

## Indoor Air Pollution and Cardiovascular Health

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Air pollution significantly increases cardiovascular morbidity and mortality [1-3]. During several past years, attention was drawn to Indoor Air Pollution (IAP) and its possible role as a risk factor for cardiovascular disease. National Human Activity Pattern Survey (sponsored by the Environmental Protection Agency) demonstrated that people spend most of their time indoors (up to 87%) [4]. While indoors, they are exposed to indoor air pollutants generated by cooking, combustion of biomass fuel (BMF, wood, charcoal, etc), use of electrical appliances, fireplaces, smoking and by ozone-associated chemical reactions involving household elements (e.g. linoleum, paint, adhesives, particle filters, ventilation ducts etc.) [5]. A significant source of Indoor Air Pollution (IAP) is smoking. Importantly, both mainstream and exhaled cigarette smoke generates fine particles (aerodynamic diameter  $\leq 2.5 \mu\text{m}$ ,  $\text{PM}_{2.5}$ ) [6]. Study of air quality in 66 US casinos revealed that in the smoking casinos means  $\text{PM}_{2.5}$  concentration was  $53.8 \mu\text{g}/\text{m}^3$  vs.  $4.3 \mu\text{g}/\text{m}^3$  outside those casinos [7].

In addition to the indoor-specific pollutants, a great deal of IAP originates from outdoor sources, and then penetrates indoors. Polidori et al. demonstrated that in two retirement homes in the Los Angeles basin, the indoor measured Organic Carbon (OC) had on average 36-44% of outdoor-generated primary OC [8]. Penetration of outdoor pollutants indoors is characterized by infiltration efficiency ( $F_{\text{inf}}$ ).  $F_{\text{inf}}$  represents the fraction of ambient particles that penetrate indoors and remains suspended. Study of home cohorts in six communities in Maryland, Illinois, North Carolina, California, New York and Minnesota found  $F_{\text{inf}}$  of  $0.62 \pm 0.21$  for  $\text{PM}_{2.5}$  [9]. These data indicate that indoor air contains considerable amount of ambient particulates from outdoors.

Significant adverse health effects are caused by ultrafine particles (aerodynamic diameter  $< 100 \text{ nm}$ , UFP). The main source of outdoor UFP is fuel combustion in motor vehicles, aircrafts and marine vessels. UFP were shown to actively penetrate buildings with closed windows ( $F_{\text{inf}}$  was 0.48 for particles  $> 90 \text{ nm}$  in diameter). With opened windows  $F_{\text{inf}}$  for these particles increased significantly and comprised 0.72 [10].

The majority of the population in developing countries and residents in rural areas in developed countries extensively use Biomass Fuel (BMF) for cooking which leads to a significant release of air pollutants and decreases indoor air quality [11]. Improvement in open-fire stoves (adding enclosed combustion chamber and chimney for ventilation) in villages in San Marcos area in Guatemala, resulted in a greater than 2 times decrease in the indoor concentration of  $\text{PM}_{2.5}$  (from  $264$  to  $102 \mu\text{g}/\text{m}^3$ ), and was associated with  $3.7 \text{ mm Hg}$  decrease in systolic blood pressure (SBP) and  $3.0 \text{ mm}$  decrease in Diastolic Blood Pressure (DBP) in women [12].

In women living in six villages in northwestern Yunan province in China, BMF combustion for cooking resulted in a median indoor concentration of  $\text{PM}_{2.5}$  of  $52 \mu\text{g}/\text{m}^3$  in summer and  $105 \mu\text{g}/\text{m}^3$  during the winter. In female cohort older than 50 years, a 1-log- $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  (i.e. increase 10 times), was associated with  $4.1 \text{ mm Hg}$  increase in SBP and  $1.8 \text{ mm Hg}$  increase in DBP. Based upon a statistical model employed by the Asia Pacific Cohort Studies Collaboration,

the authors estimated that lowering SBP by  $4 \text{ mm Hg}$  among Asian women 50-59 years of age, would result in an 18% decrease in coronary artery disease and a 22% decrease in stroke, which would result in a staggering decrease in death rate by 230,900 deaths/year [13, 14]. Study in healthy young non-smoking students in Taipei (Taiwan) revealed a SBP increase by  $1.85$ - $2.64 \text{ mm Hg}$  per interquartile range (IQR) of indoor coarse particles and by  $1.94$ - $2.99 \text{ mm Hg}$  per IQR of indoor fine particles at 1-4-hour means. Increases of comparable magnitude were also found for DBP and heart rate [15].

More studies are needed to address the link between IAP and acute myocardial infarction, coronary artery disease, atherosclerosis, hypertension and congestive heart failure. However, data already published clearly indicate that IAP is a serious risk factor for cardiovascular disease. Health risk of IAP is underscored by the fact that disability-adjusted life-years (DALYs) lost due to IAP comprise 1,100 DALYs per 100,000 population [16]. On a positive note, there is evidence suggesting that measures aimed at fighting IAP, such as Clean Indoor Air Acts (banning indoor smoking) are effective in reducing the prevalence of cardiovascular disease [17].

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