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# A review on citation amnesia in depression and inflammation research

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**Abstract**

Once original scientific results are published the author has the "intellectual property" and may claim ownership. Discovery credit is one of the most important "rewards" for scientists and thus incorrect credits undermine the reward system of science. Scientists who publish should therefore give proper credit and acknowledge the primary sources. Failure to do so is regarded as "citation negligence", "the disregard syndrome", "citation amnesia", "plagiarism by omission", "bibliographic plagiarism" or "citation plagiarism", and may range from an unconscious or conscious "failure to credit a prior discoverer so as to give an improper impression of priority" to "the appropriation of another person's ideas or results without given proper credit". False discovery credit is considered to be "a menace to honest science", "a serious transgression" or "intellectual theft, be it intentional or not".

This paper describes some examples of citation amnesia showing that scientists often fail to credit prior sources and give false discovery credit to other scientists. One example is the association between major depression and activated immuno-inflammatory pathways, a discovery by European groups and published in many papers since 1990. Now, 25 years later, it is commonplace that these theories are credited to secondary American sources whose work in "the last decade", did or did not examine these pathways in major depression. This gives an improper impression of priority of American-based scientists. Here it is proposed that this citation amnesia and plagiarism reinforced the wrong science and had negative effects on the development of immune-inflammatory biomarkers and new immune-related treatments for depression. It is concluded that journal editors should improve their citation standards to guarantee correct assignment of discovery credit for example by demanding a signed pledge from the authors that correct citations to the primary sources were made.

COMMENTARY

## INTRODUCTION

Recently, an American-based author, published a review starting with a statement that the associations between inflammation and stress and depression have been recognized for over a decade and then the author refers to American-based scientists who published their theories in 2006–2011 (Littrel 2012). Before I reviewed that paper for another journal where it was rejected. My major concerns were that a) this American author did not credit the scientists who discovered these pathways as

primary sources and did not mention other European groups as primary sources; and b) the narrow scope of the review further reinforced the wrong science. I wrote in my review, which was sent to this author, that the first sentence of the paper should read "The association between systemic *immune-inflammatory pathways and* depression has been recognized for over *two* decades" and that European authors should be credited as primary sources, i.e. Maes *et al.* (1990 and beyond); Sluzewska *et al.*; Seidel *et al.*; Song & Leonard; Xia *et al.*; etc. [Due to space limitations I will cite only two of our review articles (Leonard & Maes 2012; Maes *et al.* 2012) and give in italics the authors and publication year of the original publications, which are also listed in these two reviews]. I wrote in that review that not only the inflammatory hypothesis of depression was wrongly attributed to American-based authors, but also specific depression/stress-related pathways, including glucocorticoid resistance explained by cytokines (Maes *et al.* 1991), activation of indoleamine dioxygenase in depression and interferon-α-induced depression (Maes *et al.* 1993; 1994, 2000; etc) and lowered omega-3 PUFAs in depression and increased inflammatory responses in individuals with lowered omega-3 (Maes *et al.* 1996; 2000). Indeed, the abovementioned European authors discovered most if not all aspects of the immune-inflammatory theory of depression one decade before American scientists started with their inflammation-related research in major depression. Nevertheless, in their re-submission and publication this "bibliographic negligence" was not corrected. It should be stressed however that this American author made an attempt to cite a few European authors, albeit not the primary sources, in contrast to many other American authors who only cite American sources.

Many authors now reassign discovery credit of the immune-inflammatory theory of depression from the primary European sources to two American-based groups. It is now commonplace that original findings made by European groups, including increased IL-6 signaling in depression (Maes *et al.* 1993; 1995), are reattributed to American-based authors some of whom even never published on IL-6 in major depression.

## DEFINITIONS OF CITATION AMNESIA AND CITATION PLAGIARISM

Every scientist who publishes papers should be aware of the relevant literature and should properly credit and acknowledge the primary source (Koch 2003). Failure to do so is regarded as "the disregard syndrome" or "citation amnesia" (Garfield 2002; Koch 2003; Ginsberg 2001). Other descriptions for this kind of plagiarism are "plagiarism by omission", "citation negligence", "petty larceny plagiarism", "the appropriation of another person's ideas or results without given proper credit", "a conscious failure to credit a prior discoverer so as to give an improper impression of priority", "the deliberate presentation of another's texts or ideas as one's own" and "take insights or lines of arguments from another author and give only weak or no acknowledgments at all" (Garfield 1979; 1980; 1987; 1989; Martin 1984; LaFolette 1992; Delahunty 2009; Nature Genetics 2009). "Using another scientist's ideas or logic without due reference is intellectual plagiarism" (Nature Photonics 2009).

Once the original results are published the author may claim ownership and the ownership is reinforced by citations (Garfield 1980). The ideas resulting from the primary findings thus are the "intellectual property" of the author(s) who reported the original findings. Moreover, discovery credit is, for most scientists, the most important "reward" and therefore false credits and attributions undermine the reward system of science (Garfield 1982). "Science depends upon trust, credit and attribution" (Nature Genetics 2009). "Published ideas, thoughts, concepts and results are the tangible essence of a scientist and must be defended" (Nature Photonics 2009). Therefore, false discovery credit is considered to be a "menace to honest science", a "serious transgression" or "intellectual theft", be it intentional or not (Garfield 1991; 2002; Nylenna *et al.* 1999; Ginsburg 2001). Nature (2009) in an Editorial concludes that "Editors are obliged to act if concerns are raised about improper attribution" and that "public humiliation will act as a deterrent to those who passed off another's work as their own".

## CITATION AMNESIA VERSUS CITATION PLAGIARISM

Nevertheless, in my opinion, a distinction should be made between citation amnesia (unconscious) versus citation plagiarism (conscious). The American author of the abovementioned paper had of course no direct personal advantage by not citing the original discoverers and giving credit to American-based groups. Because the author has no direct conflict of interest it cannot be labeled as "citation plagiarism". Nevertheless, in an email response to me this author writes that she respects my work and agrees that I was the first to make seminal contributions. "You can be proud that others have entered into a field that you initiated. When others follow a lead investigator that generally attests to the importance of the initial contributions". Thus while it is confirmed that my research groups initiated the field, discoverer credit in publications is given to American-based scientists. Other primary European sources are not cited although they contributed to the discoveries and replicated the pathways one decade before American scientists initiated their research on inflammation in depression. Thus, this American author gives an improper impression of American priority and therefore it cannot be denoted as citation amnesia, which is unconsciously. Knowing that due credit is seldom if ever given by American authors to the original concepts

which arose outside of the USA, I propose to call this mechanism "American citation plagiarism". The aim of this procedure commonly applied by some, but not all, American scientists is to strengthen the impact of American versus non-American research (authors' and journals' impact factors).

## THE DYNAMICS OF CITATION AMNESIA AND CITATION PLAGIARISM

Until 2008 my discoveries and the discoveries by other European groups were reinforced by many citations bringing me in the top 100 of most cited psychiatrists-psychologists. However, as of 2008–2009 and quite suddenly two American scientific groups became the gold standard to be cited as primary sources by many American authors and later also by many (but not all) non-American authors. The dynamics of this sudden switch ?

In 2008, I got an email from one of the acting directors at one of the divisions of the National Institutes of Health (NIH, Bethesda, USA) that my inflammatory-depression theories were "co-opted" by an American-based group in a review paper in a leading neuroscience journal (2008). In this review, the American-based authors cite one editorial by Maes *et al.* (1995) and consequently discredit Maes *et al.* repeatedly: "Maes failed to attract the interest of the psychiatric community" (although Maes since 2003 has been an ISI and Tompsom Reuters highly cited author) and the "postulate of Maes that common pathophysiological mechanisms link depression to inflammation was limited".

The American authors then claim that the "postulate" of Maes *et al.* was limited because "biomarkers of inflammation in clinically depressed patients are not always elevated". These authors, however, failed at citing the relevant literature on immuno-inflammatory biomarkers and their diagnostic performance (e.g. Maes 1993; 1995). Many biomarkers are now well consolidated in meta-analyses (see Leonard & Maes 2012).

Although a key component of the review, these American-based authors do not cite Maes' discoveries (1990 and beyond) that the major pro-inflammatory and Thelper-1 cytokines are increased in depression. Two of these papers (Maes *et al.* 1990; 1991), showing upregulated IL-2-related mechanisms and thus immune activation, were published prior to what these American authors quote as the first paper on the role of cytokines in depression, i.e. a hypothesis paper published by my friend Ronald Smith (Smith 1991). In contrast to Smith's macrophage hypothesis, my original findings in 1990–1994 showed not only inflammation, but especially activation of immune-inflammatory and cell-mediated immune (Thelper-1) pathways, including lowered tryptophan and activation of indoleamine dioxygenase (IDO). Therefore, I and Ronald Smith together published an editorial on the monocyte-T-lymphocyte theory of depression, which considered all seminal findings of my research groups, the first replication studies and previous results which were not interpreted as showing activation of immuno-inflammatory pathways (Maes *et al.* 1995).

In their review paper (2008) the American authors continue and claim that "Maes did not provide proof that decreasing the inflammatory response attenuates symptoms of depression". However, they silence all primary reports of Maes and other European groups on the effects of antidepressants on immune-inflammatory pathways in humans and animal models and how the complex antidepressant-induced changes in these pathways are associated with clinical improvement or treatment resistance (Maes *et al.* 1995; 1996; 1996; 1997; 1997; 1997; 1999; 1999; 2005; Maes 2001; 2002; Lin *et al.* 2000; Kubera *et al.* 2000; 2000; 2000; 2000; 2001; 2004; 2005; 2005; Kenis & Maes 2002).

Then the American authors further discredit the primary sources : "other key components that would support this "postulate" were also missing, such as a demonstration that stimulation of the immune system induces depression-like disorders and identification of a possible common pathophysiological mechanism between the effects of cytokines and the neurobiological basis of depression". However, these American authors fail again to cite key findings of Maes *et al.* that immune activation may cause depression via specific pathophysiological mechanisms, e.g. activation of immune-inflammatory pathways and lowered levels of tryptophan through IDO activation by cytokines.

Surprisingly, the same American authors then ascertain that they discovered the key inflammatory component of depression, i.e. lowered levels of tryptophan through IDO activation by cytokines. They credit only papers by themselves, e.g. their first "landmark" paper being published in 2002 and showing that IFNα-induced reductions in tryptophan are associated with the onset of depression. However, these American-based authors fail to credit prior reports showing that this tryptophan-IDO pathway is activated in depression (Maes *et al.* 1993; 1994; 1996; 1997; 1998; 2001; 2001; 2002; Song *et al.* 1998;etc). They fail to credit my earlier papers showing that IFNα-induced changes in IDO activity or reductions in plasma tryptophan are associated with the onset of depression (Maes *et al.* 2001; Bonaccorso *et al.* 2002). They fail to cite my earlier review that the pathophysiology underpinning IFNα-induced depression is associated with lowered tryptophan and activation of the IDO pathway (Bonaccorso *et al.* 2000). Then they fail to give credits to the first original data and reviews on the new hypothesis that not tryptophan depletion but the formation of neurotoxic tryptophan catabolites is involved in the pathophysiology of mood states (postnatal), IFNα-induced depression and maybe clinical depression (Maes *et al.* 2001; 2001; 2002; 2002; Kubera & Maes 2000; Bonaccorso *et al.* 2000; 2002; Wichers *et al.* 2005; Wichers & Maes 2004; Bonaccorso & Maes 2004). These Ameri-

can authors thus have omitted the primary sources and by not citing the primary sources reassign discoverer credit to themselves and appropriate the original findings on IDO activation in depression and IFNα-induced depression.

## HOW EUROPEAN THEORIES ARE SWEPT UNDER THE CARPET AND CO-OPTED BY AMERICAN-BASED AUTHORS

Finally (as top of the bill), the same American-based authors claim to describe a "*new hypothesis, set out in their Review*, that depression can actually be caused by inflammation in vulnerable patients" and they incorrectly credit themselves and one other American research group (2006) for that "new" theory. They do not credit the discoverers who published the original findings and ensuing theories that depression may be induced by activated immuno-inflammatory pathways in some vulnerable individuals (Maes 1993; 1995; 1997; 2005; Maes *et al.* 1997; 1997; 1999; van West & Maes 1999; Bonaccorso *et al.* 2000; Kubera & Maes 2000; Maes *et al.* 2001; Bonaccorso *et al.* 2002; Wichers & Maes 2002; 2004; Schiepers *et al.* 2005; etc). They do not even mention that Maes *et al.* stated in the cited editorial (Maes *et al.* 1995): "if activation of monocytes of lymphocytes is at all related to the pathophysiology or pathogenesis of major depression, it could account for the fact that a wide diversity of etiologic factors, such as organic factors, e.g. infections, cancer, autoimmune disorders, injuries, the postpartum period, as well as psycho-social stressors can be accompanied by major depression in some vulnerable subjects". They do not cite earlier reviews by other European groups.

Thus, this review in a leading journal gives an incorrect impression of precedence, first discrediting the primary sources with incorrect statements, silencing 18 years of European research results and then claiming that they present a "new" hypothesis. Thereby these American authors reassign credit for the theories of Maes and collaborators and other European groups to themselves and one other American group. Thus, one American paper suffices to sweep 18 years of European pioneering research under the carpet.

## RESPONSES OF AUTHORS AND JOURNALS TO COMPLAINTS ABOUT CITATION AMNESIA/PLAGIARISM

Evidently, the Editors of that leading journal and the individuals who acted as reviewers did not take care to guarantee correct assignment of discovery credit. In 2013, the Group that published the paper (G) faced with my complaints decided not to take any action because "While G sympathises with your concerns, G is satisfied that your contribution is appropriately credited in the paper. G is accordingly unable to assist you any further ...". The authors' US University declared that "The complaint filed by Maes ... is disingenuous and frivolous. The complete lack of merit of the complaint and the superficiality of the basis for the complaint may indicate maliciousness". The American organization that supported the research of this review concluded that: "You alleged that the authors ... of the review paper ... have failed in their review to appropriately interpret or include/credit important work that you and other groups have done in the subject matter. While it may be reasonable to consider such a review to lack comprehensiveness and to be inadequate, such differences in opinion are specifically excluded from the PHS definition of research misconduct". Nevertheless, in my complaint letter it was stated that there are no differences in opinion at all since I fully agree with my own theories that were co-opted in the American review.

Other American scientific journals faced with citation amnesia violation complaints sometimes start their first line of defense by debating that copyright laws may not apply in the case of intellectual theft as they do not protect ideas, but only the actual expression of ideas. Another example is a paper published by an American author who attributed the inflammation and depression theory to the two abovementioned American groups. While the author has no direct advantage to credit other individuals than the discoverer, the author gives – as American – credit to American-based scientists and thus commits "American citation plagiarism". I did not receive any apologizes from the author only an aggressive letter from her American departmental head. The editor of the journal refuses to examine the case properly and thus does not adhere to good Publication Ethics Policies for Medical Journals, as have been described for example by COPE – The Committee on Publication Ethics (http://publicationethics.org/).

In another journal run by an American editor, increased levels of cytokines in depression, e.g. interleukin-6 (IL-6), are incorrectly attributed to the American authors who wrote the abovementioned review and who never published one paper on IL-6 in major depression. The editor proposed me to write a "respectful review" on IL-6 and pain, not to correct the citation amnesia. It is interesting to note that other leading American psychiatric journals, which now refuse to correct American citation amnesia in their journals, rejected the first original papers on the immune-inflammtory hypothesis of depression in the 1990s with nonsense remarks, e.g. "when there are no findings", "I don' get a message from this paper" and "badly understood immune tests".

And then finally also non-American authors start to credit the abovementioned American authors as primary sources. One funny example is a paper in a Pakistani journal attributing the inflammatory theory of depression to the two abovementioned American groups, while there is no reference at all to the Euro-

pean groups who discovered the pathways. In this case, there is clearly no direct or indirect advantage as the author is not American, thus it should be denoted as "citation amnesia." Probably a young copycat, copying what he read in one of the American leading journals that now massively credit Americans as the major contributors to the field. Nevertheless, their reactions are remarkable: the author who committed citation amnesia claims "Maes is unreasonable" and the editor of the journal writes a long letter that one has to be humble ("As one grows in academic stature, one becomes more humble") and proposes that I may write a letter-to-the-editor to explain "my version" while the authors may respond with "their version". Also this Editor refuses to examine the case and thus does not adheres to COPE rules. Luckily there are few journals, e.g. "Journal of Neuroinflammation" that promptly intend to correct and adjust the bibliographic omissions made. Clearly, there is no consensus among editors and publishers how to handle scientific intellectual property theft.

## THIS KIND OF CITATION AMNESIA HAS REINFORCED THE WRONG SCIENCE

Another issue is that this kind of citation amnesia reinforced the "wrong science". Thus, in the gold standard American literature of the "last decade" (i.e. the two abovementioned American research groups) the connection between depression, sickness behavior and inflammation and the association between IFNα-induced depression through IDO activation became the main focus of interest, while measurements of a few pro-inflammatory biomarkers (especially IL-6) and C-reactive protein (CRP) were proposed to be the gold standard biomarkers.

First, the connection between sickness behavior and depression, which became a gold standard in depression research, is non existent (Maes *et al.* 2012). Indeed, we explained somewhere else that sickness behavior and depression are two different conditions although increased cytokine levels may be related to both conditions (Maes *et al.* 2012). One wonders how much grant money has been waisted to research into a beneficial, short-lasting acute inflammtory response to acute immune injury as an incorrect model for a chronic complex disease such as major depression, characterized by chronic and detrimental (auto) immune alterations.

Second, the gold standard American research does not consider other established and more important immune-inflammatory-related pathways. Over two decades ago (1990–1991) it was already summarized that depression is a disorder characterized by an interrelated upregulation of cell-mediated immune (CMI) activation, inflammation and autoimmunity, not only "inflammation". More recent and new immune-related pathways in depression are oxidative and nitrosative stress (O&NS), lowered antioxidant levels, autoimmune reactions to neoepitopes, sensitization of immune-inflammtory pathways, increased bacterial translocation, etc. (Leonard & Maes 2012). Interestingly, not one of these pathways was discovered by American groups. By focusing too much on acute sickness behavior as a model for major depression, these new pathways were largely neglected by American scientists and are still neglected. We can only wait until also these pathways will also be co-opted in the near future.

Third, by focusing on some selected biomarkers, such as IL-6 and CRP, many other discoveries were neglected. For example, more appropriate biomarkers of clinical depression were already established in the 1990s, e.g. the acute phase reactants haptoglobin, albumin and zinc (Maes *et al.* 1991; 1992; 1996). Another example is the measurement of plasma IL-6 in depression, a discovery by Maes *et al.* now commonly attributed to American groups. Nevertheless, pro-inflammatory IL-6 signaling (now called IL-6 trans-signaling) can only be evaluated when plasma IL-6 and the soluble IL-6 receptor (sIL-6R) levels (and sgp130) are measured simultaneously (Maes *et al.* 1995; 2013). Surprisingly, the gold standard American groups only consider plasma IL-6 as a valid biomarker. Until today they did not recognize that IL-6 trans-signaling is the key phenomenon underpinning inflammatory IL-6 signaling (Maes *et al.* 1995). Thus, they lag far behind the state of the art, around 19–20 years, but nonetheless are commonly cited by other American authors as the primary sources with regard to IL-6 signaling in depression. Indeed, most if not all papers published after our papers in 1995–1997 only report on increased plasma IL-6 and not on increased IL-6 trans-signaling. All that prior and newer knowledge is largely neglected by the American literature of the "last decade" thus prioritizing the wrong science, e.g. sickness behavior or short-lasting behavioral responses as a model of major depression and plasma IL-6 (without sIL-6R measurements) and CRP (but not the more important acute phase proteins) as biomarkers.

This has additionally detracted the research focus from the big immune-inflammatory-oxidative-nitrosative stress picture that has emerged in depression and has interfered with biomarker and new treatment development as well (Leonard & Maes 2012). New combinatorial therapies for depression with negative immunoregulatory, anti-inflammatory and anti-oxidative compounds are now developed. However, American authors now target inflammatory cytokines with drugs that have too many side effects to be used in clinical depression, e.g. infliximab. Some American authors now even propose infliximab as a new possible treatment in depression. However, based on the state-of-the-art (Leonard & Maes 2012) treatments should be developed that multi-target CMI activation, inflammation, O&NS, lowered antioxidant levels, mitochondrial functions, bacterial translocation, autoimmune responses and neuroprogression (Maes *et al.* 2012; 2013).

## CONCLUSIONS

Overall, referees and journal editors should improve their citation standards to guarantee the readership full access to all available information and should demand a signed pledge that correct citations were made (Garfield 1980; 2002; Ginsberg 2001). It is time that COPE publishes stringent criteria to halt this kind of citation omissions.

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