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

Matteo Bonzini[a], Andrea Baccarelli[b], Pier Alberto Bertazzi[b], Armando Tripodi[c], Andrea Artoni[c], Pier Mannuccio Mannucci[c], Pietro Apostoli[d]

a Department of Environmental and Occupational Health, University of Milan and Fondazione IRCCS Ospedale Maggiore Policlinico, Mangiagalli e Regina Elena, Milan, Italy
b Exposure, Epidemiology and Risk Program, Harvard School of Public Health, Boston, MA, USA c Angelo Bianchi Bonomi Hemophilia and Thrombosis Center, Department of Internal Medicine, University of Milan and Fondazione IRCCS Ospedale Maggiore Policlinico, Mangiagalli e Regina Elena, Milan, Italy d Institute of Occupational Health and Industrial Hygiene, Brescia University, Italy

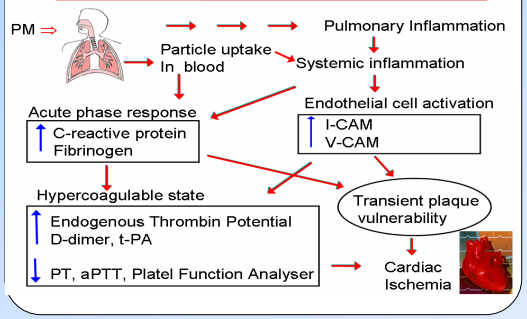
Background

- 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 a molecules (I-CAM, V-CAM) when compared with reported l healthy Caucasian populations[6], especially at the end working-week. (Table 2)
- All other coagulation/inflammatory indexes resulted in th of normality and did not increased during the workin (Table 2)
- None of the considered inflammatory/coagulation indexes clearly associated with the individual exposure to PM_{10} (Table 3)

Conclusions

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

References

1. Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL (2000) Fine particulate air poll mortality in 20 U.S. cities, 1987-1994. *N Engl J Med*, 343, 1742
2. Samoli E, Touloumi G, Zanobetti A, Le Tertre A, Schindler C, Atkinson R, Vonk J, Rossi G, Rabczenko D, Schwartz J, Katsouyanni K (2003) Investigating the dose-response relation be pollution and total mortality in the APHEA-2 multicity project. *Occup Environ Med*, 60, 977
3. Nemmar A, Hoet PH, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF, Van Mortelmans L, Nemery B (2002) Passage of inhaled particles into the blood circulation in *Circulation*, 105, 411.
4. Seaton A, Soutar A, Crawford V, Elton R, McNerlan S, Cherrie J, Watt M, Agius R, Stout Particulate air pollution and the blood. *Thorax*, 54, 1027
5. Peters A., Doring A., Wichmann H.E. & Koenig W. (1997) Increased plasma viscosity dur pollution episode: a link to mortality? *Lancet*, 349, 1582.
6. Hwang SJ, Ballantyne CM, Scharret AR, Smith LC, Davis CE, Gotto AM, Boerwinkle Circulating adhesion molecules VCAM-1 ICAM-1and E-selectin in Carotid Atherosclerosis an Coronary Heart Disease Cases *Circulation*, 96, 4219
7. Baccarelli A, Zanobetti A, Martinelli I, Grillo P, Hou L, Giacomini S, Bonzini M, Lanzani G, Man Bertazzi PA, Schwartz J, Effects of exposure to air pollution on blood coagulation, *J.Thromb* 2007 Feb, 5(2) :252-60.

Provided by AIR Universita degli studi di Milano
CORE
Metadata, citation and similar papers at core.ac.uk