TECHNICAL ATTACHMENTS

- Summary of contributions by collaborating institutions (page A2)
- Summary of gas phase, air quality measurements made in Southeast Texas (pages A3-A5)
- Summary of gas and particulate phase, air quality measurements made by the Houston Regional Monitoring (HRM) Network and the Cities of Houston and Galveston (page A6)
- Description of airborne instrumentation that will be used in the Texas Air Quality Study (pages A7-A9)
- Summary of exposure study to be conducted in the Houston region, in collaboration with the proposed Supersite (page A10)
- Summary of toxicology study to be conducted in the Houston region, in collaboration with the proposed Supersite (page A11-A12)

Summary of leveraging opportunities (letters confirming these leveraging opportunities are attached to this proposal)

Institution	**Type of support**	**In-kind Value**	**Direct Cash Support**
Texas Natural Resource Conservation Commission	In-kind monitoring support for both 16-month and 6-week sampling programs	In excess of $2,500,000	
Texas Natural Resource Conservation Commission	Direct support of infrastructure development for 6 week sampling program		to be determined
Southern Oxidants Study/TexAQS 2000	In-kind support, providing specialized instrumentation during 6 week sampling program, and subsequent data analysis	$5,000,000 - $10,000,000	
SEARCH, SCISSAP and other Southeastern U.S. sampling networks	In-kind monitoring support for 16 month sampling program		
Houston Regional Monitoring Network	In-kind monitoring support; data from monitoring sites		
Texas Hazardous Substance Research Center	Direct support of data analysis for 6-week and 16-month sampling program		$100,000
University of Texas, Houston Health Science Center	U.S. EPA Grant on PM induced lung inflammation	$674,288	
City of Houston	In-kind monitoring support for 16 month sampling program		
Mickey Leland Air Toxics Research Center	Houston portion of Mickey Leland National Urban Air Toxics Research Center and Health Effects Institute grant for “Contributions of Outdoor PM Sources to Indoor Concentrations and Personal Exposures: A Three City Study”	Houston portion of $1,132,206	
Southern California Center for Airborne Particulate Matter (SCCAPM)	In-kind support – participation on advisory committees		

Summary of gas phase, air quality measurements made in the Houston area
(Monitors run by the TNRCC, the City of Houston and the Houston Regional Monitoring Network are identified)

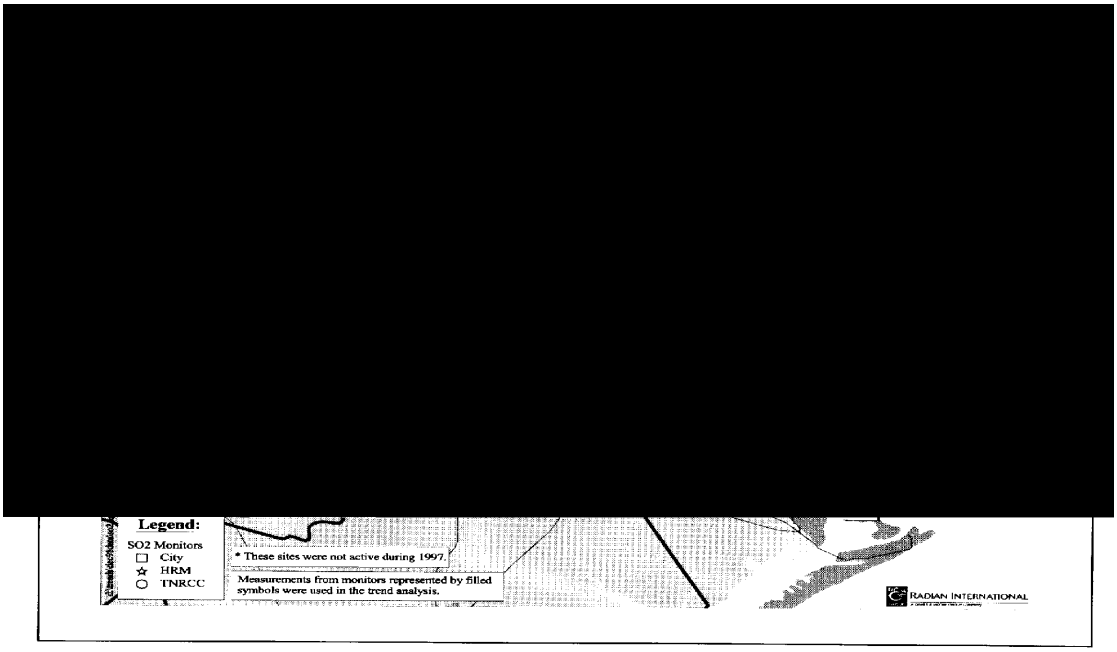

Figure A-1. Locations of SO_2 Monitoring Stations in the Houston-Galveston Area

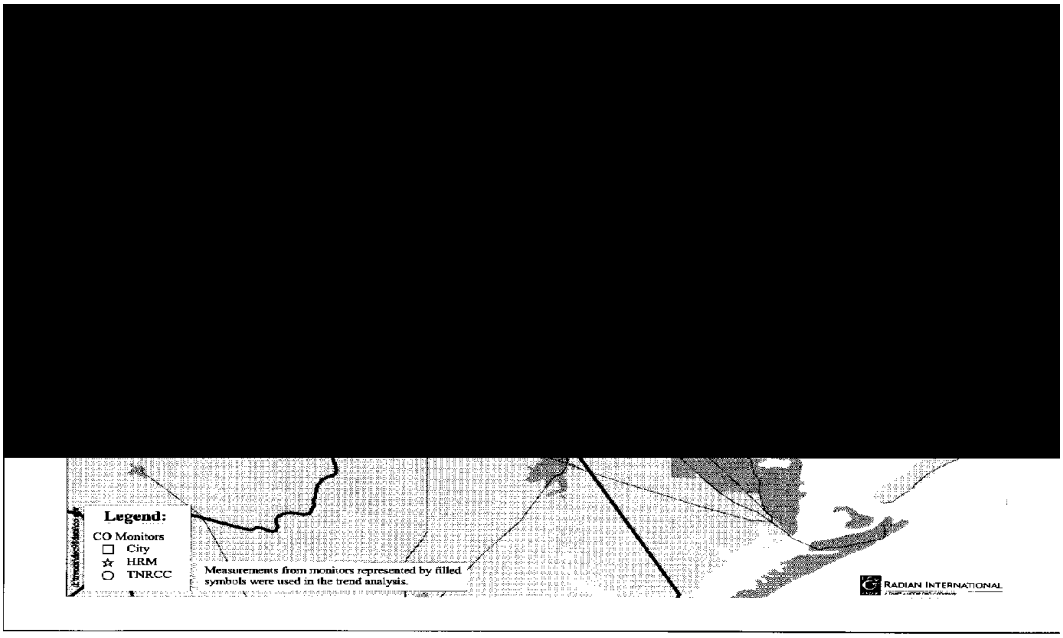

Figure A-2. Locations of CO Monitoring Stations in the Houston-Galveston Area

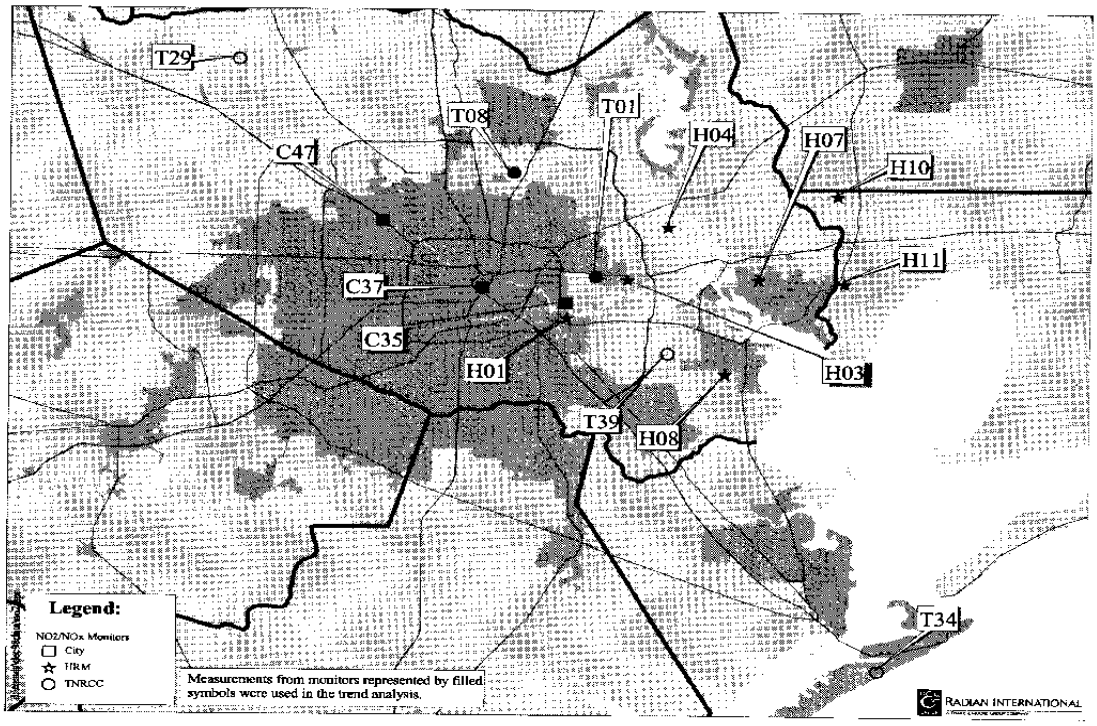

Figure A-3. Locations of NO_2/NO_x Monitoring Stations in the Houston-Galveston Area

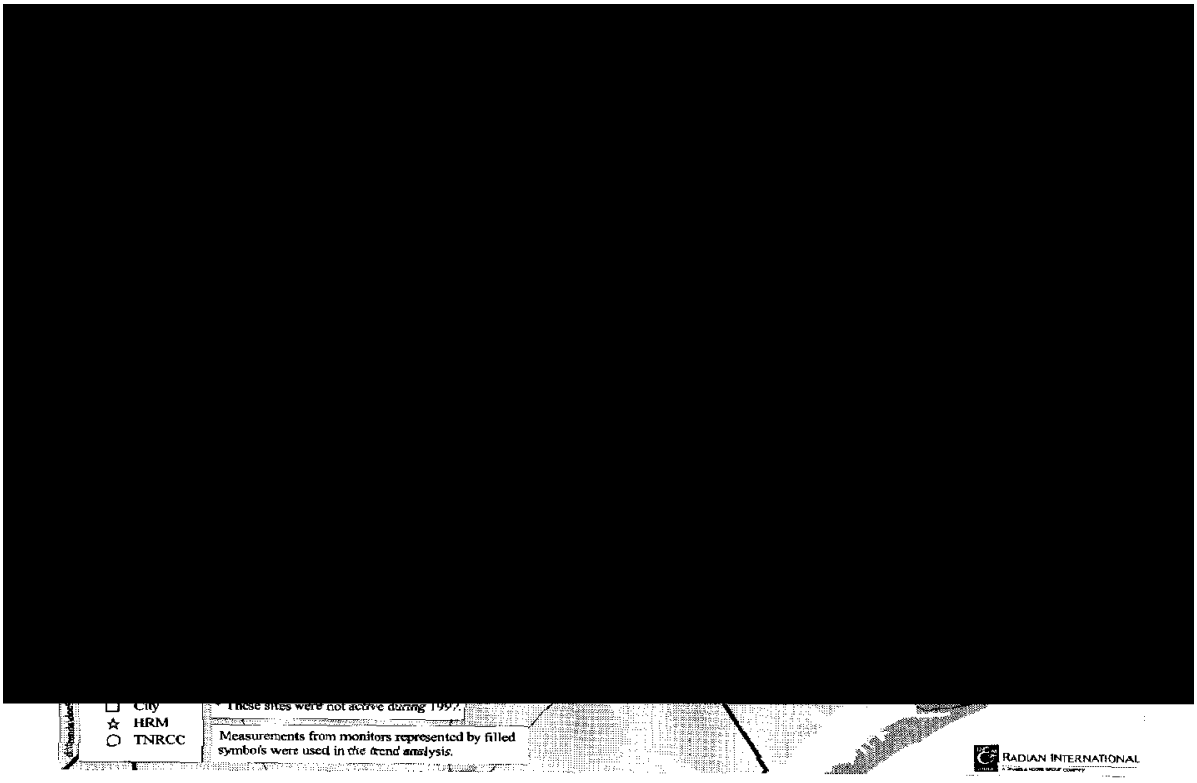

Figure A-4. Locations of PM_{10} Monitoring Stations in the Houston-Galveston Area

Figure A-6. Locations of VOC Monitoring Stations in the Houston-Galveston Area

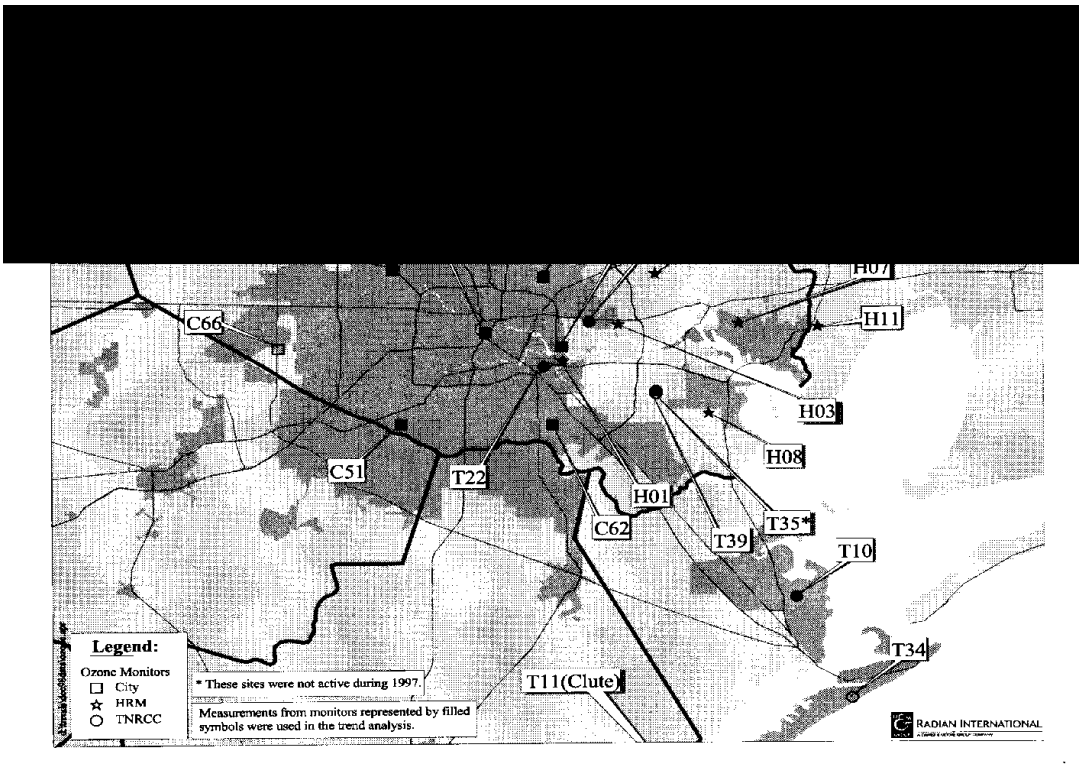

Figure A-7. Locations of O_3 Monitoring Stations in the Houston-Galveston Area

Summary of gas and particulate phase, air quality measurements made by the Houston Regional Monitoring (HRM) Network and the Cities of Houston and Galveston

City/Location	Site ID	SO_2	CO	NO/NO_x	PM_{10}	O_3	VOC
HRM Sites in Houston							
Pasadena/Central Street	HRM 1	X	X	X	X	X	X
Houston/Haden Road	HRM 3	X	X	X	X	X	X
Channelview/Miller Rd.	HRM 4	X	X	X		X	X
Baytown/West Baker	HRM 7	X	X	X	X	X	X
LaPorte/Fairmont Parkway	HRM 8	X	X	X		X	X
Mont Belvieu/Hatcherville Rd	HRM 10	X	X	X		X	X
Baytown/West Bay Rd	HRM 11	X	X	X		X	X
City of Houston Sites							
Galina Park/Clinton Dr.	C35	X	X	X	X	X	X
Houston/Crawford at Polk	C37		X	X	X	X	
Houston/Bingle	C45				X		
Houston/North Wayside	C46	X				X	
Houston/Lang	C47		X	X		X	
Houston/Croquet	C51	X				X	
Houston/Monroe	C62	X			X	X	
Houston/Highway 6	C66				X	X	
City of Galveston Sites							
Texas City/Texas Ave.	C04				X		
Texas City/5th Ave.	C53				X		

Site 3 of the Houston Regional Monitoring Network is of special interest to the program. At this site, the following measurements will be made (as described in the supporting letter attached to this proposal)

Oxides of nitrogen (NO, NO_2, NO_x)
Ozone
Sulfur dioxide
Carbon Monoxide
PM_{10} (every sixth day)
$PM_{2.5}$ (Continuous TEOM)
$PM_{2.5}$ (24 hour integrated FRM every 6th day)
Ambient carbon particulate, elemental and organic (Continuous 3 hour averaging period)
Hydrogen sulfide
Semi-continuous ambient sulfate (Aerosol Dynamics instrument)
Speciated VOC (24 hour integrated measurement every 6th day)
Wind speed, wind gust, wind direction, wind direction variance (measured at 10 m)
Net radiation
Precipitation

Description of airborne instrumentation that will be used in the Texas Air Quality Study

Aircraft Instrument Package for the DOE Grumman G-1

Parameter	*Time Resolution*	*Method*	*Det. Limit*
Ozone (O_3)	10 s	UV Absorption	25 ppb
Carbon Monoxide (CO)	20 s	NDIR	20 - 25 ppb
Fast CO (FCO)	5 s	VUV Resonance	5 ppb
Sulfur Dioxide (SO_2)	2 s	UV Pulsed Fluorescence	200-300 ppt
Nitric Oxide (NO)	< 10 s	NO/O_3 Chemiluminescence	20 ppt
Nitrogen Dioxide (NO2)	< 10 s	Photolysis, NO/O_3 Chem	50 ppt
Nitrogen Dioxide (optional)	< 10 s	Luminol Chemiluminescence	0.015 ppbv
Total Nitrogen Oxides (NO_y)	< 10 s	Mo Converter, NO/O_3 Chem	300-400 ppt
PAN	1 s / every 7 min	Cyrogenic GC	15 ppt
CH_2O (optional)	Continuous (1min delay)	Fluorescence	100 ppt
PAN	4 s	Tandem Mass Spectrometry	400 ppt
HNO_2	4 s	Tandem Mass Spectrometry	400 ppt
HNO_3	4 s	Tandem Mass Spectrometry	400 ppt
NH_3 (optional)	4 s	Tandem Mass Spectrometry	~2 ppb
Formic/Acid acids (optional)	4 s	Tandem Mass Spectrometry	100 ppt
Canister VOCs		Canister Sampling, GC/FID	0.1 ppbv
bscat	1 s	Nephelometer	0-103/Mm
Aerosol size distribution	1 s	PCASP	(0.17 - 3 µm)
Aerosol size distribution	1 s	FSSP	(2 - 47 µm)
Particle Number	1 s	CNC (two)	(> 7 nm, > 3 nm)
UV Radiation	1 s	Eppley Pyranometer	295 - 385 nm
Short-wave Irradiance	1 s	Eppley PSP	(285 - 2800 nm)
Long-wave Irradiance	1 s	Eppley PIR	(4 - 50 microns)
Water Vapor (H_2O)	1 s	Lyman Alpha Absorption	±0.1 g m-3(est.)
Air Temperature	1 s	Platinum Resistance	±0.5 ºC
Dewpoint/Frostpoint	1 s	Chilled Mirror	D.P. ±0.2 ºC, F.P. ±0.4 ºC
Wind Components (u-,v-,w-)	1 s	Gust Probe	< 0.5 m s^{-1}
Altitude	1 s	Barometric	< 1 mb
Position	1 s	GPS	< 3 m

Lockheed WP-3D Orion: NOAA Aircraft Operations Center / NOAA Aeronomy Laboratory
Endurance: 10 hrs; Ceiling: 7.6 km; Payload: >2700 kg; Research Speed: 100-150 m/s

Aircraft Instrument Package for the NOAA WP-3D Orion

Parameter	*Time Resolution*	*Method*	*Det. Limit*
Ozone (O_3)	10 s	UV Absorption	1 ppb
Fast O3 (FO_3)	1 s	NO/O_3 Chemiluminescence	0.2 ppb
Fast CO (FCO)	1 s	VUV Resonance	25 ppb
Carbon Dioxide (CO_2)	< 1 s	NDIR	0.2 ppm
Sulfur Dioxide (SO_2)	2 s	UV Pulsed Fluorescence	1 ppb
Nitric Oxide (NO)	1 s	NO/O_3 Chemiluminescence	30 ppt
Nitrogen Dioxide (NO_2)	3 s	Photolysis, NO/O_3 Chem	100 ppt
Total Nitrogen Oxides (NO_y)	1 s	Au Converter, NO/O_3 Chem	50 ppt
PAN	1 s / every 6 min	Dir. Injection, GC/ECD	< 5 ppt
PPN	1 s / every 6 min	Dir. Injection, GC/ECD	< 5 ppt
MPAN	1 s /every 6 min	Dir. Injection, GC/ECD	< 5 ppt
Nitric Acid (HNO_3)	1 s	C I Mass Spectrometry	10 ppt
NH_3	5 s	C I Mass Spectrometry	50 ppt
In-situ VOCs	1 min./every 15 min	Cryo Collection, GC/FID	< 10 ppt
Canister VOCs	< 1 min	Canister Sampling, GC/MS	< 10 ppt
CH_2O		Liquid Chromatography	
Peroxides (incl. H_2O_2)	1 min	Dual Enzymatic / Fluorimeter	30 ppt
Aerosol size distribution	1 s	NMASS	5 - 90 nm
Aerosol size distribution	1 s	ERAST	70 - 1000 nm
Total Radiation	1 s	Eppley Pyranometers – Zenith & Nadir	0.28 - 2.8 µ
UV Radiation	~10 s	Eppley Pyranometers – Zenith & Nadir	295 - 480 nm
Visible Radiation		Visible Absorption Spectrometer	420 - 700 nm
Water Vapor (H_2O)	1 s	Lyman Alpha Absorption	
Air Temperature	1 s	Platinum Thermistor	
Dewpoint/Frostpoint	< 3 s	Chilled Mirror	
Wind Speed	1 s	Derived from INE	
Wind Direction	1 s	Derived from INE	
Altitude	1 s	Barometric	
Position	1 s	GPS, INE	
Air Speed	1 s	Barometric	
Biometer		3-wavelength IR Absorption	
Atmospheric Reflectivity		C & X Band Radars	

Aircraft Instrument Package for the NOAA deHavilland Caribou

Parameter	*Time Resolution*	*Vertical Resolution*	*Method*	*Det. Limit*
Ozone	3 – 8 s	90 m	DIAL Lidar	4 - 10 ppb
Aerosol Backscatter	3 – 8 s	15 m	DIAL Lidar	5×10^{-6} $m^{-1}sr^{-1}$
Surface Temperature	1 s	NA	IR Radiometer	0.2 ºC

Summary of exposure study to be conducted in the Houston region, in collaboration with the proposed Supersite

EOHSI Personal Exposure Study (Abstract)

(C.P. Weisel, M. Morandi, T.H. Stock, B. Turpin, J. Zhang, S. Colome, D.M. Spektor)

The major objectives of the proposed research are a) to estimate the fraction that outdoor sources contribute to indoor and personal air concentrations of volatile organic compounds (VOCs), aldehydes and respirable particulate matter (PM2.5) in three distinct major urban centers in CA, TX, and NJ, and b) to estimate the exposures to populations living in the three urban centers from outdoor air toxic emissions based on residential air exchange rates and the measured relationship between outdoor air concentrations and both the indoor and personal air concentrations. The major hypotheses to be tested are that 1) in residences immediately adjacent to outdoor sources a measurable and significant proportion of the personal exposures, breath concentrations and indoor air concentrations to selected VOCs, aldehydes and PM2.5 are contributed by outdoor point and area sources under appropriate meteorological conditions, and that 2) residential air exchange rate is a major determinant of the influence of outdoor air concentration on indoor air and personal exposure; therefore the influence of outdoor air on indoor and personal air can be predicted for a large segment of the population within the proposed study areas from outdoor air concentrations and air exchange rates.

See: http://www.sph.uth.tmc.edu/www/ctr/leland/research.htm

Airborne Particulate Matter-Induced Lung Inflammation

EPA Grant Number: R826782
Title: Airborne Particulate Matter-Induced Lung Inflammation
Investigators: Andrij Holian, Maria T. Morandi, Edwin Parsley
Institution: University of Texas Houston Health Science Center
EPA Project Officer: Deran Pashayan
Project Period: October 1, 1998 - September 30, 2001
Project Amount: $674,288
Research Category: Health Effects and Exposures to Particulate Matter and Associated Air Pollutants

Description

Objectives/Hypotheses: Recent epidemiological studies have reported a statistically significant association between short-term increases in airborne respirable particulate matter (PM) and increased mortality and morbidity from respiratory and cardiovascular disease. Although toxic effects of airborne PM have been demonstrated with a variety of animal models and, to a more limited extent, with human subjects, the mechanism(s) that would explain the reported associations between exposure to PM and adverse health effects remains to be elucidated. Given the results of the epidemiology studies and some of the toxicology data, this mechanism(s) probably involves exacerbation of pre-existing cardiovascular and pulmonary chronic diseases. The very large number of individuals exposed to respirable PM and the lack of an accepted mechanistic hypothesis to explain the reported adverse health effects emphasizes the importance of the current proposed study. We propose that one of the important targets of PM-induced inflammation is the alveolar macrophage (AM). The purpose of this study is to test the hypothesis that fine PM (PM2.5) induce apoptosis of what is termed an immune suppresser population of AM that allows the remaining immune active AM population to more easily activate T helper cells resulting in activation of cytokine cascades and development of lung inflammation. We further propose that these effects would be more pronounced in individuals with chronic lung disease.

Approach: There are four major goals of this research. Goal 1: To characterize PM2.5-induced apoptosis and phenotype shifts in human AM in vitro and AM apoptosis and T helper cell activation in murine models in vivo. Goal 2: To characterize the influence of age in murine models on the bioactivity of PM2.5. Goal 3: To characterize the effects of PM2.5 on AM apoptosis and phenotype shifts in human AM isolated from patients with chronic lung disease. Goal 4: To characterize the bioactive chemical components of PM2.5 that affect apoptosis and phenotype shifts in human AM and T helper cell activation in murine models. To accomplish these goals PM2.5 will be collected on polyester membrane filters until a sufficient mass (is accumulated for groups of in vitro and in vivo studies. Sites for collection will be representative of industrial, motor vehicle, residential and background sources around Houston as well as sites in El Paso, TX. It is anticipated that approximately 30 separate filter pools will be collected during each year of the study. Particles will be collected in a 12 hr daytime (photochemically derived PM)

and nighttime formate at each site. PM collected on filters from same site sampling will be pooled for chemical analysis and biological studies. The PM will be analyzed for metals and organic components. Additional positive and negative control particles will include NIST particles 1648 and 1649, crystalline silica, titanium dioxide and ROFA particles (provided by the EPA). Studies in Goal 1 will be in vitro studies to assess the ability of the PM to cause apoptosis and necrosis of human AM, shifts in AM phenotypes, and stimulation of antigen presenting cell activity. Similar studies (apoptosis and antigen presenting activity) will be conducted in vivo by giving the particles intratracheally to C57Bl/6 and Balb/c mice and will also include measurements of Th1 and Th2 cytokines in the lung lavage fluid. Lung inflammation caused by the various PM will be assessed by differential analysis of lavaged AM and histological examination of perfusion fixed lung sections. Studies in Goal 2 will be in vivo studies with mice using optimal endpoints determined from Goal 1 and will examine very young mice and aged mice obtained from the National Institute of Aging. Studies in Goal 3 will utilize AM obtained from patients with chronic obstructive lung disease, asthma and chronic interstitial lung disease and focus on whether PM are more effective in causing apoptosis, shifts in macrophage phenotypes and stimulation of immune responses in cells from these sensitive subpopulations. Studies in Goal 4 will fractionate PM into water soluble and organic soluble components and test these on human AM in vitro and mice in vivo to determine where the biologically active component of PM is located.

Expected Results: We anticipate demonstrating that PM will cause a dose-dependent induction of apoptosis in human AM in vitro that will preferentially deplete the suppresser AM population. The remaining population of human AM will be able to more effectively stimulate T helper cells. Similar results are expected in vivo with murine models. Further, we anticipate that AM from young and old mice will be more susceptible to injury than cells from young adult mice. It is also expected that AM obtained from patients with chronic lung disease will demonstrate an even greater shifts of AM phenotypes than cells from healthy subjects. In addition, we propose that we will be able to correlate the potency of PM some component(s) or property of PM.

Improvement in Risk assessment or Risk Management: One of the major limitations in understanding the health risks associated with PM exposure (as defined by the NRC report on PM) is the lack of a well-defined toxic mechanism and uncertainty in what characteristics of PM are important in determining the toxic effects of PM. This project will address a testable hypothesis that could account for morbidity and mortality effects of PM and address the characteristics of PM that account for the toxic effects of PM. Therefore, the results from this study will be very useful for developing mechanistic-based risk assessment strategies for particulate exposure on human health.

Supplemental Keywords: ambient air, indoor air, sensitive populations, environmental chemistry, biology, analytical, Texas, TX, EPA Region 6