

Time Course of Endothelial Dysfunction and Endothelial Microparticle Generation After Acute Second Hand Smoke Exposure in Healthy Non-smokers



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BACKGROUND:

- Active smoking is a well-known cardiovascular risk factor with impaired endothelial function in chronic smokers.
- However, data about the effect of second hand smoke (SHS) exposure on the cardiovascular system are more limited.

HYPOTHESIS:

- We sought to evaluate the impact of SHS on the endothelium by monitoring the endothelial function and the level of endothelial microparticles (EMPs) formation, which is a specific marker of endothelial injury, during the 24h following a short SHS exposure.

METHODS:

- Ten healthy young non-smoking volunteers (see **Table 1**) were exposed to controlled levels of second hand smoke in a controlled human exposure chamber for 30 min.
- Endothelial function was assessed as flow mediated dilation (FMD) using ultrasound and blood was drawn before (baseline), then immediately (0 min), 60 min, 150 min, and 24 h after exposure.
- In parallel, circulating EMPs were measured using flow cytometry in platelet-free plasma samples obtained from peripheral venous blood. Specific measurements were obtained for endothelial MPs that were CD62e+ (E-selectin), CD144+ (VE-Cadherin), and CD31+ (PECAM) /CD41-.
- After a wash-out period of >1 week, n=7 of the volunteers were subjected to the same protocol with 30 min control air exposure.

Table 1 : Baseline characteristics of the subjects

Mean age (y)	30 ±1
Sex (M/F)	7 / 3
Body mass index (kg/m ²)	24.3 ± 1.1
LDL-Cholesterol (mg/dl)	121 ± 9
Fasting glucose (mg/dl)	73 ± 3
Mean blood pressure (mmHg)	74 ± 3

RESULTS

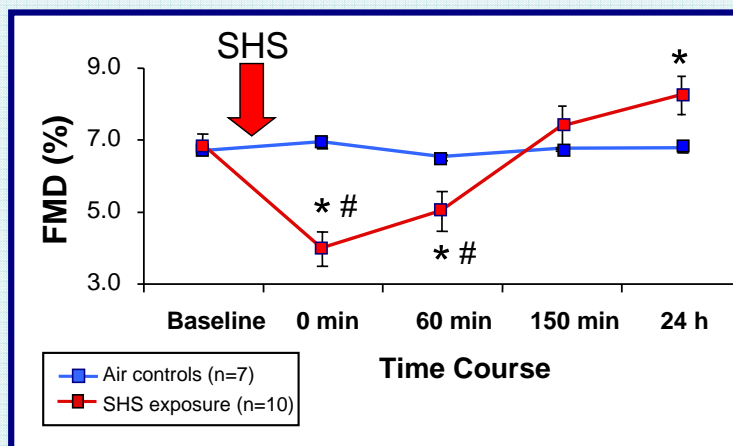


Figure 1: Time course of FMD after SHS exposure (*: p<0.05 vs. baseline of same day) (#: p<0.05 vs. respective time point on control day)

- Baseline values were not different for any of the EMPs groups between study days.
- The number of circulating EMPs after SHS significantly increased by 3-fold, suggesting endothelial activation (CD62e+ EMPs) and structural damage (CD144+ and CD31+/CD41- EMPs) (**Fig.2**).
- Peak increase was observed 60 min after exposure and remained significantly elevated throughout the study: CD31+/CD41- (1724±395 versus 512±75 events/μl, p=0.01 for 60 min vs. baseline), CD62e+ (258±66 vs. 88±20 events/μl, p=0.006) and CD144+ (643±145 vs. 196±39 events/μl, p=0.004).
- EMPs levels remained unaffected by control air exposure.

- Baseline values of FMD did not significantly differ between study days (6.8 ±0.3% vs. 6.6 ±0.1%)
- SHS transiently blunted FMD immediately and 60 min after (3.9±0.5% and 5.0±0.5 %, p<0.01 versus baseline, repeated measurements ANOVA) (**Fig.1**).
- FMD returned to baseline at 150 min and was significantly greater than baseline at 24h (8.2±0.6%, p=0.01).

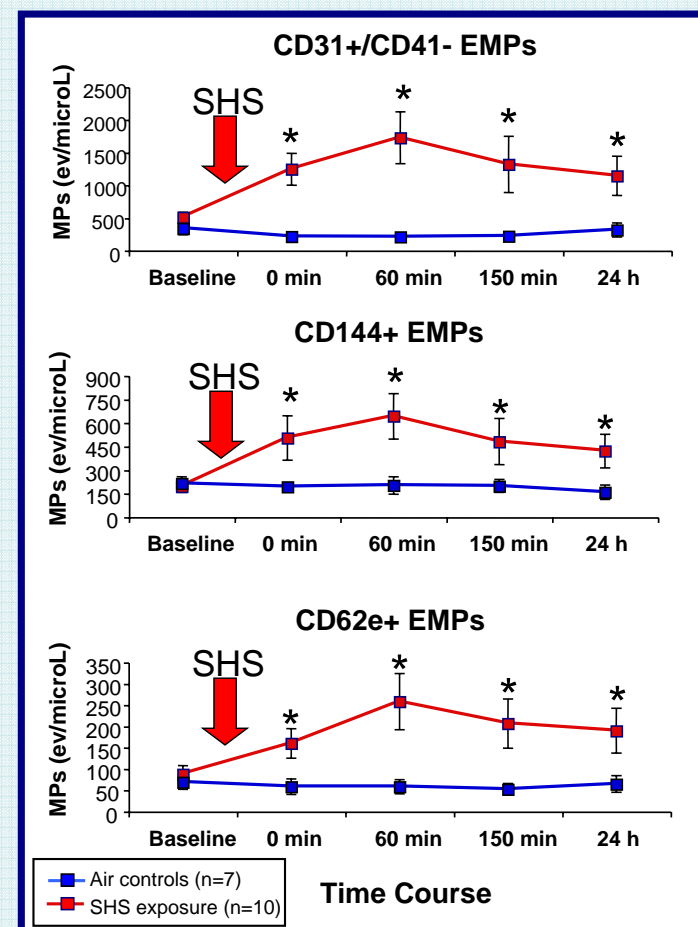


Figure 2: Time course of EMPs levels after SHS exposure (*: p<0.05 vs. baseline of same day and vs. respective time point on control day)

CONCLUSIONS:

Our results suggest that a short-term exposure to SHS induces acute endothelial injury with functional impairment (as indicated by reversible endothelial dysfunction), and structural lesion (as specified by concomitant increase in circulating endothelial microparticles levels).