

independent of oestrogen pathways, such as metabolic dysfunction (Gangwisch *et al.* 2007) and chronic inflammation (Irwin *et al.* 2006).

Again, we thank Yang *et al.* for this letter and are glad that more studies, such as the population-based case-control study in Jiujiang city mentioned by Yang *et al.*, are using objective measures along with questionnaires to better assess both the quantity and quality of sleep in relation to breast cancer risk and other health outcomes.

## REFERENCES

- Blask DE (2009) Melatonin, sleep disturbance and cancer risk. *Sleep Med Rev* 13(4): 257–264.
- Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG *et al.* (2007) Sleep duration as a risk factor for diabetes incidence in a large U.S. sample. *Sleep* 30(12): 1667–1673.

- Girschik J, Heyworth J, Fritschi L (2013) Self-reported sleep duration, sleep quality, and breast cancer risk in a population-based case-control study. *Am J Epidemiol* 177(4): 316–327.
- Irwin MR, Wang M, Campomayor CO, Collado-Hidalgo A, Cole S (2006) Sleep deprivation and activation of morning levels of cellular and genomic markers of inflammation. *Arch Intern Med* 166(16): 1756–1762.
- Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ (2008) Self-reported and measured sleep duration: how similar are they? *Epidemiology* 19(6): 838–845.
- Vogtmann E, Levitan EB, Hale L, Shikany JM, Shah NA, Endeshaw Y *et al.* (2013) Association between sleep and breast cancer incidence among postmenopausal women in the women's health initiative. *Sleep* 36(10): 1437–1444.
- Yang W-S, Wang X, Deng Q, Zhao H, Fan W-Y (2015) Sleep duration and breast cancer risk in the breast cancer detection demonstration project follow-up cohort: true associations or bias? *Br J Cancer* 112(11): 1838–1839.

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Published online 21 April 2015

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# BJC

British Journal of Cancer (2015) 112, 1840 | doi: 10.1038/bjc.2014.583

## Comment on 'Possible pro-carcinogenic association of endotoxin on lung cancer among Shanghai women textile workers'

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Sir,

In a recent article in this Journal, Checkoway *et al.* (2014) suggest that the exposure to endotoxin in industrial environments is associated with an increase in the risk of lung cancer.

A number of studies over the past 50 years has demonstrated a decreased risk in different environments involving a high exposure to endotoxin such as cotton handling and farming (Rylander, 1992; Maestrangeo *et al.*, 2005; Lenters *et al.*, 2010). Plausible cellular mechanisms for this defence have been discussed. In the data now presented there are no significant differences in risk—all are within the 95% confidence limit—and no significance for trend in relation to exposure duration. The only observation, thoroughly discussed, is a small, non-significant increase in risk in a subgroup. It is difficult to understand how such data can be used as a support to challenge a previously well-established relationship.

More serious is the lack of control of possible confounding factors. It is well known that indoor air pollution from cooking fuels is a risk factor for lung cancer. Such exposures change over the years and are closely related to socio-economic factors. The problem is discussed but in the absence of data the discussion remains speculative. Diet modulates the risk of lung cancer but is not discussed (Seow *et al.*, 2002; Rylander and Axelsson, 2006). Finally, possible changes in endotoxin exposure over the years are not dealt with. Also in China, work hygiene standards have improved over the years since the measurements were made and could result in a change of exposure to endotoxin.

In view of the above, a correct conclusion from the material presented is that 'no relation between endotoxin exposure and lung cancer risk could be detected'.

## REFERENCES

- Checkoway H, Lundin JI, Costello S, Ray R, Li W, Eisen EA, Astrakianakis G, Seixas N, Applebaum K, Gao DL, Thomas DB (2014) Possible pro-carcinogenic association of endotoxin on lung cancer among Shanghai women textile workers. *Br J Cancer* 111: 603–607.
- Lenters V, Basinas I, Beane-Freeman I, Boffetta P, Checkoway H, Coggon D, Portengen L, Sim M, Wouters IM, Heederik D, Vermeulen R (2010) Endotoxin exposure and lung cancer risk: a systematic review and meta-analysis of the published literature on agriculture and cotton textile workers. *Cancer Causes Control* 21: 523–555.
- Maestrangeo G, Grange J, Fadda E, Fedeli U, Buja A, Lange JH (2005) Lung cancer risk effect of dairy farming and the consequence of removing occupational exposure. *Am J Epidemiol* 161: 1037–1046.
- Rylander R (1992) Environmental exposures with decreased risk for lung cancer? *Int J Epidemiol* 19: S67–S71.
- Rylander R, Axelsson G (2006) Lung cancer risk in relation to vegetable and fruit consumption and smoking. *Int J Cancer* 118: 739–743.
- Seow A, Poh WT, The M, Eng P, Wang YT, Tan WC, Chia KS, Yu M, Lee HP (2002) Diet, reproductive factors, and lung cancer risk among Chinese women in Singapore: evidence for a protective effect of soy in non-smokers. *Int J Cancer* 97: 365–371.

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Published online 20 November 2014

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# BJC

British Journal of Cancer (2015) 112, 1840–1841 | doi: 10.1038/bjc.2014.584

## Reply to Comment on: 'Possible pro-carcinogenic association of endotoxin on lung cancer among Shanghai women textile workers'

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Sir,

We appreciate the thoughtful comments by Rylander and Jacobs (2015) on our paper (Checkoway *et al.*, 2014). The absence of an inverse

exposure-response relation for endotoxin and lung cancer in the extended follow-up was somewhat unexpected in view of the reported consistent findings from numerous prior studies, including our initial follow-up of the