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TRENDS IN FOOD SCIENCE & TECHNOLOGY

Letter to the Editor

Response

Please find below our response to the issues raised in the letter written by Solae Scientists regarding the following article:

Girgih, A. T., Myrie, S. B., Aluko, R. E., Jones, P. J. H. (2013). Is category A status assigned to soy protein and cardiovascular disease risk reduction health claim by the United States Food and Drug Administration still justifiable? Trends in Food Science and Technology, 30, 121–132.

1. The authors claim that the FDA "assessment of the soy protein evidence excluded the isoflavone component because of safety concerns such as altered menstrual cycle, male infertility and memory loss" (p. 122) **is untrue and misleading**. In fact, in Section 3 of the FDA Final Rule (Federal Register of October 26, 1999)1 which the authors cite, the following reasons for not including isoflavones is stated as: "Given the limited number of studies and the contradictory outcomes, FDA was not persuaded that the isoflavone component of soy protein was a relevant factor to the diet–disease relationship. Rather, FDA tentatively concluded that the evidence from a wide range of studies using differently processed soy protein was supportive of a relationship between soy protein per se and reduced risk of CHD".

Response: Please note that the intended statement was "The FDA assigned the soy protein health claim the highest ranking of an 'A' category status, but the assessment of the soy protein evidence excluded the isoflavone component ***probably*** because of safety concerns such as altered menstrual cycle, male infertility and memory loss (Fitzpatrick, 2003)". We apologize to readers if the omission of the word 'probably' caused any confusion regarding the intent of the sentence. However, please note that we cited a post-1999 reference and therefore, the sentence is simply our own assessment to indicate that the exclusion of isoflavone could have been due to the safety concerns associated with isoflavone consumption. Our inference is based on the "**contradictory outcomes**" phrase in Section 3 of the FDA Final Rule (Federal Register of October 26, 1999), which we interpreted to mean the various studies that have shown beneficial, non-beneficial and even harmful effects of dietary isoflavones. Since the FDA decided not to include the isoflavone component, a logical interpretation is that either the decision was based on its lack of biological activity or the potential to cause harmful effects. Since scientific evidence suggests potential harmful effects rather than inactivity, we inferred that the exclusion of isoflavones could have been due to safety concerns. The sentence in question was balanced by the next statement, which stated thus: "***Intact soy proteins that contain isoflavones have gained considerable attention in the last decade for their potential role in reducing the risk factors for CHD (Sagara et al., 2004), including their ability to lower serum cholesterol significantly more than soy protein without isoflavones in humans (Sacks et al., 2006a; Crouse et al., 1999)***". Therefore, we argued that while the FDA might not have considered isoflavones in their decision on health benefits of soybean proteins, evidence certainly do exist that soybean isoflavones could have positive effects on CHD.

2. As evidence of the need to demote the level of the FDA soy protein health claim, the authors point out that the AHA challenged the heart health claim in 2006[2] and that the European Food Safety Authority failed to approve the soy protein and reduction of LDL-C claim application on two occasions. With regard to the AHA challenge, one of the original authors of the AHA Advisory conducted a more recent meta-analysis and re-evaluation of the studies cited by the AHA in their 2006 Advisory and concluded that the studies overall showed that soy protein provided significant LDL cholesterol (LDL-C) reduction (−0.17 mmol/L or −4.3%) through an intrinsic mechanism and also contributed to cholesterol lowering when displacing animal derived protein (extrinsic mechanism) by an estimated 3.6–6.0%[3]. This level of LDL-C reduction is similar to that observed with other ingredients which have health claims (barley beta-glucan (−0.26 to −0.27 mmol/L)[4,5], oat beta-glucan (−0.13 to −0.21 mmol/L)[6–8] and viscous soluble fibers (−0.17 mmol/L)[7]). With regard to the failure of soy protein to obtain health claim approval by EFSA, it is among the greater than 93% of ingredients whose applications for health claims were rejected by

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this organization[9]. It is well recognized that health claims in the EU, particularly for complex ingredients such as soy protein which are mixtures and not single characterized molecular entities and for which there is not yet a single identified mechanism of action, will continue to be difficult to obtain under the current paradigm.

Response: We do not dispute the statements about American Heart Association (AHA) and European Food Safety Authority (EFSA). However, we also discussed (page 126) the recent meta-analysis and re-evaluation conducted by one of the authors of the AHA advisory (Jenkins *et al*., 2010), which showed that the average reduction in LDL-C from 22 previous studies in the AHA Advisory was 4.3%. This value is far less than the 9 to ~13% range upon which the soy protein health claim obtained its most significant support for approval. While Jenkins *et al*. (2010) have proposed intrinsic and extrinsic mechanisms to suggest that the CHD benefits of soy proteins could be as high as 10.3%, the authors stated in their conclusion that "***We can offer no new insights to explain the difference in LDL-C reduction between the pre- and post-1995 soy studies***", which agrees with our position that a discrepancy does exist between data used for the soy protein health claim approval and currently available evidence. This point is core to our review article. Indeed, the fact that the EFSA did not approve the soy protein health claim is additional indication that the scientific evidence is not strong enough.

3. The authors also present an argument that the isoflavones may be responsible for the cholesterol lowering effect of soy protein when this argument has largely been abandoned recently in light of many studies conducted since the health claim was issued that show that isolated isoflavones do not lower cholesterol (studies are summarized in Harland, J. I. *et al*., 2013)[10]. Moreover, the authors cite Taku, K. *et al*., 2007[11] as support for the role of isoflavones as being responsible for the cholesterol lowering effect of soy protein, however, the authors failed to cite a subsequent meta-analysis by Taku, K. *et al*., 2008[12] that showed that isolated isoflavones, in the absence of soy protein, do not demonstrate a cholesterol lowering effect.

Response: This review collated published arguments and scientific works on the potential role of intact soy protein (Anderson *et al*., 1995; Baum, J. *et al*., 1998), soy isoflavones (Taku *et al*., 2007; Zhuo *et al*., 2004; Song *et al*., 2007) or a mixture of both (Sagara *et al*., 2003; Sanders *et al*., 2002; Crouse *et al*., 1999) in terms of observed lipid lowering effects of soy products. The review article provides background information on the evolving nature of the role of soy protein components and not just a focus on recent data. Moreover, the fact that recent evidence suggests lack of effectiveness of isoflavones alone in LDL-C reduction by soy protein is reflected on page 126 of our article, which states that "***Another school of thought from recent meta-analyses suggests that the greatest reductions in cholesterol, especially in LDL-C levels, are achieved when soy protein and soy isoflavones are consumed concurrently rather than when eaten separately***". Therefore, the article already contains information pertaining to the lack of LDL-C reducing effect by isoflavones in the absence of soy protein.

4. The authors compiled a table of studies of emerging clinical trials and meta-analyses showing moderate to no effect of soy protein on CVD risk (Table 2); the authors cite 5 meta-analyses conducted after 1999 (Weggemans, 2003; Balk, 2005; Zhan & Ho, 2005; Reynolds, 2006; Taku, 2007) but fail to cite additional relevant meta-analyses (Zhou, 2004[13]; Harland, 2008[14]; Hooper, 2008[15]; Anderson, 2011[16]) that have been published since 1999. The authors do not provide a justification or criteria for the selection of the studies listed in Table 2. Of the studies listed, two examined the effects of isoflavones with little or no soy protein consumption (Clerici *et al*., 2007; Atteritano *et al*., 2007) and one study was included that was not a cholesterol lowering study but a report of lipid changes in a study designed to investigate the role of soy protein on bone density in post-menopausal women (Campbell *et al*., 2010). The authors failed to cite 21 clinical studies that were conducted between 1999 and 2010 on soy protein and cholesterol lowering (Allen, 2007[17]; Ashton, 2000[18]; Azadbakht, 2007[19]; Borodin, 2000[20]; Dent, 2001[21]; Evans, 2007[22]; Greany, 2004[23]; Hoie, 2005a, 2007[24]; Hoie 2005b[2526]; Maki, 2010[27]; McVeigh, 2006[28]; Meyer, 2004[29]; Pipe, 2009[30]; Radhakrishnan, 2009[31]; Takatsura, 2000[32]; Van Horn, 2001[33]; Welty, 2007[34]; West, 2005[35]; Wong, 2010[36]) (all cited in Harland *et al*., 2010)[10]. Overall it can be concluded that the authors did not conduct a comprehensive and fair review of the soy protein and cholesterol lowering literature.

Response: Even though this is a review article, due to page limitations it is impossible to cite virtually all works that are related to a topic. Moreover, most of the excluded references contained data that are similar to the contents of Table 2 in our article, so we see no additional value of reporting several references that have similar data for soy protein lipid reduction ability (usually in the range of 2–7%). The authors have conducted a comprehensive and fair review of both past and presence evidence regarding soy protein efficacy as a modulator of CHD using a representative list of published works. The comments from our colleagues at Solae would have been useful if they indicated specific references absent from our review article, but which contradict current opinion in the scientific literature that there is only a moderate reduction (2–7%) in

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