restrictive schemes). Regardless of the variant, Panels D, E, and F of Figure D1 show that baseline citations are well balanced, not only on average, but for every quantile of the distribution. Table C1—with a structure identical to Table 2

[viii] Note that Iacus et al. (2011) pioneered coarsened exact matching (CEM) as an alternative to the propensity score, in the context of estimating valid causal effects in cross-sectional regressions, under the assumption of unconfoundedness. In contrast, we are merely using it as a sensible blocking technique to delineate a control group which we will then combine with our group of treated article in a longitudinal, article-level panel dataset. In particular, any fixed difference across articles (or their authors) would be swept out by the article fixed effect in our estimation framework.

[ix] The distributions of citation at the article-level are vintage-specific, i.e., for each possible year of publication, we compute quantiles of the citation distribution after one year, after two years,..., after n years, only limited by the coverage of the *Web of Science* data at our disposal (1950-2015).

in the main body of the manuscript—provides descriptive statistics for the analytic samples corresponding to each scheme. While balance on these covariates is, for most of them, guaranteed by the blocking procedure, note that the investigator's overall citation count at baseline, which was not used in matching, is also remarkably similar for in the treated and control samples, regardless of the variant considered.

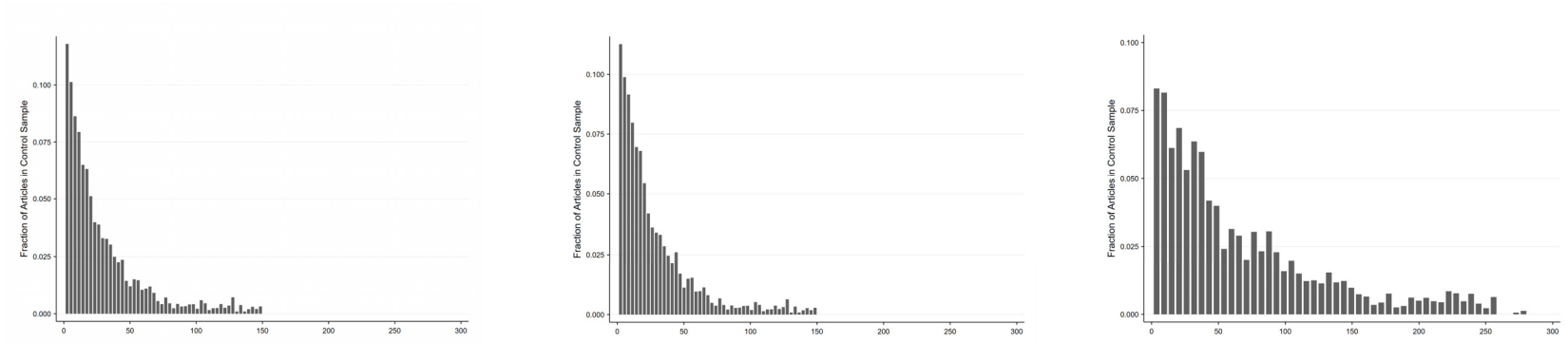
Table C2 and Figure C2 are analogs of Table 3 and Panel A of Figure 4 in the main body of the manuscript, and speak to the causal effect of death on the citation trajectories of treated articles, relative to control articles, after death, relative to before. The results are closely similar across variants, buttressing the claim that our core set of results is not an artefact of the idiosyncrasies of the matching scheme selected. In particular, in all of these variants, one cannot detect meaningful differential citation trends for treated and control articles, in the years preceding the death. Only in the few years after the year of death can one observe a meaningful increase in the rate of citations, which appears not to be sustained after the fourth or fifth year, depending on the variant. In our view, all three variants therefore result in a control group with desirable properties from the standpoint of a difference-in-differences analysis.

In light of the above, the main body of the manuscript carries out the analysis under the least restrictive blocking scheme, with its higher fraction of eligible treated articles matched, and a lower ratio of treated to control articles ($\simeq 1:17$).

Figure C1
Three Alternative Blocking Schemes to Select a Control Group

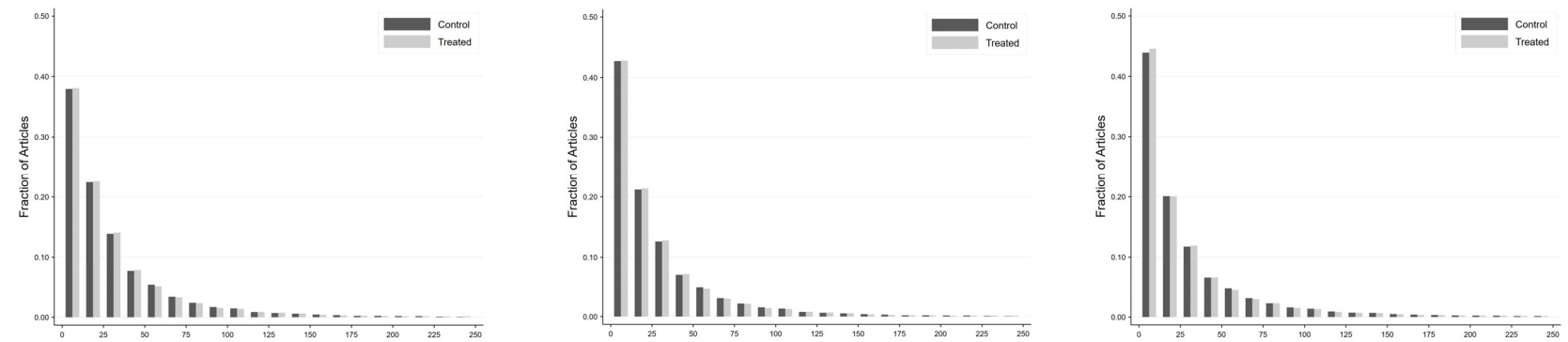
Distribution of Nb. of Controls per Treated Article

A. Most Restrictive ***B. Intermediate*** ***C. Least Restrictive***



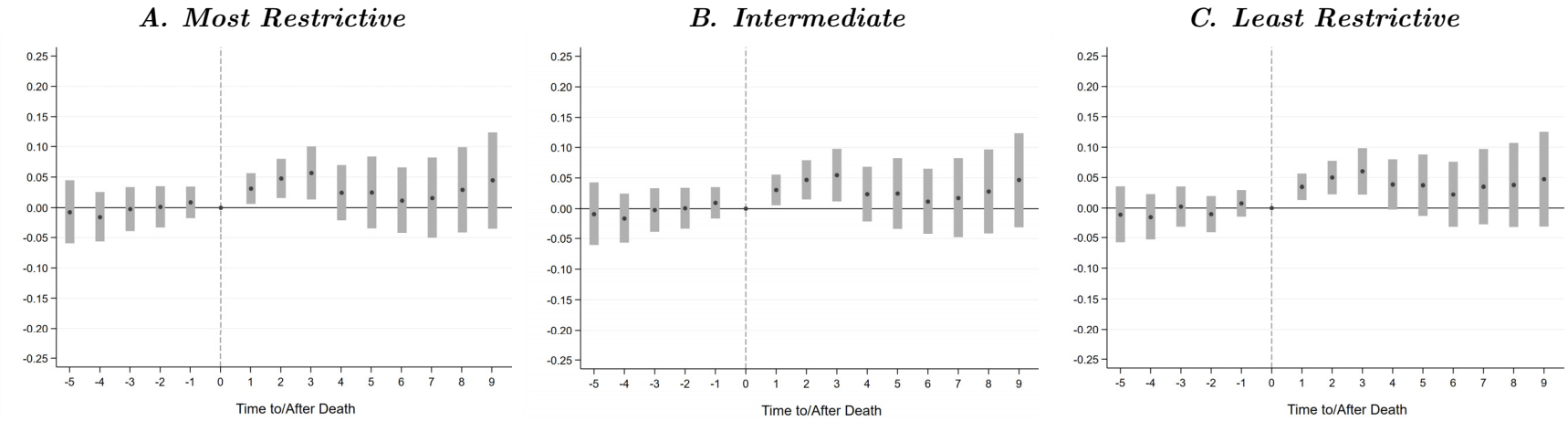
Stock of Citations at Baseline

D. Most Restrictive ***E. Intermediate*** ***F. Least Restrictive***



Note: The top three figures display the histogram for the distribution of the number of control articles matched for each treated article. Panel A corresponds to the most restrictive scheme, where 62% of the eligible treated articles are dropped because we find no controls with a matching characteristic profile (treated and control article in a ratio of 1:7 on average). Panel B corresponds to the matching scheme with an intermediate level of restrictiveness, which drops 59% of the eligible treated articles for want of a match (treated and control article in a ratio of 1:8 approximately). Finally, the least restrictive scheme (and the one used in the main body of the manuscript) drops 38% of the eligible articles by deceased scientists (treated and control article in a ratio of 1:17 approximately). Panels D, E, and, F display the distribution of the cumulative number of citations up to the baseline year for the treated and control articles respectively, under each of the three proposed matching schemes.

Figure C2
Effect of a Scientist's Death on the Reception of their Work – Event Study Graphs



Note: The dark dots in the above plots correspond to coefficient estimates stemming from conditional (scientist) fixed effects Poisson specifications in which citation flows are regressed onto year effects, article age effects, as well as 15 interaction terms between treatment status and the number of years before/after the death of the author (the indicator variable for treatment status interacted with the year of death is omitted). The specifications also include a full set of lead and lag terms common to both the treated and control articles to fully account for transitory trends around the time of the event. The 95% confidence interval (corresponding to [QML] robust standard errors, clustered at the level of the scientist) around these estimates is plotted with light grey bars. Panels A, B, and C correspond to dynamic versions of the specifications in the first column of Table C2.

Table C1: Baseline Summary Statistics for Control & Treated Articles

	Mean	Median	Std. Dev.	Min.	Max.	Mean	Median	Std. Dev.	Min.	Max.
A. Most Restrictive	Control Publications (N=143,511)					Treated Publications (N=19,111)				
Article Age in Year of Death	4.402	4	2.522	0	9	4.402	4	2.522	0	9
Article Year of Publication	1977.180	1977	10.928	1950	2002	1977.180	1977	10.929	1950	2002
Article Nb. of Authors	3.197	3	1.499	1	15	3.206	3	1.483	1	13
Article Citations at Baseline	34.652	18	68.708	0	11,505	34.183	18	63.011	0	3,138
Investigator Year of Birth	1929.125	1929	10.176	1896	1966	1928.326	1928	10.264	1897	1959
Investigator Degree Year	1955.444	1955	10.492	1923	1989	1955.191	1955	10.533	1920	1986
Investigator Death Year	1992.511	1994	8.103	1969	2003	1992.511	1994	8.103	1969	2003
Investigator Cuml. Nb. of Citations	14,128	9,771	14,558	91	188,430	14,125	10,139	12,141	77	76,231
B. Intermediate	Control Publications (N=161,748)					Treated Publications (N=20,970)				
Article Age in Year of Death	4.404	4	2.532	0	9	4.404	4	2.532	0	9
Article Year of Publication	1977.151	1977	10.986	1950	2002	1977.151	1977	10.986	1950	2002
Article Nb. of Authors	3.184	3	1.505	1	15	3.193	3	1.491	1	13
Article Citations at Baseline	32.058	16	66.129	0	11,505	31.624	16	60.725	0	3,138
Investigator Year of Birth	1929.009	1929	10.229	1896	1966	1928.228	1928	10.298	1897	1959
Investigator Degree Year	1955.328	1955	10.530	1923	1989	1955.077	1955	10.570	1920	1986
Investigator Death Year	1992.503	1994	8.152	1969	2003	1992.503	1994	8.152	1969	2003
Investigator Cuml. Nb. of Citations	13,877	9,512	14,465	53	188,430	13,864	9,961	12,086	77	76,231
C. Least Restrictive	Control Publications (N=454,599)					Treated Publications (N=27,147)				
Article Age in Year of Death	4.482	5	2.574	0	9	4.482	5	2.574	0	9
Article Year of Publication	1976.641	1977	11.243	1950	2002	1976.641	1977	11.243	1950	2002
Article Nb. of Authors	3.190	3	1.576	1	15	3.200	3	1.563	1	13
Article Citations at Baseline	35.457	16	98.945	0	18,055	33.309	15	65.275	0	3,129
Investigator Year of Birth	1928.301	1928	10.441	1895	1966	1927.535	1927	10.516	1893	1960
Investigator Degree Year	1954.588	1954	10.761	1921	1989	1954.317	1954	10.826	1920	1988
Investigator Death Year	1992.353	1994	8.316	1969	2003	1992.353	1994	8.316	1969	2003
Investigator Cuml. Nb. of Citations	13,586	9,359	14,318	17	188,430	13,799	9,895	12,264	77	76,231

Note: For each matching scheme, the sample consists of all of the publications for treated and control scientists that the procedure described in Appendix C has culled from the universe of last-authored original publications by deceased and still-alive scientists. The matching procedure is "one-to-many": each treated article is matched with zero, one, or more control articles. The descriptive statistics above are weighted by the inverse number of controls in a matching strata. All time-varying covariates are measured in the year of the scientist's death (or counterfactual year of death for the control scientist). The article-level citation counts correspond to the accumulated stock of citations up to the year of death.

Table C2: Effect of Scientist's Death on Citation Rates

	All Causes of Death	All Causes of Death		Sudden Deaths		Anticipated Deaths	
	All Ages	$<$ 65 at Death	$\geq$ 65 at Death	$<$ 65 at Death	$\geq$ 65 at Death	$<$ 65 at Death	$\geq$ 65 at Death
A. Most Restrictive							
After Death	0.081**	0.087**	0.073	0.099*	-0.003	0.075†	0.156†
	(0.030)	(0.032)	(0.060)	(0.048)	(0.076)	(0.042)	(0.087)
Nb. of Investigators	7,649	7,103	3,604	5,942	2,714	5,418	2,689
Nb. of Source Articles	162,572	104,821	57,751	47,736	22,833	54,537	32,015
Nb. of Source Article-Year Obs.	3,674,958	2,097,337	1,577,621	915,806	607,318	1,127,331	890,721
Log Likelihood	-5,424,468	-3,316,672	-2,105,942	-1,454,206	-802,541	-1,797,041	-1,210,704
B. Intermediate							
After Death	0.082**	0.088**	0.074	0.100*	-0.003	0.077†	0.158†
	(0.030)	(0.032)	(0.060)	(0.048)	(0.075)	(0.042)	(0.085)
Nb. of Investigators	7,929	7,387	3,750	6,227	2,879	5,673	2,798
Nb. of Source Articles	182,640	117,237	65,403	53,212	26,275	60,899	35,763
Nb. of Source Article-Year Obs.	4,135,623	2,350,441	1,785,182	1,023,983	698,937	1,260,766	993,601
Log Likelihood	-5,703,920	-3,484,001	-2,217,996	-1,527,749	-852,248	-1,883,884	-1,266,354
C. Least Restrictive							
After Death	0.071*	0.078*	0.068	0.103*	0.028	0.061	0.128†
	(0.032)	(0.032)	(0.056)	(0.043)	(0.078)	(0.042)	(0.075)
Nb. of Investigators	9,038	8,567	4,500	7,524	3,533	6,749	3,568
Nb. of Source Articles	481,337	309,154	172,183	138,545	70,012	161,651	93,625
Nb. of Source Article-Year Obs.	10,947,398	6,243,544	4,703,854	2,696,929	1,857,319	3,361,745	2,611,750
Log Likelihood	-17,010,037	-10,262,936	-6,741,759	-4,421,808	-2,684,950	-5,563,598	-3,754,028

Note: Estimates stem from fixed effects Poisson specifications. For each article, one observation per year is included in the sample in the window between the year of publication and ten years after the (possibly counterfactual) event or 2006, whichever comes earlier. The dependent variable is the total number of citations accrued to a publication in a particular year. All models incorporate a full suite of year effects and nine article age effects, as well as a term common to both treated and control articles that switches from zero to one after the death of the scientist, to address the concern that age, year and individual fixed effects may not fully account for transitory citation trends after death. The number of observations varies slightly across columns because the conditional fixed effects specification drops observations corresponding to articles for which there is no variation in activity across the entire observation period. Robust (QML) standard errors in parentheses, clustered at the level of the star scientist. $^{\dagger}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$.

Appendix D: Predicting Posthumous Citations

The difference-in-differences specification (eqn. (1), page 22) is the basis for the estimation of the causal effect of a scientist's premature passing on the flow of recognition at the article level. The model allows one to estimate the conditional expectation of the citation response, and does not rely on the specific features of the Poisson distribution (cf. Santos Silva and Tenreyro 2006, Wooldridge 1997).

Another goal of our study is to establish the plausibility of memorialization as a mechanism that contributes to explaining our core finding, that of a relative increase in recognition after a superstar scientist passes away. In order to do so, we must generate a measure of predicted posthumous citation for each article by a deceased scientist. To do so, we collapse the article-level panel data for both treated and control scientists onto a cross-section where the outcome of interest in the actual number of total posthumous citations, and two separate sets of covariates (also denoted by the term "predictive features" or simply "features"). The first set comprises only a parsimonious list of 140 features. The second set adds an additional 588 features.

To be more precise, the restricted set includes the number of citations that accrued to the article in the pre-death (or pre-counterfactual death) period (log transformed, with an indicator variable absorbing the zero citation cases—19,185 articles or 4% of the sample), a female scientist indicator variable, year of publication effects, indicator variables for type of degree (MD, PhD, and MD/PhD), a full suite of indicator variables for the scientists' year of (possibly counterfactual) death, a series of indicator variables for scientists' highest degree graduation years, and a series of 30 indicator variables corresponding to the article age at time of death. The expanded set of features include all the covariates in the restricted set plus (i) 472 indicator variables for each journal (427 journals who contribute less than 10 article observations to the dataset are collapsed onto a single indicator variable), (ii) 14 indicator variables for the number of authors on the paper (the top category include all authorship lists including 15 or more authors, approximately 0.15% of the sample), (iii) 20 indicator variables for each ventile of the number of trainees distribution (at the time of death), (iv) 20 indicator variables for each ventile of the number of coauthors distribution (at the time of death), (v) a dummy for intramural scientists as well as 20 indicator variables for each ventile of the cumulative NIH funding distribution (at the time of death), and (vi) 20 indicator variables for each ventile of the "self-promotion" distribution (at the time of death). Importantly, the list of features does not include an indicator for deceased scientists.

Using these features, we then perform a variety of predictive exercises using a mix of classic and more novel techniques:

(a) A negative binomial maximum likelihood procedure where posthumous citations are regressed on the restricted set of covariates;

(b) A high-dimensional fixed effects quasi-maximum likelihood Poisson routine (Correia et al. 2019) where posthumous citations are regressed on the expanded set of covariates;

(c) A penalized Poisson procedure using Lasso regularization and the expanded set of covariates. Specifically, we use the "plugin formula" (Belloni et al. 2016) to minimize the Lasso objective function. In this framework, the penalization parameters are chosen to guarantee consistent prediction and parameter estimation.

Table D1 provides a correlation table for the actual and predicted posthumous citations using these three approaches. The three prediction methods yield predicted values that are highly correlated with one another, although the correlation between actual and predicted citations is lowest using the penalized Poisson procedure. To choose among these alternatives, we compare their out-of-sample predictive power, using model deviance as the prediction metric.[x] Specifically, each model is trained on an 80% subsample of articles (clustering on investigator) and tested on the remaining 20%. The Lasso-penalized Poisson procedure exhibits

[x] The deviance is a classic goodness-of-fit measure for count data models (Cameron and Windmeijer 1996) and its use here mirrors the role of the root-mean-square error (RMSE) for prediction in the context of linear models.

by far the best out-of-sample performance, with the lowest deviance overall and very similar deviances on both the training and the testing sample. Table D2 provides the full set of diagnostics for this procedure using four different response variables: posthumous citations, posthumous citations, excluding citations from memorializers and coauthors, posthumous citations outside of the window of five years that begins with the year of death, and posthumous citations outside of the window of five years that begins with the year of death, excluding citations from memorializers and coauthors.[xi]

In the main body of the manuscript, we therefore make use of the predictions generated by the penalized Poisson procedure. We sum the article-level predictions to generate an individual-level measure of predicted citation "afterlife" for each deceased scientist. Panel A of Figure D1 displays the histogram for the distribution of this measure. Panel B of Figure D1 displays the histogram for the distribution of "excess citations," i.e., the difference between actual posthumous citations received and the predicted score.

[xi] In contrast, the negative binomial procedure exhibits a deviance ratio that is an order of magnitude higher for the testing sample, relative to the training sample. This comparison is not available for the HDFE Poisson procedure because it cannot project out-of-sample for the fixed effects that are not estimated when performing the routine on the training subsample.

Table D1: Correlations Between Predicted Posthumous Citations Measures

		Actual	**Predicted**		
			LASSO	HDFE Poisson	Negative Binomial
	Actual	1			
Predicted	LASSO	0.424	1		
Predicted	HDFE Poisson	0.629	0.783	1	
Predicted	Negative Binomial	0.989	0.808	0.877	1

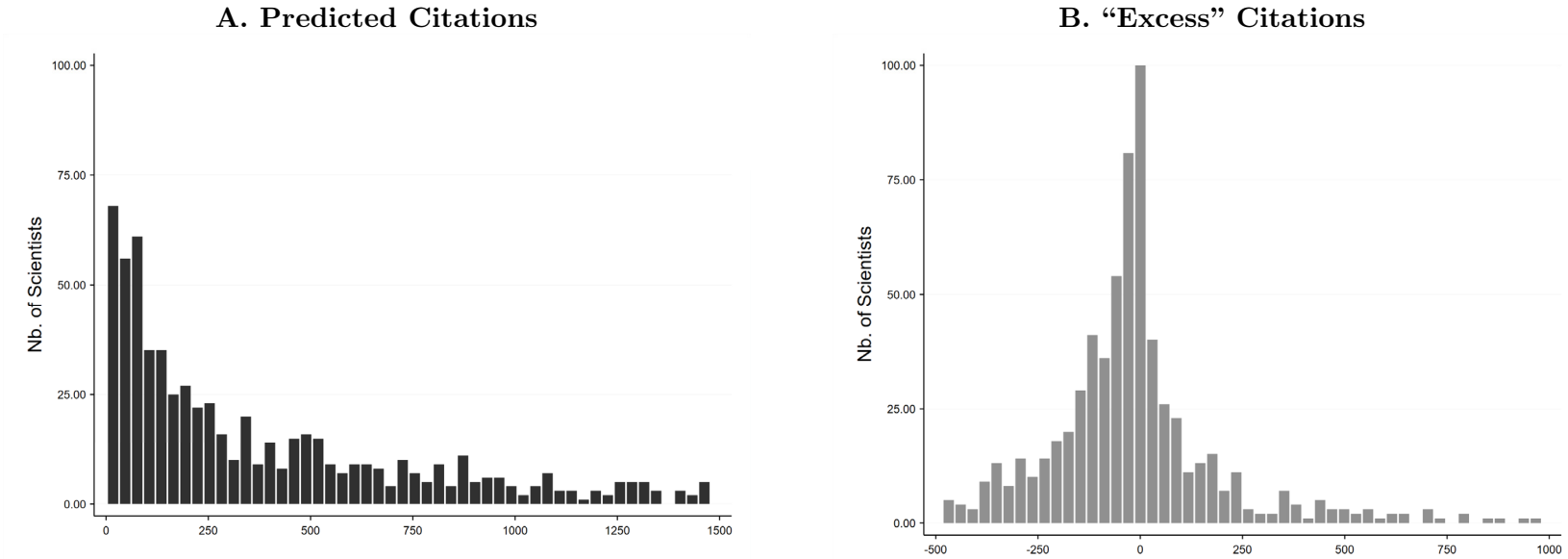
Note: We contrast three approaches to predict citations at the article level in the years after the death on the basis of covariates known at the time of death. The first approach stems from a penalized lasso Poisson procedure using the plugin method of Belloni, Chernozhukov, and Wei (2016) using an extensive set of more than 700 covariates, including a comprehensive set of journal fixed effects. The second stems from a high-dimensional fixed effects Poisson estimation routine recently proposed by Correia, Guimarães, and Zylkin (2019) using a similarly extensive of covariates, but without a penalty term to prevent overfitting. The third and final set of predictions stem from a negative binomial model estimated by maximum likelihood using a more parsimonious set of "only" 140 predictors. All the correlation coefficients reported above precisely estimated ($p<0.001$).

Table D2: Prediction Diagnostics, Lasso

Response:	Subsample	All post-death citations	All post-death citations, excl. citations from memorializers & coauthors	All citations, post-year of death+5	All citations, post-year of death+5, excl. citations from memorializers & coauthors
Nb. of Source Articles	Training	381,138	381,138	381,138	381,138
	Testing	96,705	96,367	96,705	96,367
Nb. of Investigators	Training	7,237	7,237	7,237	7,237
	Testing	1,774	1,774	1,774	1,774
Deviance	Training	23.908	22.847	16.572	15.950
	Testing	25.762	24.610	17.328	16.691
Deviance Ratio	Training	0.530	0.526	0.453	0.447
	Testing	0.521	0.517	0.456	0.449
Nb. of Non-zero Predictors		210	209	212	210
Nb. of Potential Predictors		728	728	728	728

Note: The lasso prediction model is trained on 80% of the sample of 481,746 articles (clustering at the investigator level), and tested on the remaining 20%. The deviance ratios are nearly identical across the training and testing subsample, indicating high out-of-sample predictive power.

Figure D1
Predicted and "Excess" Citations



Note: Panel A (in dark grey) displays the distribution of posthumous predicted citations for the sample of 720 treated scientists. The predictions were obtained using a Lasso Poisson procedure, using 728 covariates. Panel B (in light grey) displays the distribution of posthumous "excess" citations for the 720 treated scientists. "Excess" citations are defined by the difference, for each scientist, between the actual of citations garnered posthumously with the number of citations from our predictive model. Note that rather than an "excess," for many deceased scientists one can observe a "shortfall" of citations as they receive fewer posthumous citations than our model predicted. Sixty seven outliers with more than 1,500 predicted cumulative posthumous citations are omitted in the pictures above, solely to help its legibility (they are included in the statistical analysis).

Appendix E: Collecting Data on Recognition Events

Identifying recognition events for the deceased and the still-living. We collect events recorded in academic journals that celebrate, recognize, or memorialize the scientists in our sample, whether they are deceased or still-living. The challenge is to do so in a manner this consistent over time and does not entail a built-in recognition bias in favor of the deceased. To do so, we rely on *PubMed*, a publicly available bibliometric database curated by the Library of Medicine, which contains, as of 2019, 29 million records for the biomedical research literature, life science journals, and online books. The coverage of this database is extensive, both in its depth (with more than 5,000 journals indexed) and its longitudinal dimension (with comprehensive coverage of the english-language research literature since the early 1950s).

Helpfully, every publication in *PubMed* is tagged by one or more of 80 distinct publication types ("Letters," "Journal Articles," "Meta-Analysis," "Randomized Controlled Trial"...). Ten of these publication types could potentially denote a recognition event: Autobiography, Bibliography, Biography, Collected Works, Festschrift, Interview, Introductory Journal Article, Lectures, Personal Narrative, and Portrait. Focusing on the 413,611 articles tagged by one of these publication type contained in the 2019 version of *PubMed*, we extract 22,912 articles whose title include the last name of one of the scientist, and either his/her first name or middle name. We then handcode each of these records to filter out those that do not pertain to one of the 9,046 scientists in the sample, but rather to an homonym. The resulting dataset contains 5,850 individual articles.

We then classify each of these articles into five mutually exclusive categories: obituaries, festschrifts, interviews, awards and medals, and a residual category which include events such as a republished "classic" articles with a commentary, reminiscences about the role of a scientist in the history of his/her field, autobiographical notes, etc. The first two rows of Table E1 provide a breakdown of the number of articles by category, separately for deceased and still-living scientists. While there are more events overall in the control sample, this reflects that the ratio of deceased to control scientists is roughly $1:12$. Per scientist, there are many more events for the deceased than for the still living (1.74 vs. 0.52 on average).

An oddity is that 9% of the control scientists have an obituary written about them. Recall that in order to contribute an article to the control sample, an elite scientist must be alive five years after the year of death for the deceased scientist with whom s/he is matched. Yet, they might have died in the ensuing years. More typically, many of the other types of recognition events for the still-living scientists arrive in the twilight of their careers, or after they have retired.

In order to compare the intensity of recognition between the prematurely deceased and still-alive scientists, we leverage our research design. Recall that a byproduct of the matching procedure at the article level (cf. Appendix C) is to generate a counterfactual year of death for each elite scientist whose articles match those of treated scientists. This counterfactual year of death provides a temporal anchor to compare recognition for the deceased as well as the living. A slight complication arises since the same scientist can serve as control multiple times, for different treated scientists who passed away in different years between 1969 and 2003. As a result, there is typically more than one counterfactual year of death for each control scientist. To get around this problem, we simply select one of the possible counterfactual years of death for each living scientist at random. We then use a window of one year before until four years after the year of death (or counterfactual death) symmetrically for deceased and control scientists, and simply sum the number of recognition events for each scientist within that window.

The third and fourth rows of Table E1 break down the recognition events after filtering out events that fall outside of this design-inspired window. By construction, every control scientist is alive during that time period, which implies that the number of obituaries for these scientists is exactly zero. In fact, only 6% of the control scientists are recognized at all during the window, versus 49% of the deceased scientists.

Figure E1, Panels A and B display the corresponding histograms for the total number of recognition events, broken down by treatment status. Two facts should be emphasized. First, the distribution of recognition is

extremely skewed for deceased and still-living scientists. Second, per scientist the deceased are recognized much more intensely than the living.

A finer-grained look at memorialization for the deceased. The focus on academic memory events was justified in light of the fact that they are recorded consistently over time, and that the set of criteria used to collect them does not entail a bias that mechanically produces more events for deceased scientists. At the same time, it is clear that deceased scientists are memorialized through more diverse channels than simply by publications in the biomedical literature. The second part of this appendix attends to this diversity by systematically collecting memory events for deceased scientists, regardless of source.

To get a broader view of memorialization, we add to the academic literature search systematic internet Google searches. Specifically, we searched for the scientists name, degree, and death year (e.g, `John Gibbon, MD 1973`). We categorized the valid search results as university web posts, *New York Times* and other newspaper obituaries, *Wikipedia* pages, and miscellaneous online obituaries. We labeled these memories "popular memories," and we found an average of just over two per scientist. Table E2 reports basic statistics on the classification of memory events by type.

Below we report additional results that seek to provide more context and some nuance for understanding the results reported in Section 4.2.1. "Estimating the Determinants of Recognition." In particular, the data on recognition events including still living scientists was sparse, precluding an analysis of its intensity, and the key outcome of interest was simply the presence of at least one event in the design window of $[-1; 5]$ years around the year of death/counterfactual death. Figure E2 displays the histogram for the distribution of total memory events (i.e., "academic"+"popular") in the sample of 720 deceased scientists only. Twenty one (2.9%) scientists in the sample are never memorialized, which means that their passing was ascertained from the social security administration death index, or a mention in a publication that appeared after their death.

Figure E3 displays the memorialization-age gradient in the raw data. Older scientists do tend to get memorialized more intensely, but the difference is especially stark for the relatively small number of scientists who die at a very advanced age (75 years old and up), but before retiring from research activities. One way to interpret these findings is that scientists who remain productive and "at the top of their game" very late in life are "forces of nature" whose very longevity invites a vigorous memorialization effort.[xii]

Who are the memorializers? For the 720 deceased scientists, we examine the authors of academic memory events and identify 1,332 unique memorializer/deceased pairs for the 1,256 academic memory events in the dataset for which we can obtain a *PubMed* article identifier (74.89% of the 1,677 total academic memories). For 1,025 (76.95%) of these pairs, the full text is available from *PubMed* and we can determine the type of relationship that exist between memorializing and memorialized individuals. We consider three types of relationships. The first category is social, as in the case of a former collaborator, mentor, or trainee. The second type corresponds to intellectual linkages, as in the case of a colleague or editor of a journal in the same field. The third basis for relationships is purely organizational, as in the case of department colleagues within the same institution. This leaves a small residual category of memory events written by historians and journalists with no obvious relationship with the deceased.[xiii] The proportion of relationships that fall in each category are reported in Table E3.

Almost 60% of these relationships are social in nature, and only 15% of the memorializers appear to not have been proximate with the deceased in either the social, intellectual, or spatial dimension. The vanguard of the salesforce is therefore drawn from a fairly narrow set of "satellites" that gravitated around the star

[xii] Note that this does not contradict the results presented in Panel B of Figure 5, which demonstrated that at every age, but particularly for the young, deceased elite scientists tend to get recognized more than still-living elite scientists.

[xiii] We code these relationships to make the categories mutually exclusive: social relationships that are also intellectual or geographic are classified as social; intellectual relationships that are also organizational (but not social) are classified as intellectual; and only purely organizational relationships are classified as such. The residual category comprises all relationships for which we could exclude a social, intellectual, and organizational connection.

while s/he was alive. In contrast, in Table 6, we reported that the death event appeared to mobilize citers of all stripes; in particular, we observed no difference between the effect on the citing behavior of former collaborators versus those who had never collaborated with the departed scientist.

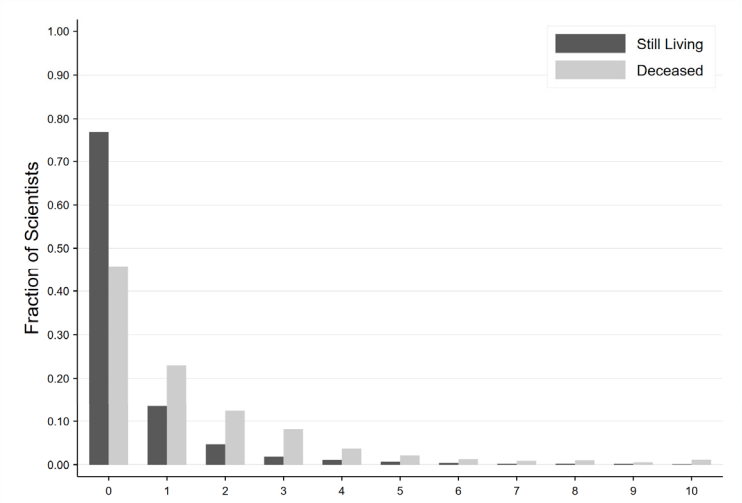
The evidence is therefore consistent with a particular sequence unfolding after the death event where close associates take on the burden of memorializing the deceased, and in certain conditions this triggers a much wider and diffuse response that expresses itself in the form of an elevated propensity to cite the work of the deceased.

Determinants of memorialization. Table E4 reproduces the analysis presented in Table 7, with two important modifications. First the sample is limited to the set of 720 deceased scientists. Second, we use Poisson specifications (with robust, quasi-maximum likelihood standard errors) rather than logit specifications since there is enough variation in this more limited sample to model the intensive memorialization margin together with the extensive memorialization margin. The results are qualitatively similar, except for the *Self-Promoter* indicator variable, which appears to correlate positively with the intensity of memorialization. Table E5 reproduces Table E4, with the small twist that NAS Biographical memoirs are omitted from the count of academic memory events—one might be concerned that the correlation between NAS membership and memorialization intensity reflects the built-in memorialization channel that the National Academy of Sciences has created to celebrate the career accomplishments of its deceased members. However, compared with Table E4, this results only in a slight attenuation of the coefficient for NAS Membership. All other coefficients remain substantively unchanged.

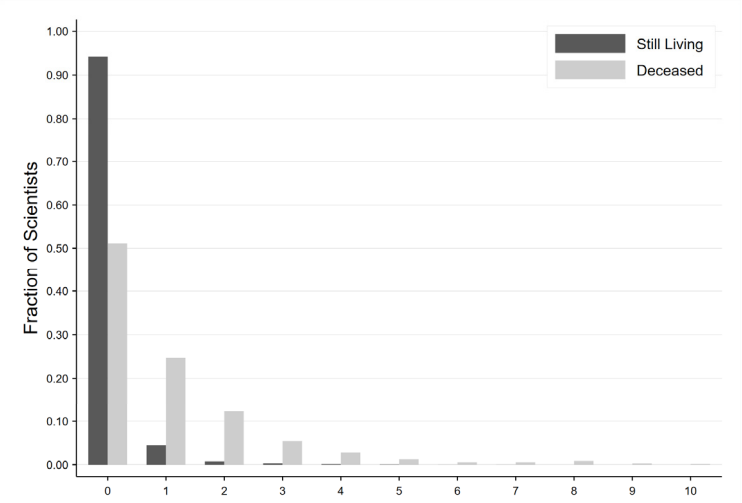
For the purposes of probing the robustness of these results, we also ran identical analyses to that presented in Table E4, but using the number of "popular" memory events, as well as the overall number of memory events (i.e., the sum of academic and "popular" events) as an outcome. The results suggest a broadly similar pattern, but with attenuated magnitudes and noisier estimates for some of the coefficients of interest. These results are reported in Tables E6 and E7.

Figure E1
Distribution of Academic Recognition Events

A. As of 2019

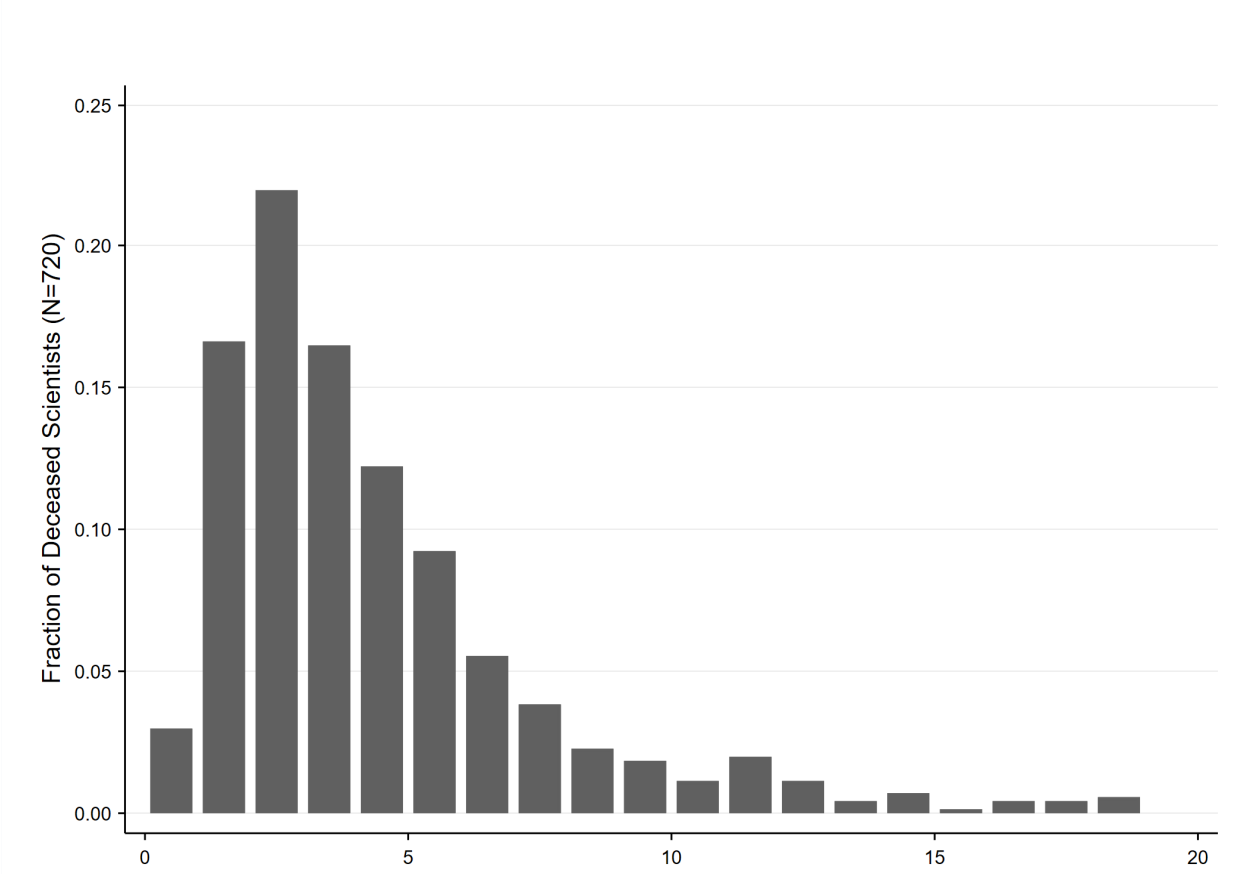


B. Within the Design Window



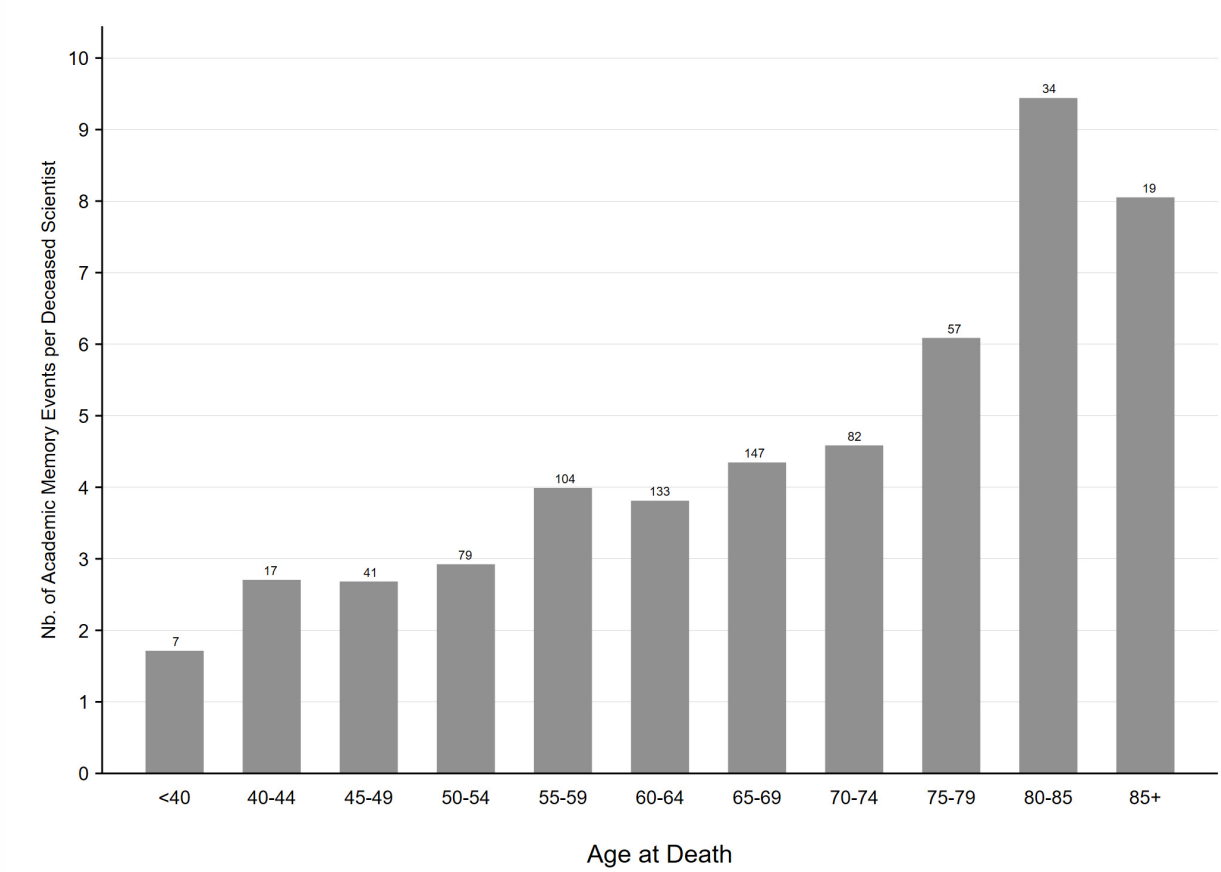
Note: These histograms depict the distribution of the number of academic "recognition events" in the sample of 720 deceased scientists and 8,326 still living scientists. An academic recognition event corresponds to an article indexed by *PubMed* that references the name of a scientist and is of the following types: obituary; festschrift; biography/autobiography; ceremony after an award/medal; interview; as well as a miscellaneous category (see the text of the appendix for more details). In Panel A, the cumulative stock of such events is tabulated over the entire career until April 2019. In contrast, Panel B leverages our research design, by anchoring the analysis around the time window between one year before death and five years after death (or counterfactual death for the matched control scientists).

Figure E2
Distribution of Memory Events for the Deceased Scientists



Note: Histogram for the total number of memory events ("academic" and "popular") in the sample of 720 deceased scientists. A memory event can be academic (e.g., an obituary published in a journal, an award or a lecture named after the scientist, a symposium organized in his/her memory, or a NAS biographical memoir) or popular (e.g., a Wikipedia page, an obituary published in a newspaper or magazine, a web posting, etc.). Nineteen scientists (2.6%) in the sample are never memorialized, which means that their passing was ascertained from the death index from the social security administration, or a mention in a publication that appeared after the death. The figure omits thirteen (1.8%) scientists with more than 20 memory events. The five most memorialized scientists in the sample are: Henry Kunkel (known for his discoveries in basic immunology, 25 events); Sidney Farber (who pioneered modern chemotherapy, 26 events); Peter Safar (who pioneered cardiopulmonary resuscitation, 27 events); John H. Gibbon, Jr., (inventor of the heart-lung machine, 35 events); and Jonas Salk (discoverer of the polio vaccine, 65 events).

Figure E3
Age and Memorialization Intensity for the Deceased Scientists



Note: Number of academic memory events per deceased scientist, by age bracket. The numbers at the top of each bar indicate the number of scientists in the sample who died in the corresponding age bracket.

Table E1: Summary Statistics for Academic Recognition Events (N=9,046 Scientists)

			Obituary	Festschrift	Interview	Award/ Medal	Misc.	At least one event	Total
As of 2019	Still Living	Total Nb. of Events	847	198	737	776	1,766	1,944	4,324
		Average per scientist	0.102	0.024	0.089	0.093	0.212	0.233	0.519
	Deceased	Total Nb. of Events	511	58	17	54	616	399	1,256
		Average per scientist	0.710	0.081	0.024	0.075	0.856	0.554	1.744
Within the design window	Still Living	Total Nb. of Events	0	55	146	153	300	479	654
		Average per scientist	0.000	0.007	0.018	0.018	0.036	0.058	0.079
	Deceased	Total Nb. of Events	464	35	4	9	265	354	777
		Average per scientist	0.644	0.049	0.006	0.013	0.368	0.492	1.079

Note: The cross-tabulations above breakdown the number of academic "recognition events" in the sample of 720 deceased scientists and 8,326 still living scientists, by type of event: Obituary, Festschrift, Interview, Award/Medal, and a miscellaneous category (see the text of the appendix for more details). In the first two rows, the cumulative stock of such events is tabulated over the entire career until June 2019. In contrast, the third and fourth rows leverage our research design, by anchoring the analysis around the time window between one year before death and five years after death (or counterfactual death for the matched control scientists).

Table E2: Summary Statistics for Memory Events, Deceased Scientists Only (N=720)

		Count	Mean	Median	Std. Dev.	Min.	Max.
Academic	Festschrift/Memorial Symposium	66	0.092	0	0.312	0	2
	Obituary in an Academic journal	1,518	2.108	1	2.997	0	24
	NAS Biographical Memoir	93	0.129	0	0.393	0	6
	Total Nb. Memory Events in Academic Publications	**1,677**	**2.329**	**1**	**3.202**	**0**	**30**
Popular	New York Times Obituary	237	0.329	0	0.479	0	3
	Other Newspaper Obituary	327	0.454	0	0.941	0	17
	University Web Post	351	0.487	0	0.713	0	5
	Misc. Web Post	382	0.531	0	1.680	0	34
	Wikipedia page	183	0.254	0	0.436	0	1
	Total Nb. of "Popular" Memory Events	**1,480**	**2.056**	**1**	**2.742**	**0**	**54**
Total	**Total Nb. of Memory Events**	**3,157**	**4.385**	**3**	**4.845**	**0**	**65**

Note: The number of academic memories listed in this table is considerably higher than that in Table E1 (1,677 versus 1,256), as this table includes academic memories which were not recorded by *PubMed*.

Table E3: Memorializers' Relationships to Deceased Elite Scientists

Type of Relationship	Specific Connection	Percentage of Sample
Social	Trainee	36.97%
	Collaborator	20.49%
	Family	0.39%
	Trained together	0.58%
		58.43%
Intellectual	Colleague in same field	17.07%
	Journal editor	1.37%
		18.44%
Organizational	Shared employer	8.10%
		8.10%
None	No social relation	9.46%
	Historian	2.54%
	Journalist	3.02%
		15.02%

Note: The percentages correspond to the fraction of 1,025 memorializer-deceased pairs that have a particular characteristic (e.g., the deceased and the memorializer are in the same institution) and for which information was available from *PubMed*. The different categories have been defined to be mutually exclusive, i.e., social relationships that are also intellectual or geographic are classified as social; intellectual relationships that are also organizational (but not social) are classified as intellectual; and only purely organizational relationships are classified as such. The residual category comprises all relationships for which we could exclude a social, intellectual, and spatial connection.

Table E4: Estimating the Determinants of Academic Memories

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Ln(cmltv. citations at death)		0.245**			0.053	0.245**	0.202**	0.158*
		(0.042)			(0.067)	(0.049)	(0.061)	(0.065)
Ln(cmltv. publications at death)			0.449**		0.385**			
			(0.054)		(0.087)			
Ln(cmltv. funding at death)				0.074†	0.007			
				(0.041)	(0.037)			
Member of the NAS		0.595**	0.675**	0.757**	0.644**	0.595**	0.625**	0.628**
		(0.101)	(0.091)	(0.092)	(0.096)	(0.101)	(0.105)	(0.108)
Ln(Nb. of past trainees)						-0.004		-0.006
						(0.053)		(0.054)
Ln(Nb. of past coauthors [non-trainees])							0.078	0.098
							(0.070)	(0.071)
Self-Promoter								0.192*
								(0.089)
Female	-0.173	-0.062	0.007	-0.155	0.001	-0.062	-0.065	-0.082
	(0.183)	(0.167)	(0.163)	(0.165)	(0.162)	(0.169)	(0.168)	(0.173)
Death is Sudden	0.125	0.141	0.145†	0.118	0.152†	0.141	0.142	0.142
	(0.092)	(0.087)	(0.088)	(0.089)	(0.087)	(0.088)	(0.087)	(0.087)
Nb. of Scientists	720	720	720	720	720	720	720	720
Pseudo-R^2	0.157	0.227	0.238	0.207	0.239	0.227	0.228	0.231

Note: Estimates stem from Poisson specifications. The sample consists of a cross-section of the 720 deceased scientists. The dependent variable is the total number of academic memory events created for a scientist posthumously. All models include—but do not report—controls for degree type (PhD and MD/PhD, MD is the omitted category), a suite of indicator variables for each death year, six indicator variables for age categories at the time of death (less than 45 years old; between 45 and 55 years old; between 55 and 60 years old; between 60 and 65 old; between 65 and 70 years old; between 70 and 75 years old; above 75 years old is the omitted category), and an indicator variable for unknown cause of death. Self-Promoter is an indicator variable equals to one for investigators above the median in term of self-promotional behavior (as assessed by the number of unrelated—i.e., out-of-subfield—self-citations as a proportion of all citations in a deceased scientist's entire body of work).

Robust standard errors in parentheses, clustered at the level of the star scientist. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table E5: Estimating the Determinants of Non-NAS Academic Memories

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Ln(cmltv. citations at death)		0.267**			0.072	0.269**	0.226**	0.181**
		(0.045)			(0.071)	(0.052)	(0.064)	(0.069)
Ln(cmltv. publications at death)			0.474**		0.388**			
			(0.058)		(0.092)			
Ln(cmltv. funding at death)				0.079†	0.006			
				(0.043)	(0.039)			
Member of the NAS		0.400**	0.490**	0.579**	0.451**	0.402**	0.429**	0.434**
		(0.107)	(0.099)	(0.101)	(0.102)	(0.107)	(0.111)	(0.114)
Ln(Nb. of past trainees)						-0.008		-0.011
						(0.056)		(0.057)
Ln(Nb. of past coauthors [non-trainees])							0.075	0.096
							(0.074)	(0.076)
Self-Promoter								0.203*
								(0.092)
Female	-0.192	-0.068	-0.001	-0.172	-0.007	-0.069	-0.072	-0.089
	(0.184)	(0.173)	(0.169)	(0.171)	(0.168)	(0.174)	(0.173)	(0.179)
Death is Sudden	0.114	0.138	0.137	0.112	0.146	0.137	0.138	0.138
	(0.095)	(0.091)	(0.091)	(0.092)	(0.091)	(0.092)	(0.091)	(0.091)
Nb. of Scientists	720	720	720	720	720	720	720	720
Pseudo-R^2	0.150	0.204	0.214	0.181	0.216	0.204	0.205	0.208

Note: Estimates stem from Poisson specifications. The sample consists of a cross-section of the 720 deceased scientists. The dependent variable is the total number of academic memory events created for a scientist posthumously, but in contrast to the results reported in Table E4, the count has been modified to exclude NAS Biographical Memoirs. All models include—but do not report—controls for degree type (PhD and MD/PhD, MD is the omitted category), a suite of indicator variables for each death year, six indicator variables for age categories at the time of death (less than 45 years old; between 45 and 55 years old; between 55 and 60 years old; between 60 and 65 old; between 65 and 70 years old; between 70 and 75 years old; above 75 years old is the omitted category), and an indicator variable for unknown cause of death. Self-Promoter is an indicator variable equals to one for investigators above the median in term of self-promotional behavior (as assessed by the number of unrelated—i.e., out-of-subfield—self-citations as a proportion of all citations in a deceased scientist's entire body of work).

Robust standard errors in parentheses, clustered at the level of the star scientist. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table E6: Estimating the Determinants of Memorialization – Popular Memories

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Ln(cmltv. citations at death)		0.049			0.006	0.064	0.042	0.039
		(0.032)			(0.062)	(0.045)	(0.056)	(0.069)
Ln(cmltv. publications at death)			0.109*		0.154†			
			(0.043)		(0.088)			
Ln(cmltv. funding at death)				-0.083	-0.103			
				(0.103)	(0.103)			
Member of the NAS		0.509**	0.516**	0.602**	0.559**	0.499**	0.511**	0.493**
		(0.146)	(0.146)	(0.103)	(0.114)	(0.128)	(0.168)	(0.152)
Ln(Nb. of past trainees)						0.068		0.069†
						(0.042)		(0.041)
Ln(Nb. of past coauthors [non-trainees])							0.011	0.013
							(0.085)	(0.077)
Self-Promoter								0.113
								(0.086)
Female	0.108	0.110	0.133	0.069	0.137	0.091	0.108	0.085
	(0.125)	(0.118)	(0.117)	(0.119)	(0.120)	(0.115)	(0.118)	(0.114)
Death is Sudden	0.020	0.015	0.016	-0.008	-0.002	0.015	0.013	0.011
	(0.099)	(0.106)	(0.106)	(0.087)	(0.086)	(0.099)	(0.106)	(0.099)
Nb. of Scientists	720	720	720	720	720	720	720	720
Pseudo-R^2	0.107	0.130	0.131	0.134	0.139	0.137	0.131	0.139

Note: Estimates stem from Poisson specifications. The sample consists of a cross-section of the 720 deceased scientists. The dependent variable is the total number of "popular" memory events created for a scientist posthumously. A popular memory is a university web post, New York Times obituary, other newspaper obituary, Wikipedia page, or miscellaneous online obituary. All models include—but do not report—controls for degree type (PhD and MD/PhD, MD is the omitted category), a suite of indicator variables for each death year, six indicator variables for age categories at the time of death (less than 45 years old; between 45 and 55 years old; between 55 and 60 years old; between 60 and 65 old; between 65 and 70 years old; between 70 and 75 years old; above 75 years old is the omitted category), and an indicator variable for unknown cause of death. Self-Promoter is an indicator variable equals to one for investigators above the median in term of self-promotional behavior (as assessed by the number of unrelated—i.e., out-of-subfield—self-citations as a proportion of all citations in a deceased scientist's entire body of work).

Robust standard errors in parentheses, clustered at the level of the star scientist. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table E7: Estimating the Determinants of Memorialization – Total Memories

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Ln(cmltv. citations at death)		0.148**			0.025	0.157**	0.119*	0.099†
		(0.031)			(0.053)	(0.038)	(0.047)	(0.054)
Ln(cmltv. publications at death)			0.284**		0.281**			
			(0.039)		(0.070)			
Ln(cmltv. funding at death)				-0.000	-0.043			
				(0.071)	(0.068)			
Member of the NAS		0.560**	0.602**	0.685**	0.605**	0.557**	0.578**	0.572**
		(0.098)	(0.095)	(0.081)	(0.087)	(0.093)	(0.109)	(0.105)
Ln(Nb. of past trainees)						0.022		0.021
						(0.038)		(0.038)
Ln(Nb. of past coauthors [non-trainees])							0.051	0.061
							(0.063)	(0.060)
Self-Promoter								0.154*
								(0.072)
Female	-0.008	0.050	0.098	-0.024	0.095	0.043	0.046	0.029
	(0.120)	(0.105)	(0.103)	(0.106)	(0.104)	(0.105)	(0.106)	(0.106)
Death is Sudden	0.077	0.082	0.082	0.061	0.078	0.083	0.080	0.080
	(0.076)	(0.077)	(0.077)	(0.072)	(0.070)	(0.076)	(0.077)	(0.075)
Nb. of Scientists	720	720	720	720	720	720	720	720
Pseudo-R^2	0.142	0.206	0.214	0.192	0.217	0.208	0.207	0.211

Note: Estimates stem from Poisson specifications. The sample consists of a cross-section of the 720 deceased scientists. The dependent variable is the total number of memory events created for a scientist posthumously. Total memories is the sum of both popular and academic memories. All models include—but do not report—controls for degree type (PhD and MD/PhD, MD is the omitted category), a suite of indicator variables for each death year, six indicator variables for age categories at the time of death (less than 45 years old; between 45 and 55 years old; between 55 and 60 years old; between 60 and 65 old; between 65 and 70 years old; between 70 and 75 years old; above 75 years old is the omitted category), and an indicator variable for unknown cause of death. Self-Promoter is an indicator variable equals to one for investigators above the median in term of self-promotional behavior (as assessed by the number of unrelated—i.e., out-of-subfield—self-citations as a proportion of all citations in a deceased scientist's entire body of work).

Robust standard errors in parentheses, clustered at the level of the star scientist. $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Appendix F: List of 720 Deceased Elite Scientists

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
Lester R. Dragstedt	[1893-1975]	MD/PhD, 1921	sudden	University of Chicago School of Medicine	Pathogenesis of Peptic and Gastric Ulcer
Jerzy Neyman	[1894-1981]	PhD, 1924	sudden	University of California — Berkeley	founder of modern theoretical statistics
Ralph D. Lillie	[1896-1979]	MD, 1926	sudden	Louisiana State University Health Sciences Center New Orleans	Histochemistry of Pigments and Carcinoid Tumors
Robert K.-S. Lim	[1897-1969]	PhD, 1920	anticipated	Miles Medical Science Research Laboratories	neurophysiology of pain
Ernst Simonson	[1898-1974]	MD, 1924	sudden	University of Minnesota School of Medicine	cardiology and physiology
Owen H. Wangensteen	[1898-1981]	MD/PhD, 1925	sudden	University of Minnesota School of Medicine	Origin and Nature of Acid Peptic Ulcer
Fritz A. Lipmann	[1899-1986]	MD/PhD, 1928	anticipated	Rockefeller University	Glucose Transport in Normal and Malignant Cells
Leo T. Samuels	[1899-1978]	PhD, 1930	unknown	University of Utah School of Medicine	Steroid Transfer in Normal and Malignant Endocrine Cells
Thomas Francis, Jr.	[1900-1969]	MD, 1925	sudden	University of Michigan School of Medicine	physician, virologist, and epidemiologist
Harold P. Morris	[1900-1982]	PhD, 1930	sudden	Howard University College of Medicine	Induction-Continuation-Genetics of Experimental Tumors
J. Murray Steele	[1900-1969]	MD, 1925	sudden	New York University School of Medicine	Bidirectional Movement of Ions Across the Intestines
Gottfried S. Fraenkel	[1901-1984]	PhD, 1925	sudden	University of Illinois at Urbana-Champaign	insect physiology and behavior
Ernest Witebsky	[1901-1969]	MD, 1926	sudden	SUNY at Buffalo School of Medicine and Biomedical Sciences	Serological Specificity of Normal and Cancer Tissues
Alexander B. Gutman	[1902-1973]	MD/PhD, 1928	sudden	Mount Sinai School of Medicine	Purine Metabolism and Gouty Arthritis
John E. Howard	[1902-1985]	MD, 1928	sudden	Johns Hopkins University School of Medicine	Calcium Metabolism and Skeletal Physiology
Sidney Farber	[1903-1973]	MD, 1927	sudden	Harvard Medical School	Chemotherapy of Cancer and Related Biological Studies
John H. Gibbon, Jr.	[1903-1973]	MD, 1927	sudden	University of Pennsylvania School of Medicine	inventor of heart-lung machine
Hans Popper	[1903-1988]	MD/PhD, 1944	anticipated	Mount Sinai School of Medicine	correlation of structure and function in liver disease
J. Herbert Conway	[1904-1969]	MD, 1930	sudden	Weill Medical College — Cornell University	Studies on the Homotransplantation of Tissues
Grace A. Goldsmith	[1904-1975]	MD, 1932	anticipated	Tulane School of Public Health and Tropical Medicine	B Group of Vitamins in Human Nutrition
James D. Hardy	[1904-1985]	PhD, 1930	anticipated	University of Mississippi Medical Center	Temperature Regulation and Brain Stem Neuronal Activity
John H. Lawrence	[1904-1991]	MD, 1930	sudden	University of California — Berkeley	Erythropoietin and Marrow By Positron Scanning
Jack Schultz	[1904-1971]	PhD, 1929	sudden	University of Pennsylvania School of Medicine	Cytochemical Studies of the Nature and Function of Genes
Wendell M. Stanley	[1904-1971]	PhD, 1929	sudden	University of California — Berkeley	Mechanism of Antibody Specificity
Cesare G. Tedeschi	[1904-1974]	MD, 1928	unknown	Metrowest Medical Center	Thymus, Lymphoid Tissue and Adipose Tissue
S. Bernard Wortis	[1904-1969]	MD, 1927	sudden	New York University School of Medicine	Sympathetic Activity and Addiction
Jacob Yerushalmy	[1904-1973]	PhD, 1930	unknown	University of California — Berkeley	Biologic & Environmental Factors in Child Development
Morris B. Bender	[1905-1983]	MD, 1930	sudden	Mount Sinai School of Medicine	Neurophysiologic Aspects of Visual Discrimination
Chandler McC. Brooks	[1905-1989]	PhD, 1931	sudden	SUNY Downstate Medical Center	Neurophysiological Study of Neuroendocrine Activity
Charles K. Friedberg	[1905-1972]	MD, 1929	sudden	Mount Sinai School of Medicine	Effects of Exercise and Drugs in Heart Block
Thomas F. Gallagher	[1905-1975]	PhD, 1931	unknown	Montefiore Medical Center	steroid hormone production and metabolism in cancer
Per F. Scholander	[1905-1980]	MD/PhD, 1934	sudden	UCSD School of Medicine	Secretion of Gases in the Swimbladder of Fishes
Tracy M. Sonneborn	[1905-1981]	PhD, 1928	sudden	Indiana University at Bloomington	Normal and Abnormal Cell Growth and Heredity
Lyman C. Craig	[1906-1974]	PhD, 1931	unknown	Rockefeller University	Purification and Structure of Active Principles
Max Delbrück	[1906-1981]	PhD, 1930	anticipated	California Institute of Technology	replication mechanism and the genetic structure of viruses
Karl A. Folkers	[1906-1997]	PhD, 1931	sudden	University of Texas at Austin	peptide antagonists of LHRH as gonadotropin inhibitors
Frank L. Horsfall, Jr.	[1906-1971]	MD, 1932	anticipated	Memorial Sloan-Kettering Cancer Center	Immunological Studies of Atypical Pneumonia
William Pomerance	[1906-1978]	MD, 1929	anticipated	NIH/NCI	Gynecologic Oncology
Berta V. Scharrer	[1906-1995]	PhD, 1930	anticipated	Albert Einstein College of Medicine	Immunocytochemical Study of Invertebrate Nervous System
Henry A. Schroeder	[1906-1975]	MD, 1933	unknown	Dartmouth Medical School	Abnormal Trace Metals in Cardiovascular Diseases
Nathan W. Shock	[1906-1989]	PhD, 1930	anticipated	NIH/NIA	Physiological Studies of Aging in the Heart, Kidneys, and Lungs
S. Smith Stevens	[1906-1973]	PhD, 1933	sudden	Harvard University	Psychophysics and Hearing
Georges Ungar	[1906-1977]	MD, 1939	unknown	University of Tennessee Health Sciences Center	Chemical Transfer of Drug Tolerance and Learned Behavior
Dan H. Campbell	[1907-1974]	PhD, 1935	sudden	California Institute of Technology	Researches on Blood and Immunochemistry
Morton J. Hamburger	[1907-1970]	MD, 1934	sudden	University of Cincinnati College of Medicine	Studies in Staphylococcal Infection
Michael J. Hogan	[1907-1976]	MD, 1930	anticipated	UCSF School of Medicine	Studies on Ocular Dystrophies and Extraocular Muscles
Leslie A. Stauber	[1907-1973]	PhD, 1937	sudden	Rutgers University	Visceral Leishmaniasis in Experimental Animals
Alexander S. Wiener	[1907-1976]	MD, 1930	anticipated	New York University School of Medicine	Blood Groups in Non-Human Primates
Harland G. Wood	[1907-1991]	PhD, 1935	anticipated	Case Western Reserve University School of Medicine	heterotrophic carbon dioxide fixation
Benjamin Alexander	[1908-1978]	MD, 1934	unknown	New York Blood Center	Coagulation, Hemorrhage, and Thrombosis
William F. Caveness	[1908-1981]	MD, 1943	anticipated	NIH	authority on head injuries
David G. Cogan	[1908-1993]	MD, 1932	sudden	NIH/NEI	Metabolism of the Normal and Abnormal Ocular Lens
John P. Fox	[1908-1987]	MD/PhD, 1936	unknown	University of Washington School of Medicine	Rhinovirus Immunology and Epidemiology
Herman M. Kalckar	[1908-1991]	MD/PhD, 1939	sudden	Boston University Medical Center	Genes, Enzymes, Nucleotides, and Carbohydrate Patterns
Maurice Lev	[1908-1994]	MD, 1934	anticipated	Rush-Presbyterian-St Luke's Medical Center	Studies of Congenital Heart Disease
Carl V. Moore	[1908-1972]	MD, 1932	sudden	Washington University in St. Louis School of Medicine	Erythropoiesis and Iron Metabolism
Alvin M. Pappenheimer, Jr.	[1908-1995]	PhD, 1932	sudden	Harvard University	Biology of Diphtheria
George K. Smelser	[1908-1973]	PhD, 1932	sudden	Columbia University College of Physicians & Surgeons	Electron Microscopy of the Eye
Abraham White	[1908-1980]	PhD, 1931	sudden	Stanford University School of Medicine	Biochemical Studies of Lymphoid Tissue
Geoffrey H. Bourne	[1909-1988]	PhD, 1943	sudden	Emory University School of Medicine	Ultrastructural Changes in Scorbutic Tissues
Jacob W. Dubnoff	[1909-1972]	PhD, 1945	anticipated	USC Keck School of Medicine	Active Forms of Vitamin B12 and Sulfhydryl Groups
R. Gordon Gould	[1909-1978]	PhD, 1933	anticipated	Stanford University School of Medicine	Cholesterol Metabolism and Hypocholesterolemic Drugs
Thomas D. Kinney	[1909-1977]	MD, 1936	anticipated	Duke University School of Medicine	Subcellular Pathology of Ferritin Transport
V. Everett Kinsey	[1909-1978]	PhD, 1937	sudden	Oakland University	Intraocular Fluid Dynamics
Koloman Laki	[1909-1983]	PhD, 1936	sudden	NIH/NIADDK	Discovery of blood-clotting Factor XIII
Carl L. Larson	[1909-1978]	MD, 1939	anticipated	University of Montana at Missoula	Nonspecific Resistance To Viral-Induced Tumors
Francis C. Lowell	[1909-1979]	MD, 1936	sudden	Harvard Medical School/Massachusetts General Hospital	Allergy of the respiratory tract
Walsh McDermott	[1909-1981]	MD, 1934	sudden	Weill Medical College — Cornell University	Latent and Dormant Microbial Infections
Erwin Neter	[1909-1983]	MD, 1934	sudden	Children's Hospital of Buffalo	Study of Bacterial Toxins and Hemagglutination
David D. Rutstein	[1909-1986]	MD, 1934	sudden	Harvard Medical School	Preventive Medicine
Robert H. Williams	[1909-1979]	MD, 1934	sudden	University of Washington School of Medicine	Diabetes Etiology, Pathogenesis, and Management
Ernest Bueding	[1910-1986]	MD, 1936	anticipated	Johns Hopkins School of Hygiene and Public Health	Comparative Biochemistry of Parasitic Helminths
Albert S. Gordon	[1910-1992]	PhD, 1934	sudden	New York University School of Medicine	Humoral Control of Blood Cell Formation and Release
David E. Green	[1910-1983]	PhD, 1934	anticipated	University of Wisconsin School of Medicine	molecular biology of membrane systems
Werner Henle	[1910-1987]	MD, 1934	anticipated	University of Pennsylvania School of Medicine	serologic response to epstein-barr virus infection
Alexander D. Langmuir	[1910-1993]	MD, 1935	anticipated	Johns Hopkins School of Hygiene and Public Health	Infectious diseases surveillance
George V. Taplin	[1910-1979]	MD, 1936	anticipated	UCLA School of Medicine	radioactive albumin macroaggregates for the detection of pulmonary embolism
Paul M. Aggeler	[1911-1969]	MD, 1937	anticipated	UCSF School of Medicine	discovery of the plasma thromboplastin component
Frank A. Beach	[1911-1988]	PhD, 1940	sudden	University of California — Berkeley	Hormonal Control Over Social Interactions
Ernest Borek	[1911-1986]	PhD, 1938	unknown	AMD Cancer Research Center	molecular biology of ethionine carcinogenesis

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
William J. Bowen	[1911-1970]	PhD, 1936	sudden	NIH/NIAMD	Studies of enzymes involved in the release of chemical energy
Edward W. Dempsey	[1911-1975]	PhD, 1937	sudden	Columbia University College of Physicians & Surgeons	Mechanisms of Formation and Destruction of Myelin
Michael Doudoroff	[1911-1975]	PhD, 1939	anticipated	University of California — Berkeley	bacteriology and immunology
Jordi Folch-Pi	[1911-1979]	MD, 1932	sudden	Harvard Medical School/Massachusetts General Hospital	Biochemistry of the Mucolipids of the Nervous System
William T. Niemer	[1911-1971]	PhD, 1946	sudden	Creighton University School of Medicine	Influence of Telencephalon nn the Hypothalamus
Charles H. Rammelkamp, Jr.	[1911-1981]	MD, 1937	sudden	Case Western Reserve University School of Medicine	early studies on the clinical application & mechanism of action of antimicrobials
Yoshio Sato	[1911-1972]	PhD, 1946	anticipated	NIH/NIAMD	Studies of Steroidal Alkaloids
Reidar F.A. Sognnaes	[1911-1984]	DDS/PhD, 1941	sudden	UCLA School of Dentistry	Studies on Forensic Dental Records
Fred H. Allen, Jr.	[1912-1987]	MD, 1938	sudden	New York Blood Center	blood grouping
Raymond T. Carhart	[1912-1975]	PhD, 1936	sudden	Northwestern University School of Medicine	audiology and otolaryngology
Albert H. Coons	[1912-1978]	MD, 1937	sudden	Harvard Medical School	bacteriology and immunology
William Likoff	[1912-1987]	MD, 1938	unknown	Hahnemann Medical College	diagnosis and prognosis of pulmonary hypertension
Daniel Mazia	[1912-1996]	PhD, 1937	anticipated	Stanford University School of Medicine	isolation of the mitotic apparatus
Hermann Rahn	[1912-1990]	PhD, 1938	anticipated	SUNY at Buffalo School of Medicine and Biomedical Sciences	Interaction of Gas Phase Diffusion and Blood Flow
Arnold M. Seligman	[1912-1976]	MD, 1937	anticipated	Johns Hopkins University School of Medicine	Experimental and Clinical Studies in Cancer Chemotherapy
Harry A. Waisman	[1912-1971]	MD/PhD, 1947	sudden	University of Wisconsin School of Medicine	Developmental Biochemistry and Mental Retardation
Arthur Cherkin	[1913-1987]	PhD, 1953	anticipated	Sepulveda VA Medical Center	neurobiology of memory
William S. Johnson	[1913-1995]	PhD, 1940	sudden	Stanford University School of Medicine	synthetic organic chemistry
Stephen W. Kuffler	[1913-1980]	MD, 1937	sudden	Harvard Medical School	Microphysiology of Synaptic Transmission
Maurice Landy	[1913-1993]	PhD, 1940	anticipated	NIH	genetic control of immune responsiveness
Choh Hao Li	[1913-1987]	PhD, 1938	anticipated	UCSF School of Medicine	isolation and synthesis the human pituitary growth hormone
Werner K. Noell	[1913-1992]	MD, 1938	unknown	University of Kansas Medical Center	Translation of Visual Cell mRNA in Model Systems
Alex B. Novikoff	[1913-1987]	PhD, 1938	anticipated	Albert Einstein College of Medicine	histochemical studies of the Golgi apparatus
Efraim Racker	[1913-1991]	MD, 1938	sudden	Weill Medical College — Cornell University	identifying and purifying Factor 1, the first part of the ATP synthase enzyme
Mindel C. Sheps	[1913-1973]	MD, 1936	anticipated	University of North Carolina at Chapel Hill School of Medicine	biostatistics and demography
Jerome R. Vinograd	[1913-1976]	PhD, 1940	sudden	California Institute of Technology	Studies of the DNA from Oncogenic Viruses
Edgar Zwilling	[1913-1971]	PhD, 1940	sudden	Brandeis University	morphogenesis of limb development in coelenterates
Frederic C. Bartter	[1914-1983]	MD, 1940	sudden	University of Texas Health Sciences Center at San Antonio	interaction between the kidney and various endocrine systems
J. Werner Braun	[1914-1972]	PhD, 1936	sudden	Rutgers University	DNA-Associated Antigens and Cancer Therapy
Paul A. Bunn	[1914-1970]	MD, 1941	sudden	University of Michigan School of Medicine	evaluation of streptomycin as a therapeutic agent for tuberculosis
Eugene P. Cronkite	[1914-2001]	MD, 1940	anticipated	Brookhaven National Laboratory	hematopoiesis and radiation injury
Thaddeus S. Danowski	[1914-1987]	MD, 1940	sudden	University of Pittsburgh School of Medicine	Serum Electrolyte Changes in Carbohydrate Metabolism
Harry A. Feldman	[1914-1985]	MD, 1939	anticipated	SUNY Upstate Medical University at Syracuse	Streptococcal Infections in a Population of Families
Aubrey Gorbman	[1914-2003]	PhD, 1940	anticipated	University of Washington School of Medicine	Hormonal Action on Central Nervous Function
Marie R. Haug	[1914-2001]	PhD, 1968	sudden	Case Western Reserve University School of Medicine	Stresses Strains and Elderly Physical Health
Fred Karush	[1914-1994]	PhD, 1938	anticipated	University of Pennsylvania School of Medicine	Interactions of Immunoglobulins
Arnost Kleinzeller	[1914-1997]	MD/PhD, 1941	anticipated	University of Pennsylvania School of Medicine	Active Sugar Transport in Renal Cells
Herschel L. Roman	[1914-1989]	PhD, 1942	sudden	University of Washington School of Medicine	Genetic Investigations in Yeast
Jonas E. Salk	[1914-1995]	MD, 1939	sudden	Salk Institute for Biological Studies	effective vaccine for polio
Klaus Schwarz	[1914-1978]	MD, 1939	sudden	UCLA School of Medicine	Selenium and Unidentified Essential Trace Elements
Sol Spiegelman	[1914-1983]	PhD, 1944	anticipated	Columbia University College of Physicians & Surgeons	nucleic acid hybridization
Edward A. Steinhaus	[1914-1969]	PhD, 1939	sudden	University of California — Irvine	The Diseases of Invertebrate Animals
Marshall R. Urist	[1914-2001]	MD, 1941	anticipated	UCLA School of Medicine	inductive substrates of tooth and bone formation
George N. Wise	[1914-1974]	MD, 1938	sudden	Albert Einstein College of Medicine	Investigation into the vascular diseases of the retina
Isadore Zipkin	[1914-1973]	PhD, 1942	anticipated	UCSF School of Medicine	Role of Fluoride in Experimental Periodontal Disease
Bernard R. Baker	[1915-1971]	PhD, 1940	sudden	University of California — Santa Barbara	Synthesis of Nucleosides for Cancer Chemotherapy
Daniel A. Brody	[1915-1975]	MD, 1940	sudden	University of Tennessee Health Sciences Center	Generator Properties of Isolated Mammalian Hearts
Marian W. Kies	[1915-1988]	PhD, 1944	sudden	NIH/NIMH	Studies of experimental allergic encephalomyelitis
Harvey C. Knowles, Jr.	[1915-1984]	MD, 1942	anticipated	University of Cincinnati College of Medicine/Children's Hospital	clinical studies of gestational diabetes
Hamish N. Munro	[1915-1994]	MD/PhD, 1956	anticipated	Tufts University School of Medicine	Nutritional Regulation of Protein Metabolism
Joseph H. Ogura	[1915-1983]	MD, 1941	sudden	Washington University in St. Louis School of Medicine	Physiology of Deglutition and Voice in Larynx Analog
John W. Porter	[1915-1984]	PhD, 1942	unknown	University of Wisconsin School of Medicine	regulation of lipogenesis by insulin and glucagon
Maurice S. Raben	[1915-1977]	MD, 1939	sudden	Tufts University School of Medicine	Humoral & Metabolic Aspects of Cardiac Function
Paul J. Scheuer	[1915-2003]	PhD, 1950	anticipated	University of Hawaii School of Medicine	The Molecular Structure of Ciguatoxin and Palytoxin
Irving J. Selikoff	[1915-1992]	MD, 1941	anticipated	Mount Sinai School of Medicine	asbestos and cancer
Elizabeth Stern	[1915-1980]	MD, 1940	anticipated	UCLA School of Medicine	effects of steroid contraception on the ovary
Earl W. Sutherland, Jr.	[1915-1974]	MD, 1942	sudden	Vanderbilt University School of Medicine	action of sympathomimetic amines and 3 5-AMP
Benjamin E. Volcani	[1915-1999]	PhD, 1941	anticipated	UCSD School of Medicine	Biochemical Studies on Siliceous Skeletal Formation
David F. Waugh	[1915-1984]	PhD, 1940	sudden	MIT	Protein Interactions and Physicochemical Properties
Christian B. Anfinsen, Jr.	[1916-1995]	PhD, 1943	sudden	Johns Hopkins University School of Medicine	protein structure and protein folding
Frederik B. Bang	[1916-1981]	MD, 1939	sudden	Johns Hopkins University School of Medicine	Upper Respiratory Antiviral Defense in Malnutrition
Joseph Cochin	[1916-1985]	MD/PhD, 1955	anticipated	Boston University Medical Center	Factors in Tolerance to the Narcotic Analgesics
Sidney P. Colowick	[1916-1985]	PhD, 1942	unknown	Vanderbilt University School of Medicine	enzymatic oxidation and phosphorylation
Norman R. Davidson	[1916-2002]	PhD, 1939	sudden	California Institute of Technology	physical chemistry of nucleic acids
Bernard D. Davis	[1916-1994]	MD, 1940	anticipated	Harvard Medical School	Membrane-Associated Ribosomes and Protein Secretion
Albert Dorfman	[1916-1982]	MD/PhD, 1944	anticipated	University of Chicago School of Medicine	biochemistry of connective tissues
Herman K. Hellerstein	[1916-1993]	MD, 1941	anticipated	Case Western Reserve University School of Medicine	Rehabilitation of cardiac patients
Henry G. Kunkel	[1916-1983]	MD, 1942	sudden	Rockefeller University	identification of MHC Class II molecules
Arnold Lazarow	[1916-1975]	MD/PhD, 1941	sudden	University of Minnesota School of Medicine	Fetal Endocrinology and Study of Diabetes and Pregnancy
Arthur E. Martell	[1916-2003]	PhD, 1941	anticipated	Texas A&M University	Reactions of Metal Chelate Compounds
Manfred M. Mayer	[1916-1984]	PhD, 1946	sudden	Johns Hopkins University School of Medicine	immunochemistry of the complement system
Frederick S. Philips	[1916-1984]	PhD, 1940	anticipated	Memorial Sloan-Kettering Cancer Center	pharmacological properties of chemotherapeutic agents and chemical carcinogenesis
David Pressman	[1916-1980]	PhD, 1940	sudden	Roswell Park Cancer Institute	membrane antigens from normal and leukemic lymphocytes
Samuel Schwartz	[1916-1997]	MD, 1943	anticipated	University of Minnesota School of Medicine	Biological and Biochemical Effects of Porphyrins
Hans-Lukas Teuber	[1916-1977]	PhD, 1947	sudden	MIT	Behavioral Effects of Brain Injury
Robert Traub	[1916-1996]	PhD, 1947	anticipated	University of Maryland School of Medicine	Studies of Certain Important Genera of Siphonaptera
Monroe E. Wall	[1916-2002]	PhD, 1941	sudden	Research Triangle Institute	isolation and chemistry of plant antitumor agents
Gregorio Weber	[1916-1997]	MD/PhD, 1947	anticipated	University of Illinois at Urbana-Champaign	application of fluorescence spectroscopy to the biological sciences
Richard J. Winzler	[1916-1972]	PhD, 1938	sudden	Florida State University	Chemistry and Metabolism of Serum Glycoproteins
Herman A. Witkin	[1916-1979]	PhD, 1939	sudden	Princeton University	Studies of Men With XYY and XXY Chromosome Complements
Murray B. Bornstein	[1917-1995]	MD, 1952	sudden	Albert Einstein College of Medicine	copolymer as a protective treatment for the exacerbation of multiple sclerosis
Abraham I. Braude	[1917-1984]	MD/PhD, 1950	sudden	UCSD School of Medicine	pathogenesis and treatment of life-threatening septic shock
James A. Campbell	[1917-1983]	MD, 1943	sudden	Rush-Presbyterian-St Luke's Medical Center	cardiac catheterization laboratory
Thomas C. Chalmers	[1917-1995]	MD, 1943	anticipated	Mount Sinai School of Medicine	Studies in Chronic Liver Disease
Ephraim Donoso	[1917-1988]	MD, 1941	anticipated	Mount Sinai School of Medicine	Cooperative Study of Drugs and Coronary Heart Disease
Alfred S. Evans	[1917-1996]	MD, 1943	anticipated	Yale Medical School	Epidemiological Studies of EB virus in Hodgkin's Disease

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
Eugene M. Farber	[1917-2000]	MD, 1943	sudden	Stanford University School of Medicine	Biologic Effects of Photochemotherapy in Psoriasis
Michelangelo G.F. Fuortes	[1917-1977]	MD, 1941	sudden	NIH/NINDS	Neurophysiological studies of motoneurons and electrical activity
Max Halperin	[1917-1988]	PhD, 1950	anticipated	Georgetown University Medical Center	Statistical Methods for Clinical Trials in Chronic Diseases
Philip Handler	[1917-1981]	PhD, 1939	anticipated	Duke University School of Medicine	Sulfite Oxidation in Biological Systems
T. C. [Tao-Chiuh] Hsu	[1917-2003]	PhD, 1951	anticipated	University of Texas MD Anderson Cancer Center	Cytogenetic Assays of Human Genetic Instability
Nathan O. Kaplan	[1917-1986]	PhD, 1943	sudden	UCSD School of Medicine	isolation and structure determination of coenzyme A
Albert S. Kaplan	[1917-1989]	PhD, 1952	anticipated	Vanderbilt University School of Medicine	Metabolism of Cells Infected With Nuclear DNA Viruses
Edward H. Kass	[1917-1990]	MD/PhD, 1947	anticipated	Harvard Medical School/Brigham & Women's Hospital	mechanism of toxic shock syndrome
Tsoo E. King	[1917-1990]	PhD, 1949	unknown	University of Pennsylvania School of Medicine	bioenergetic apparatus in heart mitochondria
Albert L. Lehninger	[1917-1986]	PhD, 1942	anticipated	Johns Hopkins University School of Medicine	Structure and function of mitochondria
Jessica H. Lewis	[1917-2003]	MD, 1942	sudden	University of Pittsburgh School of Medicine	Blood Coagulation and Hemorrhagic Disease
John P. Merrill	[1917-1984]	MD, 1942	sudden	Harvard Medical School/Brigham & Women's Hospital	role of the immune system in kidney transplantation
Jack Metcoff	[1917-1994]	MD, 1942	unknown	Chicago Medical School	Maternal Malnutrition and Fetal Development
Edwin D. Murphy	[1917-1984]	MD, 1943	unknown	NIH/NCI	Stages of Carcinogenesis of the Cervix Uteri in Mice
Albert Segaloff	[1917-1985]	MD, 1942	sudden	Tulane University School of Medicine	hormonal treatment of advanced breast cancer
Merton F. Utter	[1917-1980]	PhD, 1942	sudden	Case Western Reserve University School of Medicine	structure and function of pep carboxykinase isozymes
Robert B. Woodward	[1917-1979]	PhD, 1936	sudden	Harvard University	studies in the chemistry of natural products
Elijah Adams	[1918-1979]	MD, 1942	unknown	University of Maryland School of Medicine	Metabolism of Tyrosinases and Tyrosine Hydroxylases
Solomon A. Berson	[1918-1972]	MD, 1945	sudden	Mount Sinai School of Medicine	Studies of the use of radioisotopes in clinical investigation and diagnosis
Walter E. Brown	[1918-1993]	PhD, 1949	anticipated	American Dental Association Health Foundation	chemistry of calcium phosphates
Frederick H. Carpenter	[1918-1982]	PhD, 1944	anticipated	University of California — Berkeley	mechanism of leucine aminopeptidase
George C. Cotzias	[1918-1977]	MD, 1944	anticipated	Weill Medical College — Cornell University	Chemical Dissection and Therapy of Brain Disorders
Joseph F. Foster	[1918-1975]	PhD, 1943	sudden	Purdue University	Physicochemical Basis of Biological Stability
Dexter French	[1918-1981]	PhD, 1942	anticipated	Iowa State University	Mechanism of Amylase Action
Henry S. Kaplan	[1918-1984]	MD, 1940	anticipated	Stanford University School of Medicine	radiation-induced leukemia in the C57BL mouse
George E. Murphy	[1918-1987]	MD, 1943	anticipated	Weill Medical College — Cornell University	Rheumatic Disease and Glomerulonephritis
Harvey M. Patt	[1918-1982]	PhD, 1942	anticipated	UCSF School of Medicine	ultra-high dose rates in experimental radiotherapy
Henry Rapoport	[1918-2002]	PhD, 1943	sudden	University of California — Berkeley	total synthesis of heterocyclic drugs
Ruth Sager	[1918-1997]	PhD, 1948	anticipated	Harvard Medical School/Dana Farber Cancer Institute	role of tumor suppressor genes in breast cancer
Cornelius A. Tobias	[1918-2000]	PhD, 1942	anticipated	University of California — Berkeley	biological effects of cosmic rays and other ionizing radiation
Charles W. Todd	[1918-1987]	PhD, 1943	anticipated	City of Hope Medical Center	Immunology and Immunochemistry of Tumor Antigens
E. Jack Wylie	[1918-1982]	MD, 1943	sudden	UCSF School of Medicine	development of techniques for the treatment and management of chronic visceral ischemia
William G. Dauben	[1919-1997]	PhD, 1944	sudden	University of California — Berkeley	ultraviolet irradiation of natural products
Lloyd J. Filer, Jr.	[1919-1997]	MD/PhD, 1952	sudden	University of Iowa College of Medicine	Growth Patterns and Body Composition of Pigs
Thomas B. Fitzpatrick	[1919-2003]	MD/PhD, 1952	anticipated	Harvard Medical School/Massachusetts General Hospital	dynamics of epidermal pigmentation
Bernard G. Greenberg	[1919-1985]	PhD, 1949	anticipated	University of North Carolina School of Public Health	biostatistics related to health services
Morton I. Grossman	[1919-1981]	MD/PhD, 1944	anticipated	UCLA School of Medicine	studies on the etiology of peptic ulcer
Daniel S. Lehrman	[1919-1972]	PhD, 1954	sudden	Rutgers University	Psychobiological Studies of Behavior
Alvin Nason	[1919-1978]	PhD, 1952	unknown	Johns Hopkins University School of Medicine	Enzymology of Nitrate Respiration and Assimilation
Carl M. Pearson	[1919-1981]	MD, 1946	anticipated	UCLA School of Medicine	studies in adjuvant-induced arthritis
Judith G. Pool	[1919-1975]	PhD, 1946	anticipated	Stanford University School of Medicine	Pathophysiology of Hemophilia
Cyril S. Stulberg	[1919-1977]	PhD, 1947	anticipated	Wayne State University School of Medicine	Bacterial and Viral Agents in the Diarrheas of Infancy
Donovan J. Thompson	[1919-1991]	PhD, 1951	sudden	University of Washington School of Medicine	Biostatistics: Sampling Designs for Field Studies
Russell J. Barrnett	[1920-1989]	MD, 1948	sudden	Yale Medical School	Relation of Fine Structure To Biochemical Function
Mones Berman	[1920-1982]	PhD, 1957	anticipated	NIH/NCI	mathematical modeling of biological systems
Leo K. Bustad	[1920-1998]	DVM/PhD, 1960	anticipated	Washington State University	Veterinary Physiology
Ernest Cotlove	[1920-1970]	MD, 1943	sudden	NIH/NHI	Studies of Kidney and Electrolyte Metabolism
Harriet P. Dustan	[1920-1999]	MD, 1944	anticipated	University of Vermont College of Medicine	Mechanisms of Hypertension
Fred I. Gilbert, Jr.	[1920-1995]	MD, 1945	unknown	University of Hawaii School of Medicine	clinical studied of hyper- and hypothyroidism
William F. Harrington	[1920-1992]	PhD, 1952	sudden	Johns Hopkins University School of Medicine	myosin thick filament structure and assembly
Charles D. Heidelberger	[1920-1983]	PhD, 1946	anticipated	USC Keck School of Medicine	effects of fluorinated pyrimidines on tumors
Henry Kamin	[1920-1988]	PhD, 1948	anticipated	Duke University School of Medicine	Biological Oxidations in Mitochondria and Microsomes
Peter Kellaway	[1920-2003]	PhD, 1947	anticipated	Baylor College of Medicine	clinical investigations of childhood epilepsy
Teruzo Konishi	[1920-1984]	MD/PhD, 1955	anticipated	NIH/NIEHS	physiological and biophysical functions of the inner ear
Toichiro Kuwabara	[1920-1991]	MD/PhD, 1952	sudden	Harvard Medical School	Ultrastructure of Retina and Retinal Disease
Abraham M. Lilienfeld	[1920-1984]	MD, 1944	sudden	Johns Hopkins School of Hygiene and Public Health	epidemiological methods for the study of chronic diseases
Ardie Lubin	[1920-1976]	PhD, 1951	anticipated	Naval Health Research Center	Repeated Measurement Design in Psychopharmacology
Philip R.A. May	[1920-1986]	MD, 1944	anticipated	UCLA School of Medicine	controlled clinical studies of schizophrenia
Jean Mayer	[1920-1993]	PhD, 1948	sudden	Tufts University School of Medicine	Metabolic Aspects of Obesity
Elizabeth C. Miller	[1920-1987]	PhD, 1945	anticipated	University of Wisconsin School of Medicine	carcinogenesis and reactive electrophilic metabolites
Edgar E. Ribi	[1920-1986]	PhD, 1948	sudden	NIH/NIAID/Rocky Mountain Laboratory	Identification of microbial adjuvants for cancer immunotherapy
Marion I. Barnhart	[1921-1985]	PhD, 1950	sudden	Wayne State University School of Medicine	blood disorders
Lawrence Bogorad	[1921-2003]	PhD, 1949	sudden	Harvard University	Organelle Genes and Gene Regulation
John C. Cassel	[1921-1976]	MD, 1946	anticipated	University of North Carolina School of Public Health	cultural change, blood pressure, and heart disease
C. Clark Cockerham	[1921-1996]	PhD, 1952	anticipated	North Carolina State University	The Statistics of Genetic Systems
Allen S. Fox	[1921-1977]	PhD, 1948	unknown	University of Wisconsin School of Medicine	Immunogenetic studies of drosophila melanogaster
Charlotte Friend	[1921-1987]	PhD, 1950	anticipated	Mount Sinai School of Medicine	tissue studies of murine virus-induced leukemia
Donald B. Hackel	[1921-1994]	MD, 1946	anticipated	Duke University School of Medicine	Diabetes Mellitus in Psammomys Obesus
Harold Koenig	[1921-1992]	MD/PhD, 1949	unknown	Northwestern University School of Medicine	Molecular Pathology of Blood-Brain Barrier Breakdown
Marian E. Koshland	[1921-1997]	PhD, 1949	anticipated	University of California — Berkeley	biochemical methods to examine the immune response
Grant W. Liddle	[1921-1989]	MD, 1948	sudden	Vanderbilt University School of Medicine	Pituitary-Adrenal Physiology and Pharmacology
Mortimer B. Lipsett	[1921-1985]	MD, 1951	anticipated	NIH	steroid metabolic conversions in human subjects
Peter N. Magee	[1921-2000]	MD, 1945	anticipated	Thomas Jefferson University	genetic basis of carconogenesis
Henry R. Mahler	[1921-1983]	PhD, 1948	anticipated	Indiana University at Bloomington	Studies of the Structure, Function, and Biosynthesis of Respiratory Enzymes
Jack Orloff	[1921-1988]	MD, 1943	anticipated	NIH/NHLBI	cyclic AMP and the cellular response to antidiuretic hormone
Andrew C. Peacock	[1921-1985]	PhD, 1949	anticipated	NIH/NCI	Invention of of the polyacrylamide gel electrophoresis process
Seymour Perry	[1921-2000]	MD, 1947	anticipated	Georgetown University Medical Center	evaluation of medical technology
Sidney Riegelman	[1921-1981]	PhD, 1948	sudden	UCSF School of Medicine	intersubject variation in first pass effect of drugs
Griff T. Ross	[1921-1985]	MD/PhD, 1945	anticipated	NIH/NICHD	radioimmunoassay for human chtionic gonadotropin
Belding H. Scribner	[1921-2003]	MD, 1945	sudden	University of Washington School of Medicine	dialysis in the treatment of chronic uremia
David Spiro	[1921-1974]	MD/PhD, 1956	sudden	New York Medical College	Ultrastructure and Contractile Mechanisms of Mammalian Cardiac Muscle
David H.P. Streeten	[1921-2000]	MD/PhD, 1951	sudden	SUNY Upstate Medical University at Syracuse	thyroid and parathyroid hormones in hypertension
Samuel Sutton	[1921-1986]	PhD, 1955	sudden	University of Chicago School of Medicine	Drug Effects on Psychophysiological Functions
Jack E. White	[1921-1988]	MD, 1944	anticipated	Howard University School of Medicine	epidemiology and treatment of cancer among african-americans
David Zeaman	[1921-1984]	PhD, 1950	unknown	University of Connecticut Storrs	Retardate Discrimination Learning and Attention

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
Harold Edelhoch	[1922-1986]	PhD, 1947	anticipated	NIH/NIDDK	physical chemistry of thyroglobulin
Mortimer M. Elkind	[1922-2000]	PhD, 1953	anticipated	Colorado State University	cell radiation response of cultured mammalian cells
Seymour Fisher	[1922-1996]	PhD, 1948	sudden	SUNY Upstate Medical University at Syracuse	The Role of Body Attitudes in Behavior
Robert A. Good	[1922-2003]	MD/PhD, 1947	anticipated	University of South Florida College of Medicine	role of the thymus in immune system development
Carl W. Gottschalk	[1922-1997]	MD, 1945	sudden	University of North Carolina at Chapel Hill School of Medicine	micropuncture studies of mammallian renal system
Susumu Hagiwara	[1922-1989]	PhD, 1951	sudden	UCLA School of Medicine	evolutionary and developmental properties of calcium channels in cell membranes
Lucille S. Hurley	[1922-1988]	PhD, 1950	sudden	University of California — Davis	genetic and nutritional interactions in development
David T. Imagawa	[1922-1991]	PhD, 1950	sudden	Harbor-UCLA Medical Center	morphological conversion with leukemia viruses
Shirley A. Johnson	[1922-1970]	PhD, 1949	unknown	George Washingron University School of Medicine/VA Hiospital of Washington,	hemophilia B and the differentiation of prothrombin activation
C. Henry Kempe	[1922-1984]	MD, 1945	unknown	University of Colorado Health Sciences Center	immunological problems of smallpox
S. Morris Kupchan	[1922-1976]	PhD, 1945	anticipated	University of Virginia School of Medicine	Chemistry of Tumor-Inhibitory Natural Products
Herbert G. Langford	[1922-1991]	MD, 1945	sudden	University of Mississippi Medical Center	electrolyte intake and blood pressure in hypertension
Sidney Leskowitz	[1922-1991]	PhD, 1950	anticipated	Tufts University School of Medicine	Cellular Aspects of Tolerance and Delayed Sensitivity
Sol Levine	[1922-1996]	PhD, 1953	sudden	Harvard School of Public Health	targets for worksite prevention of alcohol problems
Cyrus Levinthal	[1922-1990]	PhD, 1951	anticipated	Columbia University College of Physicians & Surgeons	colinearity of genes and proteins, and the nature of messenger RNA
David M. Maurice	[1922-2002]	PhD, 1951	anticipated	Columbia University College of Physicians & Surgeons	interference theory of corneal transparency
Alton Meister	[1922-1995]	MD, 1945	anticipated	Weill Medical College — Cornell University	amino acid and glutathione biochemistry
James Olds	[1922-1976]	PhD, 1952	sudden	California Institute of Technology	Pharmacology of Motivational Mechanisms
J. David Robertson	[1922-1995]	MD/PhD, 1952	anticipated	Duke University School of Medicine	electron microscopy of cell membranes
Bertram Sacktor	[1922-1988]	PhD, 1949	sudden	NIH/NIA	Mechanisms of hormonal regulation of cellular pH
Mearl F. Stanton	[1922-1980]	MD, 1948	anticipated	NIH/NCI	Carcinogenecity of Fibers
Kwan C. Tsou	[1922-1985]	PhD, 1950	sudden	University of Pennsylvania School of Medicine	Cytochemical Substrates and Anticancer Agents
Charles A. Waldron	[1922-1995]	DDS, 1945	sudden	Emory University School of Medicine	Oral Pathology
Ernst L. Wynder	[1922-1999]	MD, 1950	anticipated	American Health Foundation	epidemiologic studies of tobacco control
William S. Beck	[1923-2003]	MD, 1946	anticipated	Harvard Medical School	biochemistry of blood cell formation
Arnold F. Brodie	[1923-1981]	PhD, 1952	unknown	USC Keck School of Medicine	Mechanisms of Oxidative Energy Generation in Bacteria
Josiah Brown	[1923-1985]	MD, 1947	sudden	UCLA School of Medicine	biochemical studies of lipid and carbohydrate metabolism
James M. Felts	[1923-1988]	PhD, 1955	sudden	UCSF School of Medicine	synthesis and processing of plasma lipoproteins
Samuel B. Guze	[1923-2000]	MD, 1945	anticipated	Washington University in St. Louis School of Medicine	neurobiology, genetics, and epidemiology of alcoholism
Eugene C. Jorgensen	[1923-1981]	PhD, 1953	sudden	UCSF School of Medicine	structure/activity relationships of compounds related to thyroxin
Norman Kretchmer	[1923-1995]	MD/PhD, 1952	anticipated	University of California — Berkeley	Metabolism Regulation During Development
M. Powell Lawton	[1923-2001]	PhD, 1952	anticipated	Philadelphia Geriatric Center	studies of mental health, quality of life, and caregiving of the elderly
Paul Margolin	[1923-1989]	PhD, 1956	sudden	Public Health Research Institute of the City of New York	Mutation and Suppressor Studies of a Bacterial Gene
Kehl Markley, 3rd	[1923-1979]	MD, 1947	sudden	NIH/NIAMDD	burn treatment specialist
William W. Montgomery	[1923-2003]	MD, 1947	anticipated	Harvard Medical School	Methods of Correcting Dysfunctions of the Human Larynx
Andrew G. Morrow	[1923-1982]	MD, 1946	sudden	NIH/NHLBI	surgical correction of idiopathic hypertrophic subaortic stenosis
Peter W. Neurath	[1923-1977]	PhD, 1950	sudden	Tufts University School of Medicine	Chromosomal Variants of Cells Converted By Viruses
John Rankin	[1923-1981]	MD, 1947	unknown	University of Wisconsin School of Medicine	development of a pragmatic stroke outcome scale
Herbert J. Rapp	[1923-1981]	PhD, 1955	sudden	NIH/NCI	Immunology and immunotherapy of annimal cancers
Lewis W. Wannamaker	[1923-1983]	MD, 1948	sudden	University of Minnesota School of Medicine	clinical and epidemiologic aspects of streptococcal infections
Alfred P. Wolf	[1923-1998]	PhD, 1953	anticipated	Brookhaven National Laboratory	synthesis of simple molecules in pure form and high specific activity for PET
Constantine S. Anast	[1924-1987]	MD, 1947	unknown	Harvard Medical School/Children's Hospital	hormonal regulation of mineral metabolism
C. Andrew L. Bassett	[1924-1994]	MD/PhD, 1955	anticipated	Columbia University College of Physicians & Surgeons	Bioelectric Phenomena Controlling Bone Growth
Myron L. Bender	[1924-1988]	PhD, 1948	sudden	Northwestern University School of Medicine	Mechanism of Action of Proteases
David H. Blankenhorn	[1924-1993]	MD, 1947	anticipated	USC Keck School of Medicine	control of risk factors in atherosclerosis
Eli Chernin	[1924-1990]	PhD, 1951	sudden	Harvard School of Public Health	Biology and Biological Control of Schistosomiasis
Wallace H. Clark, Jr.	[1924-1997]	MD, 1947	sudden	Harvard Medical School	Biology of Human Cutaneous Malignant Melanoma
Adolph I. Cohen	[1924-1996]	PhD, 1954	anticipated	Washington University in St. Louis School of Medicine	Cytology and Physiology of the Retina
Donald S. Fredrickson	[1924-2002]	MD, 1949	sudden	NIH/NHI	structure and metabolism of plasma lipoproteins and their role in lipid transport
Clarence J. Gibbs, Jr.	[1924-2001]	PhD, 1962	sudden	NIH/NINDS	infectuous diseases of the nervous system
Victor A. Gilbertsen	[1924-1990]	MD, 1953	anticipated	University of Minnesota School of Medicine	Development of cost-effective methods to diagnose presymptomatic cancers
Menek Goldstein	[1924-1997]	PhD, 1955	sudden	New York University School of Medicine	purification of enzymes in the catecholamine synthetic pathway
Herbert F. Hasenclever	[1924-1978]	PhD, 1953	anticipated	NIH/NIAID	Polysaccharides of pathogenic fungi
Edward W. Hook, Jr.	[1924-1998]	MD, 1949	sudden	University of Virginia School of Medicine	Host Resistance Unrelated To Specific Immunity
Thomas R. Johns, 2nd	[1924-1988]	MD, 1948	sudden	University of Virginia School of Medicine	physiological studies of myasthenia gravis
William B. Reed	[1924-1976]	MD, 1952	sudden	USC Keck School of Medicine	Clinical studies of epidermolysis bullosa
Timothy J. Regan	[1924-2001]	MD, 1952	anticipated	University Hospital of Newark, NJ	myocardial function and metabolism in chronic disease
Lucien J. Rubinstein	[1924-1990]	MD, 1948	sudden	University of Virginia School of Medicine	differentiation and stroma-induction in neural tumors
Peter Safar	[1924-2003]	MD, 1948	anticipated	University of Pittsburgh School of Medicine	clinical studies of brain resuscitation
Joseph Stokes, 3rd	[1924-1989]	MD, 1949	anticipated	Boston University Medical Center	epidemiological studies of coronary heart disease
Robert J. Stoller	[1924-1991]	MD, 1948	sudden	UCLA School of Medicine	clinical studies of gender identity
W. Dean Warren	[1924-1989]	MD, 1950	anticipated	Emory University School of Medicine	Cirrhosis, Shunt Surgery, and Nitrogen Metabolism
Emanuel M. Bogdanove	[1925-1979]	PhD, 1953	sudden	Medical College of Virginia	Endocrine-Influencing Centers in the Hypothalamus
Arend Bouhuys	[1925-1979]	MD/PhD, 1956	sudden	Yale Medical School	Physiological Studies on Air Pollution and Bysinosis
Margaret O. Dayhoff	[1925-1983]	PhD, 1948	sudden	Georgetown University Medical Center	computer study of sequences of amino acids in proteins
Ernst Freese	[1925-1990]	PhD, 1954	sudden	NIH/NINDS	mutations, membrane transport, and cell differentiation
Sidney H. Ingbar	[1925-1988]	MD, 1947	anticipated	Harvard Medical School/Beth Israel Medical Center	physiology of the thyroid gland and its clinical diseases
Milton Kern	[1925-1987]	PhD, 1954	anticipated	NIH/NIADDK	Ribonucleic Acids of Specifically Isolated Ribosomes
Philip R. Kimbel	[1925-1990]	MD, 1954	anticipated	University of Pennsylvania School of Medicine	causes of emphysema and other pulmonary diseases
Werner H. Kirsten	[1925-1992]	MD, 1953	sudden	NIH/NCI	Pathogenesis of Induced Leukemia and Tumors in Rats
Ariel G. Loewy	[1925-2001]	PhD, 1951	sudden	Haverford College	Distribution and Function of the Isopeptide Bond
William H. Oldendorf	[1925-1992]	MD, 1947	sudden	UCLA School of Medicine	x-ray shadow radiography and cerebral angiography
N. Raphael Shulman	[1925-1996]	MD, 1947	anticipated	NIH/NIDDK	Physiology and biochemistry of platelets
Paul A. Srere	[1925-1999]	PhD, 1951	sudden	University of Texas Southwestern Medical Center at Dallas	cell metabolism and the krebs tca cycle
Michel M. Ter-Pogossian	[1925-1996]	PhD, 1950	sudden	Washington University in St. Louis School of Medicine	multislice PET scanning technology
William H. Tooley	[1925-1992]	MD, 1949	anticipated	UCSF School of Medicine	prevention and treatment of respiratory distress in neonates
Kelly M. West	[1925-1980]	MD, 1948	sudden	University of Oklahoma School of Medicine	Causes of Diabetes and Obesity in Oklahoma Indians
George Winokur	[1925-1996]	MD, 1947	anticipated	University of Iowa College of Medicine	genetics of bipolar disease, mania, alcoholism and other psychiatric diseases
Robert H. Abeles	[1926-2000]	PhD, 1955	anticipated	Brandeis University	rational design of small-molecule inhibitors of enzymes
J. Weldon Bellville	[1926-1983]	MD, 1952	anticipated	UCLA School of Medicine	dynamic isolation studies of control of respiration
Nemat O. Borhani	[1926-1996]	MD, 1949	anticipated	University of Nevada at Reno	multicenter clinical studies of hypertension and cardiovascular disease
Zanvil A. Cohn	[1926-1993]	MD, 1953	sudden	Rockefeller University	macrophage in cell biology and resistance to infectious disease
Russell L. De Valois	[1926-2003]	PhD, 1952	sudden	University of California — Berkeley	brain mechanisms underlying color vision
Giovanni Di Chiro	[1926-1997]	MD, 1949	anticipated	NIH	interventional neuroradiology
Nicholas R. DiLuzio	[1926-1986]	PhD, 1954	anticipated	Tulane University School of Medicine	Role Recognition Factors and Macrophages in Neoplasia
Fritz E. Dreifuss	[1926-1997]	MD, 1950	anticipated	University of Virginia School of Medicine	clinical investigations of childhood epilepsy
Edward V. Evarts	[1926-1985]	MD, 1948	sudden	NIH	electrophysiological activity of in vivo neurons in waking and sleeping states

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
Norbert Freinkel	[1926-1989]	MD, 1949	sudden	Northwestern University School of Medicine	metabolic regulation in normal and diabetic pregnancies
Norman Geschwind	[1926-1984]	MD, 1951	sudden	Harvard Medical School/Beth Israel Medical Center	relationship between the anatomy of the brain and behavior
Richard Gorlin	[1926-1997]	MD, 1948	anticipated	Mount Sinai School of Medicine	studies of coronary blood flow and myocardial metabolism
Edward C. Heath	[1926-1984]	PhD, 1955	anticipated	University of Iowa College of Medicine	chemistry and metabolism of polysaccharides
Edward Herbert	[1926-1987]	PhD, 1953	anticipated	Oregon Health & Science University	regulation of expression of opioid peptides and receptors
Roger T. Kelleher	[1926-1994]	PhD, 1955	anticipated	Harvard Medical School	drug effects on behavior controlled by aversive stimuli
William B. Kinter	[1926-1978]	PhD, 1955	unknown	Mount Desert Island Biological Lab	Physiology and Morphology of Cell Transport
John A. Kirkpatrick, Jr.	[1926-1994]	MD, 1949	anticipated	Harvard Medical School/Children's Hospital	studies of esophageal atresia
Julius Marmur	[1926-1996]	PhD, 1951	anticipated	Albert Einstein College of Medicine	genetics and biochemistry of cellular regulation
Vincent Massey	[1926-2002]	PhD, 1953	sudden	University of Michigan School of Medicine	biological oxidation mechanisms of proteins that contain riboflavin
Brigitte A. Prusoff	[1926-1991]	PhD, 1978	unknown	Yale Medical School	follow-up of maintenance treatment for depression
Wallace P. Rowe	[1926-1983]	MD, 1948	anticipated	NIH	genetic basis of disease in murine leukemia viruses
Kiichi Sagawa	[1926-1989]	MD/PhD, 1958	anticipated	Johns Hopkins University School of Medicine	modelling the mechanics of cardiac chamber contraction
Norman P. Salzman	[1926-1997]	PhD, 1953	anticipated	Georgetown University Medical Center	Role in the Immune Response of the Glycosylation of SIV Gp120
Frederick Stohlman, Jr.	[1926-1974]	MD, 1947	sudden	Tufts University School of Medicine	Dissociation Curve and Erythropoietin Production
Gordon M. Tomkins	[1926-1975]	PhD, 1953	anticipated	UCSF School of Medicine	post-transcriptional control of gene expression
Irwin M. Weinstein	[1926-2002]	MD, 1949	sudden	UCLA/Cedars-Sinai Medical Center	Influence of the Pancreas on Iron Absorption
Robert D. Allen	[1927-1986]	PhD, 1953	anticipated	Dartmouth Medical School	cytoplasmic rheology of motile cells
Gerald D. Aurbach	[1927-1991]	MD, 1954	sudden	NIH	bone metabolism and calcium homeostasis
Leonard R. Axelrod	[1927-1975]	PhD, 1952	unknown	Environmental Protection Agency	Studies in Steroid Intermediate Metabolism
Sarah H. Broman	[1927-1999]	PhD, 1965	sudden	NIH/NINDS	Interventions for Verbal and Motor Deficits in Children
Gustavo Cudkowicz	[1927-1982]	MD, 1952	sudden	SUNY at Buffalo School of Medicine and Biomedical Sciences	controls of proliferation specific for leukemias
Donnell D. Etzwiler	[1927-2003]	MD, 1953	anticipated	University of Minnesota School of Medicine	Influence of Diabetes Control on Vascular Complications
Pierre M. Galletti	[1927-1996]	MD/PhD, 1954	sudden	Brown Medical School	Synthesis of artificial lung and kidney systems
Paul M. Gallop	[1927-1996]	PhD, 1953	anticipated	Harvard Medical School/Children's Hospital	Protein structure and collagen maturation
George G. Glenner	[1927-1995]	MD, 1953	anticipated	UCSD School of Medicine	molecular structure of the amyloid protein
Lauran D. Harris	[1927-1987]	MD, 1951	anticipated	Boston University Medical Center	Control of Sphincter Strength
Victor D. Herbert	[1927-2002]	MD, 1952	anticipated	Veterans Administration Hospital, Bronx, NY	Vitamin B12 and Folic Acid Metabolism
William H. Hildemann	[1927-1983]	PhD, 1956	anticipated	UCLA School of Medicine	mechanisms of immunoblocking versus tumor immunity
Peter D. Klein	[1927-2001]	PhD, 1954	sudden	Baylor College of Medicine	Metabolism of 13C Compounds in Digestive Diseases
Dorothy T. Krieger	[1927-1985]	MD, 1949	anticipated	Mount Sinai School of Medicine	CNS-pituitary-adrenal interactions
Richard C. Lillehei	[1927-1981]	MD/PhD, 1960	sudden	University of Minnesota School of Medicine	Cardiac Dynamics in Experimental Cardiogenic Shock
Richard L. Lyman	[1927-1975]	PhD, 1957	anticipated	University of California — Berkeley	Characterization and Isolation of Lecithins
Charles G. Moertel	[1927-1994]	MD, 1953	anticipated	Mayo Clinic	clinical treatments of gastrointestinal cancer
Hans J. Müller-Eberhard	[1927-1998]	MD, 1953	anticipated	Scripps Research Institute	identification of proteins and reaction mechanisms of the complement system
Murray Rabinowitz	[1927-1983]	MD, 1950	anticipated	University of Chicago School of Medicine	mitochondrial assembly and replication
Frank Restle	[1927-1980]	PhD, 1954	sudden	Indiana University at Bloomington	Experiments on Multi-Stage Models of Learning
Gerald P. Rodnan	[1927-1983]	MD, 1949	sudden	University of Pittsburgh School of Medicine	renal transport if uric acid and protein
Daniel Rudman	[1927-1994]	MD, 1949	sudden	Medical College of Wisconsin	adipokinetic substances of the pituitary gland
Dante G. Scarpelli	[1927-1998]	MD/PhD, 1960	anticipated	Northwestern University School of Medicine	metabolism of pancreatic carcinogens
George Streisinger	[1927-1984]	PhD, 1953	sudden	University of Oregon	genetic mutations and the nervous system development in lower vertebrates
Robert Thompson	[1927-1989]	PhD, 1955	anticipated	University of California — Irvine	neural systems subserving learning and memory
Nina S. Braunwald	[1928-1992]	MD, 1952	anticipated	Harvard Medical School/Brigham & Women's Hospital	development of prosthetic heart valves for children
Alberto DiMascio	[1928-1978]	PhD, 1966	sudden	Tufts University School of Medicine	Evaluation of Psychotherapy in Treating Depression
John L. Doppman	[1928-2000]	MD, 1953	anticipated	NIH/CC	Flow Dynamics in Anterior Spinal Artery
Edward C. Franklin	[1928-1982]	MD, 1950	anticipated	New York University School of Medicine	structure and properties of rheumatoid antibodies
Erhard Gross	[1928-1981]	PhD, 1958	sudden	NIH/NICHD	Structure determinations of the peptide antibiotics
Lucien B. Guze	[1928-1985]	MD, 1951	sudden	UCLA School of Medicine	pathogenesis of experimental pyelonephritis
Thomas P. Hackett, Jr.	[1928-1988]	MD, 1952	sudden	Harvard Medical School/Massachusetts General Hospital	Denial and Mortality/Morbidity in Myocardial Infarction
Gerald L. Klerman	[1928-1992]	MD, 1954	anticipated	Weill Medical College — Cornell University	phsychological studies of depression, schizophrenia and panic and other anxiety disorders
Robert S. Krooth	[1928-1979]	MD/PhD, 1957	sudden	Columbia University College of Physicians & Surgeons	gene action in cultured human and other mammalian cells
Carl C. Levy	[1928-1981]	PhD, 1957	anticipated	NIH/NCI	Studies of the regulation of intracellular mRNA
Christopher L. Longcope	[1928-2003]	MD, 1953	anticipated	Umass Medical School	reproductive function and gonadal steroid dynamics
William J. Mellman	[1928-1980]	MD, 1952	anticipated	University of Pennsylvania School of Medicine	Biochemical Genetics of Cultured Human Cells
Carl Monder	[1928-1995]	PhD, 1956	sudden	The Population Council	corticosteroid metabolism in juvenile hypertension
Leo J. Neuringer	[1928-1993]	PhD, 1957	anticipated	MIT	NMR studies of normal and transformed cell membranes
Edward W. Purnell	[1928-1993]	MD, 1957	anticipated	Case Western Reserve University School of Medicine	Study of Eye Physiology and Disease by Ultrasound
Jay P. Sanford	[1928-1996]	MD, 1952	anticipated	University of Texas Southwestern Medical Center at Dallas	Host Factors in Chronic Pyelonephritis
Alfred A. Smith	[1928-1980]	MD, 1956	unknown	New York Medical College	Autonomic Activity and Addiction
D. Eugene Strandness, Jr.	[1928-2002]	MD, 1954	sudden	University of Washington School of Medicine	ultrasonic duplex scanner for noninvasive vascular disease diagnosis
Howard E. Freeman	[1929-1992]	PhD, 1956	sudden	UCLA School of Medicine	Studies on the Social Organization of Medical Care
Sidney Futterman	[1929-1979]	PhD, 1954	anticipated	University of Washington School of Medicine	biochemistry of the retina and pigment epithelium
Edgar C. Henshaw	[1929-1992]	MD, 1956	sudden	University of Rochester School of Medicine & Dentistry	intermediary metabolism in animals and in man
Lubomir S. Hnilica	[1929-1986]	PhD, 1952	sudden	Vanderbilt University School of Medicine	nuclear antigens in human colorectal cancer
Charles E. Huggins	[1929-1990]	MD, 1952	anticipated	Harvard Medical School/Massachusetts General Hospital	human blood storage procedures
Peter W. Lampert	[1929-1986]	MD, 1955	anticipated	UCSD School of Medicine	pathogenesis of virus-induced brain disease
David J.L. Luck	[1929-1998]	MD/PhD, 1962	anticipated	Rockefeller University	microtubular systems in human cells
James W. Maas	[1929-1995]	MD, 1954	sudden	University of Texas Health Sciences Center at San Antonio	MHPG Excretion, Catecholamine Metabolism, and Depression
A. Louis McGarry	[1929-1985]	MD, 1955	anticipated	Nassau County Department of Mental Health	Competency To Stand Trial and Mental Illness
Kenneth M. Moser	[1929-1997]	MD, 1954	anticipated	UCSD School of Medicine	clinical outcomes after pulmonary thromboendarterectomy
Milton Orkin	[1929-1999]	MD, 1954	anticipated	University of Minnesota School of Medicine	ttreatment of skin infestations and scabies
J. Kiffin Penry	[1929-1996]	MD, 1955	anticipated	Bowman Gray School of Medicine at Wake Forest University	Studies of the control of epileptoc seizures
John J. Pisano	[1929-1985]	PhD, 1955	sudden	NIH/NHLBI	Isolation of active peptides
Felix T. Rapaport	[1929-2001]	MD, 1954	sudden	SUNY Health Sciences Center at Stony Brook	induction of unresponsiveness to allografts
Russell Ross	[1929-1999]	DDS/PhD, 1962	anticipated	University of Washington School of Medicine	response-to-injury origins of atherosclerosis
Miriam M. Salpeter	[1929-2000]	PhD, 1953	anticipated	Weill Medical College — Cornell University	neurobiology of myasthenia gravis
Laurence M. Sandler	[1929-1987]	PhD, 1956	sudden	University of Washington School of Medicine	cytogenetics of meiosis and development in drosophila
Robert C. Schlant	[1929-2002]	MD, 1951	anticipated	Emory University School of Medicine	Hepatic Venography
G. Jeanette Thorbecke	[1929-2001]	MD/PhD, 1954	sudden	New York University School of Medicine	histologic and functional aspects of lymphoid tissue development
Lester Baker	[1930-2000]	MD, 1959	anticipated	University of Pennsylvania School of Medicine/CHOP	clinical studies of type I diabetes control and complications
Edwin L. Bierman	[1930-1995]	MD, 1955	anticipated	University of Washington School of Medicine	Metabolism of particulate fat in diabetes and atherosclerosis
Amico Bignami	[1930-1994]	MD, 1954	anticipated	Harvard Medical School	brain specific protein in astrocytes
Barbara H. Bowman	[1930-1996]	PhD, 1959	anticipated	University of Texas Health Sciences Center at San Antonio	genetic control of the structure of human proteins

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
L. Rao Chervu	[1930-1988]	PhD, 1962	sudden	Albert Einstein College of Medicine	Improved Radiopharmaceuticals For Nephrology and Urology
Gerald Cohen	[1930-2001]	PhD, 1955	anticipated	Mount Sinai School of Medicine	Catecholamine-Derived Alkaloids in Alcoholism
Joseph E. Coleman	[1930-1999]	MD/PhD, 1963	anticipated	Yale Medical School	structure and function of metalloenzyme synthesis
George B. Craig, Jr.	[1930-1995]	PhD, 1956	sudden	University of Notre Dame	genetics and reproductive biology of aedes mosquitoes
David Garfinkel	[1930-1990]	PhD, 1955	anticipated	University of Pennsylvania	computer modeling of complex biological systems
J. Calvin Giddings	[1930-1996]	PhD, 1955	anticipated	University of Utah School of Medicine	Biomedical Separations--Field Flow Fractionation
Michael J. Goldstein	[1930-1997]	PhD, 1957	anticipated	UCLA School of Medicine	contributing factors to the onset of schizophrenia
DeWitt S. Goodman	[1930-1991]	MD, 1955	sudden	Columbia University College of Physicians & Surgeons	lipid metabolism and its role in the development of heart and artery disease
Richard J. Herrnstein	[1930-1994]	PhD, 1955	sudden	Harvard University	Quantification and Control of Smoking
Aaron Janoff	[1930-1988]	PhD, 1959	anticipated	SUNY Health Sciences Center at Stony Brook	pathology of smoking and emphysema
Frank Lilly	[1930-1995]	PhD, 1965	anticipated	Albert Einstein College of Medicine	role of hereditary factors in governing susceptibility to cancer-causing agents
Gilda H. Loew	[1930-2001]	PhD, 1957	anticipated	Molecular Research Institute	computational investigation of the structural and functional aspects of heme proteins and enzymes
Leah M. Lowenstein	[1930-1984]	MD/PhD, 1958	unknown	Jefferson Medical College	Regulation of Renal Compensatory Adaptation
Paul C. MacDonald	[1930-1997]	MD, 1955	anticipated	University of Texas Southwestern Medical Center at Dallas	origin and interconversion of gonadal and adrenal streoid hormones
Charles W. Mays	[1930-1989]	PhD, 1958	anticipated	NIH/NCI	Reducing Cancer Risk By Radionuclide Chelation
William M. McKinney	[1930-2003]	MD, 1959	anticipated	Bowman Gray School of Medicine at Wake Forest University	application of ultrasonic energy to study the nervous system
Edward W. Moore	[1930-1999]	MD, 1955	anticipated	Medical College of Virginia	Pathophysiology of the billiary tract and gallbladder
Gisela Mosig	[1930-2003]	PhD, 1959	anticipated	Vanderbilt University School of Medicine	DNA Replication and Recombination in Bacteriophage
Jerry D. Niswander	[1930-1984]	DDS, 1955	anticipated	NIH/NIDR	genetics of oral and facial disorders
Hymie L. Nossel	[1930-1983]	MD/PhD, 1962	sudden	Columbia University College of Physicians & Surgeons	causes of thrombosis and the nature of hemostasis
Donald A. Pious	[1930-1998]	MD, 1956	anticipated	University of Washington School of Medicine	somatic cell genetic analysis of human immune response genes
F. Brantley Scott, Jr.	[1930-1991]	MD, 1955	sudden	Baylor College of Medicine	inflatable penile prosthesis
F. Blair Simmons	[1930-1998]	MD, 1956	sudden	Stanford University School of Medicine	development of a cochlear prothesis system for hearing loss
Dennis Slone	[1930-1982]	MD, 1956	anticipated	Boston University Medical Center	Comprehensive Surveillance of Marketed Drugs
Andrew P. Somlyo	[1930-2003]	MD, 1956	sudden	University of Virginia School of Medicine	vasomotor function of smooth muscle and their relation to heart disease
Muriel R. Steele	[1930-1979]	MD, 1957	anticipated	UCSF School of Medicine	Management of splenic injuries
Thoralf M. Sundt, Jr.	[1930-1992]	MD, 1959	anticipated	Mayo Clinic	surgical techniques for intracranial aneurysms
Charles L. Wittenberger	[1930-1987]	PhD, 1959	sudden	NIH/NIDR	Regulation of enzymes involved in transport and netabolism of sugars
Sheldon M. Wolff	[1930-1994]	MD, 1957	anticipated	Tufts University School of Medicine	treatment of fevers from infectious diseases like wegener's granulomatosis
Janine André-Schwartz	[1931-1995]	MD, 1959	anticipated	Tufts University School of Medicine	Studies of the proliferative response of lymphocytes to allografts
Louis V. Avioli	[1931-1999]	MD, 1957	anticipated	Washington University in St. Louis School of Medicine	mineral and skeletal metabolism in diabetes, kidney, and gastrointestinal disorders
Harold A. Baltaxe	[1931-1985]	MD, 1960	sudden	University of California — Davis	development of new coronary angiographic techniques
Paul P. Carbone	[1931-2002]	MD, 1956	sudden	University of Wisconsin School of Medicine	treatment and prevention of hodgkin's disease and early breast cancer
Richard A. Carleton	[1931-2001]	MD, 1955	anticipated	Brown Medical School	clinical studies of diet and smoking as cardiovascular disease risk factors
Sidney R. Cooperband	[1931-1979]	MD, 1956	unknown	Boston University Medical Center	Lymphocyte Proliferation Inhibitory Factor
Julian M. Davidson	[1931-2001]	PhD, 1959	anticipated	Stanford University School of Medicine	physiological bases of human sexuality
Gareth M. Green	[1931-1998]	MD/PhD, 1957	anticipated	Harvard University School of Public Health	role of alveolar macrophages in pulmonary defense mechanisms
John A. Gronvall	[1931-1990]	MD, 1956	sudden	University of Michigan School of Medicine	Pathology
James K. McDougall	[1931-2003]	PhD, 1971	anticipated	University of Washington/FHCRC	role of DNA viruses in cancer
Ernst A. Noltmann	[1931-1986]	MD, 1956	anticipated	University of California — Riverside	biochemical and physical characterization of phosphoglucose isomerase
Paul A. Obrist	[1931-1987]	PhD, 1958	anticipated	University of North Carolina at Chapel Hill School of Medicine	Studies of Heart Rate Conditioning
Guillermo H. Pacheco	[1931-1974]	PhD, 1961	anticipated	NIH/NIAID	Studies of Filariasis
James W. Prahl	[1931-1979]	MD/PhD, 1964	sudden	University of Utah School of Medicine	structural basis of the functions of human complement
Frank J. Rauscher, Jr.	[1931-1992]	PhD, 1957	sudden	NIH/NCI	Discovery of the Rauscher Murine Leukemia Virus
Donald J. Reis	[1931-2000]	MD, 1956	anticipated	Weill Medical College — Cornell University	neural control of blood circulation
Kenneth W. Sell	[1931-1996]	MD/PhD, 1968	anticipated	Emory University School of Medicine	Blood and Tissue Banking
Thomas G. Smith, Jr.	[1931-1998]	MD, 1960	sudden	NIH/NINDS	Studies of glial cell morphology in vivo and in vitro
Edward A. Smuckler	[1931-1986]	MD/PhD, 1963	anticipated	UCSF School of Medicine	cytochemical studies in liver injury
George F. Solomon	[1931-2001]	MD, 1955	sudden	UCLA School of Medicine	psychiatry and biobehavioural sciences
W. Alden Spencer	[1931-1977]	MD, 1956	anticipated	Columbia University College of Physicians & Surgeons	Plasticity of the Simplest Neuronal Pathways
Joseph W. St. Geme, Jr.	[1931-1986]	MD, 1956	anticipated	University of Colorado Health Sciences Center	pediatric infectious diseases
Wigbert C. Wiederholt	[1931-2000]	MD, 1955	anticipated	UCSD School of Medicine	age related neurodegenerative diseases in micronesia
Henryk M. Wisniewski	[1931-1999]	MD/PhD, 1960	sudden	SUNY Downstate Medical Center	pathogenesis of inflammatory demyelinating diseases
Richard P. Bunge	[1932-1996]	MD, 1960	anticipated	University of Miami School of Medicine	schwann cell biology and human spinal cord injury
Ralph R. Cavalieri	[1932-2001]	MD, 1956	sudden	UCSF School of Medicine	utilization of tyrosine by the thyroid gland
Robert A. Cooper, Jr.	[1932-1992]	MD, 1958	sudden	University of Rochester School of Medicine & Dentistry	radiation studies of the mouse distal lung
Ramzi S. Cotran	[1932-2000]	MD, 1956	anticipated	Harvard Medical School/Brigham & Women's Hospital	mechanisms of immune, infectious, and vascular renal injury
Dominick E. Gentile	[1932-1997]	MD, 1957	sudden	St Joseph Hospital–Orange, CA	Studies of hemo- and peritoneal dialysis
Richard K. Gershon	[1932-1983]	MD, 1959	anticipated	Yale Medical School	immunologic responses to tumor grafts
John P. Glynn	[1932-1971]	PhD, 1960	sudden	NIH/NCI	Immunosupression and the course of viral-induced Neoplasms
Edgar Haber	[1932-1997]	MD, 1956	anticipated	Harvard University School of Public Health	biological regulation of the renin-angiotensin system
Frank A. Oski	[1932-1996]	MD, 1958	anticipated	Johns Hopkins University School of Medicine	Erythrocyte Metabolism in the Premature Infant
Lawrence H. Piette	[1932-1992]	PhD, 1957	anticipated	Utah State University	electron spin resonance spectroscopy
George J. Schroepfer, Jr.	[1932-1998]	MD/PhD, 1961	sudden	Rice University	regulation of the formation and metabolism of cholesterol
Jurgen Steinke	[1932-1973]	MD, 1956	sudden	USC Keck School of Medicine/Rancho Los Amigos Hospital	Dynamics of Rat Fetal Insulin Secretion
Robert L. Summitt	[1932-1998]	MD, 1955	unknown	University of Tennessee Health Scences Center	clinical and chromosomal variation in children
Richard M. Asofsky	[1933-2000]	MD, 1958	anticipated	NIH/NIAID	T-cells in graft-versus-host disease
Marilyn Bergner	[1933-1992]	PhD, 1970	anticipated	Johns Hopkins School of Hygiene and Public Health	Cost and Efficacy of Home Care For COPD Patients
Vincent L. DeQuattro	[1933-2001]	MD, 1960	sudden	USC Keck School of Medicine	Role of Catecholamines in Hypertension
G. Harrison Echols, Jr.	[1933-1993]	PhD, 1959	anticipated	University of California — Berkeley	Genetic and chemical studies of phage lambda development
Julio H. Garcia	[1933-1998]	MD, 1958	sudden	Case Western Reserve University School of Medicine	Reperfusion in Experimental Brain Infarct
Gordon Guroff	[1933-1999]	PhD, 1959	sudden	NIH/NICHD	molecular mechanism of amino-acid conversion to neurotransmitters
E. Carwile LeRoy	[1933-2002]	MD, 1960	sudden	University of South Carolina School of Medicine	Structure and Immunology of Basement Membrane
Richard N. Lolley	[1933-2000]	PhD, 1961	sudden	USC Keck School of Medicine	Maturation of Metabolism in Normal and Dystrophic Retina
William J. Meyers	[1933-1970]	PhD, 1960	anticipated	University of Louisville School of Medicine	Autonomic Correlates of Attention in Infants
Sheldon D. Murphy	[1933-1990]	PhD, 1958	anticipated	University of Washington School of Medicine	Biochemical and Physiologic Response to Toxic Stress
Thomas F. Necheles	[1933-1984]	MD/PhD, 1961	sudden	NIH/NCI	Computer Assisted Classification of Acute Leukemia
Jerome T. Pearlman	[1933-1979]	MD, 1957	anticipated	UCLA School of Medicine	laboratory studies of retinal degenerations
Edward J. Sachar	[1933-1984]	MD, 1956	anticipated	Columbia University College of Physicians & Surgeons	chemicals in mental illness
John E. Salvaggio	[1933-1999]	MD, 1957	sudden	Tulane University School of Medicine	Mechanisms of immediate and delayed sensitivity in pulmonary disease
John C. Seidel	[1933-1988]	PhD, 1961	sudden	Boston Biomedical Research Institute	actin-myosin interaction in pulmonary smooth muscle
Donald C. Shreffler	[1933-1994]	PhD, 1961	sudden	Washington University in St. Louis School of Medicine	organization and functions of H-2 gene complex
Mehdi Tavassoli	[1933-1993]	MD, 1961	anticipated	University of Mississippi Medical Center	hematopoietic stem cell purification and biology
John R. Williamson	[1933-2000]	PhD, 1960	anticipated	University of Pennsylvania School of Medicine	molecular mechanisms of hormonal signal transduction

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
Ronald S. Wilson	[1933-1986]	PhD, 1959	sudden	University of Louisville School of Medicine	Heritability and Mental Development
Issa Yaghmai	[1933-1992]	MD, 1959	sudden	UCLA-Olive View Medical Center	radiological diagnosis of musculoskeletal disorders
Edwin H. Beachey	[1934-1989]	MD, 1962	anticipated	University of Tennessee Health Sciences Center	chemistry and immunology of streptococcal m proteins
Peggy J. Copple	[1934-1997]	MD, 1959	sudden	University of Arizona College of Medicine	Pediatrics Neurology
Sandra A. Daugherty	[1934-2000]	MD/PhD, 1966	anticipated	University of Nevada at Reno	studies of chronic fatigue syndrome, women's health initiative
Wylie J. Dodds	[1934-1992]	MD, 1960	anticipated	Medical College of Wisconsin	esophageal motor function in health and disease
Roger O. Eckert	[1934-1986]	PhD, 1960	anticipated	UCLA School of Medicine	ionic and metabolic mechanisms in neuronal excitability
Philip J. Fialkow	[1934-1996]	MD, 1960	sudden	University of Washington School of Medicine	origins of myeloid leukemia tumors
John Gibbon	[1934-2001]	PhD, 1967	anticipated	Columbia University College of Physicians & Surgeons	CNS functions underlying the interval time sense in animals and humans
Joram Heller	[1934-1980]	MD/PhD, 1965	anticipated	UCLA School of Medicine	biochemical and biophysical investigation of rhodopsin
James R. Klinenberg	[1934-1999]	MD, 1959	sudden	UCLA School of Medicine	pathophysiology of gout and hyperuricemia
Kenneth L. Melmon	[1934-2002]	MD, 1959	sudden	Stanford University School of Medicine	autacoids as pharmacologic modifiers of immunity
Gerald P. Murphy	[1934-2000]	MD, 1959	sudden	Roswell Park Cancer Institute	detection, immunotherapy, and prognostic indicators of prostate cancer
Harold C. Neu	[1934-1998]	MD, 1960	anticipated	Columbia University College of Physicians & Surgeons	surface enzymes in bacteria
George Némethy	[1934-1994]	PhD, 1962	anticipated	Mount Sinai School of Medicine	methods to analyze and predict the structures of protein molecules
John S. O'Brien	[1934-2001]	MD, 1960	anticipated	UCSD School of Medicine	discovery of the gene responsible for Tay-Sachs disease
Jiri Palek	[1934-1998]	MD, 1958	anticipated	Tufts University School of Medicine	membrane properties of abnormal red cells
Demetrios P. Papahadjopoulos	[1934-1998]	PhD, 1963	sudden	UCSF School of Medicine	phospholipid-protein interactions, lipid vesicles, and membrane function
Paul B. Sigler	[1934-2000]	MD/PhD, 1967	sudden	Yale Medical School	structural analysis of biological macromolecules
Donald F. Summers	[1934-2001]	MD, 1959	anticipated	NIH	composition, assembly and replication of RNA viruses
Howard M. Temin	[1934-1994]	PhD, 1959	anticipated	University of Wisconsin School of Medicine	molecular biology and genetics of tumor viruses
Lois W. Tice	[1934-1985]	MD, 1959	sudden	NIH/NIADDK	Anatomical and Physiological Structure of Cells and Tissues
Allan C. Wilson	[1934-1991]	PhD, 1961	anticipated	University of California — Berkeley	use of molecular approaches to understand evolutionary change
Alvito P. Alvares	[1935-2001]	PhD, 1966	sudden	Uniformed Services University of the Health Sciences	Biochemical Manifestations of Toxicity in Gold Therapy
John C. Liebeskind	[1935-1997]	PhD, 1962	anticipated	UCLA School of Medicine	behavioral and electrophysiological studies of pain
James R. Neely	[1935-1988]	PhD, 1966	sudden	Pennsylvania State University College of Medicine	effects of diabetes and oxygen deficiency in regulation of metabolism in the heart
Takis S. Papas	[1935-1999]	PhD, 1970	sudden	Medical University of South Carolina	characterization of ETS genes and retroviral onc genes
Ora M. Rosen	[1935-1990]	MD, 1960	anticipated	Memorial Sloan-Kettering Cancer Center	Cloning and characterization of gene for human insulin receptor
Bernard Sass	[1935-1989]	DVM, 1961	anticipated	NIH/NCI	Veterinary Pathology
James C. Steigerwald	[1935-1988]	MD, 1961	sudden	University of Colorado Health Sciences Center	Basica and clinical studies of scleroderma
Roy H. Steinberg	[1935-1997]	MD/PhD, 1965	anticipated	UCSF School of Medicine	pigment epithelium interactions with neural retina
Donald T. Witiak	[1935-1998]	PhD, 1961	sudden	University of Wisconsin School of Medicine	stereochemical studies of hypocholesterolemic agents
D. Martin Carter	[1936-1993]	MD/PhD, 1971	sudden	Rockefeller University	susceptibility of pigment and cutaneous cells to DNA injury by UV
Catherine Cole-Beuglet	[1936-1987]	MD, 1962	anticipated	University of California — Irvine	ultrasonography of the breast
James N. Gilliam	[1936-1984]	MD, 1964	anticipated	University of Texas Southwestern Medical Center at Dallas	cutaneous lupus erythematosus pathogenesis mechanisms
Oscar A. Kletzky	[1936-1994]	MD, 1961	anticipated	UCLA School of Medicine	ameliorating effects of estrogen replacement therapy on cerebral blood flow and sleep
Loretta L. Leive	[1936-1986]	PhD, 1963	anticipated	NIH/NIADDK	Role of bacterial cell surface in microbial physiology and pathogenesis
Shu-Ren Lin	[1936-1979]	MD, 1962	sudden	University of Rochester School of Medicine & Dentistry	imaging studies of cerebral blood flow after cardiac arrest
Dale E. McFarlin	[1936-1992]	MD, 1961	sudden	NIH	neuroimmunological studies of multiple sclerosis
Arnold M. Mordkoff	[1936-1971]	PhD, 1963	sudden	New York University School of Medicine	Physiological Patterns and Performance Efficiency
Sydney E. Salmon	[1936-1999]	MD, 1962	anticipated	University of Arizona College of Medicine	quantitative method for evaluating changes in myeloma tumor mass
Roy D. Schmickel	[1936-1990]	MD, 1961	sudden	University of Pennsylvania School of Medicine	isolation and characterization of human ribosomal DNA
Thomas W. Smith	[1936-1997]	MD, 1965	anticipated	Harvard Medical School/Brigham & Women's Hospital	Mechanism and reversal studies of digitalis
Joseph B. Warshaw	[1936-2003]	MD, 1961	anticipated	University of Vermont College of Medicine	developmental neurobiology of respiratory control
Nelson M. Butters	[1937-1995]	PhD, 1964	anticipated	UCSD School of Medicine	cognitive deficits related to chronic alcoholism
Thomas P. Dousa	[1937-2000]	MD/PhD, 1968	sudden	Mayo Clinic	cellular action of vasopressin in the kidney
Victor J. Ferrans	[1937-2001]	MD/PhD, 1963	sudden	NIH	myocardial and vascular pathobiology
David W. Fulker	[1937-1998]	PhD, 1967	anticipated	University of Colorado at Boulder	adoption studies of development in middle childhood
Patricia S. Goldman-Rakic	[1937-2003]	PhD, 1963	sudden	Yale Medical School	development and plasticity of the primate frontal lobe
A. Arthur Gottlieb	[1937-1998]	MD, 1961	sudden	Tulane University School of Medicine	role of macrophage nucleic acid in antibody production
Caroline T. Holloway	[1937-1998]	PhD, 1964	sudden	NIH/NCRR	Unsaturated Fatty Acid Biosynthesis in the Aorta
Jon I. Isenberg	[1937-2003]	MD, 1963	anticipated	UCSD School of Medicine	duodenal mucosal bicarbonate secretion in human
Chaviva Isersky	[1937-1986]	PhD, 1967	anticipated	NIH/NIDDK	characterization of mast cell receptors for immunoglobulin E
John J. Jeffrey, Jr.	[1937-2001]	PhD, 1965	sudden	Albany Medical College	mechanism of action and the physiologic regulation of mammalian collagenases
Takeo Kakunaga	[1937-1988]	PhD, 1966	sudden	NIH/NCI	Molecular carcinogenesis
Allastair M. Karmody	[1937-1986]	MD, 1963	anticipated	Albany Medical College	In situ vein bypass technique in femorodistal bypass surgery
Sandy C. Marks, Jr.	[1937-2002]	DDS/PhD, 1968	sudden	Umass Medical School	bone cell biology
William L. McGuire	[1937-1992]	MD, 1964	sudden	University of Texas Health Sciences Center at San Antonio	mechanisms of hormonal control and growth and regression of mammary carcinoma
Eva J. Neer	[1937-2000]	MD, 1963	anticipated	Harvard Medical School/Brigham & Women's Hospital	regulation and cellular levels of G protein subunits
Roland L. Phillips	[1937-1987]	MD/PhD, 1971	sudden	Loma Linda University School of Medicine	role of lifestyle in cancer and cardiovascular disease among Adventists
Mette Strand	[1937-1997]	PhD, 1964	anticipated	Johns Hopkins University School of Medicine	parasite immunochemistry and vaccine development
John J. Stuart	[1937-1986]	MD/PhD, 1971	unknown	Bowman Gray School of Medicine	cancer clinical trials
Roderich Walter	[1937-1979]	PhD, 1964	anticipated	University of Illinois at Chicago	Biofunctional Conformation of Peptide Hormones
Theodore S. Zimmerman	[1937-1988]	MD, 1963	anticipated	Scripps Research Institute	platelet/plasma protein interaction in blood coagulation
Merton R. Bernfield	[1938-2002]	MD, 1961	anticipated	Harvard Medical School/Children's Hospital	nature and interactions of cell surface proteoglycans during morphogenesis
Thomas F. Burks, 2nd	[1938-2001]	PhD, 1967	sudden	University of Texas Health Sciences Center at Houston	central and peripheral neuropeptide pharmacology
Cornelia P. Channing	[1938-1985]	PhD, 1966	anticipated	University of Maryland School of Medicine	Studies of the Mechanism of Luteinization in Vitro and in Vivo
Verne M. Chapman	[1938-1995]	PhD, 1965	sudden	Roswell Park Cancer Institute	development of cumulative multilocus map of mouse chromosomes
William L. Chick	[1938-1998]	MD, 1963	anticipated	Umass Medical School	studies of islet and beta cells in pancreatic transplantation
Bernard N. Fields	[1938-1995]	MD, 1962	anticipated	Harvard Medical School/Brigham & Women's Hospital	genetic and molecular basis of viral injury to the nervous system
J. Christian Gillin	[1938-2003]	MD, 1966	anticipated	UCSD School of Medicine	serotenergic mechanisms in sleep and depression
Walter F. Heiligenberg	[1938-1994]	PhD, 1964	sudden	UCSD School of Medicine	neuroethological studies of electrolocation
Lawrence D. Jacobs	[1938-2001]	MD, 1965	anticipated	SUNY at Buffalo School of Medicine and Biomedical Sciences	recombinant b interferon as treatment for Multiple Sclerosis
Emil T. Kaiser	[1938-1988]	PhD, 1959	sudden	Rockefeller University	mechanism of carboxypeptidase action
Irving Kupfermann	[1938-2002]	PhD, 1964	anticipated	Columbia University College of Physicians & Surgeons	Behavioral and neural analysis of learning in aplaysia
Samuel A. Latt	[1938-1988]	MD/PhD, 1971	sudden	Harvard Medical School/Children's Hospital	genetic and cytogenetic studies of mental retardation
Tai-Shun Lin	[1938-1994]	PhD, 1970	anticipated	Yale Medical School	synthesis and development of nucleoside analogs as antiviral and anticancer compounds
Barbara J. Lowery	[1938-2002]	PhD, 1973	anticipated	University of Pennsylvania School of Medicine	understanding stress responses of people who were physically ill
Harold A. Menkes	[1938-1987]	MD, 1963	sudden	Johns Hopkins University School of Medicine	occupational and environmental lung disease
Jane Pitt	[1938-2003]	MD, 1964	anticipated	Columbia University College of Physicians & Surgeons	perinatal transmission of HIV and retroviral infections
Theodore Reich	[1938-2003]	MD, 1963	anticipated	Washington University in St. Louis School of Medicine	genetic aspects of mental illness
Eleanor M. Saffran	[1938-2002]	PhD, 1968	anticipated	Temple University School of Medicine	cognitive deficits in brain-damaged patients
Elizabeth M. Smith	[1938-1997]	PhD, 1978	anticipated	Washington University in St. Louis School of Medicine	psychiatric problems among disaster survivors
John H. Walsh	[1938-2000]	MD, 1963	sudden	UCLA School of Medicine	gastrointestinal hormones, gastric acid production and peptic ulcer disease
Abraham Worcel	[1938-1989]	MD, 1963	sudden	University of Rochester School of Medicine & Dentistry	structure of interphase and metaphase chromosomes

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
Dolph O. Adams	[1939-1996]	MD/PhD, 1969	sudden	Duke University School of Medicine	Development and regulation of macrophage activation
James N. Davis	[1939-2003]	MD, 1965	sudden	SUNY Health Sciences Center at Stony Brook	mechanisms underlying neuronal injury after brain ischemia
Robert J. Fass	[1939-2002]	MD, 1964	anticipated	Ohio State University	In vitro methods to test antimicrobial susceptibility of infectious agents
Marian W. Fischman	[1939-2001]	PhD, 1972	anticipated	Columbia University College of Physicians & Surgeons	behavioral pharmacology of cocaine
Andreas R. Gruentzig	[1939-1985]	MD, 1964	sudden	Emory University School of Medicine	coronary angioplasty
Eric Holtzman	[1939-1994]	PhD, 1964	sudden	Columbia University College of Physicians & Surgeons	intracellular circulation of photoreceptor membranes
Donald J. Magilligan, Jr.	[1939-1989]	MD, 1965	sudden	Henry Ford Health Sciences Center	natural history and limitations of porcine heart valves
W. Frederick Sample	[1939-1979]	MD, 1966	unknown	UCLA School of Medicine	anatomic correlation of ultrasound
David S. Sigman	[1939-2001]	PhD, 1965	anticipated	UCLA School of Medicine	enzymology and gene targeting
C. Richard Taylor	[1939-1995]	PhD, 1963	anticipated	Harvard University	Energetics of animal locomotion
Kenneth J.W. Taylor	[1939-2003]	MD/PhD, 1975	anticipated	Yale Medical School	diagnostic ultrasound imaging
Richard J. Wyatt	[1939-2002]	MD, 1964	anticipated	NIH	biochemistry of schizophrenia
Nathaniel A. Young	[1939-1979]	MD, 1962	sudden	NIH/NCI	molecular biology of enteroviruses
Marshall H. Becker	[1940-1993]	PhD, 1968	anticipated	University of Michigan School of Medicine	elaboration of the Health Belief Model
Allan Beigel	[1940-1996]	MD, 1965	anticipated	Arizona State Hospital	therapeutic effectiveness of halfway house programs
Priscilla A. Campbell	[1940-1998]	PhD, 1968	anticipated	University of Colorado HSC/Nat. Jewish center	cell biology of the immune response to bacteria
Donald J. Cohen	[1940-2001]	MD, 1966	anticipated	Yale Medical School	Tourette's syndrome and autism in children
Anthony Dipple	[1940-1999]	PhD, 1964	sudden	NIH	metabolic activation and DNA interactions of polycyclic aromatic hydrocarbon carcinogens
D. Michael Gill	[1940-1990]	PhD, 1967	sudden	Tufts University School of Medicine	biochemistry of cholera toxin and other pathogenic toxins
Jeffrey A. Gottlieb	[1940-1975]	MD, 1966	anticipated	MD Anderson Cancer Center	combination chemotherapy regimens for treatment of soft tissue sarcomas
Keith Green	[1940-2001]	PhD, 1964	anticipated	Medical College of Georgia	ion and water movement in ocular tissues, ocular response to drugs
James L. Lehr	[1940-1989]	MD, 1968	anticipated	University of Chicago School of Medicine	Computer-mediated Radiology System
Robert M. Macnab	[1940-2003]	PhD, 1969	sudden	Yale Medical School	sequence analysis and function of bacterial flagellar motor
Melvin L. Marcus	[1940-1989]	MD, 1966	anticipated	Umass Medical School	cardiology, heart disease, coronary vascular adaptations to myocardial hypertrophy
David G. Marsh	[1940-1998]	PhD, 1964	anticipated	Johns Hopkins University School of Medicine	genetics of allergy and asthma
John N. Whitaker	[1940-2001]	MD, 1965	sudden	University of Alabama School of Medicine	Myelin Basic Protein Peptides in Body Fluids
Thomas S. Whitecloud, 3rd	[1940-2003]	MD, 1966	sudden	Tulane University School of Medicine	navigation techniques for minimal access spine surgery
Roger M. Brown	[1941-2002]	PhD, 1972	sudden	NIH/NIDA	Behavioral Sciences Research
Robert M. Joy	[1941-1995]	PhD, 1969	anticipated	University of California — Davis	pesticide induced changes in central nervous function
Robert A. Mendelson, Jr.	[1941-2001]	PhD, 1968	anticipated	UCSF School of Medicine	molecular mechanism of muscle contraction
Ethan R. Nadel	[1941-1998]	PhD, 1969	anticipated	Yale Medical School	thermoregulation during exercise and heat exposure
Samuel W. Perry, 3rd	[1941-1994]	MD, 1967	anticipated	Weill Medical College — Cornell University	psychological course of prolonged infection among AIDS patients
Harvey D. Preisler	[1941-2002]	MD, 1965	anticipated	Rush-Presbyterian-St Luke's Medical Center	clinical and biological studies of myeloid leukemias
Charles E. Putman	[1941-1999]	MD, 1967	sudden	Duke University School of Medicine	NMR Imaging Studies
Helene S. Smith	[1941-1997]	PhD, 1967	anticipated	UCSF School of Medicine	malignant progression of the human breast/predictors of breast cancer prognosis
Ronald G. Thurman	[1941-2001]	PhD, 1967	sudden	University of North Carolina at Chapel Hill School of Medicine	hepatic metabolism, alcoholic liver injury and toxicology
Philip G. Weiler	[1941-1991]	MD, 1965	anticipated	University of California — Davis	coronary heart disease & stroke in the elderly
Bruce M. Achauer	[1942-2002]	MD, 1967	sudden	University of California — Irvine	non-invasive methods to assess the depth of burn wounds
Laird S. Cermak	[1942-1999]	PhD, 1968	anticipated	Boston University Medical Center	psychological studies of memory and cognitive deficits related to chronic alcoholism
Christopher A. Dawson	[1942-2003]	PhD, 1969	sudden	Medical College of Wisconsin	pulmonary hemodynamics
Howard J. Eisen	[1942-1987]	MD, 1969	sudden	NIH/NICHD	Mechanism of action of glucocorticoid hormones
Bruce W. Erickson	[1942-1998]	PhD, 1970	anticipated	University of North Carolina at Chapel Hill School of Medicine	engineering of nongenetic beta proteins
Ronald D. Fairshter	[1942-1988]	MD, 1968	anticipated	University of California — Irvine	clinical studies in chronic obstructive pulmonary disease
Ira M. Goldstein	[1942-1992]	MD, 1966	anticipated	UCSF School of Medicine	pancreatitis, complement and lung injury
Richard E. Heikkila	[1942-1991]	PhD, 1969	sudden	UMDNJ Robert Wood Johnson Medical School	oxidation-reduction reactions and the dopamine receptor system
Pokar M. Kabra	[1942-1990]	PhD, 1972	sudden	UCSF School of Medicine	application of liquid chromatography to therapeutic drug monitoring
Michale E. Keeling	[1942-2003]	DVM, 1966	sudden	University of Texas MD Anderson Cancer Center	Resocialization of Chimpanzees
Henry C. Krutzsch	[1942-2003]	PhD, 1968	sudden	NIH/NCI	Studies of protein purification and sequencing
Joachim G. Liehr	[1942-2003]	PhD, 1968	anticipated	University of Texas Medical Branch at Galveston	mechanism of estrogen-induced carcinogenesis
Gregory Mooser	[1942-2003]	DDS/PhD, 1972	anticipated	USC Keck School of Medicine	characterization of glucosyltranserase enzymes secreted by oral bacteria
Alan S. Morrison	[1942-1992]	PhD, 1972	anticipated	Brown Medical School	hormones in the epidemiology of prostatic hyperplasia
Simon J. Pilkis	[1942-1995]	MD/PhD, 1971	sudden	University of Minnesota School of Medicine	carbohydrate metabolism and diabetes
B. Frank Polk	[1942-1988]	MD, 1967	anticipated	Johns Hopkins University School of Medicine	epidemiology of HIV infection
Robert M. Pratt, Jr.	[1942-1987]	PhD, 1970	sudden	NIH/NIEHS	Molecular studies of fetal cranofacial development
Julio V. Santiago	[1942-1997]	MD, 1967	sudden	Washington University in St. Louis School of Medicine	role of social factors, lifestyle practices, and medication in the onset of type II diabetes
Bruce S. Schoenberg	[1942-1987]	MD, 1980	anticipated	NIH	prevention and control of neurological disorders
Susan M. Sieber	[1942-2002]	PhD, 1971	anticipated	NIH/NCI	biochemical epidemiology and cancer
Michael Solursh	[1942-1994]	PhD, 1968	anticipated	University of Iowa College of Medicine	extracellular matrix and cell migration
Matthew I. Suffness	[1942-1995]	PhD, 1970	anticipated	NIH/NCI	Development of Taxol
Arthur T. Winfree	[1942-2002]	PhD, 1970	anticipated	University of Arizona College of Medicine	Principles of Temporal Organization
Ann L. Brown	[1943-1999]	PhD, 1967	sudden	University of California — Berkeley	learning and transfer processes in knowledge acquisition
Ahmad I. Bukhari	[1943-1983]	PhD, 1971	sudden	Cold Spring Harbor Laboratory	life cycle of mutator phage μ
Roland D. Ciaranello	[1943-1994]	MD, 1970	sudden	Stanford University School of Medicine	molecular neurobiology and developmental disorders
Fredric S. Fay	[1943-1997]	PhD, 1969	sudden	Umass Medical School	generation and regulation of force in smooth muscle
Charles A. Janeway, Jr.	[1943-2003]	MD, 1969	anticipated	Yale Medical School	innate immunity and T lymphocyte biology
George Khoury	[1943-1987]	MD, 1970	anticipated	NIH	genetics of simian virus 40, human papovavirus and HIV
Lee A. Lillard	[1943-2000]	PhD, 1972	sudden	University of Michigan School of Medicine	elderly health and health care utilization
Jonathan M. Mann	[1943-1998]	MD, 1974	sudden	Harvard University School of Public Health	AIDS prevention
Thomas A. McMahon	[1943-1999]	PhD, 1970	sudden	Harvard University	orthopedic biomechanics
William D. Nunn	[1943-1986]	PhD, 1972	sudden	University of California — Irvine	regulation of fatty acid/acetate metabolism in e. coli
James S. Seidel	[1943-2003]	MD/PhD, 1976	sudden	Harbor-UCLA Medical Center	clinical studies in pediatric life support and cardiopulmonary resuscitation
Donald L. Shapiro	[1943-1989]	MD, 1968	unknown	University of Rochester School of Medicine and Dentistry	Isolation and study of human type II pneumocytes
Milton H. Stetson	[1943-2002]	PhD, 1970	anticipated	University of Delaware	Comparative Endocrinology
Gerald L. Stoner	[1943-2002]	PhD, 1974	sudden	NIH/NINDS	neuropathology and molecular epidemiology of the human polyomavirus
James E. Bailey	[1944-2001]	PhD, 1969	anticipated	California Institute of Technology	basic measurements of genetically engineered cells and immobilized enzyme biocatalysts
G. Scott Giebink	[1944-2003]	MD, 1969	sudden	University of Minnesota School of Medicine	pathogenesis of otitis media and immunizations
Norton B. Gilula	[1944-2000]	PhD, 1971	anticipated	Scripps Research Institute	cell junction biosynthesis and biogenesis/cell-cell communication
Michael A. Kirschenbaum	[1944-1997]	MD, 1969	anticipated	University of California — Irvine	prostaglandins and kidney medicine
Peter A. Kollman	[1944-2001]	PhD, 1970	anticipated	UCSF School of Medicine	free energy perturbation calculations and their application to macromolecules
Joel D. Meyers	[1944-1991]	MD, 1970	anticipated	University of Washington/FHCRC	infections caused by suppression of the immune system in organ transplant and AIDS patients
Joaquim Puig-Antich	[1944-1989]	MD, 1967	sudden	University of Pittsburgh School of Medicine	psychobiology and treatment of child depression
Lonnie D. Russell, Jr.	[1944-2001]	PhD, 1974	sudden	Southern Illinois University School of Medicine	filament regulation of spermatogenesis
Don C. Wiley	[1944-2001]	PhD, 1971	sudden	Harvard University	viral membrane and glycoprotein structure
Roger R. Williams	[1944-1998]	MD, 1971	sudden	University of Utah School of Medicine	genetics and epidemiology of coronary artery diseases
Laurence A. Mack	[1945-1995]	MD, 1971	anticipated	University of Washington School of Medicine	use of ultrasound, computed tomography, and MRI in medical diagnosis

Investigator Name			Cause of death	Institution at the time of death	Scientific domain
John P. Merlie	[1945-1995]	PhD, 1973	sudden	Washington University in St. Louis School of Medicine	molecular genetics of the acetylcholine receptor
Lois K. Miller	[1945-1999]	PhD, 1972	anticipated	University of Georgia	genetics and molecular biology of baculoviruses
Peter M. Steinert	[1945-2003]	PhD, 1972	sudden	NIH	structures and interactions of the proteins characteristic of epithelial cells
Howard S. Tager	[1945-1994]	PhD, 1971	sudden	University of Chicago School of Medicine	biochemical structure, action, regulation and degradation of the insulin and glucagon molecules
David Tapper	[1945-2002]	MD, 1970	anticipated	University of Washington School of Medicine	Detection of Ocular Tumors
Harold M. Weintraub	[1945-1995]	MD/PhD, 1973	anticipated	University of Washington/FHCRC	characterization and function of MyoD gene
Gerald T. Babcock	[1946-2000]	PhD, 1973	anticipated	Michigan State University	bioenergetic mechanisms in multicenter enzymes
Mary Lou Clements	[1946-1998]	MD, 1972	sudden	Johns Hopkins University School of Medicine	AIDS Vaccine Evaluation
John M. Eisenberg	[1946-2002]	MD, 1972	anticipated	Georgetown University Medical Center	Derived Thresholds in Medical Decision Making
Ira Herskowitz	[1946-2003]	PhD, 1971	anticipated	UCSF School of Medicine	genetics of yeast mating type
Stanley R. Kay	[1946-1990]	PhD, 1980	sudden	Albert Einstein College of Medicine	symptoms and diagnostic tests of schizophrenia
Sukdeb Mukherjee	[1946-1995]	MD, 1971	sudden	Medical College of Georgia	Neuroleptic Effects on Regional Cerebral Blood Flow
John J. Wasmuth	[1946-1995]	PhD, 1973	sudden	University of California — Irvine	human-hamster somatic cell hybrids/localization of Hnyington's disease gene
Elizabeth A. Bates	[1947-2003]	PhD, 1974	anticipated	UCSD School of Medicine	cross-linguistic studies of language development, processing and breakdown in aphasia
John G. Gambertoglio	[1947-2001]	PharmD, 1972	anticipated	UCSF School of Medicine	pharmacokinetics in healthy volunteers and subjects with renal insufficiency and on hemodialysis
Janis V. Giorgi	[1947-2000]	PhD, 1977	anticipated	UCLA School of Medicine	cellular immunology of resistance to HIV
Leonard N. Horowitz	[1947-1992]	MD, 1972	anticipated	University of Pennsylvania School of Medicine	diagnosing and treatment of ventricular arrythmia
Jeffrey M. Isner	[1947-2001]	MD, 1973	sudden	Tufts University School of Medicine	therapeutic angiogenesis in vascular medicine, cardiovascular laser phototherapy
Markku Linnoila	[1947-1998]	MD/PhD, 1974	anticipated	NIH	studies on the biological bases of impulsivity and aggression
John B. Penney, Jr.	[1947-1999]	MD, 1973	sudden	Harvard Medical School/Massachusetts General Hospital	receptor mechanisms in movement disorder pathophysiology
Lynn M. Wiley	[1947-1999]	PhD, 1975	sudden	University of California — Davis	morphogenesis in early mammalian embryos
Michael E. Burt	[1948-1997]	MD/PhD, 1981	sudden	Memorial Sloan-Kettering Cancer Center	Isolated lung perfusion for patients with unresectable metastases from sarcoma
Larry C. Clark	[1948-2000]	PhD, 1981	anticipated	University of Arizona College of Medicine	nutritional prevention of cancer
Terry L. Thomas	[1948-2002]	PhD, 1986	anticipated	NIH/NCI	radiation health effects
Trudy L. Bush	[1949-2001]	PhD, 1977	sudden	University of Maryland School of Medicine	postmenopausal estrogen/progestins interventions
Neil S. Jacobson	[1949-1999]	PhD, 1977	sudden	University of Washington School of Medicine	marital therapy, domestic violence, and the treatment of depression
John L. Kemink	[1949-1992]	MD, 1975	sudden	University of Michigan School of Medicine	Clinical studies of cochlear implantations
Richard P. Nordan	[1949-1998]	PhD, 1983	sudden	FDA/CBER	discovery of interleukin 6
Eva U.J. Paucha	[1949-1988]	PhD, 1976	anticipated	Harvard Medical School/Dana Farber Cancer Institute	mechanism of transformation by SV40 large T antigen
Tsumao Saitoh	[1949-1996]	PhD, 1977	sudden	UCSD School of Medicine	altered protein kinases in alzheimer's disease
Robert F. Spencer	[1949-2001]	PhD, 1974	anticipated	Medical College of Virginia	neuroanatomy of the oculomotor system
Kiertisin Dharmsathaphorn	[1950-1990]	MD, 1972	anticipated	UCSD School of Medicine	intestinal secretory mechanisms and antidiarrheal drugs
JoAnn E. Franck	[1950-1992]	PhD, 1981	anticipated	University of Washington School of Medicine	hippocampal damage as a cause of epilepsy
Gary J. Miller	[1950-2001]	MD/PhD, 1978	sudden	University of Colorado Health Sciences Center	vitamin D receptors in the growth regulation of prostate cancer cells
Elizabeth A. Rich	[1951-1998]	MD, 1977	sudden	Case Western Reserve University School of Medicine	natural history of lymphocytic alveolitis in hiv disease
Nava Sarver	[1951-2001]	PhD, 1978	anticipated	NIH/NIAID	Targeted AIDS Drug Discovery
Jeffrey M. Hoeg	[1952-1998]	MD, 1977	sudden	NIH/NHLBI	Studies of familial hypercholesterolemia
Thomas K. Tatemichi	[1952-1995]	MD, 1978	anticipated	Columbia University College of Physicians & Surgeons	mechanisms and syndromes of dementia related to stroke
Roberta D. Shahin	[1953-1997]	PhD, 1985	sudden	FDA/CBER	Studies of Protective Immunity in Pertussis
Matthew L. Thomas	[1953-1999]	PhD, 1981	sudden	Washington University in St. Louis School of Medicine	function and regulation of leukocyte surface glycoproteins
Mu-En Lee	[1954-2000]	MD/PhD, 1984	sudden	Harvard Medical School/Massachusetts General Hospital	characterization of vascular smooth muscle LIM protein
Thomas L. O'Donohue	[1954-1987]	PhD, 1980	sudden	NIH/NIMH	discovery of new central peptidergic pathways
Ernest G. Peralta	[1959-1999]	PhD, 1986	anticipated	Harvard University	signal transduction mechanisms of muscarinic receptors
Alan P. Wolffe	[1959-2001]	PhD, 1984	sudden	NIH	role of DNA methylation in regulating gene expression in normal and pathological states
Eugenia Spanopoulou	[1960-1998]	PhD, 1988	sudden	Mount Sinai School of Medicine	Biochemistry and Regulation of V(D)J Recombination

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