of those professionally concerned in the enterprise of medical research.

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1 Smith R. Hype from journalists and scientists. *BMJ* 1992;304:730 (21 March.)

SIR,—Rarely do the medical journals mention the work of lay medical correspondents in the national press; rarer still do they devote an editorial to press stories.¹ It was therefore surprising that Richard Smith was unable to point to any inaccuracy in my stories on AIDS and the gene for asthma published in the *Sunday Times*.² Instead, his attack relied on accusations of “appalling hype,” which he failed to substantiate. In fact, both stories were followed up prominently by virtually every serious newspaper and television company; clearly, the vast majority of my journalist colleagues disagreed with Smith’s judgment.

Smith’s real objection seems to be that research papers—the *BMJ*’s future exclusives—have received a premature airing in the full glare of national newspaper coverage. Though I do not doubt the necessity of peer review, I am sure that the *BMJ* does not wish to put the professional’s right to publish above the public’s right to know.

The research on asthma showed a cumulative lod score—the scientific measurement of likelihood—of 10¹⁴, a figure of many millions to one against the gene for asthma not having been identified.³ When scientists of the eminence of Sir Walter Bodmer and Dr Bridget Ogilvie tell me that such important research is a medical milestone they are not contradicting the more cautious doctors leading the research team; they are simply using their experience to give the wider perspective demanded by my readers. How then can Smith say they are doing nothing for the public understanding of science?

The multimillion pound research programmes into combating AIDS and mapping the human genome depend more than most on public and charitable funding; the paying public, who in the main are not the *BMJ*’s readers but mine, deserve to be kept up to date. To do otherwise would be censorship, wouldn’t it?

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1 Smith R. Hype from journalists and scientists. *BMJ* 1992;304:730. (21 March.)

2 Ballantyne A. British breakthrough likely to end asthma suffering. *Sunday Times* 1992 March 8:1-3.

3 Ballantyne A. AIDS death rate halved by new drug. *Sunday Times* 1991 December 29:1-3.

4 Shirakawa T, Morimoto K, Hashimoto T, Furuyama J, Yamamoto M, Takai F. Linkage between IgE responses underlying asthma and rhinitis (atopy) and chromosome 11q in Japanese families. *Cytogenet Cell Genet* 1991;58:1970-1.

5 Young RP, Sharp PA, Lynch JR, Faux JA, Lathrop GM, Cookson WO, Hopkin JM. Confirmation of linkage between atopic IgE responsiveness and chromosome 11q in 64 nuclear families. *Cytogenet Cell Genet* 1991;58:1974-5.

SIR,—Richard Smith’s editorial expresses concern that the publication in newspapers of scientific advances related to disease may raise false hopes in patients suffering from the diseases concerned.¹ The editorial was provoked by reports of the work of Dr Julian Hopkin, who is not only a scientist but also a consultant physician responsible for patients suffering from asthma and related conditions. Dr Hopkin believes that he and his team have shown that asthma seems to develop mainly because of a change in a particular gene on one particular chromosome. He has stated publicly that the identification of this gene offers hope for much improved prevention and treatment in the future when new drugs can be developed. In speaking to journalists I took the lead from Dr Hopkin.

I believe it is essential that scientific work should be aired in the press and so made available to a wide audience, not just disseminated by scientists to their peers who read specialist journals. Therefore I am happy to discuss with journalists the scientific results of work funded by the Wellcome Trust, knowing that they are likely to quote a small fragment of what is said in a long interview. We know that patients included in Dr Hopkin’s studies are delighted that the work has received this publicity. They, and I, are confident that the study of genetic disease, which is the concern of so many outstanding biomedical scientists today, will eventually lead to better treatments for diseases such as asthma, but none of us believes that we can confidently state when knowledge of the genetic basis of any disease will have this happy outcome.

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1 Smith R. Hype from journalists and scientists. *BMJ* 1992;304:730. (21 March.)

SIR,—Richard Smith is right to accuse some journalists and scientists of hyperbole.¹ It is not enough, however, to blame it on an “unholy alliance” of individual sinners from the professions of journalism and medicine.

At the root of the problem is the need for medical scientists to put across their findings to a public that is both ill informed about health and hungry for knowledge about it. In theory, with highly sophisticated techniques in science and journalism, this should be easy. But in practice it is increasingly bedevilled by mutual suspicion and stereotyping.

The solution is not, as Smith seems to suggest, that scientists should be more careful about what they say to the press; in reality this will mean that they will increasingly say nothing. Instead, attention should focus on more fundamental issues, such as understanding the structural reasons why this type of communication has failed and improving the standards of health education generally. Far from adopting a rule of silence, doctors should actively seek to understand the communication process and to improve their mass communications skills, which, alas, are often sadly lacking. The alternative, it seems, would be the re-establishment of a sorcerer élite, which is in nobody’s interest.

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1 Smith R. Hype from journalists and scientists. *BMJ* 1992;304:730. (21 March.)

SIR,—Richard Smith’s trenchant editorial on the media’s hype of the discovery of the gene for asthma¹ is particularly relevant because the science behind the finding, which is not new, is highly controversial.

The article in the *Sunday Times* not only failed to distinguish between atopy and asthma or to mention that adult asthma is usually not allergic. It also failed to mention that other workers have tried to replicate this work without success. The article appeared when virtually every British expert on allergy was at the annual conference of the American Academy of Allergy and Immunology in Orlando, Florida. It was picked up by other news media and beamed around the world, even making breakfast television in Orlando. “On the Monday morning,” said one conference delegate, “we woke up to hear of the ‘breakthrough’ on CBS news. Then we went into the conference and heard a paper that said the exact opposite.”² The paper was given by Professor Malcolm Blumenthal of the University of Minnesota, who said, “Our group is familiar with Hopkin and Cookson’s work, which

was published in the *Lancet* in 1988³ and 1989,⁴ saying that atopy was associated with the 11q chromosome. We duplicated—or at least tried to duplicate—their studies using the same probe and could not find any evidence for this linkage. We all feel there is a genetic basis for asthma, so finding the gene is of major importance. My prejudice is that it’s caused by more than one gene. However, the type of genetics involved and where the genes are, nobody has a good idea.”

Among Professor Blumenthal’s audience was Tak Lee, the National Asthma Campaign professor of allergy at Guy’s Hospital. He too has been unable to replicate Cookson and Hopkin’s work, and his group’s findings were in print for all to see before the report in the *Sunday Times*.⁵ He said, “In 1988 and 1989 the Oxford group made what at the time was a very important discovery of an autosomal dominant gene for allergy. This was interesting because genetic studies have been under way for years. Our group repeated their work because we wanted to see if the site of chromosome 11 also has an association with bronchial asthma. We had no reason to doubt that their work was correct. We did our work very carefully, used the same reagents and the same probe and a similar number of families, and we got a completely negative answer. It’s inexplicable: it’s not only that many of us can’t find the association, many of us can’t even find the band they are reporting.”

There are at least three other studies in press or being written up that also fail to confirm Cookson and Hopkin’s two papers. The only one that does confirm it, a Japanese study, found the association in only 10% of patients studied. As one expert said, “Allergy’s a common problem. If allergy is a major gene you shouldn’t need to exclude 90% of your subjects. Until the Oxford work is confirmed by more than one centre it’s meaningless.”

The hype angered American workers by claiming that the Oxford group’s work was done on far less money than comparable American research teams have received.

The scientists themselves have kept fairly quiet, being understandably reluctant to engage in what might be misconstrued as a slanging match. As a science journalist I have been alarmed at the way the hype over allergy invaded professional science publications and I am scandalised by the reluctance of science news editors to consider publishing a follow up article pointing out that the science itself is controversial. I am therefore glad to be able to report this controversy in the *BMJ*, albeit only in the correspondence columns. Otherwise the hype would succeed in lowering the already dismal public understanding of science.

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1 Smith R. Hype from journalists and scientists. *BMJ* 1992;304:730. (21 March.)

2 Roitman-Johnson B, Rich S, Greenberg B, Moberg D, Roberts S, Blumenthal M. Genetics of atopy [Abstract 492]. *J Allergy Clin Immunol* 1992;89:267.

3 Cookson W, Hopkin J. Dominant inheritance of atopic immunoglobulin-E responsiveness. *Lancet* 1988;i:86-8.

4 Cookson W, Sharp PA, Faux JA, Hopkin J. Linkage between immunoglobulin-E responses underlying asthma and rhinitis and chromosome 11q. *Lancet* 1989;i:1292-5.

5 Lympny P, Welsh K, MacCochrane G, Kemeny DM, Lee TH. Genetic analysis, using DNA polymorphism, of the linkage between chromosome 11q13 and atopy and bronchial hyper-responsiveness to methacholine. *J Allergy Clin Immunol* 1992;89:619-28.

Advice to authors

Priority will be given to letters that are less than 400 words long and are typed with double spacing. All authors should sign the letter. Please enclose a stamped addressed envelope for acknowledgment.