

# How citation distortions create unfounded authority: analysis of a citation network

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## ABSTRACT

**Objective** To understand belief in a specific scientific claim by studying the pattern of citations among papers stating it.

**Design** A complete citation network was constructed from all PubMed indexed English literature papers addressing the belief that  $\beta$  amyloid, a protein accumulated in the brain in Alzheimer's disease, is produced by and injures skeletal muscle of patients with inclusion body myositis. Social network theory and graph theory were used to analyse this network.

**Main outcome measures** Citation bias, amplification, and invention, and their effects on determining authority.

**Results** The network contained 242 papers and 675 citations addressing the belief, with 220 553 citation paths supporting it. Unfounded authority was established by citation bias against papers that refuted or weakened the belief; amplification, the marked expansion of the belief system by papers presenting no data addressing it; and forms of invention such as the conversion of hypothesis into fact through citation alone. Extension of this network into text within grants funded by the National Institutes of Health and obtained through the Freedom of Information Act showed the same phenomena present and sometimes used to justify requests for funding.

**Conclusion** Citation is both an impartial scholarly method and a powerful form of social communication. Through distortions in its social use that include bias, amplification, and invention, citation can be used to generate information cascades resulting in unfounded authority of claims. Construction and analysis of a claim specific citation network may clarify the nature of a published belief system and expose distorted methods of social citation.

## INTRODUCTION

Biomedical knowledge arises from scientific data. The means by which this occurs within individual scientific papers is a generally accepted process whereby papers report rationale, methods, results, and conclusions. How an entire belief system shared by a scientific community ultimately evolves from data across all papers within a specialty is less well understood. I describe and apply methods for the analysis of such belief systems using a specific example.

The belief system studied is that a protein,  $\beta$  amyloid, known for its role in injuring brain in Alzheimer's disease, is also produced by and injures skeletal muscle fibres in the muscle disease sporadic inclusion body myositis. This belief system was chosen for analysis because of its importance to the care of patients with inclusion body myositis, as this view seems to be accepted by many as likely or established fact (at least 200 different journal articles have stated such), with  $\beta$  amyloid production often reported to be a central element in the pathogenesis of the disease (see web extra note 1), and directs research and treatment trials in the specialty. The approach taken here was simply to collect all statements in the medical literature on this belief system and to study the pattern of citation among them—that is, how each statement is supported by reference to other papers.

## METHODS

The methods are fully described in web extra note 2. Briefly, queries identified all English language PubMed indexed articles potentially containing statements pertaining to any of three related molecules ( $\beta$  amyloid precursor protein, its transcript, or one of its potential cleaved protein products,  $\beta$  amyloid) and muscle disease. These 766 papers (see web extra table 1) were searched for statements addressing the belief that these molecules are abnormally and specifically present in muscle fibres of patients with inclusion body myositis among many other muscle diseases, identifying 302 papers<sup>1-302</sup> addressing the broad category of "amyloid" and inclusion body myositis of which 242 papers discussed these specific molecules (see web extra table 2). I collected all statements addressing the belief and citations supporting these statements. Each paper was classified as primary data (containing experimental data addressing the specific and abnormal presence of these molecules in inclusion body myositis muscle), myositis review (review papers with the term myositis or the equivalent in their title), model (reporting cell culture or animal model experiments), or other (all other papers). I classified each citation as supportive, neutral, or critical according to how its underlying statement supported the belief. A network was then constructed representing papers as nodes and citations as links from one node to another.

Another investigator (Anthony Amato) validated text and citation extraction for 17% of the papers, including all primary data papers.

This citation network was further extended into research proposals funded by the US National Institutes of Health, obtained through the Freedom of Information Act in accordance with National Institutes of Health policy.

This claim specific citation network was then analysed using graph theory<sup>303</sup> (see methods in web extra). Briefly, custom MATLAB software (MathWorks; Natick, MA) and the MatlabBGL package (written by David Gleich) were used for the analysis of adjacency matrices representing these networks. A centrality measure<sup>304</sup> on the papers was defined (called the citation path index; similar to other variants of centrality measure<sup>305</sup>). Authority was measured according to the method of Kleinberg.<sup>306</sup> Visualisation of networks was carried out using Pajek (<http://vlado.fmf.uni-lj.si/pub/networks/pajek/>). The maximum likelihood estimate method<sup>307</sup> was implemented in MATLAB, with code available from [www.santafe.edu/aaronc/powerlaws/](http://www.santafe.edu/aaronc/powerlaws/).

## RESULTS

### Authority and belief in a claim specific citation network

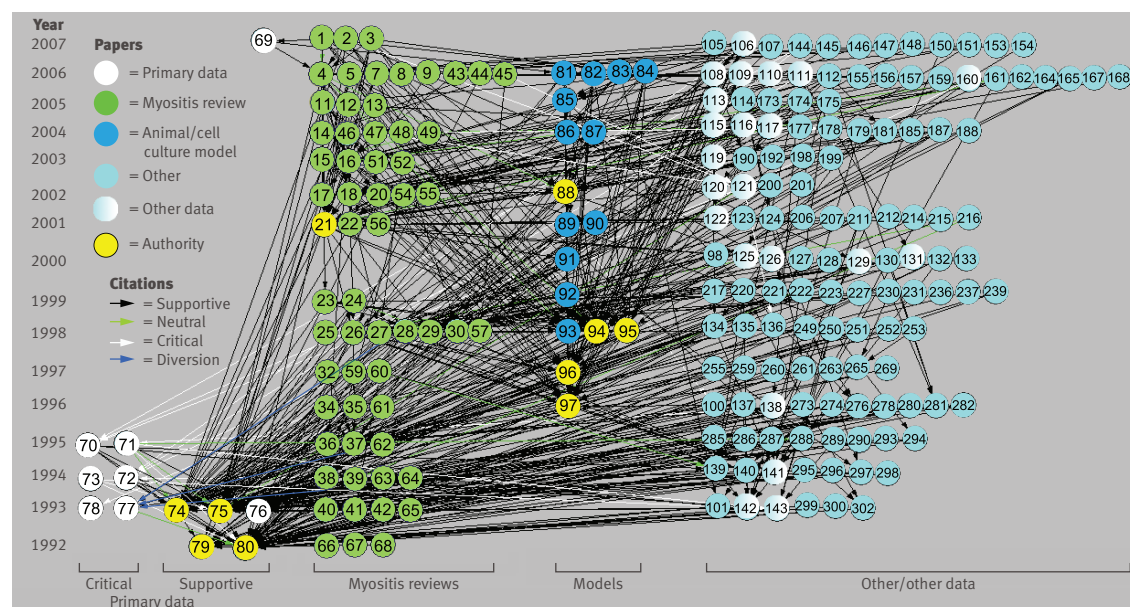
The claim that  $\beta$  amyloid and its precursors are abnormally and specifically present in inclusion body myositis muscle fibres among many other muscle diseases was studied. The 242 papers containing statements addressing it (all exact text provided in web extra table 3) and the 675 citations (not counting duplicates

from one paper to another; see web extra table 4) supporting these statements were used to construct a claim specific citation network (fig 1). This network contained 220 609 citation paths, with chains of citations flowing from one paper to the next representing the entire National Library of Medicine PubMed indexed discourse on the claim as of 26 October 2007. The historical growth and various mathematical properties<sup>308</sup> of this network are discussed in web extra note 3.

Within networks certain nodes may be recognised as “authorities”,<sup>306</sup> receiving large amounts of network traffic. Such authorities can be identified by computational methods alone through examining the patterns of connections among the nodes; this is how many internet search engines identify authoritative web pages. Because citation is in part an act of communication within a community of people, social network theory<sup>309</sup> in particular can be used to analyse it. Under social network theory, authority of a claim indicates the community’s net belief about it. Using these computational methods,<sup>306</sup> four primary data papers, five model papers, and one review paper constituted the 10 most authoritative papers. All these papers expressed the view that the claim was true.

### Citation bias against critical primary data

Of the 10 most authoritative papers, four provided experimental data addressing the claim, reporting the presence of these molecules in inclusion body myositis muscle fibres.<sup>74 75 79 80</sup> All four papers were from the same laboratory, two of which<sup>79 80</sup> probably reported mostly the same data without citing each other, a



**Fig 1** Claim specific citation network. Citations regarding claim that  $\beta$  amyloid precursor protein mRNA or protein, or  $\beta$  amyloid protein, is abnormally present in inclusion body myositis muscle. The network is organised according to paper category and year of publication. Authority status (yellow) was defined computationally by network theory. Many citations flow to supportive primary data but not critical data. Papers are represented as nodes (n=218) and citations as directed edges (supportive n=636, neutral n=18, critical n=21, diversion n=3). Twenty four papers contain statements pertaining to claim but do not make or receive citations about it (not shown).

practice currently viewed as one that distorts available evidence (see web extra note 4). Major technical weaknesses were present in these papers, most notably a lack of quantitative data as to how many affected muscle fibres were seen and a lack of specificity of reagents for distinguishing  $\beta$  amyloid protein from  $\beta$  amyloid precursor protein (see web extra note 5).

Inspection of the network disclosed six primary data papers that were relatively isolated, receiving no or few citations (fig 1). These papers contained data that refuted or weakened the claim. Three papers<sup>71 73 77</sup> from independent laboratories reported that in a combined 35 patients with inclusion body myositis studied, 28 had no affected muscle fibres while the remaining seven had five or fewer affected muscle fibres (typical biopsy sections contain thousands of muscle fibres). Two papers<sup>70 72</sup> by the laboratory that wrote the four authority papers reported that two of these molecules ( $\beta$  amyloid precursor protein transcript and protein) were not specific to inclusion body myositis but were present in muscle fibres during regeneration in all diseased controls (up to 43 patients in seven disease categories, including polymyositis, dermatomyositis, Duchenne muscular dystrophy, and amyotrophic lateral sclerosis). These findings weaken the view that abnormal amounts of these molecules have any specificity to inclusion body myositis and that they cause degeneration of myofibre in patients with inclusion body myositis. One of these papers reported that all three molecules, including  $\beta$  amyloid, were produced by muscle invading macrophages in inclusion body myositis and all other inflammatory myopathies,<sup>70</sup> offering an alternative source than myofibre production for them and indicating that  $\beta$  amyloid was non-specifically present in other inflammatory myopathy muscle (see web extra note 6 for a detailed discussion of these papers).

To understand why supportive but not critical data achieved authority over the ensuing 12 years since publication of all of these data, the number of citations received by each paper was analysed (fig 2). The supportive papers received 94% of the 214 citations to these primary data, whereas the six papers containing data that weakened or refuted the claim received only 6% of these citations (differing citation frequency,  $P=0.01$ ). Citation bias, here defined as statistically significant differences in the number of citations received among primary data papers, seemed to be specifically against critical data not the laboratory producing it, as two papers<sup>70 72</sup> that were biased against were written by the same research group that wrote four of the highly cited supportive papers. For example, one of the papers<sup>70</sup> addresses a crucial question in the specialty, the relation between inflammation and degeneration,<sup>1-39</sup> but reported data that potentially conflicted with the belief that  $\beta$  amyloid is produced by inclusion body myositis myofibres or is uniquely present in inclusion body myositis muscle (reporting that  $\beta$  amyloid is produced by muscle invading macrophages in all inflammatory myopathies). These data have never been cited by their authors despite them

having made 104 citations about  $\beta$  amyloid to other primary data papers.

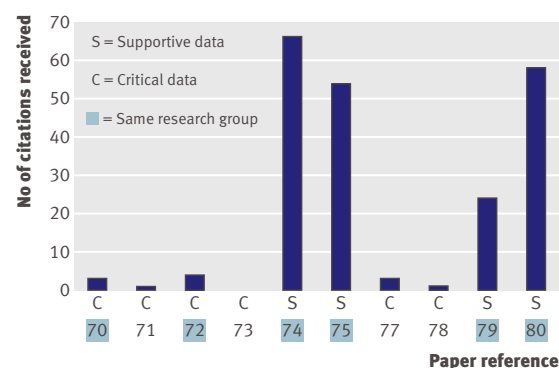
### Citation bias to justify models

Citation bias has also been used to claim that animal and cell culture experiments are valid models of inclusion body myositis, in 17 papers.<sup>81-97</sup> Of the 32 citations to primary data from these papers, 31 (97%) flowed to the four highly supportive papers,<sup>74 75 79 80</sup> whereas only one citation (3%) was made to any of the six papers that presented data weakening or refuting these as valid models for inclusion body myositis (fig 3). For example, one paper<sup>83</sup> cited another<sup>74</sup> in support of “abnormal accumulation of A $\beta$ -containing inclusions are present in skeletal muscle of IBM patients” but not papers that found no<sup>71</sup> or little<sup>73 77</sup>  $\beta$  amyloid protein. Similarly, the same paper<sup>83</sup> cited a paper<sup>75</sup> in support of “there is evidence that APP [amyloid precursor protein] mRNA levels are selectively enhanced in human IBM [inclusion body myositis] samples thereby providing physiological justification for the overexpression of this protein in transgenic mice,” but not the paper<sup>73</sup> that found no  $\beta$  amyloid precursor protein mRNA or the paper,<sup>72</sup> by the same authors as the paper,<sup>75</sup> that found  $\beta$  amyloid precursor protein mRNA not “selectively enhanced” in inclusion body myositis but present in muscle fibres in all other muscle diseases examined. The uncited data<sup>72</sup> suggest that the animal and cell culture experiments are no more models of inclusion body myositis than any other neuromuscular disease in which muscle regeneration occurs.

### Citation diversion

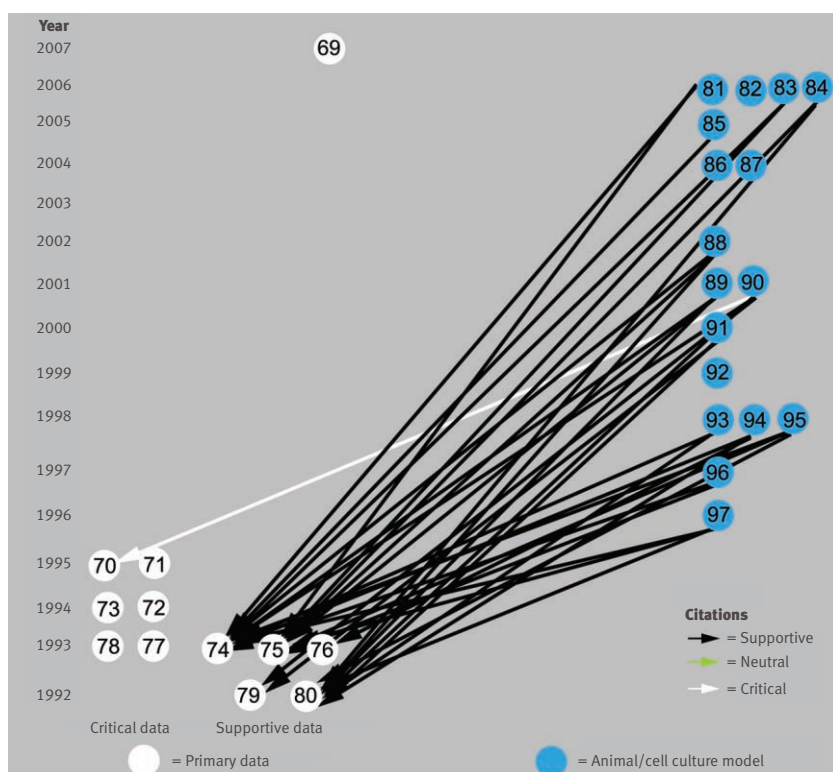
Some papers cited content but distorted it. This is not citation bias, as papers are cited, but rather a different process called here “citation diversion”—that is, the citing of content but the altering of its meaning in a manner that diverts its implications.

One primary data paper<sup>77</sup> reported no  $\beta$  amyloid precursor protein or  $\beta$  amyloid in three of five patients with inclusion body myositis and its presence in only a “few fibres” in the remaining two patients. Three



**Fig 2** | Citation bias against content critical of claim. Shown are citation frequencies to four authoritative supportive primary data papers and six primary data papers<sup>70-75 77-80</sup> containing data critical of claim





**Fig 3** Citations from animal and cell culture model papers to primary data papers supporting rationale for overproduction of  $\beta$  amyloid precursor protein mRNA as a valid model of inclusion body myositis. Only one of 32 citations flows to papers<sup>70-73 77 78</sup> that present data that conflict with the validity of these models

papers<sup>28 37 38</sup> cited these data (fig 1) reporting that they “confirmed” the claim (for example, one paper<sup>38</sup> said “ $\beta$ APP<sup>76 77</sup> in s-IBM fibers has been confirmed by others”). Whether such data confirm the claim is perhaps open to interpretation. At the least these data are exaggerated and generalised into a view that  $\beta$  amyloid precursor protein is “accumulated in vacuolated muscle fibers of s-IBM patients<sup>77[other]</sup>” as stated by one paper,<sup>28</sup> supported by an erroneous citation because three patients in the paper<sup>77</sup> had 1.4% to 5% of their myofibres vacuolated but all lacked  $\beta$  amyloid precursor protein. Over the ensuing 10 years, these three supportive citations developed into 7848 supportive citation paths—chains of false claim in the network created by citation diversion.

In another example of citation diversion one paper<sup>81</sup> stated “Thus, it has been widely accepted that intracellular accumulation of  $\beta$ APP, A $\beta$  [ $\beta$  amyloid] and other  $\beta$ APP proteolytic fragments play an important role in the pathogenesis of IBM,<sup>86 89</sup>” although one of the papers<sup>89</sup> had not widely accepted this claim, stating “A $\beta$ -intracellular deposition may be an epiphenomenon unrelated to myofiber death.”

#### Amplification through influential papers and citations

Between 1996 and 2007 support for the claim grew exponentially, with the number of supportive citations and citation paths increasing sevenfold and 777-fold, to

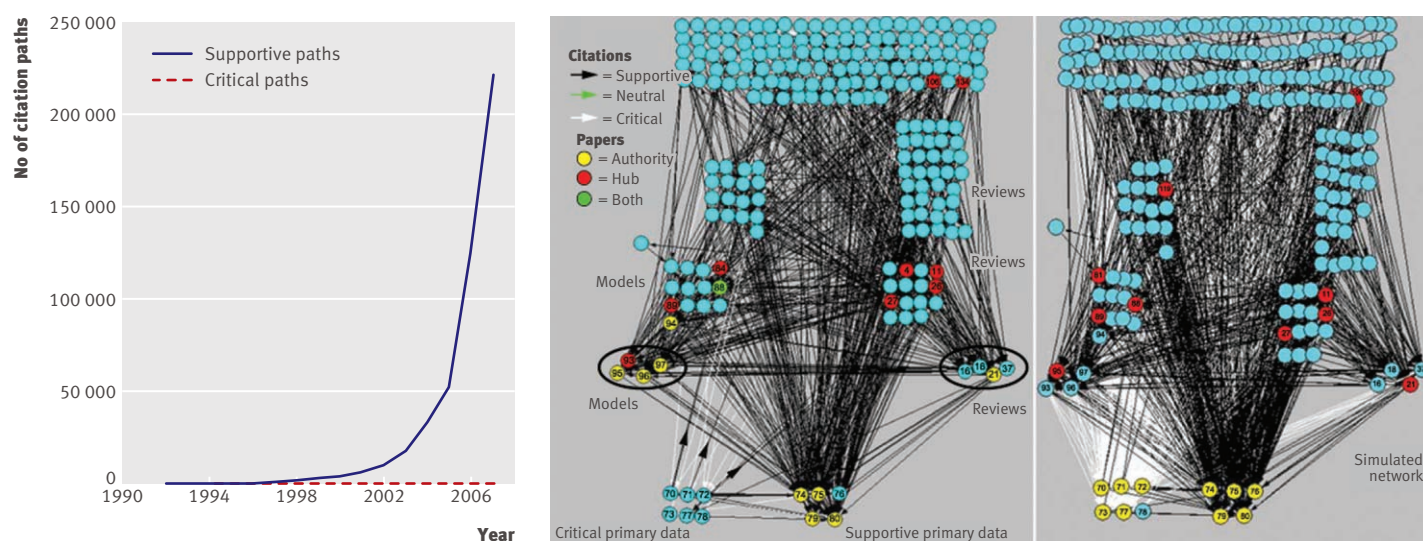
636 citations and 220 553 citation paths. In contrast, the critical view grew to only 21 citations and 28 citation paths (fig 4). No papers refuted or critiqued the critical data, but instead the data were just ignored. Analysis of a claim specific citation network can identify exactly which papers and citations have been most influential in pushing forward belief<sup>304 305</sup> (see web extra note 7). The increased support was facilitated by a small number of papers, not reporting any primary data, through which large amounts of traffic (citation paths) flow in the network. For example, 63% of all citation paths (n=139 391) flow through one review paper<sup>21</sup> (compared with 2% of citation paths flowing through randomly selected other papers); 95% of all citation paths flow through four review papers<sup>16 18 21 37</sup> by the same research group (8% through four randomly selected other papers).

A lens effect was present in which a small number of these influential review papers and model papers containing no data on claim validity collected and focused citation (similar to a magnifying lens collecting light) on particular primary data papers supportive of the belief, while isolating others that weakened it (fig 4). Such papers have a network property known as high betweenness centrality.<sup>304</sup>

The term amplification can be used to describe the expansion of a claim’s belief system by citation to papers lacking any data addressing it, the phenomenon observed here. Amplification is not inherent to published belief systems. Authors could choose to cite only primary data when making claims, resulting in amplification minimal networks. Amplification of a claim is instead introduced into belief systems through the citing of review papers and other papers that lack data addressing the claim. Certainly such papers may be cited for other reasons; amplification only arises when they are cited to support claims of experimental results reported elsewhere. (See web extra note 8 for further discussion of amplification and methods for quantifying it.)

#### Network authority emerges through bias against critical content and amplification

Papers may be biased against for many potential reasons. To examine the role of bias exclusively against critical content in establishing authority, a simulated network was constructed in which all statements making a supportive claim were amended to recognise critical views of equivalent content and temporal availability. Removing bias against critical content was sufficient to result in authority status for five of the six infrequently cited primary data papers (fig 4), indicating that authority status of the claim emerges from the citation bias against critical content. The claim cannot be both true and false; the resulting balanced authority of supportive and refuting papers indicates that without citation bias there would be balanced belief in its truth and falseness (see web extra note 9).



**Fig 4 | Amplification and authority of claim.** (Left) Historical growth of supportive and critical citations in network. (Middle) Rearrangement of network (fig 1) to visualise a lens effect in which eight key papers (surrounded by ovals; seven by same research group; 97% of all network traffic passes through them) create citation flows among each other, and both amplify claim and focus citations to supportive data papers. Net effect results in network authority status<sup>306</sup> (yellow) of supportive data papers. (Right) Computational elimination of citation bias results in balanced authority of both support for claim and its refutation through additional recognition of critical data papers<sup>70-73,77</sup> as authorities

### Invention

Distinct from citation bias and amplification, certain types of fact developed and spread through the belief system. These particular facts were not those that arose from restatement of published claims, but rather involved different mechanisms either deliberate or through scholarly negligence, herein called invention. For example, a subclaim (that the accumulation of  $\beta$  amyloid occurs early and precedes other abnormalities) has variously been stated as hypothesis, likelihood, or fact in 27 papers supported by 37 citations (see web extra note 10). Nine of these citations (24%), used to support text making these claims, in fact flowed to papers that contained no statement on the temporal relation of  $\beta$  amyloid to other abnormalities in inclusion body myositis muscle (dead end citations). This subclaim had transformed from hypothesis to “fact” through citation alone, a process that might be called citation transmutation (fig 5). Thus one paper<sup>5</sup> contained it as fact (“The appearance of A $\beta$ -positive, non-conophilic deposits precedes vacuolization in IBM muscle fibers<sup>80</sup>”) supporting this statement by citing the paper<sup>80</sup> where it had only been proposed as hypothesis (“may represent early changes of IBM”). Similarly, another paper<sup>134</sup> reported this as fact (“our previous studies demonstrated that abnormalities of  $\beta$ APP precede other changes including congophilia<sup>74,80,141</sup>”) even though the cited papers stated it only as hypothesis<sup>74,80</sup> or made no statement at all<sup>141</sup> about the accumulation of  $\beta$  amyloid precursor protein preceding other abnormalities.

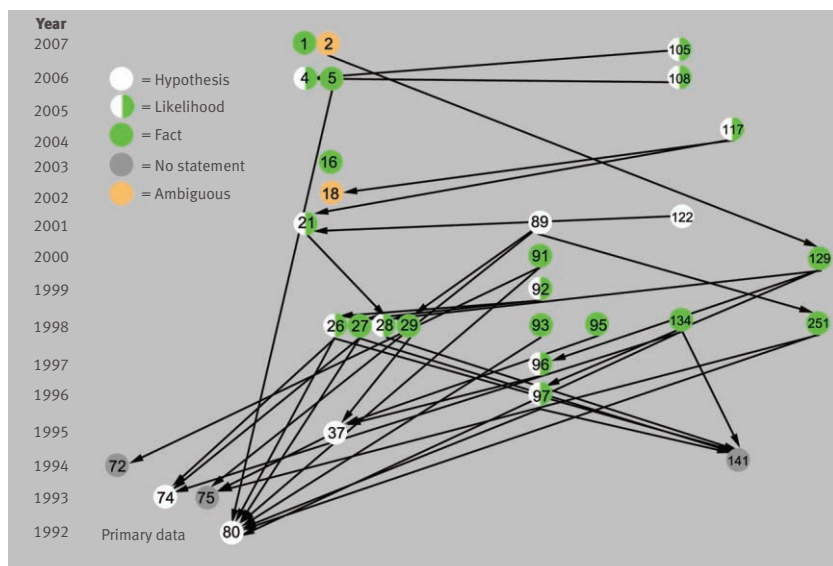
In another form of invention, claims are introduced as fact through a “back door” that bypasses peer review and publication of methods and data. This is accomplished by repeated misrepresentation of abstracts as papers (seven different papers, 17 citations to 12

different misrepresented abstracts; for example, citation to *Neurol* 2003;60:333-334, an abstract with correct listing *Neurol* 2003;60(suppl 1):A333-4; see web extra note 11). The claim that “ $\beta$ -amyloid<sub>42</sub> isoform [is] more common than  $\beta$ -amyloid<sub>40</sub>”<sup>74</sup> is supported in this manner and accepted by peers as fact (paper 2 states this citing paper 4) (see web extra note 12 for another form of invention called title invention).

### Bias and invention in National Institutes of Health funded research proposals

Through the publication of scientific papers and the demonstration of these publications as evidence of productivity, the elements of bias, amplification, and invention can be used indirectly to support requests for research funding. To determine if these mechanisms were used directly to support such requests, the claim specific citation network was extended from the PubMed indexed literature into the research sections and bibliographies of National Institutes of Health funded grant proposals containing text addressing the claim, obtained under the Freedom of Information Act according to National Institutes of Health policy.<sup>310</sup> Of 27 grant proposals requested (identified through searches of the National Institutes of Health CRISP database as described in web extra note 13), nine were released by the National Institutes of Health. These seemed to be the proposals most pertinent to the belief system.

Citation bias or invention was present in eight of nine of these proposals (fig 6). Of 23 citations to primary data (not counting multiple citations from one proposal to a single paper) addressing the claim’s validity, 20 were made to supportive primary data (19 supportive citations and one neutral citation), two were instances of citation diversion (one paper<sup>77</sup> again



**Fig 5** | Conversion of hypothesis to fact through citation alone. Citations on statement that accumulation of  $\beta$  amyloid "precedes" other abnormalities in inclusion body myositis muscle. Statement as fact is supported through citation to papers that only state it as hypothesis (for example, references 5 to 80, 91 to 80, 134 to 74) or sometimes supported by citation to papers that contain no statements addressing it (for example, references 91 to 72, 251 to 75; dead end citations). This phenomenon might be called citation transmutation (see web extra note 10 for statements)

cited for supporting the claim when it weakens it), and one was made to critical content. Invention of fact supported through citation to hypothesis, dead end citation, and abstracts misrepresented as papers were similarly present in these funded proposals. These were sometimes used directly to justify requests for funding of the proposed studies (for example, "The accumulation of epitopes of  $\beta$ APP is an early event in the disease relative to the other changes,<sup>37,96</sup> justifying our focused investigation of A $\beta$ "; one paper<sup>37</sup> stated this only as hypothesis; the other paper<sup>96</sup> stated this as likelihood not fact, supporting that view also through citation to the other paper,<sup>37</sup> stated as hypothesis (see web extra note 13 for further discussion).

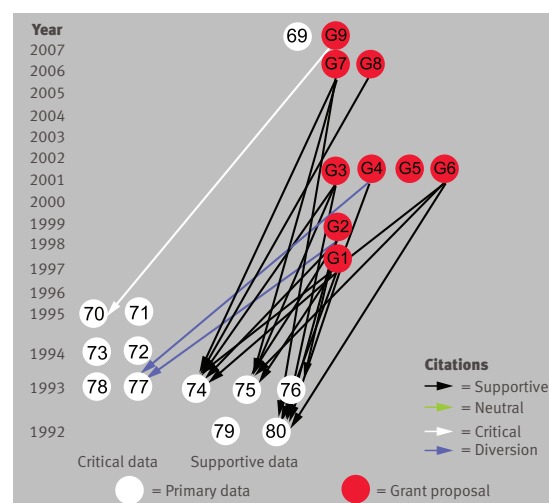
## DISCUSSION

Citation, the act of connecting text statements through reference to the broader literature, is not simply an impartial scholarly method for joining related published knowledge. Citation may be used for self serving purposes<sup>311</sup> or as a tool for persuasion<sup>312</sup> (see web extra note 14). These aspects of citation might be called social citation. I studied how distortions of the persuasive aspect of social citation may result in broad acceptance of unfounded claims as fact. These distortions can be detected and interpreted through social network theory<sup>309</sup> because citation as persuasion is a social behaviour. Network theory applied to citation networks constructed from entire paper bibliographies, such as the science citation network,<sup>313</sup> can disclose societal attitudes to journals and specific papers (for example, impact factors), but these networks are not

suitable for understanding the foundation for belief in specific claims. When networks are instead confined to citation pertaining to one set of related claims (a claim specific citation network), they become sharply focused tools for understanding social communication pertaining to the claims—what is in effect the published record of a belief system shared by a community. These allow for study of not just what is said about a belief (the traditional scope of review papers), but also who hears it and how it is retold.

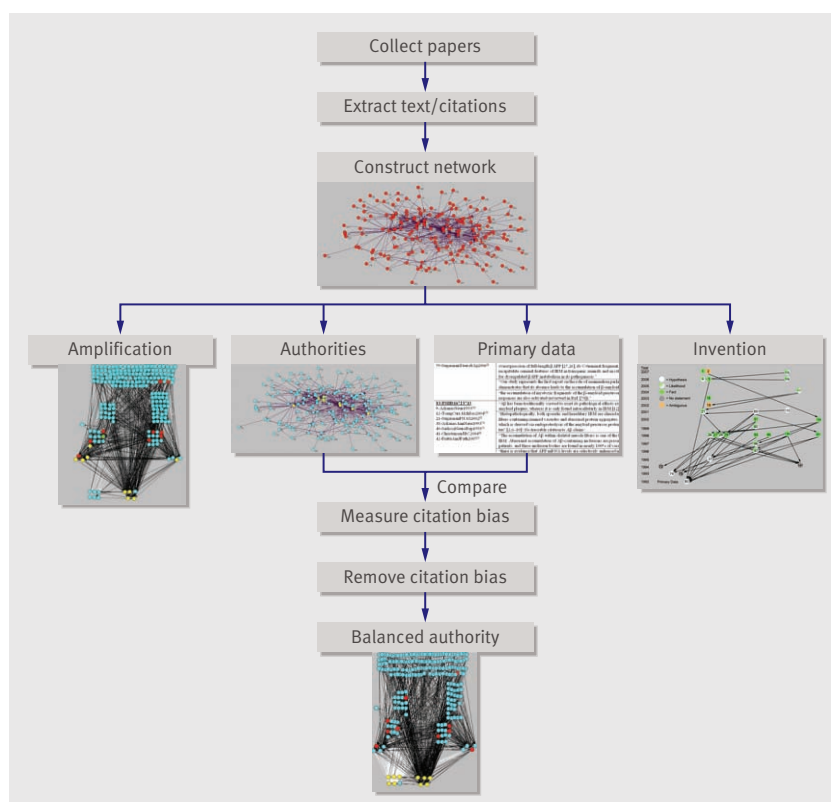
The general approach taken here (fig 7) addressed belief in claims; no experiments were done addressing their truth. The computational analysis of the claim specific citation network representing this belief system detected certain distortions in the patterns of citation that would not have been expected had only scholarly citation been used. Primary data that weakened or refuted claims on which the belief was based were ignored (citation bias) and a small number of influential papers and citations exponentially amplified supportive claim over time without presenting new primary data (amplification). Certain related claims were invented as fact. The combined effects of these citation distortions resulted in authority of the belief (acceptance of it) according to social network theory.

There are varied forms and consequences of distorted persuasive citation seen in this study (see box). Citation bias against critical content can be used for the systematic support of claim,<sup>314</sup> results in the loss of implications of isolated data (see web extra note 15), and can be used to justify construction of animal models, which can then be circularly used to amplify claim (see web extra note 16). Such animal models have enormous appeal, and some publications describing them achieved authority status in this network (fig 1)



**Fig 6** | Extension of PubMed claim specific citation network into National Institutes of Health funded research proposals. Nine funded research grants (G1-G9; see web extra note 13) contain statements and citations addressing claim; their citations to primary data are shown. Citation bias and citation diversion are present





**Fig 7 | Overview of approach.** After construction of the claim specific citation network, a combined manual and computational endeavour, steps on left (determination of authorities [yellow papers] and identification of amplification) require only computational algorithms; right half (identifying which papers contain actual data addressing claim validity and identifying invention) requires careful reading of paper content. Combining results of authority identification with data identification allows for recognition of citation bias and subsequent steps for its simulated removal and assessment of effects on network

despite reporting no data addressing the claim—that is, whether these  $\beta$  amyloid related molecules are present in human inclusion body myositis muscle. Amplification involves repetitive citation of review papers or other papers lacking data, often through self citation, features noted previously in a variation of a claim specific citation network.<sup>315</sup> Invention has multiple variations.

Three factors may account for how citation distortions created authority in this belief system. Foremost is the power of citation through the choice of which papers to cite and which to ignore (citation bias), by citing but distorting content (citation diversion), and by using citation to invent fact (citation transmutation, dead end citation, and back door invention).

Second is an inherent property of negative results, which failed to spread through the network. These were not repeatedly cited by their authors in subsequent papers (only one instance was present<sup>274</sup>) as perhaps there was simply nothing further to say about them. Unlike “positive results” there is nothing exciting to be repeatedly written about how something was not found in an experiment. Thus the progression from data to accepted claim is different within a single paper

compared with across many papers in a specialty. Within a single paper readers generally view new claims as false until proved true through convincing methods and results. Across a network of papers, however, the barrier to the propagation of negative results biases claims as being viewed as true until proved false.

Thirdly, this belief system is possibly an information cascade (also called an informational cascade),<sup>316,317</sup> an entity resulting when people perceive advantage in accepting the prevailing view over any private information they may have when making choices. Indeed certain mathematical properties of information cascades (preferential attachment) would be expected to produce a network with properties seen here (a biased network with a power law distribution of node degrees; see web extra note 3). Many authors may just not be aware of the critical data, as these data are effectively isolated from the discourse about this claim and not mentioned in any review articles. Although unsound information cascades are in theory fragile and fall apart quickly when exposed,<sup>316</sup> this may not occur in biomedical belief systems, where contradicted claims may persist.<sup>318</sup>

Many published biomedical belief systems may be information cascades because repetition of claims is ubiquitous in the biomedical literature. Many are built on sound data, with authors repeating claims after trusting the published expert opinion of their colleagues. However, there are incentives for generating and joining information cascades regardless of their soundness. Joining an information cascade aids publication as articles have to say something and negative results are biased against.<sup>319</sup> Generating and joining an information cascade may improve the likelihood of obtaining research funding because hypothesis driven research is an essential requirement<sup>320</sup> at many research funding agencies such as the National Institutes of Health, and successful funding generally requires a “strong hypothesis . . . based on current scientific literature”<sup>320</sup>—that is, the published belief system of a claim. Chances for successful funding may therefore be increased through joining the cascade (repeating the claim and proposing experimental plans around it). In the extension of this citation network into text within grant proposals that have been funded by the National Institutes of Health, citation bias, diversion, or invention were often present. Once research funding has been used to join a cascade there are further incentives to interpret results through confirmation bias (“in a way that confirms one’s preconceptions and to avoid information and interpretations which contradict prior beliefs”<sup>321</sup>) to demonstrate success of the research for subsequent funding. Although joining an information cascade may be an optimal behaviour for some people, it reduces the likelihood that future investigators can discover whether it is sound.<sup>317</sup>

Methods for the construction and analysis of comprehensive claim specific citation networks present challenges and limitations. These include interpreting meaning of text, as people may reasonably interpret

## Vocabulary of citation distortions

### Citation

Both scholarly and social forms: the scholarly form connects statements to the broader medical literature, the social form (social citation) includes self serving and persuasive subtypes

### Citation distortions

Self serving citation is always a distortion

Persuasive citation may be necessary to communicate new, sound claims to the scientific community; it may, however, have distorted uses—citation bias, amplification, and invention

### Citation bias

Systematic ignoring of papers that contain content conflicting with a claim

Bolster claim; justifying animal models to provide opportunities to amplify claim

### Amplification

Expansion of a belief system without data

Citation made to papers that don't contain primary data, increasing the number of citations supporting the claim without presenting data addressing it

### Invention

*Citation diversion*—citing content but claiming it has a different meaning, thereby diverting its implications

*Citation transmutation*—the conversion of hypothesis into fact through the act of citation alone

*Back door invention*—repeated misrepresentation of abstracts as peer reviewed papers to fool readers into believing that claims are based on peer reviewed published methods and data

*Dead end citation*—support of a claim with citation to papers that do not contain content addressing the claim

*Title invention*—reporting of “experimental results” in a paper’s title, even though the paper does not report the performance or results of any such experiments

text differently, and understanding the distinct phenomena observed (see web extra note 17 for a discussion of these issues). In principle many biomedical claims have an associated citation network, the study of which provides a powerful approach to detecting citation bias, amplification, and invention, and understanding the nature of the authority of the claim.

## WHAT IS ALREADY KNOWN ON THIS TOPIC

In addition to its scholarly use, citation has social uses, both self serving and as a tool for persuasion

One distortion of this persuasive aspect of citation, citation bias, has been recognised in clinical trial reporting where it may lead to false belief about a therapy’s efficacy

## WHAT THIS STUDY ADDS

Distortions in the persuasive use of citation—bias, amplification, and invention—can be used to establish unfounded scientific claims as fact

Categorising these distorted uses of citation and having vocabulary for them aids in their recognition

How scientific data evolve into entire published biomedical belief systems around specific claims can be studied through a device called a claim specific citation network and the use of social network theory

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**Competing interests:** SAG had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

**Ethical approval:** Not required.

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