Effect of PM exposure on coagulation indexes among steel workers in Italy

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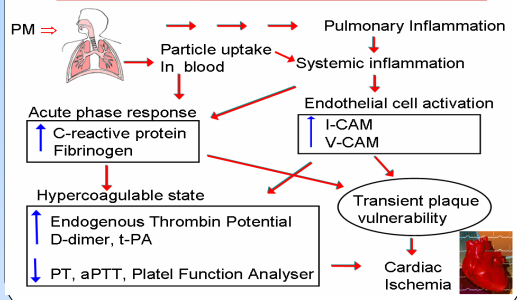
Background

- A growing body of evidence suggests that particulate air pollution leads to increased cardiovascular mortality and morbidity in the general population[1,2].
- The exact mechanisms linking the inhalation of ambient air particles to acute cardiovascular effects are not completely understood. The main current hypothesis is that particles produces alveolar and systemic inflammation which increases blood coagulation, leading to an increased risk of ischemic events [3,4,5] .
- Few study investigated population with a well characterized exposure assessment and with bio-markers of inflammation and coagulation.
- Workers exposed to well defined levels of micro-particles represent an ideal condition to investigate the effect of micro-particles on coagulation regulative mechanisms and on cardiovascular system.

Aim of the Study

To verify if occupational exposure to measured levels of PM causes alteration in inflammatory and coagulation indexes in a group of healthy steel workers.

PM - Potential Mechanisms of Action



Methods

- We enrolled 63 male workers employed in a steel production plant near Brescia. For each workers were obtained:
  - Blood drawing in the morning of the first day and of the last day of the working-week.
  - Detailed information about smoking habits, non-occupational exposure to PM, working and health history.
  - Personal exposure to PM_{10} and PM_1 , calculated considering environmental monitoring in the work place and the specific tasks of each subject.
- Multiple regression models (with adjustment for age, BMI, education, smoking and NSAIDs consumption) were performed to analyze the relation between PM exposure and coagulation/inflammatory parameters considering:
  - The end of the working-week
  - Variations of the parameters during the working week (end-beginning)

Results

- Workers enrolled were exposed to PM levels from 2 to 20 times higher than the adopted levels of action for the general population (50 µg/m³ for PM_{10} , EC dir. 30/1999) (table 1).

Table 1:Averagel exposure of enrolled subjects

Exposure	Mean (SD)	Min	Max
PM_{10} (µg/m³)	233 (214)	73	1220
PM_1 (µg/m³)	8 (6)	1.7	30

Table 2: Coagulation and inflammatory indexes. Comparison between beginning and end of the working-week

Outcomes	N	mean (SD)		paired t test
		first day	last day	
ETP	56	790 (308)	820 (283)	p=0.24
PFA (w. epineph.)	59	130 (34)	135 (33)	p=0.21
PFA (w. ADP)	59	90 (28)	88 (15)	p=0.60
t-PA	57	7.03 (3.8)	7.13 (4.0)	p=0.69
d-dimer	57	161 (72)	169 (88)	p=0.12
PT	56	11.1 (0.7)	11.1 (0.8)	p= 0.70
aPTT	56	29 (2.9)	29 (2.4)	P=0.44
PCR	57	1.61 (2.10)	1.42 (1.57)	p=0.18
WBC	62	7.31 (0.20)	7.30 (0.22)	p=0.91
I-CAM	57	309 (42)	321 (48)	p=0.03
V-CAM	57	1035 (111)	1126 (205)	p=0.001

Table 3: PM_1 exposure and variations of coagulation and inflammatory indexes during the working-week.

Outcomes	N	Multiple regression model	
		adj. ß†,	p value
ETP	56	-11.8	p=0.81
PFA (w. epinephrine)	60	-0.01	p=0.83
PFA (w. ADP)	61	+0.07	p=0.16
t-PA	57	-0.06	p=0.38
d-dimer	57	-3.84	p=0.72
PT	56	-0.003	p=0.97
aPTT	56	-0.23	p=0.62
PCR	57	+0.03	p=0.92
WBC	62	-0.08	p=0.78
I-CAM	50	+4.52	p=0.64
V-CAM	50	-85.26	p=0.14

† variation for a 10 µg/m³ increase of PM_1 exposure. Multiple regression model adjusted for BMI, smoking, age, education, NSAIDs consumption.

- Our workers presented higher levels of circulating adhesion molecules (I-CAM, V-CAM) when compared with reported levels of healthy Caucasian populations[6], especially at the end of the working-week. (Table 2)
- All other coagulation/inflammatory indexes resulted in the range of normality and did not increased during the working-week. (Table 2)
- None of the considered inflammatory/coagulation indexes resulted clearly associated with the individual exposure to PM_{10} or PM_1. (Table 3)

Conclusions

- Our study conducted among workers exposed to high concentration of PM found increased levels of circulating adhesion molecules, established risk factors of cardiovascular diseases
- We found no relation between personal exposure and other inflammatory or coagulation indexes.
- Small sample size or possible misclassifications of the exposure could have affect the power of the study to detect small changes in some considered biological indexes.
- Another possible explanation of our negative results is that our healthy workers were not comparable with the general population in which adverse effects were observed [5,7](*healthy worker effect*).
- If this hypothesis will be confirmed, our results will suggest that PM effect is concentrated on population with reduced capacity to respond to toxic agents. To individuate which are conditions of hyper-susceptibility will be a major public health priority.

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