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Impact of secondhand smoke exposure on smoking cessation in cardiac patients

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Secondhand smoke (SHS) may act as a smoking cue and prime dose of nicotine by activating acetylcholine receptors in brains to trigger smoking (1). SHS exposure at home and having a smoking spouse or peers predicted lower likelihood of smoking cessation in cancer patients (2). Similar cross-sectional findings were found in cardiac patients (3). This study prospectively investigated the roles of SHS exposure at home and outside home on smoking cessation among cardiac out-patients in Hong Kong (19.1% men and 3.1% women smoked daily in 2012).

We used data from a randomized controlled trial (RCT), which found no difference on smoking cessation between stage-matched intervention and control groups (4). A total of 1495 (80.4%) adult smokers attending the cardiac out-patient clinics in major hospitals in Hong Kong were interviewed and followed at 12-month. SHS exposure at home was reported at baseline and categorized as none, ≤1 hour/day and >1 hour/day. The number of smoking family members and exposure to SHS outside home were categorized as none and any. Smoking cessation was defined as self-reported abstinence in the past 30 days at 12-month follow-up. Socio-demographic characteristics, alcohol drinking, smoking quantity and duration, long-term medication use and cardiac surgery received were recorded and treated as potential confounders. Associations of having smoking family member(s), SHS exposure at home and SHS exposure outside home with smoking cessation were analyzed adjusting for intervention allocation (Model 1), demographic characteristics and smoking behaviors (Model 2) and clinical variables (Model 3), using logistic regressions to yield adjusted odds ratios (AORs).

The subjects (91.2% male, mean age 59 ± 13.7) smoked for an average of 39.3 (± 15.1) years and consumed cigarettes for 41 (± 27.4) pack-years. Having smoking family member(s)

(24.3%) was associated with a lower AOR (95% CI) of 0.64 (0.49-0.83) for smoking cessation with the same effect size after adjusting for various potential confounders in different models (Table 1). Any SHS exposure at home (20.2%) was associated with an AOR (95% CI) of 0.65 (0.48-0.89) for smoking cessation, and increasing duration of SHS exposure at home was associated with decreasing odds of smoking cessation (P for trend <0.001). SHS exposure outside home (89.6%) was not significantly associated with smoking cessation. Repeated analyses using 7-day abstinence as smoking cessation yielded similar results (tables not shown).

We found a prospective, dose-response and robust association between SHS exposure at home and lower likelihood of smoking cessation in cardiac out-patients. The magnitude of the association was in line with our studies on adolescents, but is smaller than studies on patients with cancer (AOR 0.19-0.25) and coronary heart disease (AOR 0.26). SHS exposure outside home in our subjects was likely and mostly from indoor workplaces, restaurants and streets where the exposure should be less intense than at home. This may explain the non-significant association between SHS exposure outside home and smoking cessation.

Smoking cessation is one of the most effective strategies for primary and secondary prevention of cardiovascular disease (CVD) but received far less attention than other CVD risk factors such as hypertension, hyperlipidemia and diabetes (5). Cardiologists play an important role in assisting smokers to quit particularly at teachable moments of CVD diagnosis and hospitalization. Current practice guidelines for smoking cessation based on the evidence from pharmacological and behavioral trials achieved lower than 50% abstinence at 12-month follow-up. In addition to standard smoking cessation practices, cardiologists should assess SHS exposure in smokers and advise smokers to avoid being exposed to SHS and

smoking cues. Including family members in smoking cessation counseling may be warranted particularly for CVD patients who usually require lifestyle and behavioral modifications using a family-centered approach. Future interventions including counseling on SHS reduction may produce extra beneficial effects on smoking cessation in cardiac patients in addition to the beneficial effects of avoiding SHS on cardiovascular function in smokers.

All information are self-reported and subjected to reporting bias. Using biological markers of SHS exposure could not distinguish places of exposure which were differentially associated with smoking cessation. The generalizability of this study might be limited as it was originally designed as a trial and adopted a non-random sampling method, although the subjects were screened based on a large pool of patients (N=60588).

We found SHS exposure at home was associated with lower likelihood of smoking cessation among cardiac out-patients. Interventions to reduce SHS exposure may increase quitting among cardiac patients.

References

- 1. Brody AL, Mandelkern MA, London ED et al. Effect of secondhand smoke on occupancy of nicotine acetylcholine receptors in brain. Arch Gen Psychiatry 2011;68:953-60.
- 2. Eng L, Su J, Qiu X et al. Second-hand smoke as a predictor of smoking cessation among lung cancer survivors. J Clin Oncol 2014;32:564-70.
- 3. Prugger C, Wellmann J, Heidrich J et al. Passive smoking and smoking cessation among patients with coronary heart disease across Europe: results from the EUROASPIRE III survey. Eur Heart J 2014;35:590-8.
- 4. Chan SS, Leung DY, Abdullah AS, Wong VT, Hedley AJ, Lam TH. A randomized controlled trial of a smoking reduction plus nicotine replacement therapy intervention for smokers not willing to quit smoking. Addiction 2011;106:1155-63.
- 5. Rigotti NA, Clair C. Managing tobacco use: the neglected cardiovascular disease risk factor. Eur Heart J 2013;34:3259-67.

Table 1 Associations of SHS exposure and smoking cessation at 12-month follow up

Family smoking and SHS	Model 1 (N=1495)	Model 2 (N=1469)	Model 3 (N=1359)
exposure	AOR (95% CI)	AOR (95% CI)	AOR (95% CI)
Smoking family member(s)			
None	1	1	1
Any	0.64 (0.49-0.83)***	0.62 (0.47-0.82)***	0.64 (0.48-0.86)**
SHS exposure at home			
None	1	1	1
≤1 hour/day	0.74 (0.54-0.99)*	0.73 (0.54-0.99)*	0.73 (0.53-1.00)
>1 hour/day	0.24 (0.08-0.67)**	0.26 (0.09-0.74)*	0.14(0.03-0.61)**
P for trend	< 0.001	< 0.01	< 0.001
Any	0.66 (0.50-0.88)**	0.67 (0.49-0.90)**	0.65 (0.48-0.89)**
Family smoking and home SHS			
None smoked without SHS	1	1	1
Any smoked without SHS	0.88 (0.55-1.4)	0.81 (0.50-1.30)	0.89 (0.53-1.47)
Any smoked with SHS	0.56 (0.4-0.77)***	0.55 (0.39-0.77)**	0.57 (0.40-0.81)**
P for trend	< 0.001	< 0.001	< 0.01
SHS exposure outside home			
None	1	1	1
Any	0.87 (0.61-1.24)	0.88 (0.61-1.27)	0.85 (0.58-1.24)

Mode 1 adjusted for group assignment (intervention or control). Model 2 adjusted for model 1 variable, baseline sex, age, self-rated health and lifetime cigarette consumption. Model 3 adjusted for model 2 variables, baseline duration of heart disease, long term medication and ever had heart surgery. AOR = adjusted odds ratio.

^{*}P<0.05, **P<0.01