

Forskningsprogram			
SNAP <input checked="" type="checkbox"/>		REPROSAFE <input type="checkbox"/>	
FLIPP		Inriktning: Ekonomiska styrmedel <input type="checkbox"/>	
Inriktning: Informationssystem och indikatorer IPP <input type="checkbox"/>			
Projekttitel (svensk): Exponering för luftföroreningar och luftvägsinflammation (APOLLON)			
Projekttitel (engelsk): Air POLLution and airway inflammatiON (APOLLON)			
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<p>Sammanfattning på svenska strukturerad enligt följande: 1) Projektets betydelse för programmet 2) Miljörelevans och förväntad betydelse för miljöpolitiken 3) Mål och hypotes 4) Metodik och genomförande 5) Kommunikationsinsatser i relation till programmet:</p> <p>1) SNAP-programmet fokuseras bland annat på hälsoeffekter av luftföroreningar genererade av biltrafik. Huvuddelen av utsläpp av partiklar och en del andra luftföroreningar härrör just från biltrafiken.</p> <p>2) Det föreligger sannolikt ett orsakssamband mellan exponering för fina partiklar och koronar hjärtsjukdom, och inflammation i de små luftvägarna kan vara en viktig länk i denna orsakskedja. Hjärt-kärlsjukdomar är en av de stora folksjukdomarna. Även om luftföroreningar endast måttligt bidrar till denna sjuklighet, så är det av betydelse för folkhälsan.</p> <p>3) Det övergripande målet för projektet är att minska på ohälsan relaterad till exponering för luftföroreningar. Det görs speciellt i detta projekt genom att försöka förbättra förståelsen för de mekanismer som gör att luftföroreningar ökar risken för hjärtsjukdom via en inflammation i luftvägarna. Den specifika hypotesen är att undersöka om exponering för yttre luftföroreningar är förknippade med en förhöjd koncentration av eNO vid högt utandningsflöde och/eller en ökad koagulationsförmåga hos blodet.</p> <p>4) Projektet genomförs på ett slumpmässigt urval av 10.000 personer i åldrarna 35-75 år, som får besvara en postal enkät och därefter undersökas avseende eNO, lungfunktion och blodprover. Exponeringen för PM₁₀, PM_{2,5}, kvävedioxid, ozon och svaveldioxid kommer för varje att modelleras utgående från befintliga luftföroreningsdata i relation till eNO och olika blod/plasma parametrar. I modellen kommer att tas hänsyn till meteorologiska data, rökvanor, luftvägssymptom m m, och dessutom kommer olika lag-tider att användas.</p> <p>5) Kan man visa att även dagens förhållandevis låga nivåer av yttre luftföroreningar är förknippade med luftvägsinflammation och påverkan på blodet så talar det för att befolkningens exponering för luftföroreningar må minska. Ur ett folkhälsoperspektiv är detta viktig kunskap.</p>			
		År 2004	År 2005
Summa sökta medel per år i kr:		743,4 kkr	826 kkr

Miljöforskningsnämnden
Ansökan om projektbidrag inom Naturvårdsverkets forskningsprogram

Sökta projektmedel fördelade på kostnadslag	År 2004 (kr)	År 2005 (kr)
Personalkostnad inkl. soc. avgifter * Sjuksköterska 12 mån á 350.000 kr Statistiker/doktorand 12 mån á 350.000 kr	350 kkr -	350 kkr 350 kkr
Övriga omkostn exkl moms (förbrukningsmtrl, analyser, resor etc)** Aerocrine NO-analysator	280 kkr	-
Delsumma av ovanstående poster:	630 kkr	700 kkr
Förvaltningspåslag: 18 %	113,4 kkr	126 kkr
Totalsumma per år: (införs sid. 1):	743,4 kkr	826 kkr

*) Specificera namn, tjänst **) Specificera

Samtliga övriga miljörelaterade projekt för vilka de sökande har beviljats anslag eller söker anslag för 2004-2006. OBS Även EU-finansiering.

Projekttitel	Finansiär	Tidsperiod	Sökt kr	Beviljat kr
ADONIX (NO)	FAS	2004-2006	3.100 kkr	

**Miljörelaterade projekt för vilka sökande har beviljats anslag för 2000-2003
OBS Även EU-finansiering**

Projekttitel	Finansiär	Tidsperiod	Beviljat Kr
WOODPART	SNAP	2002-2004	214 kkr
ADONIX (NO)	FAS	2000-2003	2.800 kkr
APOLLON	FORMAS	2001-2003	500 kkr
ADONIX/APOLLON	Hjärt-Lungfonden	2003-2005	600 kkr
ECRHS 2 (inomhusmiljö)	EU	2000-2001	500 kkr

Datum och sökandes underskrift, vilken samtidigt ger Naturvårdsverket tillåtelse att publicera sökandes namn på sin webbplats:	Datum och underskrift av prefekt eller motsvarande med namnförtydligande: Mats Hagberg, professor/överläkare/examinator
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Ansökan skall bestå av detta formulär jämte högst sex sidor lång projektbeskrivning på **engelska** (strukturerad som den svenska sammanfattningen samt en redovisning av kunskapsläget). Referenser till egna publikationer ges med sifferhänvisning till CV. Andra referenser ges i löpande text. Sökandes och eventuell medsökandes CV får omfatta högst två sidor. Inga bilagor kommer att beaktas vid bedömningen. Ansökan (max 10 A4-sidor, 12 punkters teckenstorlek) skall inlämnas i **original + 15 kopior samt elektroniskt** till ansok@naturvardsverket.se. Häfta ihop ansökan och använd hålrat papper. Ansökan skall ha inkommit senast den 15 oktober 2003 till Naturvårdsverket, Forskningssektariatet, 106 48 STOCKHOLM.

Air POLLution and airway inflammatiON (APOLLON)

Kjell Torén, MD (main applicant)
Bertil Forsberg, PhD (fellow applicant)

The project is carried out from Sahlgrenska University Hospital, Depts of Occupational and environmental medicine (Kjell Torén, Gerd Sällsten, associate professor, Anna-Carin Olin, MD, PhD, Lars Barregård, professor), and preventive cardiology (Dag Thelle, professor, and Annika Rosengren, professor) and Umeå University, Dept of Public Health and clinical medicine (Bertil Forsberg, Bo Segerstedt, MA).

Significance for the SNAP programme

The Swedish National Air Pollution and Health Effects Program have its focus on air pollution from road traffic, a major source of emissions of fine particles and certain other pollutants in Sweden. There is probably a causally association between exposure to air pollution and coronary heart disease. The proposed mechanism is that air pollution gives rise to airway inflammation, especially in the distal airways and alveoli. This inflammation enhances the production of pro-inflammatory cytokines causing an increased coagulability of the blood. Exhaled nitric oxide (eNO) is elevated among subjects with airway inflammation.

Environmental relevance and significance for environmental policy

Mortality due to coronary heart disease is of major public health concern. Due to the large numbers even a modest influence of air pollution will be of major public health relevance.

Background

Since the London smog in December 1952 it has been recognized that episodes of air pollution are associated with excess numbers of deaths. During the last years, it has also been shown that these associations even persist at the lower concentrations of air pollution such as those which are occurring in Western Europe and USA. A number of studies have shown that each increase in the PM₁₀ level of 10µg/m³ increased the rate of death from all causes by 0.4 to 1.0 percent (Katsouyanni 1997, Samet 2000). This has mainly been shown for all cause mortality, but death due to cardiovascular diseases is a major part of the deceased. In a recent Dutch study was living close to a major road associated with a doubled mortality from cardiopulmonary diseases (Hoek 2002). In a prospective study it was also shown that reduction of the levels of black smoke reduced the cardiovascular deaths by ten percent (Clancy 2002).

It has also been reported an association between hospital admissions and air pollution (Schwartz 1999), as well as an association between air pollution and cardiac arrhythmias (Peters 2000, Gold 2000). The lag time in relation to the increase of the air pollution is small, the most obvious effect is seen during the following 24 hours, or during the next day.

The importance of inflammatory processes in the development of arteriosclerosis has been highlighted during the last decade. Different pro-inflammatory cytokines, such as IL-6, are key actors inducing the production of CRP, fibrinogen and other protein

that participate in the inflammatory process. IL-6 is produced by macrophages in the airways, and it has been suggested that pulmonary inflammation caused by fine particles deposited in the alveoli may cause increased blood coagulability (Seaton 1999). It has also been shown that small particles cause an alveolar inflammation (Ghio 2000), and exposure to particles containing Cu/Zn/V have been associated with increased blood fibrinogen (Huang 03). It has, as well, been shown that the plasma viscosity increased threefold during periods with high levels of air pollution (Peters 1997), when mainly the concentrations of SO₂ that was increased, 200 µg/m³ compared with 48 µg/m³. In the Whitehall II study in London it has been shown that fibrinogen in plasma was associated with the concentrations of NO₂ and PM₁₀, however the latter only during summer (Pekkanen 2000). The median concentration of PM₁₀ was 28 µg/m³. Seaton et al (1999) modelled air pollution exposure for 112 subjects and observed unexpectedly a negative association between PM₁₀ and the number of erythrocytes and factor VIII. There was no association between PM₁₀ and IL-6, but a positive association with CRP. It may be that increased levels of fibrinogen increase the risk for coronary heart disease during episodes of high levels of air pollution.

It is also of interest to note the old observation about the connection between impaired lung function and coronary heart disease. It is independent of smoking habits, and was already shown in the Framingham study, and has been confirmed in several subsequent studies (Friedman 1976).

Hence, there seem to be some support for a connection between airway inflammation and coronary heart disease, which to some extent may be mediated via exposure to air pollution.

A simple way of measuring airway inflammation is to measure exhaled nitric oxide (eNO). In two small random population studies subjects with asthma had increased eNO (Salome 1999, Henrikssen 1999), and we have found similar findings in one cohort study of pulp mill workers (Olin 2003). In several small studies an association between eNO and ambient air pollution has been observed (Steenberg 1999, Amsterdam 1999a, b, Fischer 2002). In the latter study PM₁₀ and black smoke were associated to eNO, but the lung function measures were not. However, these studies have one major methodological disadvantage, mainly that the exhalation flow rates were low, 10 mL/s.

It has been proposed that eNO should be measured with an exhalation flow rate of 50 mL/s (ATS 1999), but higher flow rates is more representative for the distal airways and alveoli (Lehtimäki 2000, Högman 2000). The intra-individual variability (C.V.) of eNO in one minute is 5% and during two weeks 11% (Andersson 2003). In two studies there were been no signs of diurnal variability of eNO (ten Hacken 1998, Lehtimäki 2000).

The personal exposure (particles, NO₂, etc) has generally in the cited air pollution studies been estimated by modelling based on data from fixed sampling stations. For example in the Whitehall II study from London data was sampled from five sampling stations used for modelling (Pekkanen 2000). The validation of the models was performed. The modelling will introduce a significant misclassification of the true

exposure, probably a non-differential causing an underestimation of the true association. This should be further explained and elaborated.

Objective and hypothesis

The overall aim of the present study is to achieve a better understanding of the mechanisms which may exist between ambient air pollution, airway inflammation and an increased risk for coronary heart disease. The specific aim is to investigate whether exposure to air pollution is associated with increased eNO and/or increased coagulability of the blood.

Methods

The study will be performed in the context of an on-going population based epidemiological study (INTERGENE, PI Dag Thelle). The project is on a random population-sample consisting of 10.000 subjects in 35 to 75 years of age. They will be investigated with a general questionnaire dealing with different health related outcomes, diet, occupations and stress-related items.

They will be investigated with pulmonary function (flow-volume curve) which makes it possible to estimate MEF_{25} and MEF_{75} , which better reflects the small airways. eNO will be measured with three exhalation flows, 50mL/s, 100 mL/s and 300 mL/s. Weight, height, blood and plasma samples are also included, and the samples are stored for further biochemical analyses. There is also blood samples taken for analyses of genetic polymorfism (ethical permission has been obtained)

As a part of the APOLLON study all subjects are answering an additional detailed questionnaire about the activities during the last three days, including geographical location and residence. There are also questions about contact with motor vehicle emissions such as motorised lawn-mover and periods spending in traffic jams.

The field part of the study will be finished during 2005. Air pollution data will be obtained from Göteborgs Miljöförvaltning, where they have data regarding PM_{10} , $PM_{2.5}$, nitric oxides, ozone and SO_2 . The field part of the study will be finished during 2005. Temporal air pollution data will be obtained from Göteborgs miljöförvaltning, where they have data regarding PM_{10} , nitric oxides, ozone and SO_2 . The exposure for each subject during the last days before the examination will modelled by Bertil Forsberg and his research group, taking into account actual levels which will be individually adjusted using modelled average gradients in local air pollution levels (traffic pollutants indicated by nitric oxides), meteorological data, home address and questionnaire information. The use of short-term exposure data have previously been described (Forsberg 1998 a, b). The obtained dispersion model will be validated (compared) with measurements of NO_2 outside the individuals' home, as well as personal measurements of NO_2 . The exposure assessment of the project will also gain from the experience from other exposure modelstudies with within the group (Gerd Sällsten).

The study population is a cohort and the study has a longitudinal aspect, as well as a cross-sectional design. In the cross-sectional situation eNO will be related to ambient levels of air pollution. The exposure to PM_{10} , $PM_{2.5}$, NO_x , ozone and sulphur dioxide

will modelled using data from adjacent sampling stations and related to different outcomes such as eNO and fibrinogen.

The longitudinal cohort study will consist of continuous registration of hospitalised cases of cardiovascular disease events, cancer and chronic obstructive lung disorders. The event rates will be analysed according to the basic information collected during 2001-2005. The length of the follow-up will at least be three- and ten-year period. The estimated number of coronary events during the first three years is about 150, whereas the number of pulmonary cases is likely to be higher.

We are already equipped with one NO analyser and the fundings from SNAP will be used for buying one more NO analyser and for employing one more research nurse working with NO measurements. In addition, the Umeå group will start with the modelling of air pollution exposure applied to the subjects in the validation study, hence we apply for statistician during five months.

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Presentation of results

Dissemination will follow that of the SNAP programme, i.e. results will be presented on the regional, national, and international levels, to authorities, stake-holders, in journals, and at workshops and conferences.

CURRICULUM VITAE

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Läkarexamen 1976, legitimerad läkare 1980. Specialist i yrkesmedicin januari 1989, specialist i yrkes- och miljömedicin 1994 och specialist i allergisjukdomar 2000

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Forskarutbildning

leg. läk. Johan Hellgren, disputerad 2001, leg. läk. Eva Andersson, disputerad 2002

leg. läk. Anna-Carin Olin, disputerad 2002, fil. kand. Monika Lärstad (reg. 1999, halvtidskontroll 2002), leg. sjuksköterska Rosita Sundberg (reg. 2001), leg. läk Torbjörn Gustafsson (reg. 2003).

Originalpublikationer från 2002

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CV (kort) Bertil Forsberg

Bertil Forsberg är medicine doktor i epidemiologi och folkhälsovetenskap och universitetslektor vid Institutionen för folkhälsa och klinisk medicin, Umeå universitet, och arbetar ca 2/3 med externfinansierad forskning och i övrigt med undervisning, främst på Miljö- och hälsoskyddsprogrammet, samt fristående föreläsningar. Bertil Forsberg har varit ledare för Umeågruppen i de europeiska multicenterstudierna PEACE och APHEA2 med fokusering på effekter av korttidsexponering för luftföroreningar, och ingår nu i EU-projektet APHEIS med syfte att skapa ett övervakningssystem för sådana effekter. Han deltar för närvarande även i luftföroreningsstudier inom EU-projektet ECRHS ("Europastudien luftvägar och hälsa") samt i HEATWAVE, en internationell studie om studie om akuta effekter av värmeböljor. Bertil Forsberg har ansvar/at för en rad andra undersökningar och forskningsprojekt inom luftföroreningsområdet, bl a för Naturvårdsverket, Energimyndigheten, Socialstyrelsen, Folkhälsoinstitutet, länsstyrelser och kommuner.