

Editorial

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The acronym BLAME could potentially describe the phenomenon of biology-linked anxiety-mediated evasion, whereby particular food products evade consumption due to consumer perception of adverse outcomes of one sort or another. It doesn't, by the way, I made this one up!¹ In a general sense, the desire to blame someone or something for everything that goes wrong appears to be a very common human trait, encouraged by the legal profession (litigation culture), media, marketers and politicians. In the specific case of dairy foods, the examples that spring to mind include the supposed links between cardiovascular disease and dairy fats, mastitis antibiotic therapy and antimicrobial resistance, and belched methane and environmental catastrophe. And of course, lactose intolerance, for which dairy must inevitably accept blame. At this point I need to digress slightly. According to Heine *et al.* (2017) there are 'common misconceptions' between lactose intolerance and cow's milk allergy, regrettably not restricted to the lay public. I have personal experience of a family member who was breastfeeding being advised by a paediatrician that she should herself avoid dairy products because her baby was prone to digestive upsets, perhaps indicating a lactose intolerance. It is extremely worrying to me that someone who has studied medicine should have such a rudimentary knowledge of the processes of lactose synthesis exclusively in the mammary cell and lactose hydrolysis in the digestive tract. That worry extends to blaming myself and the rest of the lactation research community for failing to get important messages into the academic and general public domains. So, back to my examples. How have we, as a scientific community, responded to each of them, and did we get it right? By which I mean, right for ourselves as scientists, but more importantly right for consumers, production animals and the environment. The diet-heart hypothesis linking saturated fats to cardiovascular disease arose in the 1950s and, despite almost certainly being wrong, persists into much official nutritional advice to this day (Teicholz, 2023). Dairy fats figured prominently in the 'foods to avoid list' and prominent cardiologists and cardiovascular surgeons were perfectly happy to denounce dairy products without any evidence to support their claims. A major collaboration between Ian Givens (Reading, UK) and Arne Astrup (Copenhagen, Denmark) was at the forefront of new research and re-examination of old that effectively debunked the claims (Thorning *et al.*, 2016), and there are now very many review articles showing clearly that dairy foods are, if anything, protective. It is not my intention to examine the scientific arguments in detail, but I do draw the reader's attention to the existence of so many reviews (more than 20, according to Teicholz). Might this be overkill? I have long-since lost count of the number of articles submitted to the Journal that include comprehensive analysis of milk fatty acid profiles (more than 1500, it turns out!), the majority driven by the prospect of producing 'healthier milk'. To my knowledge, no such product exists, not because the fat cannot be manipulated (I cite the existence of a 'soft butter' produced by Irish dairy farmers for the UK market) but because, as we see now, it is most unlikely that a manipulated product would actually be healthier. The important question, 'are dairy products harmful to consumers' has been answered in the negative (although the jury is still out to some extent where butter is concerned). Will this stop the research from being done? As an Editor, I see no evidence of that. Whilst the prospect exists, funders can be persuaded to fund, cows can be persuaded to eat modified diets and Editors can be persuaded to publish. In other words, the blame erroneously attached to dairy foods has been turned to advantage by the scientific community. The same is true of antimicrobial resistance and mastitis. This single issue of the Journal carries two papers concerned with the topic, one in cattle in China, the other (where there was little evidence of resistance) in sheep in Scotland. In 2018 I was lucky enough to listen to a series of presentations from Bristol Vet School academics that were focused on antimicrobial resistance (AMR) in a One Health context. I was struck by the fact that, whilst the speakers could agree on the threat posed by AMR in a general veterinary sense, there was no agreement (amongst eminent colleagues, remember) when it came to mastitis. It is perhaps important to remember two things. Firstly, in dairy production systems throughout the developed world, efficient measures have been in place for decades to protect dairy processing plants by keeping mastitic milk out of the food chain. Inevitably, there are potential knock-on benefits for consumers. Secondly, over the last few decades the rise in AMR has been accompanied by a fall in the number of dairy cows, but a major rise in domestic pet numbers. I do not wish to be accused of complacency. I recognize that successfully containing mastitis could become more problematic in the future and I endorse efforts to limit antibiotic use on dairy farms. But in this case, I would

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contend that the important questions have not been answered. ‘Is there evidence that AMR has compromised the control of mastitis in dairy animals’ I don’t think there is, mastitis does not seem to be on the rise. ‘Is there definitive evidence that antibiotic use as mastitis therapy and/or prophylaxis has compromised human health’. Again, I think not. ‘Has the focus on AMR compromised a more holistic approach to the use of veterinary drugs, including for example antihelminthics’. The issue of ivermectin resistance in human health is disputed and unresolved (Simonart and Lam Hoai, 2024). But there are two other questions that worry me: ‘Is it perhaps the case that focus on mastitis has detracted from a more general effort to avoid veterinary-related AMR on livestock farms and, importantly, in domestic homes?’ Maybe! ‘Has bovine mastitis achieved too much research focus relative to mastitis in general, and especially mastitis in breastfeeding women’. If you follow my Editorials you will know that I have been here before (Knight, 2022), and you will know my views! Turning to methane. Since the millenium there has been an exponential rise in the number of research papers concerned with dairy cows and methane, and PubMed lists 250 in the last year alone. The FAO Report ‘Livestock’s Long Shadow’ (Steinfeld *et al.*, 2006) laid the basis for this research explosion. I have searched for evidence of direct scientific questioning of the Report and, whilst it was a rather simplistic search, I found only one article that mentioned the report by name (Glatzle, 2014). I shall not attempt to rebut either the Report or the rebuttal, and I shall admit to significant personal bias largely consequential on witnessing a huge expansion in the use of internal combustion engines during my lifetime. Once again, it is clear that dairy scientists have jumped on the bandwagon of the issue. They were able to do so largely because methane emission by ruminants is essentially from a point source (the mouth) and hence reasonably easy to measure in a research scenario. So consider this. Methane is not produced metabolically in any significant quantity by mammals, it is primarily a product of anaerobic methanogenic bacteria. In addition to the rumen, these bacteria inhabit an abundance of aquatic environments, paddy fields and landfill waste disposal sites being good examples. In contrast to dairy cattle, these sources are extremely diffuse, and not yet particularly amenable to quantification. I am happy to predict that this will change over the next decade, as satellite imaging technologies become more adept at monitoring methane emissions (it is happening already). I suggest that FAO should fairly soon commission a new report, *Lifestyle’s Long Shadow*. In closing, I might appear to have been critical of dairy research scientists for (forgive the

pun) milking the opportunities provided by the blame culture, and if so, I apologize. The blame does not really reside with the scientists, but much more so with the higher-level decision makers and funders who have encouraged or even driven the trend. I was surprised to discover that my last Editorial (Knight, 2024) gained significant social media exposure as a result of being picked up by podcasters. It would be nice if this were repeated, such that my concerns become known at political and strategic research management levels. It is high time that we reexamined the prevalent notion that decisions regarding how science can best advance society need to be made by non-scientists.

Note

¹ Twenty or so years ago the acronym BLAME was ascribed to a novel member of the CD2 family, B lymphocyte activator macrophage expressed, but its possible role in B cell activation appears to have been discounted since.

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