LETTER TO THE EDITOR

BJC

British Journal of Cancer (2013) 108, 1389–1390 | doi: 10.1038/bjc.2013.98

False positive result in study on hookah smoking and cancer in Kashmir: measuring risk of poor hygiene is not the same as measuring risk of inhaling water filtered tobacco smoke all over the world

K Chaouachi*,[1]

[1]DIU Tabacologie, University Paris XI, Paris, France

Sir,

All users of the fashionable hookah across the world, except in rare cases, do change the water of the pipe basin after each smoking session (or get it changed). This is a well-established fact, as the following note a physician in Nepal early wrote in the *British Medical Journal* shows: 'water is changed each time smoking is carried out' (Pande, 1962). As for the cancer risk, there is actually an extensive literature suggesting a null or weaker association than the one usually measured when it comes to tobacco use in general (Chaouachi and Sajid, 2010). Among others, let us cite an early study published in this very journal (Rakower and Fatal, 1962), another one in India (Sanghvi, 1981) and the most recent one in Punjab, Pakistan (Sajid *et al*, 2008). Interestingly, the latter, which was carried out on the other side of the Indian border, actually represents the same geographical, human and cultural environment as Kashmir, in which the Dar *et al* (2012) study was conducted.

However, Dar *et al*'s (2012) paper offers a false positive result regarding hookah smoking. It is based on a serious misinterpretation, which could have easily been avoided if the authors had paid more attention to a recent lung cancer study in the very same region, Kashmir, and a subsequent important clarification published by its senior author (Koul and Chaouachi, 2011; Koul *et al*, 2011). None of the Kashmiri participants changed the water after each session. A rarely cited 84-page WHO Egyptian report on shisha smoking was quoted, highlighting the filtering effect of water (Koul and Chaouachi, 2011). Amazingly, in a recent relevant review, Maziak (2012) cites that very study in Kashmir while ignoring the above important clarification by Koul and Chaouachi (2011). Furthermore, because the setup (e.g., smoking mixture not described by Dar *et al* (2012)), smoke chemistry and smoking behaviour of a Kashmiri hookah are completely different from those of the fashionable shisha used these days in Washington or London, both Dar *et al* (2012) and Maziak (2012) in his biased review extrapolate false positive findings to the whole world. Such a wide confusion is also fuelled by the widespread nominalist use of the 'waterpipe' neologism, as all water pipes of the world have almost nothing in common but that name imposed one decade ago (Chaouachi and Sajid, 2010).

Yet, the water 'detail' already led astray Maziak (2012) in a previous review when warning against aspergillosis. In fact, the cited study (Szyper-Kravitz *et al*, 2001) showed that the patient had not changed the water of his hookah for weeks. In sum, measuring a risk due to the blatant lack of primary hygiene (water, but also suction hose) is one thing and measuring the real 'corrected' risk (that of inhaling hookah smoke) is something else. Similarly, the study on ESCC, opium and tobacco use in Iran cited by Dar *et al* (2012), and on which we already commented (Chaouachi, 2009), may have simply measured the same lack of hygiene (water, hose).

Another source of confusion in Dar *et al*'s (2012) study appears when its authors state that 'hookah smokers seem to be exposed to (as) many toxic compounds as cigarette smokers', citing exclusively for this purpose studies by the US-American University of Beirut (US-AUB). First, the Kashmiri hookah is not the growingly popular shisha targeted by antismoking research (different smoking product, chemistry, puffing, etc.). Also, the cited experiments rely on machine smoking, not on human subjects. Cigarette-smoking machines (used for about 5 min at a pace of 1 puff per minute on average) have been widely criticised over the past half century (including by the WHO) for greatly distorting the

*Correspondence: Dr K Chaouachi; E-mail: kamcha@gmail.com

Published online 7 March 2013

reality of actual human cigarette smoking. Quite astoundingly, their use for hookah smoking 'simulation' (about 60 min of puffs arbitrarily and periodically drawn every 17 s) has been virtually imposed to research for almost one decade now. Here, the first unavoidable question is: where does the demonstrated relevance of the latter machine lie if the former one (for much shorter periods) has failed?

Furthermore, the setup (cramming of the smoking mixture inside the bowl, its systematic charring with a piece of charcoal kept in place over the same point for 1 full hour, etc.) represents at best a model about how not to smoke a hookah (Chaouachi and Sajid, 2010).

Admitting (absurd hypothesis) that such a 'protocol' were realistic, why did a German team found acrolein at levels 66 times lower (6 times lower for formaldehyde and acetaldehyde) and benzo[a]pyrene 20 times lower than in the studies by the US-AUB cited by Dar *et al* (2012) and Maziak (2012)? Yet, both used a shisha-smoking machine with similar parameters (notably, inter-puff time). The German scientists said they have 'fully validated (their) approach' (Schubert *et al*, 2011b, 2012). In these conditions, is it scientifically acceptable to hype this way the hazards of hookah smoking on a global scale?

The assessment of related biomarkers supports the need for questioning such methods as studies in the United States, Germany and Pakistan show (Sajid *et al*, 2008; Jacob *et al*, 2011; Schubert *et al* 2011a, 2012). Biological levels of blood nicotine and urine cotinine of shisha (with Moassel) users are generally similar to those found in cigarette smokers after having smoked 1 single (2 at the most) cigarette(s), which obviously does not mean that a hookah session is equivalent to smoking 20 cigarettes or more. As for nitrosamines, polycyclic aromatic hydrocarbons, primary aromatic amines, carbonyls, and so on, their metabolites are generally found in very much lower quantities in shisha users than among cigarette smokers (Jacob *et al*, 2011; Schubert *et al*, 2011a, 2011b, 2012). The German group even found no difference in urinary NNAL between shisha smokers and non-smokers (Schubert *et al*, 2011b). As for urinary concentrations of PAH metabolites, they were much less important in shisha smokers than in cigarette smokers by factors ranging from about 1.5 to 5 (Jacob *et al*, 2011). From there, statements asserting 'the similarity of biological consequences of waterpipe and cigarette smoking' (Dar *et al*, 2012) are groundless.

Finally, 'systematic reviews' and 'meta-analyses' have fallen prey to the same errors discussed above. Dar *et al* (2012) cite one of them but ignore a methodological critique of such biased literature and the global confusion it has contributed to fuel in this field of research (Chaouachi, 2011).

REFERENCES

Chaouachi K (2011) More rigor needed in systematic reviews on 'waterpipe' (hookah, narghile, shisha) smoking. *Chest* **139**(5): 1250–1251.

Chaouachi K, Sajid KM (2010) A critique of recent hypotheses on oral (and lung) cancer induced by water pipe (hookah, shisha, narghile) tobacco smoking. *Med Hypotheses* **74**: 843–846.

Chaouachi K (2009) Hookah, opium and tobacco smoking in relation to oesophageal squamous cell carcinoma. *Br J Cancer* **100**(6): 1015.

Dar NA, Bhat GA, Shah IA, Iqbal B, Makhdoomi MA, Nisar I, Rafiq R, Iqbal ST, Bhat AB, Nabi S, Shah SA, Shafi R, Masood A, Lone MM, Zargar SA, Najar MS, Islami F, Boffetta P (2012) Hookah smoking, nass chewing, and oesophageal squamous cell carcinoma in Kashmir, India. *Br J Cancer* **107**(9): 1618–1623.

Jacob P, Abu Raddaha A, Dempsey D, Havel C, Peng M, Yu L, Benowitz NL (2011) Nicotine, carbon monoxide, and carcinogen exposure after a single use of a waterpipe. *Cancer Epidemiol Biomarkers Prev* **20**(11): 2345–2353.

Koul PA, Hajni MR, Sheikh MA, Khan UH, Shah A, Khan Y, Ahangar AG, Tasleem RA (2011) Hookah smoking and lung cancer in the Kashmir valley of the Indian subcontinent. *Asian Pac J Cancer Prev* **12**(2): 519–524.

Koul PA, Chaouachi K (2011) Important clarifications about peculiarities of hookah smoking and lung cancer in Kashmir. *Asian Pac J Cancer Prev* **12**(8): 2145–2146.

Maziak W (2012) The waterpipe: An emerging global risk for cancer. *Cancer Epidemiol* **37**(1): 1–4.

Pande BR (1962) Hubble-bubble Smoking. *BMJ* **1**: 1556.

Rakower J, Fatal B (1962) Study of narghile smoking in relation to cancer of the lung. *Br J Cancer* **16**: 1–6.

Sajid KM, Chaouachi K, Mahmood R (2008) Hookah smoking and cancer: carcinoembryonic antigen (CEA) levels in exclusive/ever hookah smokers. *Harm Reduct J* **5**: 19.

Sanghvi LD (1981) Cancer epidemiology: the Indian scene. *J Cancer Res Clin Oncol* **99**: 1–14.

Schubert J, Heinke V, Bewersdorff J, Luch A, Schulz TG (2012) Waterpipe smoking: the role of humectants in the release of toxic carbonyls. *Arch Toxicol* **86**(8): 1309–1316.

Schubert J, Kappenstein O, Luch A, Schulz TG (2011a) Analysis of primary aromatic amines in the mainstream waterpipe smoke using liquid chromatography-electrospray ionization tandem mass spectrometry. *J Chromatogr A* **1218**(33): 5628–5637.

Schubert J, Hahn J, Dettbarn G, Seidel A, Luch A, Schulz TG (2011b) Mainstream smoke of the waterpipe: Does this environmental matrix reveal as significant source of toxic compounds? *Toxicol Lett* **205**: 279–284.

Szyper-Kravitz M, Lang R, Manor Y, Lahav M (2001) Early invasive pulmonary aspergillosis in a leukemia patient linked to Aspergillus contaminated marijuana smoking. *Leuk Lymphoma* **42**: 1433–1437.