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Invited commentary

How to save six million people per year

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In this issue of the journal, Chen et al. [1] report an association between increased intima-media thickness (IMT) of the carotid artery and second-hand smoke (SHS) exposure in non-smoking adults. Furthermore, the SHS exposure in childhood showed a relatively stronger association with increased carotid IMT than the exposure in adulthood. In another recent report, children of parents that smoke had increased risk of developing carotid atherosclerotic plaque in adulthood [2]. The same study group have previously demonstrated that exposure to parental smoking in childhood was associated with lower endothelial-dependent flow-mediated dilatation [3] and higher carotid IMT [4] in adulthood. However, these measures are surrogate end-points and needs to be interpreted carefully. In a large recent meta-analysis no association was observed between cIMT progression and cardiovascular events, whereas mean carotid IMT was positively and robustly associated with future cardiovascular risk [5]. The last observation increases the relevance of the reports mentioned above.

Apart from surrogate end-point studies, there are studies indicating that SHS is associated with a significant increase of cardiovascular disease among non-smokers [6–8]. Thus, it appears to be clear that even passive smoking increases the risk of coronary heart disease. The 2006 Spanish partial smoke-free legislation was associated with a decrease in population acute myocardial infarction incidence and mortality, particularly in women, in people aged 65–74 years, and in passive smokers [9]. Thus, the effectiveness of smoking regulations in preventing CHD has already been proved.

We know that tobacco use is the most preventable cause of death, worldwide. WHO states [10] that tobacco kills up to half of its users. Tobacco kills nearly six million people each year, of whom more than 5 million are users and ex users and more than 600,000 are nonsmokers exposed to second-hand smoke. Unless urgent action is taken, the annual death toll could rise to more than eight million by 2030. Consumption of tobacco products is increasing globally, though it is decreasing in some high-income and upper middle-income countries.

People may argue that society should not interfere in choice of life style. But health care and sick leave are expensive [11] and preventing disease means, in addition to a decrease in individual suffering and shortened lives, a significant economic gain. There is need for political action: one can compare with that we in the traffic accept social edicts and some restriction of personal freedoms such as law on seat belts and speed limits to save lives and prevent injuries. We do not allow people to use narcotics, despite these drugs kill much less people than tobacco smoking. Furthermore, the present study and numerous others, make it clear that smoking not only kills the smoker, it also causes harm to the surrounding people, such as family members and working colleagues.

The progress of treating patients with myocardial infarction has been fantastic during the last decades. Numerous research projects have been performed with the aim to improve the outcome of the acute coronary syndrome [12]. A smoking ban would probably have a larger benefit on the number of saved lives than the pharmacological progress and introduction of percutaneous coronary intervention in the treatment of myocardial infarction.

A common European decision to ban smoking from 2030 should be taken. A first step is to increase tobacco tax all over Europe as soon as possible and the second step is a smoking ban.

How long can we accept that smoking six million people each year? And how long can we accept that people earn money on these deaths?

References

[1] W.C. Chen, M. Yun, C. Fernandez, S. Li, D. Sun, C.C. Lai, Y. Hua, F. Wang, T. Zhang, S.R. Srinivasan, C.C. Johnson, G.S. Berenson, Secondhand smoke exposure is associated with increased carotid artery intima-media thickness: the Bogalusa heart study, Atherosclerosis 240 (2015) 374–379.

[2] H.W. West, M. Juonala, S.L. Gall, M. Kähönen, T. Laitinen, L. Taittonen, J.S. Viikari, O.T. Raitakari, C.G. Magnussen, Exposure to parental smoking in

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childhood is associated with increased risk of carotid atherosclerotic plaque in adulthood: the cardiovascular risk in young Finns study, Circulation 131 (2015) 1239–1246.
[3] Children of parents that smoke have increased risk of developing carotid atherosclerotic plaque in adulthood. However, parents who exercise good smoking hygiene can lessen their child's risk of developing plaque M. Juonala, C.G. Magnussen, A. Venn, S. Gall, M. Kähönen, T. Laitinen, L. Taittonen, T. Lehtimäki, E. Jokinen, C. Sun, J.S. Viikari, T. Dwyer, O.T. Raitakari, Parental smoking in childhood and brachial artery flow-mediated dilatation in young adults: the cardiovascular risk in Young Finns study and the childhood determinants of adult health study, Arterioscler. Thromb. Vasc. Biol. 32 (2012) 1024–1031.
[4] S. Gall, Q.L. Huynh, C.G. Magnussen, M. Juonala, J.S. Viikari, M. Kahonen, T. Dwyer, O.T. Raitakari, A. Venn, Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults, Eur. Heart J. 35 (2014) 2484–2491.
[5] M.W. Lorenz, J.F. Polak, M. Kavousi, E.B. Mathiesen, H. Völzke, T.P. Tuomainen, D. Sander, M. Plichart, A.L. Catapano, C.M. Robertson, S. Kiechl, T. Rundek, M. Desvarieux, L. Lind, C. Schmid, P. DasMahapatra, L. Gao, K. Ziegelbauer, M.L. Bots, S.G. Thompson, D. Yanez, M. Juraska, S.R. Srinivasan, G.S. Berenson, R.L. Sacco, J.C. Witteman, M.M. Breteler, A. Hofman, S.H. Johnsen, E. Stensland, S. Agewall, M. Sitzer, H. Steinmetz, M. Dörr, U. Schminke, H. Poppert, H. Bickel, J. Kauhanen, K. Ronkainen, J.P. Empana, P. Ducimetiere, G.D. Norata, L. Grigore, J. Price, G. Fowkes, J. Willeit, L. Bokemark, B. Fagerberg, Carotid intima-media thickness progression to predict cardiovascular events in the general population (the PROG-IMT collaborative project): a meta-analysis of individual participant data, Lancet 379 (2012) 2053–2062.
[6] S.A. Glantz, W.W. Parmley, Passive smoking and heart disease: epidemiology, physiology, and biochemistry, Circulation 83 (1991) 1–12.
[7] J. He, S. Vupputuri, K. Allen, M.R. Prerost, J. Hughes, P.K. Whelton, Passive smoking and the risk of coronary heart disease – a meta-analysis of epidemiologic studies, N. Engl. J. Med. 340 (1999) 920–926.
[8] J. Barnoya, S.A. Glantz, Cardiovascular effects of secondhand smoke: nearly as large as smoking, Circulation 111 (2005) 2684–2698.
[9] F. Agüero, I.R. Dégano, I. Subirana, M. Grau, A. Zamora, J. Sala, R. Ramos, R. Treserras, J. Marrugat, R. Elosua, Impact of a partial smoke-free legislation on myocardial infarction incidence, mortality and case-fatality in a population-based registry: the REGICOR Study, PLoS One 8 (1) (2013) e53722.
[10] WHO Fact Sheet 339. http://www.who.int/mediacentre/factsheets/fs339.
[11] P. Lundborg, Does smoking increase sick leave? Evidence using register data on Swedish workers, Tob. Control 16 (2007) 114–118.
[12] E. Braunwald, Cardiovascular pharmacology: a look back and a glimpse into the future, Eur. Heart J. Cardiovasc. Pharmacother. 1 (2015) 7–9.