



OPEN

Messaging in Biological Psychiatry: Misrepresentations, Their Causes, and Potential Consequences

Estelle Dumas-Mallet, PhD, and Francois Gonon, PhD

Abstract: Most experts in the field of psychiatry recognize that neuroscience advances have yet to be translated into clinical practice. The main message delivered to laypeople, however, is that mental disorders are brain diseases cured by scientifically designed medications. Here we describe how this misleading message is generated. We summarize the academic studies describing how biomedical observations are often misrepresented in the scientific literature through various forms of data embellishment, publication biases favoring initial and positive studies, improper interpretations, and exaggerated conclusions. These misrepresentations also affect biological psychiatry and are spread through mass media documents. Exacerbated competition, hyperspecialization, and the need to obtain funding for research projects might drive scientists to misrepresent their findings. Moreover, journalists are unaware that initial studies, even when positive and promising, are inherently uncertain. Journalists preferentially cover them and almost never inform the public when those studies are disconfirmed by subsequent research. This explains why reductionist theories about mental health often persist in mass media even though the scientific claims that have been put forward to support them have long been contradicted. These misrepresentations affect the care of patients. Indeed, studies show that a neuro-essentialist conceptualization of mental disorders negatively affects several aspects of stigmatization, reduces the chances of patients' healing, and overshadows psychotherapeutic and social approaches that have been found effective in alleviating mental suffering. Public information about mental health should avoid these reporting biases and give equal consideration to the biological, psychological, and social aspects of mental health.

Keywords: data misrepresentation, journalism, media coverage, mental health policy, publication biases

On 31 October 2019, two psychiatrists—Caleb Gardner and Arthur Kleinman—published an opinion article in the *New England Journal of Medicine*.¹ They wrote:

Ironically, although these limitations [of “biologic treatments”] are widely recognized by experts in the field, the prevailing message to the public and the rest of medicine remains that the solution to psychological

problems involves matching the “right” diagnosis with the “right” medication. Consequently, psychiatric diagnoses and medications proliferate under the banner of scientific medicine, though there is no comprehensive biologic understanding of either the causes or the treatments of psychiatric disorders.

Like other leaders in the field, Gardner and Kleinman admit that the neurobiological analysis of psychiatric disorders has not yet been translated into improved clinical care.^{2,3} Moreover, they acknowledge, for the first time in a prestigious biomedical journal, that psychiatrists deliver another message to their non-psychiatric peers in medicine and to laypeople.

Gardner and Kleinman¹ suggest that this misleading messaging negatively affects patients, caregivers, and society at large. We believe, however, that most psychiatrists are not aware that they contribute to it or passively accept it. They do not intend to deceive patients and the public. Therefore, in the absence of convincing evidence supported by observational studies, they might miss Gardner and Kleinman's important message. Here we review the academic literature describing the misrepresentation of biological psychiatry, its sources, diffusion through mass media, and social consequences. Indeed, as

From the Institute of Neurodegenerative Diseases and CNRS UMR, University of Bordeaux.

Original manuscript received 5 May 2020; revised manuscript received 21 July 2020, accepted for publication 28 July 2020.

Supported, in part by, Agence National de la Recherche grant no. ANR-18-FRAL-0003-01 (Dr. Dumas-Mallet).

Correspondence: Francois Gonon, PhD, Institute of Neurodegenerative Diseases, University of Bordeaux, 146, rue Léo Saignat, 33076 Bordeaux, France. Email: francois.gonon@u-bordeaux.fr

Copyright © 2020 The Author(s). Published by Wolters Kluwer Health, Inc. on behalf of the President and Fellows of Harvard College. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

DOI: 10.1097/HRP.0000000000000276

documented in numerous academic studies, there is often a huge gap between the observations reported in biomedical publications and their representation in mass media. In the first part of our review, we describe the misrepresentations of the scientific observations that are already present in the biomedical literature and spread in the media, with a focus on psychiatry. In the second part we review academic works that have examined how mass media cover biomedical research, with examples related to psychiatry. The third section briefly describes the consequences of these misrepresentations. The last section discusses the possible reasons why journalists, scientists, and scientific institutions contribute to the misrepresentation of biological psychiatry.

To summarize our approach here, we do not aim to fuel the debates that split psychiatry and related disciplines involved in mental health—in particular, the relationships between social sciences, psychology, neuroscience, and biological psychiatry (see Kendler [2005],⁴ Miller [2010],⁵ and Rose & Abi-Rached [2013]⁶ for thorough discussions). The multi-causal etiology of mental disorders is gaining acceptance, although mono-causal theories are still influential in psychiatry.^{4,7} Therefore, the contribution of genetic and environmental factors to the etiology of mental disorders is discussed here insofar as the mono-causal thinking still predominates in mass media. More specifically, we do not intend to question biological psychiatry per se, but the message delivered to the public. Psychotropic medications do alleviate psychiatric symptoms. For example, psychostimulants effectively treat attention-deficit/hyperactivity disorder (ADHD). It is questionable, however, to promote this treatment by asserting that it corrects an underlying dopamine deficit, although this claim has been often put forward by mass media.^{8–10} In the same way, thousands of biological studies have effectively helped to improve our understanding of mental disorders, even though the outcome of this knowledge in terms of diagnostic and treatment is still modest. In particular, the value of negative findings is often underestimated, although they serve the goal of identifying and then discarding unfounded hypotheses, such as the serotonin deficit theory of depression, which has often been invoked in advertisements for antidepressants.¹¹

THE DISTORTIONS ALREADY PRESENT IN THE SCIENTIFIC LITERATURE

Scientific publications and their corresponding press releases represent the raw material used by journalists. If these publications already misrepresent or misinterpret scientific observations, these distortions are very likely to spread into the corresponding media documents.¹² These distortions therefore are a concern not only for the scientific community but also for the public.

Data Embellishments

Fraud consists in fabricating observations to validate a theory, but cases of blatant fraud are extremely rare.¹³ Several types of data embellishment are common in the biomedical

literature.¹⁴ Researchers' surveys have revealed that 2% of them admit to having falsified their data at least once. Moreover, 14% of the interviewees said they were aware of colleagues who had embellished their data.¹⁵ Based on a survey of 390 statisticians, Wang and colleagues¹⁶ drew up a list of questionable requests from biomedical scientists. The most frequent ones were: (1) the deletion or alteration of some data in order to obtain a significant effect, (2) the termination of data collection as soon as a significant effect is observed, and (3) the multiplication of statistical tests until obtaining a positive effect and its selective reporting. During the past five years, more than three-quarters of these biostatisticians have received at least one request for partial falsification.¹⁶ These practices, called *p-hacking*, are therefore common and explain why the *p* values just below the critical threshold of .05 are abnormally frequent in biomedical science.¹⁷

Preferential Publication of Positive Results

All disciplines combined, the percentage of scientific articles reporting results that confirmed researchers' hypotheses increased from 70% in 1990 to 86% in 2007.¹⁸ Considered together, psychiatry and psychology have the highest positive outcome rate of all scientific disciplines examined by Fanelli.¹⁹ The preferential publication of positive biomedical findings might result from two trends: either researchers choose not to submit their negative results for publication, or editors reject them more frequently.^{20–22} For example, Ioannidis examined 41 meta-analyses reporting an association between brain volume abnormalities and psychiatric disorders. He showed that the number of primary studies reporting a significant difference between patients and controls was twice as high as what should have been observed on the basis of the corresponding meta-analyses.²³

Clinical trials reporting the beneficial effect of a medication are more often published than those reporting no effect, and studies about psychotropic drugs are not immune to this publication bias.^{22,24,25} This bias has been revealed by analyzing the outcome data of the clinical trials that have been registered with the Food and Drug Administration (FDA) but not published in medical journals. For example, among a total of 74 randomized, controlled trials of antidepressants registered with the FDA, 37 of the 38 trials reporting a positive effect were published in peer-reviewed journals. By contrast, among the 36 trials judged as negative by the FDA, 22 had not been published, 11 were published but reported positive outcomes, and only 3 trials published results in agreement with the FDA's judgments.²⁶

Inaccurate Description of the Methods

In scientific publications, the methods must be described in sufficient detail to allow other researchers to replicate the observations and to provide readers with all the information needed to judge the quality and implications of the results. Unfortunately, the description of the methods is often vague or incomplete.^{14,16} For example, a study published in 1999

concluded that the brain level of the dopamine transporter is 70% higher in patients suffering from ADHD.²⁷ This article was widely covered by the lay press because it claimed to reveal the biological cause of ADHD and to demonstrate the merits of psychostimulant medication, which inhibits the dopamine transporter.²⁸ In their 1999 article, the authors failed to specify that four of their six patients had a previous history of psychostimulant therapy.^{27,29} Subsequent studies have shown that the brain level of the dopamine transporter is similar in controls and in untreated ADHD patients and that prolonged psychostimulant treatment increases this level.³⁰

Misrepresentation of the Data

Within individual scientific articles there is often a huge gap between the observations per se and their presentation or interpretation—in particular, in the summaries. Numerous studies have characterized and quantified these embellishments,^{14,31,32} called *spin* in the specialized literature, and psychiatry journals are not immune to them.³³ The most misleading form of spin consists of an obvious inconsistency between the observations described earlier in the article and the conclusions drawn at the end of the article or in the summary. For example, a study reported that treating children with ADHD with a psychostimulant does not improve their reading performance and does not decrease their risk of early school dropout.³⁴ On the sole basis of a slightly lower grade-repetition rate, however, the authors concluded that this treatment improves their academic performance in the long term. This conclusion spread in newspapers.¹² Indeed, on 21 September 2007, the *Washington Post* wrote: “This is the first study that shows that taking stimulants for ADHD improves long-term school performance.”

Less extreme forms of beautification are much more frequent in biomedical publications—in particular (again), in their summaries.³² A common form consists in highlighting a statistically significant effect without mentioning the figures that question its clinical significance.³¹ For example, 159 summaries asserted a statistically significant association between ADHD and the 7R allele of the gene coding for the dopamine D4 receptor, but only 25 summaries mentioned the size of this association,¹² which is actually weak: 23% of the children diagnosed with ADHD are carriers of this allele versus 17% of control children. The omission of this quantitative information in 84% of the summaries was found in the same proportion in newspaper articles.¹²

Many articles reporting a correlation between a pathology and a risk factor improperly suggest that it is a causal factor.^{32,35} When this improper interpretation also appears in the corresponding press release, it is likely to be found in the press articles covering the study.³⁵ For example, a brain-imaging study published in 2017 reported that some subcortical brain areas are smaller in patients with ADHD.³⁶ The largest difference between patients and controls was related to the volume of the amygdala, and it was much smaller (mean difference = 1.5%) than the natural variability within healthy controls

(SD = 9.4%). The authors concluded that “our results confirm that ADHD patients truly have altered brains, i.e. that ADHD is a disorder of the brain.” This conclusion, which also appears in a corresponding press release, implicitly suggests a causal link between these brain abnormalities and ADHD. In some newspapers this causal interpretation was more explicit. Indeed, on 16 February 2017, the *Daily Telegraph* (UK) headlined: “ADHD is result of brain disorder, not bad parenting.” Inside the article the journalist wrote: “The scientists behind the study say their findings prove for the first time that the condition has a physical cause.” Yet, structural changes in certain brain areas are not necessarily the cause of mental disorders, as the authors acknowledged in a comment on their study.³⁷ For example, the most robust difference between the brains of depressed patients and those of healthy controls relates to the volume of the hippocampus. A large, international study showed that this volume reduction is modest (1.2%) and appears only after years of depression.³⁸ It is not observed in patients diagnosed with a first depressive episode. Consequently, this minimal atrophy of the hippocampus might be the consequence of chronic depression rather than its cause.³⁸

MASS MEDIA ARE UNAWARE OF THE RESEARCH PROCESS

Initial Scientific Findings Are Uncertain

Because positive findings are preferentially published in biomedical journals, it follows that the first study on a new question most often reports a larger effect size than subsequent studies on the same issue.^{39,40} This devaluation of initial studies by subsequent studies is frequent and affects all domains of biomedical research.^{39–41} For example, among 43 initial studies asserting the effectiveness of a psychotropic medication, 16 of them were invalidated by subsequent studies and 11 others reported effect sizes much larger than those of subsequent studies.⁴² Regarding research associating a risk factor with a pathology, we conducted a large comparative study on 663 initial studies.⁴³ We observed that, on average, one in two initial studies was either contradicted or strongly attenuated by the corresponding meta-analysis. This replication rate was highly variable, however, depending on subdomains. For example, among 46 initial studies reporting a significant association between a genetic risk and a psychiatric disorder, only three (7%) were confirmed, whereas this percentage was larger (38%) regarding neurological diseases. The sample sizes of these initial genetic studies were smaller for psychiatry than neurology. Thus, most initial studies in psychiatry appeared underpowered to detect small genetic risks.⁴³

Mass Media Preferentially Cover Initial Studies

The low confirmation rate of initial studies is not shocking in itself: science is a cumulative process that evolves from promising but uncertain initial studies, toward a consensus based on a corpus of independent studies confirming, or not, the initial studies. Unfortunately, mass media do not take this

process into account. We observed, on a sample of 5029 association studies, that newspapers favored initial studies (13% were covered) over subsequent studies (2.4%) and meta-analyses (1.6%).⁴⁴ As a result, half of the studies covered by newspapers were actually disconfirmed by subsequent studies. The confirmation rate was lower for psychiatry (26%) than for neurology (63%).⁴⁴ In addition, newspapers hardly ever inform the public when the studies they have covered are contradicted. For example, among the top ten scientific studies about ADHD that attracted the largest media interest during the 1990s, seven were initial studies, and all of them were either fully disconfirmed or strongly attenuated by subsequent studies.²⁸ These seven initial studies were covered by 158 newspaper articles, whereas, among the 43 subsequent studies following up the initial ones, only 2 were echoed by the press (3 articles each). All but one failed to mention that the subsequent study disconfirmed a previous study.²⁸

Journalistic standards, which favor novelty and attractiveness, explain in part the journalists' preference for initial studies. Moreover, it is exacerbated by the higher visibility of initial studies compared to subsequent studies. For example, four of the seven highly covered initial studies mentioned above were published in prestigious journals (*Lancet*, *New England Journal of Medicine*, *Science*), whereas all but one of the 43 subsequent studies were published in journals with lower impact factors.²⁸

This example illustrates a general observation: newspapers strongly favor studies published by prestigious scientific journals,⁴⁴ even though the initial studies that they publish are as often disconfirmed by subsequent studies, as are the initial studies published by journals with lower impact factors.⁴³ Newspapers preferentially cover these initial studies because these prestigious journals also produce press releases highlighting the studies they publish.⁴⁵ Indeed, these press releases are the direct source of more than 80% of the press articles reporting biomedical findings.^{45,46} Moreover, most newspaper articles are very closely inspired by these press releases and take up their biases and exaggerations without criticism.^{31,35,47,48} Finally, newspapers further accentuate publication biases by almost exclusively covering studies reporting a positive effect.⁴⁴

Mass Media Rarely Inform the Public About the Uncertainty of Initial Studies

If the initial studies published in scientific journals are, as we have seen, uncertain, communications in conferences are even more so. Newspapers do not hesitate, however, to cover them: out of 734 front page articles covering biomedical findings from 2000 to 2002, 43% reported communications at conferences.⁴⁹ Of these, only half resulted in subsequent publication in peer-reviewed journals. Among newspaper articles covering these conference communications, less than one in five informed the reader of their preliminary and uncertain nature.⁴⁹ Similarly, among press articles covering initial studies published in peer-reviewed biomedical journals, only one

in five mentioned that these discoveries were preliminary and should be confirmed by subsequent studies.^{50,51}

THE CONSEQUENCES OF MISREPRESENTING BIOLOGICAL PSYCHIATRY

Implications for Attitudes and Beliefs About Mental Disorders

Misrepresenting biological psychiatry to the public bolsters the view that "mental disorders are increasingly conceptualized as biomedical diseases, explained as manifestations of genetic and neurobiological abnormalities."⁵² Indeed, the percentage of American people who are convinced that schizophrenia and depression are genetic brain diseases increased from 61% in 1996 to 71% in 2006.⁵³ Similar increases were also documented in Austria, Germany, and Scotland.⁵⁴ The effects of this biogenetic belief on public attitudes toward mental disorders have been reviewed.^{52,55-58} Although laypeople adhering to this belief tend to blame patients less for their symptoms, they perceive them as more dangerous and are more pessimistic about a possible recovery.^{52,55} For caregivers, this neuro-essentialist conceptualization dampens their empathy toward patients.⁵² Its effects on patients' self-blame and guilt are mixed, depending on the pathology.^{52,58} Patients adhering to this view are more pessimistic about recovery, however, and focus their expectations on psychotropic medications.^{52,58} Moreover, some studies⁵⁹⁻⁶¹ showed that patients with less endorsement of biogenetic beliefs about depression seem more likely to recover, although other studies reported no relationship.⁶² This specific point has yet to be discussed in a systematic review, however; it needs further investigation. Finally, expected stigmatization discourages patients from seeking help.^{56,57} Altogether, this neuro-essentialist conceptualization negatively affects several aspects of stigmatization, reduces the chances of patients' healing, and overshadows psychotherapeutic and social approaches that have been found effective in alleviating mental suffering.¹

Social Prevention Is Kept in the Background

Mental disorders run in families. Until recently, this indisputable fact has been mainly interpreted as evidence that mental disorders are genetic diseases. At first glance, twin and family studies had fueled this view by showing that mental disorders are highly heritable.⁶³ Actually, some rare genetic variants appear to be strongly associated with intellectual disability, autism, and schizophrenia, but they explain only a small percentage of cases.⁶³

Recent genetic and epidemiologic studies have softened the view that genetic defects play a major role in the etiology of mental disorders. First, extensive genome-wide association studies have demonstrated that common mental disorders are not the product of one or a few DNA sequence variants of high penetrance.^{3,64} Instead, the genetic risks for most psychiatric disorders result from the additive effect of numerous common variants of very small effect.^{3,63} Moreover, it has been

shown that genetic risk factors can, in some cases, be either detrimental or protective, depending on the environmental conditions.^{65,66} Indeed, genetic diversity is favored by the evolutionary forces that shape species; it increases species' abilities to cope with changing environmental conditions.⁶⁶

Second, although the heritability of mental disorders is high, the concordance between monozygotic twins is usually low (e.g., 30% for schizophrenia).⁶⁷ This suggests either that the genetic component of heritability is overestimated or that environmental conditions are required to trigger the expression of mental disorders.^{67,68}

Third, interactions between gene and environment have been thoroughly investigated since the highly popularized study by Caspi and colleagues,⁶⁹ who reported a genetic susceptibility to depression induced by stressful life events. Subsequent studies and meta-analyses have disconfirmed this genetic susceptibility and reaffirmed that social stress (e.g., job loss, previous history of child maltreatment) is strongly associated with depression.^{70,71} Although the genetic susceptibility hypothesis seems plausible in psychiatry, it is not yet supported by sound experimental evidence.^{72,73} "There are few (if any) clear examples of gene by environment interactions in psychiatry, and their scope for informing either our understanding of disease pathology or clinical practice remains limited at present."^{72(p 1092)}

Fourth, recent attempts to disentangle genetic from environmental components in the etiology of mental disorders have taken advantage of natural experiments and provided convincing evidence that psychosocial conditions play a major causal role, at least for some disorders, such as the familial transmission of major depression.⁷⁴

Altogether, recent genetic and epidemiologic studies confirm the importance of environmental factors in the etiology of common mental disorders.⁷⁵ This would represent a major advance for preventive psychiatry and a great hope for society because, unlike genetic risks, psychosocial risk factors are amenable to social policies. Unfortunately, when mass media cover studies investigating together the genetic and environmental factors conferring risks for common mental disorders, such as depression or ADHD, most of them—in particular, TV programs and websites—emphasize genetic risks.^{10,76,77} Many newspapers also elaborate on psychological factors, but social factors (e.g., socioeconomic status, children born to teenage mothers, preterm birth)⁹ are almost never mentioned by mass media.^{77–79} Some psychiatrists cited by mass media have a role in generating this biased coverage;¹⁰ when communicating with journalists about their findings, biomedical scientists tend to go beyond the implications stated in their publications.^{50,80,81}

Implications on Health and Research Policies

In democratic societies scientific arguments are often mobilized to legitimize political decisions. A qualitative survey among decision makers in the German political and administrative system showed that mass media have a substantial impact

on policy processes.⁸² Indeed, political and administrative institutions strongly invest in media observation. It allows policy makers to identify topical questions and provides them a repertoire of scientific arguments that will be used to legitimize decisions. Mass media also offer decision makers both feedback about their activities and a way to influence public opinion. A survey of Belgian, Canadian, and Israeli politicians showed "that a piece of information gets more attention from politicians when it comes via the media rather than an identical piece of information coming via a personal e-mail."^{83(p 153)} In the field of research policy, interviews with 35 German policymakers working in organizations from politics, science, and research funding showed that they increasingly adapt to media logics.⁸⁴ Some interviewees even suggested that "funding organizations select research projects according to their attractiveness to the mass media."^{84(p 725)} It is likely that these interactions between mass media and political decisions also apply to mental health policies and to the funding of psychiatric research, although this hypothesis remains, to our knowledge, to be investigated. If shown to be true, it would imply that these decisions might still be largely influenced by simplistic views about mental disorders conveyed by most mass media.

POSSIBLE CAUSES

Scientists Are Pushed to Publish Exciting Findings and to Embellish Them

Biomedical publications often state that: (1) previous studies on the same issue have not produced convincing results, (2) the results provide the first proof, and (3) the discovery is clinically and socially relevant. Although often excessive, this rhetoric could be seen as rational from the point of view of scientists and their institutions. Indeed, a scientist's career mainly depends on the number and quality of his or her publications. A study published in a prestigious journal ensures authors a lasting reputation and makes their grant applications more likely to be funded.⁸⁵ To gain publication in prestigious journals, researchers might benefit from exaggerating the interest of their work.¹² On their side, the editors of prestigious journals reject, before peer review, the vast majority of the manuscripts they receive. They select the most exciting results likely to interest a large audience.⁸⁶ In fact, mass media preferentially cover studies published by prestigious journals because they believe them to be the most reliable,⁸⁷ although many of them are disconfirmed by subsequent studies.^{39,43} Surveys of researchers and journalists have shown that the relationships between them are increasingly smooth and positive;⁸⁸ interviewed scientists expressed their feeling that the coverage of their research positively affects their careers and facilitates the funding of their research.^{88,89}

Scientific institutions have contributed to this rhetorical escalation. First, they favor researchers who publish in prestigious journals.⁹⁰ Second, they encourage researchers to communicate with journalists and the public.⁸⁸ Third, scientific institutions

have considerably strengthened their press services in recent years and are flooding national journalists with press releases.^{87,88} This promotional activity appears to be effective. Indeed, national newspapers preferentially cover biomedical publications whose authors are working in that country.⁹¹ Fourth, because research is mainly funded on project-based proposals, researchers are pushed to over-promise in their grant applications and, then, to embellish their results.^{90,92–94} Finally, in all biomedical domains, including biological psychiatry, the volume of the scientific literature published each year has sharply increased during the last two decades. Thus, research in psychiatry is more and more competitive, technically sophisticated, and conceptually complex. These changes exacerbate the specialization of research fields. Because most scientists believe that they must be highly specialized to survive the academic competition, they have less time to acquaint themselves with related disciplines and might become less sensitive to ethical concerns.

Journalists Unwittingly Exacerbate the Distortions Already Present in the Biomedical Literature

As described in the first section, the presentation of biomedical observations in scientific publications is often altered by different forms of distortion: partial falsification of the results, various data embellishments, publication biases favoring initial and positive studies, citation biases, improper interpretation, and exaggerated conclusions. Press releases, issued by prestigious biomedical journals and scientific institutions, often exacerbate these distortions.^{95,96} Because most newspaper articles are closely inspired by these press releases, journalists are not the main source of these distortions.⁴⁷ Nevertheless, journalists enhance their dissemination by preferentially covering initial studies and those reporting a positive effect. Consequently, the journalistic ideal of independent and objective investigation of the facts seems to apply poorly to the media coverage of biomedical findings.⁸⁷

Two surveys of scientific journalists highlight this situation. In the first, the investigators asked journalists what items they deemed essential when covering a disease-related genetic discovery.⁹⁷ Among these items, the replication issue was often mentioned. Indeed, 79% of the interviewees considered that the press coverage of a genetic study must mention its replication status: initial study to be confirmed or subsequent study confirming or not the previous studies.⁹⁷ Facts do not necessarily match these good intentions; replication status is rarely mentioned in newspaper articles.^{50,51} In the second survey, most of the journalists also said that the replication validity is an important issue.⁸⁷ They expressed their total confidence, however, regarding the robustness of the data published in prestigious scientific journals.^{87,98} When asked about the replication validity of biomedical findings, most journalists erroneously ascribed the lack of validity to blatant fraud. Even those with a strong scientific background seemed to ignore the uncertainty inherent to initial observations.⁸⁷ The few others, who were aware of the evolving research

process, acknowledged that they encountered difficulties in accurately reporting on scientific uncertainty.⁸⁷ Indeed, compared to media immediacy, science is advancing slowly, and journalists have no time to follow up—for years, if necessary—on the initial studies they had covered.⁸⁷

All these observations explain why mass media almost never inform the public when a study that they have covered is disconfirmed by subsequent studies—which often happens, including for psychiatry.^{28,44} For example, the genetic susceptibility to depression reported by Caspi and colleagues⁶⁹ was covered by 50 newspapers articles during the week following its publication, whereas subsequent studies that disconfirmed it received no media attention. Only four newspapers covered the meta-analysis published in 2009⁷⁰ and informed the public that this interaction was disconfirmed.⁴⁴ Moreover, even elite newspapers continued citing Caspi and colleagues' conclusion years after its invalidation. For example, on 30 November 2014, the *New York Times* wrote: “What distinguishes children who prove more versus less susceptible to developmental experiences? There is no single factor, but genetics seems to play a role. For instance, short alleles of the gene 5-HTTLPR, which transports serotonin, have been linked to depression.”

Why Does the Neuro-Essentialist Discourse Gain Ground in Democratic Societies?

The neuro-essentialist discourse pushes aside a more integrated conceptualization of psychiatric disorders (often referred to as the biopsychosocial model), although neuroscience advances have not yet contributed to improve the diagnosis and treatment of mental disorders.^{1–3} Therefore, one might wonder why this reductionist discourse is so successful in democratic societies, especially since we now have strong evidence that psychosocial risk factors play an important role in the etiology of common mental disorders. Most of these factors are, in fact, linked to the relative economic level of the subjects: the greater the inequalities in a territory, the higher the prevalence of mental disorders in the most disadvantaged population.⁹⁹ Some biological correlates of this relationship have been described and further reinforce the plausibility of a causal relationship between relative poverty and mental disorders.^{99,100} The neuro-essentialist discourse pushes aside this relationship by suggesting that academic and social failures, which are actually more frequent in disadvantaged children, result from their intrinsic neurodevelopmental dysfunctions of genetic origin. Therefore, this discourse could mask the contradiction between the democratic ideal of equal opportunity at birth and the fact that disadvantaged children are even less likely than before to climb the social ladder.¹⁰¹

CONCLUSION

In 2014, Allen Frances advocated for “resuscitating the biopsychosocial model.”² He pointed out that psychotropic medications are still useful for alleviating severe psychiatric symptoms, although they are not silver bullets. Moreover,

certain biological risk factors do contribute to the etiology of specific mental disorders, such as autism.¹⁰² Research efforts regarding biological psychiatry must therefore go on. In the present state of the scientific knowledge, however, the psychosocial understanding of mental disorders is at least as important as the biological one to guide mental health professionals.^{1,2} Public information about mental health should reflect this view.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the article.

The authors thank Thomas Boraud for his support and advice.

REFERENCES

- Gardner C, Kleinman A. Medicine and the mind—the consequences of psychiatry’s identity crisis. *N Engl J Med* 2019;381:1697–9.
- Frances A. Resuscitating the biopsychosocial model. *Lancet Psychiatry* 2014;1:496–7.
- Hyman SE. The daunting polygenicity of mental illness: making a new map. *Philos Trans R Soc Lond B Biol Sci* 2018;373.
- Kendler KS. Toward a philosophical structure for psychiatry. *Am J Psychiatry* 2005;162:433–40.
- Miller GA. Mistreating psychology in the decades of the brain. *Perspect Psychol Sci* 2010;5:716–43.
- Rose N, Abi-Rached J. *Neuro: the new brain sciences and the management of the mind*. Princeton, NJ: Princeton University Press, 2013.
- Kendler KS. From many to one to many—the search for causes of psychiatric illness. *JAMA Psychiatry* 2019 Jun 19 [Epub ahead of print].
- Gonon F. The dopaminergic hypothesis of attention-deficit/hyperactivity disorder needs re-examining. *Trends Neurosci* 2009;32:2–8.
- Gonon F, Bezard E, Boraud T. What should be said to the lay public regarding ADHD etiology. *Am J Med Genet B Neuropsychiatr Genet* 2011;156:989–91.
- Bourdaa M, Konsman JP, Secail C, Venturini T, Veyrat-Masson I, Gonon F. Does television reflect the evolution of scientific knowledge? The case of attention deficit hyperactivity disorder coverage on French TV. *Public Underst Sci* 2015;24:200–9.
- Healy D. Serotonin and depression. *BMJ* 2015;350:h1771.
- Gonon F, Bézard E, Boraud T. Misrepresentation of neuroscience data might give rise to misleading conclusions in the media: the case of attention deficit hyperactivity disorder. *PLoS One* 2011;6:e14618.
- Fang FC, Steen RG, Casadevall A. Misconduct accounts for the majority of retracted scientific publications. *Proc Natl Acad Sci U S A* 2012;109:17028–33.
- Boutron I, Ravau P. Misrepresentation and distortion of research in biomedical literature. *Proc Natl Acad Sci U S A* 2018;115:2613–9.
- Fanelli D. How many scientists fabricate and falsify research? A systematic review and meta-analysis of survey data. *PLoS One* 2009;4:e5738.
- Wang MQ, Yan AF, Katz RV. Researcher requests for inappropriate analysis and reporting: a U.S. survey of consulting biostatisticians. *Ann Intern Med* 2018;169:554–8.
- Head ML, Holman L, Lanfear R, Kahn AT, Jennions MD. The extent and consequences of p-hacking in science. *PLoS Biol* 2015;13:e1002106.
- Fanelli D. Negative results are disappearing from most disciplines and countries. *Scientometrics* 2012;90:891–904.
- Fanelli D. “Positive” results increase down the hierarchy of the sciences. *PLoS One* 2010;5:e10068.
- Emerson GB, Warme WJ, Wolf FM, Heckman JD, Brand RA, Leopold SS. Testing for the presence of positive-outcome bias in peer review: a randomized controlled trial. *Arch Intern Med* 2010;170:1934–9.
- Dwan K, Gamble C, Williamson PR, Kirkham JJ. Systematic review of the empirical evidence of study publication bias and outcome reporting bias—an updated review. *PLoS One* 2013;8:e66844.
- Turner EH. Publication bias, with a focus on psychiatry: causes and solutions. *CNS Drugs* 2013;27:457–68.
- Ioannidis JP. Excess significance bias in the literature on brain volume abnormalities. *Arch Gen Psychiatry* 2011;68:773–80.
- Rising K, Bacchetti P, Bero L. Reporting bias in drug trials submitted to the Food and Drug Administration: review of publication and presentation. *PLoS Med* 2008;5:e217.
- McGauran N, Wieseler B, Kreis J, Schüler YB, Kölsch H, Kaiser T. Reporting bias in medical research—a narrative review. *Trials* 2010;11:37.
- Turner EH, Matthews AM, Linardatos E, Tell RA, Rosenthal R. Selective publication of antidepressant trials and its influence on apparent efficacy. *N Engl J Med* 2008;358:252–60.
- Dougherty DD, Bonab AA, Spencer TJ, Rauch SL, Madras BK, Fischman AJ. Dopamine transporter density in patients with attention deficit hyperactivity disorder. *Lancet* 1999;354:2132–3.
- Gonon F, Konsman JP, Cohen D, Boraud T. Why most biomedical findings echoed by newspapers turn out to be false: the case of attention deficit hyperactivity disorder. *PLoS One* 2012;7:e44275.
- Spencer TJ, Biederman J, Madras BK, et al. In vivo neuroreceptor imaging in attention-deficit/hyperactivity disorder: a focus on the dopamine transporter. *Biol Psychiatry* 2005;57:1293–300.
- Fusar-Poli P, Rubia K, Rossi G, Sartori G, Balottin U. Striatal dopamine transporter alterations in ADHD: pathophysiology or adaptation to psychostimulants? A meta-analysis. *Am J Psychiatry* 2012;169:264–72.
- Yavchitz A, Boutron I, Bafeta A, et al. Misrepresentation of randomized controlled trials in press releases and news coverage: a cohort study. *PLoS Med* 2012;9:e1001308.
- Chiu K, Grundy Q, Bero L. ‘Spin’ in published biomedical literature: a methodological systematic review. *PLoS Biol* 2017;15:e2002173.
- Jellison SA-O, Roberts W, Bowers A, et al. Evaluation of spin in abstracts of papers in psychiatry and psychology journals. *BMJ Evid Based Med* 2019 Aug 5 [Epub ahead of print].
- Barbarese WJ, Katusic SK, Colligan RC, Weaver AL, Jacobsen SJ. Long-term school outcomes for children with attention-deficit/hyperactivity disorder: a population-based perspective. *J Dev Behav Pediatr* 2007;28:265–73.
- Sumner P, Vivian-Griffiths S, Boivin J, et al. Exaggerations and caveats in press releases and health-related science news. *PLoS One* 2016;11:e0168217.
- Hoogman M, Bralten J, Hibar DP, et al. Subcortical brain volume differences in participants with attention deficit hyperactivity disorder in children and adults: a cross-sectional mega-analysis. *Lancet Psychiatry* 2017;4:310–9.
- Hoogman M, Buitelaar JK, Faraone SV, Shaw P, Franke B. Subcortical brain volume differences in participants with attention deficit hyperactivity disorder in children and adults—authors’ reply. *Lancet Psychiatry* 2017;4:440–1.

38. Schmaal L, Veltman DJ, van Erp TG, et al. Subcortical brain alterations in major depressive disorder: findings from the ENIGMA major depressive disorder working group. *Mol Psychiatry* 2016;21:806–12.
39. Ioannidis JP. Why most published research findings are false. *PLoS Med* 2005;2:e124.
40. Ioannidis JP. Contradicted and initially stronger effects in highly cited clinical research. *JAMA* 2005;294:218–28.
41. Ioannidis JP, Ntzani EE, Trikalinos TA, Contopoulos-Ioannidis DG. Replication validity of genetic association studies. *Nat Genet* 2001;29:306–9.
42. Tajika A, Ogawa Y, Takeshima N, Hayasaka Y, Furukawa TA. Replication and contradiction of highly cited research papers in psychiatry: 10-year follow-up. *Br J Psychiatry* 2015;207:357–62.
43. Dumas-Mallet E, Button K, Boraud T, Munafo M, Gonon F. Replication validity of initial association studies: a comparison between psychiatry, neurology and four somatic diseases. *PLoS One* 2016;11:e0158064.
44. Dumas-Mallet E, Smith A, Boraud T, Gonon F. Poor replication validity of biomedical association studies reported by newspapers. *PLoS One* 2017;12:e0172650.
45. Stryker JE. Reporting medical information: effects of press releases and newsworthiness on medical journal articles' visibility in the news media. *Prev Med* 2002;35:519–30.
46. De Semir V, Ribas C, Revuelta G. Press releases of science journal articles and subsequent newspaper stories on the same topic. *JAMA* 1998;280:294–5.
47. Schwartz LM, Woloshin S, Andrews A, Stukel TA. Influence of medical journal press releases on the quality of associated newspaper coverage: retrospective cohort study. *BMJ* 2012;344:d8164.
48. Sumner P, Vivian-Griffiths S, Boivin J, et al. The association between exaggeration in health related science news and academic press releases: retrospective observational study. *BMJ* 2014;349:g7015.
49. Lai WY, Lane T. Characteristics of medical research news reported on front pages of newspapers. *PLoS One* 2009;4:e6103.
50. Holtzman NA, Bernhardt BA, Mountcastle-Shah E, Rodgers JE, Tambor E, Geller G. The quality of media reports on discoveries related to human genetic diseases. *Community Genet* 2005;8:133–44.
51. Dumas-Mallet E, Smith A, Boraud T, Gonon F. Scientific uncertainty in the press: how newspapers describe initial biomedical findings. *Sci Commun* 2018;40:124–41.
52. Lebowitz MS, Appelbaum PS. Biomedical explanations of psychopathology and their implications for attitudes and beliefs about mental disorders. *Annu Rev Clin Psychol* 2019;15:555–77.
53. Pescosolido BA, Martin JK, Long JS, Medina TR, Phelan JC, Link BG. “A disease like any other”? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *Am J Psychiatry* 2010;167:1321–30.
54. Schomerus G, Schwahn C, Holzinger A, et al. Evolution of public attitudes about mental illness: a systematic review and meta-analysis. *Acta Psychiatr Scand* 2012;125:440–52.
55. Kvaale EP, Gottdiener WH, Haslam N. Biogenetic explanations and stigma: a meta-analytic review of associations among laypeople. *Soc Sci Med* 2013;96:95–103.
56. Clement S, Schauman O, Graham T, et al. What is the impact of mental health-related stigma on help-seeking? A systematic review of quantitative and qualitative studies. *Psychol Med* 2015;45:11–27.
57. Angermeyer MC, van der Auwera S, Carta MG, Schomerus G. Public attitudes towards psychiatry and psychiatric treatment at the beginning of the 21st century: a systematic review and meta-analysis of population surveys. *World Psychiatry* 2017;16:50–61.
58. Larkings JS, Brown PM. Do biogenetic causal beliefs reduce mental illness stigma in people with mental illness and in mental health professionals? A systematic review. *Int J Ment Health Nurs* 2018;27:928–41.
59. Sullivan MD, Katon WJ, Russo JE, et al. Patient beliefs predict response to paroxetine among primary care patients with dysthymia and minor depression. *J Am Board Fam Pract* 2003;16:22–31.
60. Bann CM, Parker CB, Bradwejn J, Davidson JR, Vitiello B, Gadde KM. Assessing patient beliefs in a clinical trial of hypericum perforatum in major depression. *Depress Anxiety* 2004;20:114–22.
61. Lynch J, Moore M, Moss-Morris R, Kendrick T. Do patients' illness beliefs predict depression measures at six months in primary care; a longitudinal study. *J Affect Disord* 2015;174:665–71.
62. Manber R, Chambers AS, Hitt SK, McGahuey C, Delgado P, Allen JJ. Patients' perception of their depressive illness. *J Psychiatr Res* 2003;37:335–43.
63. Nurnberger JI Jr, Austin J, Berrettini WH, et al. What should a psychiatrist know about genetics? Review and recommendations from the Residency Education Committee of the International Society of Psychiatric Genetics. *J Clin Psychiatry* 2019;80:17nr12046.
64. Kendler KS. “A gene for . . .”: the nature of gene action in psychiatric disorders. *Am J Psychiatry* 2005;162:1243–52.
65. Manuck SB, McCaffery JM. Gene-environment interaction. *Annu Rev Psychol* 2014;65:41–70.
66. Holmes AJ, Patrick LM. The myth of optimality in clinical neuroscience. *Trends Cogn Sci* 2018;22:241–57.
67. Hilker R, Helenius D, Fagerlund B, et al. Heritability of schizophrenia and schizophrenia spectrum based on the nationwide Danish Twin Register. *Biol Psychiatry* 2018;83:492–8.
68. Gottesman II, Bertelsen A. Confirming unexpressed genotypes for schizophrenia. Risks in the offspring of Fischer's Danish identical and fraternal discordant twins. *Arch Gen Psychiatry* 1989;46:867–72.
69. Caspi A, Sugden K, Moffitt TE, et al. Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science* 2003;301:386–9.
70. Risch N, Herrell R, Lehner T, et al. Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: a meta-analysis. *JAMA* 2009;301:2462–71.
71. Culverhouse RC, Saccone NL, Horton AC, et al. Collaborative meta-analysis finds no evidence of a strong interaction between stress and 5-HTTLPR genotype contributing to the development of depression. *Mol Psychiatry* 2017;23:133.
72. Munafo MR, Zammit S, Flint J. Practitioner review: a critical perspective on gene-environment interaction models—what impact should they have on clinical perceptions and practice? *J Child Psychol Psychiatry* 2014;55:1092–101.
73. Border R, Johnson EC, Evans LM, et al. No support for historical candidate gene or candidate gene-by-interaction hypotheses for major depression across multiple large samples. *Am J Psychiatry* 2019;176:376–87.
74. Thapar A, Rutter M. Do natural experiments have an important future in the study of mental disorders? *Psychol Med* 2019;49:1079–88.
75. Sonuga-Barke EJ. ‘It's the environment stupid!’ On epigenetics, programming and plasticity in child mental health. *J Child Psychol Psychiatry* 2010;51:113–5.
76. Horwitz AV. Media portrayals and health inequalities: a case study of characterizations of gene × environment interactions. *J Gerontol B Psychol Sci Soc Sci* 2005;60:48–52.

77. Mitchell J, Read J. Attention-deficit hyperactivity disorder, drug companies and the internet. *Clin Child Psychol Psychiatry* 2012;17:121–39.
78. Horton-Salway M. Repertoires of ADHD in UK newspaper media. *Health (London)* 2011;15:533–49.
79. Ponnou S, Gonon F. How French media have portrayed ADHD to the lay public and to social workers. *Int J Qual Stud Health Well-being* 2017;12:1298244.
80. Ransohoff DF, Ransohoff RM. Sensationalism in the media: when scientists and journalists may be complicit collaborators. *Eff Clin Pract* 2001;4:185–8.
81. Brechman JM, Lee CJ, Cappella J. Distorting genetic research about cancer: from bench science to press release to published news. *J Commun* 2011;61:496–513.
82. Petersen I, Heinrichs H, Peters HP. Mass-mediated expertise as informal policy advice. *Sci Technol Hum Values* 2010;35:865–87.
83. Sevenans J. How mass media attract political elites' attention. *Eur J Polit Res* 2018;57:153–70.
84. Scheu AM, Volpers A-M, Summ A, Blöbaum B. Medialization of research policy: anticipation of and adaptation to journalistic logic. *Sci Commun* 2014;36:706–34.
85. Reich ES. Science publishing: the golden club. *Nature* 2013;502:291–3.
86. Chew M, Villanueva EV, Van Der Weyden MB. Life and times of the impact factor: retrospective analysis of trends for seven medical journals (1994–2005) and their editors' views. *J R Soc Med* 2007;100:142–50.
87. Dumas-Mallet E. Regard des journalistes scientifiques sur l'actualité de la recherche biomédicale. *Hermes* 2019;83:227–33.
88. Peters HP. Gap between science and media revisited: scientists as public communicators. *Proc Natl Acad Sci U S A* 2013;110 suppl 3:14102–9.
89. Peters HP. Scientific sources and the mass media: forms and consequences of medialization. In: Rödder S, Franzen M, Weingart P, eds. *The sciences' media connection—public communication and its repercussions*. Dordrecht, Netherlands: Springer, 2012:217–39.
90. Edwards MA, Roy S. Academic research in the 21st century: maintaining scientific integrity in a climate of perverse incentives and hypercompetition. *Environ Eng Sci* 2017;34:51–61.
91. Dumas-Mallet E, Tajika A, Smith A, Boraud T, Furukawa TA, Gonon F. Do newspapers preferentially cover biomedical studies involving national scientists? *Public Underst Sci* 2019;28:191–200.
92. Ioannidis JP. Research needs grants, funding and money—missing something? *Eur J Clin Invest* 2012;42:349–51.
93. Ioannidis JP, Greenland S, Hlatky MA, et al. Increasing value and reducing waste in research design, conduct, and analysis. *Lancet* 2014;383:166–75.
94. Baker M. 1,500 scientists lift the lid on reproducibility. *Nature* 2016;533:452–4.
95. Woloshin S, Schwartz LM. Press releases: translating research into news. *JAMA* 2002;287:2856–8.
96. Woloshin S, Schwartz LM, Casella SL, Kennedy AT, Larson RJ. Press releases by academic medical centers: not so academic? *Ann Intern Med* 2009;150:613–8.
97. Mountcastle-Shah E, Tambor E, Bernhardt BA, et al. Assessing mass media reporting of disease-related genetic discoveries: development of an instrument and initial findings. *Sci Commun* 2003;24:458–78.
98. Hansen A. Journalistic practices and science reporting in the British press. *Public Underst Sci* 1994;3:111–34.
99. Pickett KE, Wilkinson RG. Income inequality and health: a causal review. *Soc Sci Med* 2015;128:316–26.
100. Hackman DA, Farah MJ, Meaney MJ. Socioeconomic status and the brain: mechanistic insights from human and animal research. *Nat Rev Neurosci* 2010;11:651–9.
101. Gonon F. La psychiatrie biologique: une bulle spéculative? *Esprit* 2011;Novembre:54–73. Available in English translation at <https://www.cairn-int.info/revue-esprit-2011-11-page-54.htm>
102. Christensen J, Gronborg TK, Sorensen MJ, et al. Prenatal valproate exposure and risk of autism spectrum disorders and childhood autism. *JAMA* 2013;309:1696–703.