Table 1. Literature Review Matrix for Epidemiology

Status: PR = peer-reviewed; NPR = non-peer-reviewed; OG = on-going

		Texas (outside HGA)			Houston Galveston Area (HGA)		
		PR	NPR	OG	PR	NPR	OG
Health Effects	Cancer				Buffler et al, 1988; Walker et al, 1982; Marmor, 1978; MacDonald, 1976		
	Non-cancer						
	Respiratory	Samet et al, 2000; Legator et al, 2001, 1998; Winchester, 1989			Samet et al, 2000; Kim et al, 1996	Gehan, 1988; Stock et al, 1988; Selwyn et al, 1985; Holguin, 1985	Hanania; Delclos et al.; Macias
	Cardiovascular	Samet et al, 2000			Samet et al, 2000; Morris et al, 1995		
	Immunological						
	Reproductive	Ihrig et al, 1998					
	Developmental						
	Symptoms, non-specific						
Exposure Factors	Local air pollution sources						
	Traffic						
	Industrial	Ihrig et al, 1998; Legator et al, 2001, 1998					
	Dry cleaners						
	Other/general					Smith et al, 1998; MacDonald, 1976	Morandi
	Personal exposure				Weisel, 2002; Naumova et al, 2002ab; Morandi et al, 1988	Gehan, 1988; Stock et al, 1988; Holguin et al, 1985	Weisel et al (RIOPA & NHANES)
	Housing characteristics						
	HVAC					Langenstein, 1997; Hail, 1991	Weisel et al (RIOPA)
	Indoor sources *)				Weisel, 2002; Naumova et al, 2002ab; Stock et al, 1986; Kim & Stock, 1986		Weisel et al (RIOPA)
	Other						
	Individual activities						
	Smoking *)						
	Hobbies					Hopkins et al, 1998; Selwyn et al, 1985	
	Occupation *)						
	Other						
	Time activity pattern						
	Indoor/outdoor time					Rifai et al, 1999; Stock & Morandi, 1988	
	Modes of transport				Long et al, 2002		
	Other						
Other	Temperature						
	Humidity						
	Socioeconomic/race/cultural					Harper et al, 2001; Gehan et al, 1989	
	Susceptibility						

*) In relation to outdoor air pollution health effects only

Table 2. Summaries of recent reviews on air pollution health effects (2 of 18)

Reference	Health End Points	Pollutants	Affected Population	Summary
Finlayson-Pitts and Pitts, 1997	Mutagenicity and other	VOCs, NO_x, ozone, PAHs, and PM	General	Reactive intermediates in the oxidation of VOCs and NO_x, i.e. OH^-, NO_3^-, and ozone, play a central role in the formation and fate of airborne toxic chemicals, PAHs, and fine particles. The PAHs, in particular, are mutagenic substances that exists as volatile, semivolatile, and particulate pollutants and formed as a result of incomplete combustion. The PAHs contribute to the mutagenicity in respirable particles found in diverse regions of the world. Whether these mutagens are also human carcinogens remains a key question. Particles have also been associated with premature mortality through epidemiological studies. The chemical composition of the particulates does not seem to affect the association. The smaller particles (e.g. $PM_{2.5}$) are especially of concern as they reach the deep lung. The smallest particles tend to be those formed by combustion processes and by gas-to-particle conversions.
Brunekreef and Holgate, 2002	Respiratory / Cardiovascular	PM_{10} and other	General	Ozone and PM exposure has been associated with respiratory and cardiovascular mortality and morbidity. Short-term time-series studies have demonstrated the association between PM_{10} and mortality, asthma, COPD, and CVD. A European study found traffic-related air pollution to be associated with mortality, but another large-scale US study did not find association between non-particulate gaseous pollutants with mortality. Long-term cohort studies in Europe, US, and Canada also demonstrated association between exposure to PM_{10}, NO_2, and SO_2 with lung function and symptoms of bronchitis, but not asthma. Early time-series studies have been criticized for their approach and methodology, however recent sophicated re-analysis by independent researchers have largely confirmed their results on the association of air pollution with respiratory and cardiovascular diseases.
Leikauf, 2002	Respiratory / Immunology	SO_2, NO_2, O_3, PM, HAPs	Persons with asthma	Compared to the general population, persons with asthma are susceptible to lower doses of inhaled irritants such as SO_2, NO_2, acidic sulfates, ozone, and DEPs. Interractions between the irritants and inhaled antigens may contribute to asthma exacerbations. In epidemiological studies, $PM_{2.5}$ and sulfate are most often implicated. Many of the HAPs are also respiratory irritants which may uncover asthma (possibly among susceptible individuals). HAPs constitute the toxic ingredients of fine particulates and tobacco smoke, which are associated with asthma exacerbations, but whether ambient HAP exposures can induce asthma remains unclear.
Peden, 2002, 2001; Ring et al, 2001	Respiratory / Immunology	NO_x, O_3, PM, SO_2, VOCs	General	SO_2, NO_2, ozone, and PM has been shown to enhance allergic responses in animal studies. For human subjects, SO_2 and coarse particles are associated with viral and bacterial airway inflammation and infectious diseases, but not allergy. Allergic diseases are associated primarily with NO_x, VOCs, ozone, and fine particles. Airborne irritants, including ozone, PM, and endotoxin, are known to induce neutrophilic inflamation in the airways of healthy volunteers. These pollutants enhance response to allergen especially in persons with asthma.
Dixon, 2002	Respiratory / Developmental	Ozone and other	Children	Children are particularly vulnerable to ambient air pollution due to high respiration rates, more time for outdoor activities, rapid growth rate and critical periods of development. In particular, ozone has been shown to exacerbate asthma in children, increase school absentiesm especially for respiratory illness, and cause deficits in growth of lung function.
Carlisle and Sharp, 2001	Respiratory	CO, NO_x, O_3, PM_{10}, SO_2, VOCs	Exercising population	Athletes are at special risk of inhaling pollutants due to high respiration rates, high fraction of mouth inhalation during exercise, and increased pulmonary diffusion capacity. Specifically, CO exposure has been found to be detrimental to athlectic performance. Repetitive high-level exposure to the other pollutants may result in changes in pulmonary functions. Asthmatic athletes are particularly vulnerable. In general, athletes are recommended not to excercise near pollution sources (such as busy traffic) and during periods of high ozone.
Schwartz and Neas, 2000	Respiratory	PM	Children	Pulmonary toxicity of urban particles varied with size, with the greatest toxicity in those smaller than 1.7 μm. Sulfate particles, which are smaller than 1 μm, also have stronger effects. Recent longitudinal studies found that measures of fine particles ($PM_{2.5}$ and sulfates) are better predictors of acute asthma-related symptoms among schoolchildren.

Table 2. Summaries of recent reviews on air pollution health effects (3 of 18)

Reference	Health End Points	Pollutants	Affected Population	Summary
Pope, 2000	Respiratory / Cardiovascular	PM	General, susceptible population	Short-term time-series studies have observed the association of acute PM exposure with elevated respiratory and cardiovascular mortality as well as hospitalizations, emergency visits, and clinic/outpatient visits for respiratory and cardiovascular disease. Increase in the occurance of lower respiratory symptoms, cough, and exacerbation of asthma and decline in lung function are also associated with acute exposure. Epidemiologic evidence from population-based cross-sectional and cohort-based studies have also demonstrated the association between chronic PM exposure to increased risk of respiratory and cardiovascular mortality in adults and respiratory, sudden infant death syndrome, chronic cough, bronchities, chest illness (but not asthma), as well as small but often statistically significant declines in lung functions. Elderly, infants, children, and people with chronic cardiopulmonary disease, influenza, and asthma are particularly susceptible to acute exposure. Chronic exposure, however, affect all segments of the population.
Dockery, 2001	Cardiovascular	PM	General	The association between PM and mortality are real, robust, not confounded by weather, and independent of other pollutants. The relative risks are larger for respiratory than for cardiovascular deaths. However, the number of deaths caused by PM are much larger for cardiovascular causes. Furthermore, PM exposure is associated also with myocardial infarctions and ventricular fibrillation, increased heart rate, decreased heart rate variability, and increased cardia arrhythmias.
Sram, 1999; Perera et al, 1999	Reproductive	PM, SO_2, HAPs	Fetusses (prenatal) and parents	Fetus may be prone to genetic damage and clears toxicants less efficiently than adults. In particular, the developing nervous system is an extremely sensitive target. Only a few studies has been done on the effect of air pollution on reproductive outcomes. However, epidemiological studies have associated exposure to PM and SO_2 with neonatal and postneonatal mortality and low birth weight. Intrauterine growth retardation (IUGR) has been observed when PM_{10} concentration during the first month of pregnancy exceeds 40 mg/m^3, which is common in major cities and industrial areas. PAHs are particularly harmful to reproductive outcomes. Prenatal exposure to PAHs has been shown to increase PAH-DNA adducts and reduce head circumference of newborns, which is associated with cognitive development. Epidemiological studies have also associated prenatal PAH exposure with low birth weight and premature births. Furthermore, many chemicals may influence the number, morphology, and motility of sperm. PAHs have been shown to alter male reproductive functions.
Thurston and Ito, 2001	Mortality (non-specific)	Ozone	General	Many newer aggregate population time-series epedemiology studies have confirmed the significant association between ozone and premature mortality. Models that incorporate the nonlinearity and the humidity interaction of the termperature-health effect association yield greater and more statistically significant ozone-mortality effect estimates.

Table 3. Epidemiological studies on air pollution health effects in Texas (4 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
Weisel, 2002 (UMDNJ)	Exposure	Los Angeles, Houston, and Elizabeth, NJ	100 homes of non-smokers	VOCs	Residential	1999-2001	---	Ambient VOC emissions contribute to exposure of air toxics indoor as well as outdoor. However, indoor sources also contribute significantly to personal exposure, and must be accounted for in epidemiological studies.	PR
Naumova et al, 2002a (Rutgers)	Exposure	Los Angeles, Houston, and Elizabeth, NJ	100 homes of non-smokers	PM (PAHs)	Residential	1999-2001	---	Residential indoor concentration of high molecular weight (5-7 ring) PAHs are dominated by outdoor sources, while indoor sources have more significant contribution to the low MW PAHs.	PR
Naumova et al, 2002b (Rutgers)	Exposure	Los Angeles, Houston, and Elizabeth, NJ	100 homes of non-smokers	PM (PAHs)	Residential	1999-2001	---	An assessment of gas-particle distribution of polycyclic aromatic hydrocarbons in coupled outdoor/indoor atmospheres.	PR
Long et al, 2002 (TRJ Environ.)	Exposure	Houston	Commuters (n = 758 cars)	---	In vehicle	2 days (mid-Sep 2000)	---	The frequency of open windows in motor vehicles are weakly affected by the time of day, vehicle type, vehicle color, vehicle speed, and heat index (HI). In particular, open windows occur more frequently when vehicle speed is less than 50 mph and HI is greater than 99.9 ^{o}F, and when the vehicle is a minivan or passenger van.	PR
Levy et al, 2001 (Harvard)	Meta-analysis, economic valuation	Houston	Houston residents	Ozone	AC and other personal exposure factors		Premature mortality, hospital admissions, and restricted activity days	Due to high level of air conditioning, Houston has lower exposure (and thus lower dose-response function) compared to other cities where comprehensive epidemiologic studies have been performed. Based on these considerations, the annual monetary value of health benefit from reducing ozone in Houston was estimated to be approximately $10 per person per $\mu g/m^3$ reduced.	PR

Table 3. Epidemiological studies on air pollution health effects in Texas (5 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
Legator et al, 2001 (UTMB)	Case-control	Odessa, TX and Puni, HI	Exposed residents (n = 223) and reference (n = 170)	H_2S	Industrial and volcanic sources		Multi symptoms	Self-reported symptoms of adverse health effects in two communities impacted by low-level H_2S were compared with those in three reference communities. Symptoms with the highest odd ratios are in the categories of central nervous system (12.7), respiratory (11.9), and blood (8.1).	PR
Harper et al, 2001	Ecological	Houston	Low-income minority residents		Socioeconomic disparity		Asthma	Preliminary study showed that the prevalence of childhood asthma is exceedingly high in South Park, a low-income minority community in Houston. The area has high levels of air pollution due to proximity to freeways, a concrete crushing plant, and petrochemical plants.	NPR
Samet et al, 2000 (Johns Hopkins & Harvard)	Time series	Largest U.S. cities, incl. D/FW, Houston, and San Antonio	Urban population (elderly only for morbidity)	PM_{10}	Non-specific	10 years (1985-1994)	Respiratory and cardiovascular deaths (county records); Pneumonia and cardivascular hospital admissions (Medicare)	Analyses for both 20 and 90 largest U.S. cities showed an average of 0.5% increase in overall mortality per 10 $\mu g/m^3$ increase in PM_{10}, primarily due to cardiorespiratory deaths. Among the 90 cities, the largest effect was in the Northeast. In contrast, Dallas/Ft. Worth and Houston were among those with the smallest effects. Other pollutants did not have significant effect on the PM_{10}-mortality relation. Medicare hospital admissions in a subset of 14 of the 90 largest cities (none in Texas) showed approx. 1% increase for cardiovascular and 2% increase for pneunomia per 10 $\mu g/m^3$ increase in PM_{10}.	PR

Table 3. Epidemiological studies on air pollution health effects in Texas (6 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
Lurmann et al, 1999 (Sonoma Tech)	Meta-analysis, economic valuation	Houston	Houston population	Ozone, PM, HAPs	Various personal exposure factors	2007 (projected)	Various respiratory problems, non-specific symptoms, restricted activity days	Achieving compliance with the 1-hr ozone and $PM_{2.5}$ NAAQS in 2007 will result in approximately $3 billion of economic benefits of reduced exposure in Houston. The economic benefit is dominated by that of fine particles, as they result in more severe health effects.	PR
Rifai et al, 1999 (UH)	Exposure	Houston	Children	---	Time activity pattern		---	Children 0-4 years of age in Houston spend more time outdoor compared to National Human Time Activity pattern Survey (NHAPS) data used in the Sonoma study, resulting in approximately 10% overall increase in exposure hours.	NPR
Ihrig et al, 1998 (TAMU)	Case-control	Central Texas	Hospital births (119 cases, 267 controls)	Arsenic	Industrial proximity (agri industry)	10 years (1983-1993)	Stillbirths (hospital records)	A hospital-based case-control study of stillbirths in a community exposed to a facility producing arsenic-based agricultural products showed increased cases of stillbirth with the prevalence odds ratio of 8.4 for Hispanics in the high-exposure group.	PR
Legator et al, 1998 (UTMB)	Ecological	Midlothian, TX	Impacted community residents (n = 58), and reference (n = 54)	PM, SO_2	Industrial proximity (cement kilns)	1996-1997	Respiratory symptoms	Significant elevations in self-reported respiratory symptoms in the study community in comparison to a reference community. Limitation: small sample size, self reported.	PR
Hopkins et al, 1998 (Rice U.)	Exposure modeling	Houston	Woman athletes	Ozone	Personal, athletic activities	6 weeks	---	Personal ozone exposure of women on the Rice University track team was measured for six weeks and compared with predictions from an ozone exposure model. The model accounts for time, space, and activity profiles and provide accurate estimates.	NPR

Table 3. Epidemiological studies on air pollution health effects in Texas (7 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
Smith et al, 1998 (TNRCC)	Exposure modeling	Houston	Houston residents	Ozone	Local sources		---	Several metrics based on spatial extent, temporal duration, and magnitude of ozone concentrations are established to measure the severity of ozone exposure to human population. A method for identifying potential 'hot spots' was presented.	NPR
Langenstein, 1997 (UTH-HSC)	Exposure	Houston	4 Houston homes	CO, CO_2, ozone, VOCs	Ventilation, AC	2 x 10 hrs	---	Neither hour of day nor air-conditioning usage was found to significantly affect the indoor pollutant levels, although air exchange rates are lower with air-conditioning use.	NPR
Kim et al, 1996.	Time series	Houston	Adults and children	SO_2	Non-specific	3 years (1990-1993)	Invasive pneumococcal disease	The occurrence of pneumococcal disease is significantly correlated with air pollution as measured by SO_2 levels.	PR
Morris et al, 1995 (Med Co of Wisconsin)	Time series	Seven major U.S. cities, incl. Houston	Urban population, elderly	CO and other criteria pollutants	Non-specific	4 years (1986-1989)	Congestive heart failure (hospitalization, Medicare)	The study showed clear and consistent correlation between Medicare hospital admissions for congestive heart failure and ambient CO level. Houston demonstrated one of the weakest associations compared to the other cities (RR = 1.11), but the strongest association at a lag of 3 days (RR = 4.1). No association was found with the other criteria pollutants.	PR
Hail, 1991 (UTH-HSC)	Exposure	Houston	A 10-story building	Ozone	Ventilation	July - Aug 1991	---	Very little outdoor air was entering the indoor environment in a Houston-area academic building. This resulted in very low risk of ozone exposure, but raised concerns on indoor pollution sources. The building's ventilation practice may be commonplace in the region.	NPR

Table 3. Epidemiological studies on air pollution health effects in Texas (8 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
Winchester, 1989 (Flo. St. U.)	Ecological	Southeastern and Western Plain states, incl. Texas	City residents, white	Acid PM	Non-specific	1979-1981	COPD mortality	COPD mortality rates exhibit a geographic pattern which may be related to the regional atmospheric conditions. Metropolitan areas in northern Texas (San Angelo, Amarillo, Lubbock) have among the highest COPD mortality rates in the U.S. Dusts generated by strong wind and dry soils coated or reacted with urban pollutants may be the cause.	PR
Gehan et al, 1989 (UTH-HSC)	Panel	Houston	Asthmatics	Ozone			Asthma	Younger, black, and non-smoking subjects experienced large decrements in peak expiratory flow (PEF) with increasing ozone exposure compared to other demographic groups.	NPR
Buffler et al, 1988 (UTH-HSC)	Ecological	Houston	Harris County residents, white male, aged 30-79 yrs	TSP	Non-specific	3 years (1979-1981)	Lung cancer deaths (county records)	Although lung cancer mortality rates are correlated to air pollution indices in Harris County, the correlation is not statistically significant when demographic factors are considered. Lung cancer mortality is strongly correlated with income or poverty, which in turn correlate with cigarette smoking rate. Limitation: Effects of cigarette smoking overwhelmed the analysis.	PR
Stock et al, 1988 (UTH-HSC)	Reanalysis	Houston	Adult asthmatics (n=42)	Ozone	Personal (indoor, outdoor, in vehicle)	6 months (May-Oct 1981)	Asthmatic episodes	Reanalysis of Holguin et al (1985) shows significant association between exposures to both increased ozone and decreased temperature on the probability of an asthmatic attack.	NPR

Table 3. Epidemiological studies on air pollution health effects in Texas (9 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
Gehan, 1988 (UTH-HSC)	Panel	Houston	Asthmatic children and adolescents (n=39)	Ozone	Personal activities, demography	6 months (May-Oct 1988)	Asthma symptoms and pulmonary peak flow	Daily pulmonary peak flow measurements corrleated with maximum hourly ozone levels at neighborhood monitoring sites (r = -0.018). The response to ozone exposure was particulary strong in the subjects who were female, black, under 12 yrs old, or unexposed to environmental tobacco smoke in the home.	NPR
Morandi et al, 1988 (UTH-HSC)	Exposure	Houston		PM	Personal		---	A comparative study of respirable particulate microenvironmental concentrations and personal exposures.	PR
Stock and Morandi, 1988 (UTH-HSC)	Exposure	Houston		---	Time activity pattern		---	A comparative evaluation of self-reported and independently observed activity patterns in an air pollution health effects study	NPR
Contant et al, 1987 (UTH-HSC)	Exposure	Houston	12 homes in 2 Houston neighborhoods	Ozone, NO_2	Residential	6 months (May-Oct 1981)	---	There is a large difference between indoor and outdoor ozone. Accurate ozone exposure estimates must account for microenvironments with no AC, time spent in different microenvironments, and hour-specific personal location of study subjects.	PR
Kim & Stock, 1986 (UTH-SPH)	Exposure	Houston	12 homes in 2 Houston neighborhoods	PM	Residential	6 months (May-Oct 1981)	---	Most indoor aerosols appears to be affected by infiltration from otudor air as well as indoor generation, with the most important determinant being cigarette smoking.	PR
Stock & Mendez, 1985 (UTH-HSC)	Exposure	Houston	78 homes	Formaldehyde	Residential	Summer 1980	---	Formaldehyde concentration is much greater indoor than outdoor. The levels depend on both age of dwelling and structural classifaction of the residence.	PR

Table 3. Epidemiological studies on air pollution health effects in Texas (10 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
Stock et al, 1985 (UTH-HSC)	Exposure	Houston	2 Houston neighborhoods	O_3, NO_2, NO_x, SO_2, CO, PM	Indoor/ outdoor	6 months (May-Oct 1981)	---	Indoor and outdoor pollutant levels were measured by a monitoring van in two Houston-area neighborhoods. Ozone was the only measured pollutant that exceeded the NAAQS during the study period.	PR
Selwyn et al, 1985 (UTH-HSC)	Panel	Houston	Vigorously exercising adults (n = 24)	Ozone	Outdoor (during exercise)	6 months (May-Oct 1981)	Pulmonary functions (pre and post exercise)	Outdoor aerobic excercises during periods of increased ambient ozone concentration up to 0.135 ppm produces at least a short-term decrease in pulmonary function.	NPR
Holguin et al, 1985 (UTH-HSC)	Panel	Houston	Adult asthmatics (n=42)	Ozone	Personal (indoor, outdoor, in vehicle)	6 months (May-Oct 1981)	Asthmatic episodes	The risk of an asthma attack was positively and significantly correlated with ozone exposure. Limitation: small number of subjects.	NPR
Walker et al, 1982 (UTMB)	Ecological	Houston	---	PM (samples' mutagen-icity, via Ames test)	Various	1 year (1977-1978)	Lung cancer mortality	Mutagenicity of Houston air particulates is highly correlated with the geographic pattern of lung cancer mortality in Houston. The mutagenicity were assessed using Ames mutagen detection system for particulate mixtures periodically collected from seven sites in Houston. The geographic variation depends only on the particulates' mutagenicity, and not to smoking. Highest mutagenicity observed in industrial areas and areas of high traffic volume.	PR
Marmor, 1978	Ecological	Houston		Air pollutants (general)			Cancer	Air & industrial pollutants have demonstrable effects in increasing regional mortality from cancer of the respiratory tract.	PR

Table 3. Epidemiological studies on air pollution health effects in Texas (11 of 18)

Reference (Affiliation)	Study Type	Location(s)	Subjects/ Population	Pollutant (s)	Exposure Factor	Analysis Time Frame	Health Outcomes	Summary of findings	Status
MacDonald, 1976 (UT-MDACC)	Ecological	Houston	Houston residents, excluding Spanish-surnamed	Air pollutants (general)	Local sources	30 years (1940-1969)	Cancer, cardiovascular, stroke, and respiratory mortality (city records)	Environmental factors of exposure over time to air and industrial pollutants in Houston has demonstrable effect in increasing regional mortality from cancer of the respiratory tract and from heart disease. Respiratory mortality generally correlates with the number of industrial installations in an area.	PR

Table 4. On-going epidemiological/exposure assessment studies in Houston (12 of 18)

Researchers (Affiliation)	Location(s)	Pollutant(s)	Exposure Factor	Health Outcomes	Description
Hanania, N.A. (Baylor CoM)	Houston	Ozone		Asthma	The study examines the correlation between ozone level and emergency room visit.
Delclos G.L. et al. (UTH-HSC)	Houston	Oxygenated air toxics (total carbonyl concentration)		Asthma	Oxygenated Urban Air Toxics and Asthma Variability in Middle School Children: A Panel Study
Macias, C. (Baylor CoM)	Houston	Various		Asthma	Part of a multi-city study which examines risk factors for ER visits related to asthma. Approximately 10,000 children will be enrolled during the two-year restrospective and prospective enrollment period at four area hospitals that began January 1, 2002. Risk factors and pollution levels will be analyzed for the entire cohort.
Morandi, M.T. (UTH-HSC)	Houston	Air Toxics		---	Ambient Air Toxics in the Houston-Galveston Area with High and Low TRI Emissions - A Pilot Study of Temporal and Spatial Concentrations Using Passive Sampling Devices
Weisel, C.P. et al. (Rutgers, UT SPH)	Los Angeles, Houston, and Elizabeth, NJ	Residential exposures to VOCs and $PM_{2.5}$	Residential	---	The RIOPA study investigates residential indoor/outdoor exchange rates especially near outdoor pollutant sources.
Weisel C.P. et al. (Rutgers, UTH SPH, UDMNJ, Clayton Labs)	Los Angeles, Houston, and Elizabeth, NJ	Personal exposure to VOCs	Personal	---	The NHANES study is to characterize the distribution of exposures to selected volatile organic compounds (VOCs) for a subset of the U.S. population.

Table 5. Toxicological studies on air pollution health effects performed by Texas researchers (13 of 18)

Reference (Affiliation)	Health End Point	Pollutants	In Vitro/Vivo; Subjects	Summary of findings	Status
Hamilton et al, 2001 (UTH-HSC)	Respiratory/Immunological: lung inflamation	PM	In Vitro; Alveolar Macrophages	Silica and PM1648, a model urban particulate, demonstrated selective toxicity to suppressor macrophages.	PR
Chao et al, 2001 (UTH-HSC)	Respiratory/Immunological: lung inflamation	PM	Murine lung cells	Scavenger receptors are necessary for the caspase activation and subsequent apoptosis and neurosis caused by silica particulates.	PR
Li et al, 2000 (UTH-HSC)	Respiratory/Immunological: lung inflamation	Ozone	In Vivo; Mice	Heme oxygenase-1 (HO-1), an antioxidant enzyme, protects against ozone-induced pulmonary inflamation on mice. It may contribute to the development of cellular adaptation to chronic ozone exposure.	PR
Li et al, 1999 (UTH-HSC)	Respiratory/Immunological: lung inflamation	Acrolein	In Vitro; Alveolar Macrophages	Acrolein-inhibited endotoxin-induced NF-κB activation may be responsible for the inhibition of cytokine release and the induction of apoptosis in human alveolar macrophages.	PR
Haney et al, 1999 (UTH-HSC)	Respiratory/Molecular Genetic: lung inflamation	Ozone	In Vivo; Mice	DNA strand breaks observed in murine brochoalveolar lavage (BAL) cells exposed to ozone. The mice were exposed to 3 hrs of 0.25 ppm or 0.5 ppm ozone before the BAL cells were collected.	PR
Hamilton et al, 1998 (UTH-HSC)	Respiratory: lung inflamation	Ozone	In Vivo; Humans	Significant apoptosis of human lung cells observed after ozone exposure. The human subjects were exposed to 0.4 ppm ozone for 1 hr with exercise, and the cells were collected 6 hrs after exposure from the bronchoalveolar lavage.	PR
Hanania et al, 1998 (Baylor CoM, U Toronto)	Respiratory: asthma execarbation	Ozone	In Vivo; Humans	Human subjects exposed to a low level of ozone at 120 ppb showed no signifcant changes in airway allergen responsiveness.	PR
Holian et al, 1998 (UTH-HSC)	Respiratory/Immunological: lung inflamation	PM	In Vitro; Alveolar Macrophages	Urban particles and residual oil fly ash induced apoptosis in human alveolar macrophages in vitro.	PR
Iyer and Holian, 1997 (UTH-HSC)	Respiratory/Immunological: lung inflamation	PM	In Vitro; Alveolar Macrophages	Apoptosis of human alveolar macrophages induced by silica dusts may involve the activation of the interleukin-converting enzyme (ICE) family of proteases.	PR
Holian et al, 1997 (UTH-HSC)	Immunological: lung inflamation	PM	In Vivo; macrophages	Fibrogenic particulates such as chrysotile, crocidolite asbestos, and crystalline silica, shift the phenotypic ratio of human alveolar macrophages to a more inflammatory condition.	PR

Table 5. Toxicological studies on air pollution health effects performed by Texas researchers (14 of 18)

Reference (Affiliation)	Health End Point	Pollutants	In Vitro/Vivo; Subjects	Summary of findings	Status
Li et al, 1997 (UTH-HSC)	Immunological: lung macrophages	Acrolein	In Vitro; Lung cells	Acrolein caused dose-dependent cytotoxicity to alveolar macrophages as demonstrated by apoptosis and necrosis. Inhibition of cytokine release and cytotoxicity may in part be responsible for acrolein-induced immunosuppression of the lung.	PR
Kirichenko et al, 1996 (UTH-HSC)	Respiratory: lung inflamation	Ozone	In Vitro; Lung cells	HNE (4-hydroxy-2-nonenal), which is toxic to cells, was formed as a result of ozone exposure and may contribute to lung cell apoptosis observed after exposure.	PR
Hamilton et al, 1996a (UTH-HSC)	Respiratory/Molecular genetic; adduct formation.	Ozone	In Vitro; Lung cells	Ozone exposure results in HNE adduct formation in vitro, which might suggest a role of this process in the cellular toxic effects of ozone.	PR
Randerath et al, 1995 (Baylor CoM)	Molecular genetic; adduct formation	PM (Diesel Exhaust)	In Vivo; Rats	Postexposure levels of some adducts and the total level of adducts were higher in exposed animals.	PR
Hazbun et al, 1993 (UTH-HSC)	Respiratory: lung inflamation	Ozone	In Vivo; Humans	Ozone exposure results in increased concentration of substance P, PGF2α and C3a in the bronchoalveolar (BAL) fluid obtained from volunteers postexposure, which might suggest mechanism of action of inflammatory and toxic effects of Ozone.	PR
Fouke et al, 1990 (Sw Found. Biomed Res)	Respiratory; Lung compliance	Ozone	In Vivo; Baboons	Pulmonary resistance increased after ozone exposure of 0.5 ppm, but the effect was partially blocked with cromolyn pretreatment.	PR

Table 6. Air Quality Data Sources (15 of 18)

Data Type	Organization	Source	Parameters	Method	Descriptions	Limitations
Monitoring Data	TCEQ	Air monitoring data	Hourly monitoring data; surface meteorology.	Collected from monitoring stations.	Hourly monitoring data for criteria pollutants, $PM_{2.5}$, and to a more limited extent VOCs and HAPs. Surface meteorological conditions (temperature, wind, radiation, and precipitation) are also recorded. Data are reported to EPA's AQS database. http://www.tnrcc.state.tx.us/air/monops/	Monitoring location chosen by modeling of criteria pollutants maximum impact prediction. Averaging times chosen to coincide with regulations not health effects results.
	Houston Regional Monitoring (HRM)	Air monitoring data	Hourly monitoring data; surface meteorology.	Collected from monitoring stations.	The HRM network records ambient levels of criteria pollutants, $PM_{2.5}$, sulfate, and 150 VOCs at seven monitoring sites. A portion of the collected information are reported to TCEQ and EPA's AQS database. http://hrm.radian.com/	All data not generally publically available. Specific case by case requests considered.
	U.S. EPA	Air Quality System (AQS)	Hourly/daily monitoring data; surface meteorology.	Collected and reported by local and state agencies and EPA offices.	Formerly part of Aerometric Information Retrieval System (AIRS), AQS contains raw monitoring data for ambient air pollutants (criteria pollutants, their precursors, and HAPs) collected by the EPA and state/local agencies. In the Houston-Galveston area, data are obtained from monitoring stations maintained by the City of Houston, TCEQ, Harris County Pollution Control, and URS Corp.'s HRM. Data are typically collected hourly. http://www.epa.gov/ttn/airs/airsaqs/	In addition to the same limitation for the TCEQ monitoring data, this data does not contail all sites. Only sites with AIRS numbers are included. Some differences in QA/QC procedures in included data. Data collected for ambient monitoring purposes not for health effects evaluations.
Monitoring Data (cont'd)	U.S. EPA	Enhanced Ozone Monitoring (PAMS)	Hourly monitoring data on ozone, NOx, VOCs; surface meteorology.	Reports from monitoring stations.	In accordance with the 1990 Clean Air Act Amendments, EPA has required more extensive monitoring of ozone and its precursors in areas with persistently high ozone levels (mostly large metropolitan areas). In these areas, the States have established ambient air monitoring sites called Photochemical Assessment Monitoring Stations (PAMS) which collect and report detailed data for volatile organic compounds, nitrogen oxides, ozone and meteorological parameters http://www.epa.gov/oar/oaqps/pams/	Data collected not designed for use in health effects studies. Primary pupose was understanding atmospheric chemistry of ozone formation.
Emission Inventory	TCEQ	Point Source Air Emissions Inventory	Emmission sources; annual emmision estimates for criteria pollutants, particulates, VOCs, and HAPs.	Scientific or vendor estimates as reported by companies.	The point source emissions inventory is an annual survey of chemical plants, refineries, electric utility plants and other industrial sites that meet the reporting criteria in the TCEQ emissions inventory rule. Compositions of VOCs, particulates, and HAPs are also estimated. Average daily emissions of VOCs during ozone season must be estimated and reported. http://www.tnrcc.state.tx.us/air/aqp/psei.html	Most data is calculated or estimated. Little historical or monitored data. Several different methods used.

Table 6. Air Quality Data Sources (16 of 18)

Data Type	Organization	Source	Parameters	Method	Descriptions	Limitations
	U.S. EPA	National Emissions Inventory (NEI)	Emission sources; stationary and mobil emission data and estimates.	Collected and reported by local and state agencies and from the TRI.	The NEI database is an emission inventory that contains data on point, area, and mobile sources emissions of criteria air pollutants and their precursors (including VOCs), and HAPs. An emission inventory lists, by source, emissions of air pollutants in a specific area for a specific time. http://www.epa.gov/air/data/netdb.html	This is a database combined from several other databases (TRI, AAM, etc). The limitations of the NEI database mirror the same limitations as the component databases. Additionally, the mobile source emissions are based on earlier versions of EPA's Mobile modeling program.
Emission Inventory (cont'd)	U.S. EPA	Toxic Release Inventory (TRI)	Emission sources; stationary and mobil emission data and estimates.	Reported by companies.	The TRI is a publicly available EPA database that contains information on specific toxic chemical releases and other waste management activities reported annually by certain covered industry groups as well as federal facilities. http://www.epa.gov/tri/	Source specific but not location or temporal specific. Annual and hourly maxes based on calculations, estimates and some monitoring.
Compliance Data	U.S. EPA	Air Facility Subsystem (AFS)	Plant- and equipment-level compliance data; operating permit data.	Collected and reported by local and state agencies.	Formerly part of Aerometric Information Retrieval System (AIRS), AFS contains compliance and permit data for stationary sources of regulated air pollutants. http://www.epa.gov/Compliance/planning/data/air/afssystem.html	Plant and equpment level data for regulatory compliance purposes (permitting). Data based on various compliance and rule methods not actual emissions monitoring.
Air quality index	TCEQ	Air Quality Index			Provides the air quality index in the metro areas based on EPA's scale for measuring air quality http://www.tnrcc.state.tx.us/cgi-bin/monops/psi_rpt	System for comparing criteria pollutants to a standard scale for ease of public notice on daily air quality. Related only to NAAQS not specific for health effects.
Other	U of Texas	Texas Air Quality Study 2000 (TexAQS 2000)	Gaseous, particulate, and hazardous air pollutant profiles.	Ground stations and aircraft.	Involving six weeks of intensive sampling in August - September 2000, it includes study on ozone and chemical makeup of fine particulates in Houston, where they come from, and how they behave in the atmosphere. http://www.utexas.edu/research/ceer/texaqs/	Data collected not designed for use in health effects studies. Primary pupose was understanding atmospheric chemistry of ozone formation.

Table 8. Data Sources on Exposure and Lifestyle (17 of 18)

Data Type	Source	Geo. Scope	Parameters	Methodology	Potentially Useful Indices	Limitations
Personal exposure assessment (cohort)	Relationship Between Indoor, Outdoor, and Personal Air (RIOPA)	Houston, Los Angeles, and central New Jersey	Indoor/outdoor conc'ns of VOCs, aldehydes, and $PM_{2.5}$; residential Air Exchange Rate; in-vehicle exposure to aldehydes; regional and seasonal variation.	Personal air sampling monitors: 48-hour samples collected three months apart from 1/1999 to 6/2001; 100 person sample size in each city.	Personal exposure to VOCs, PAHs, $PM_{2.5}$, etc; indoor/outdoor ratios of air pollutants	The lack of a probability-weighted design limits the ability to extrapolate to larger populations than the sample itself.
Health and environ'l exposure profiles (cohort)	National Health and Nutrition Examination Survey (NHANES) http://www.cdc.gov/nchs/nhanes.htm	Nationwide (on-going VOC exposure measurements in Houston, Los Angeles, and Elizabeth, NJ)	Health conditions, health & dietary lifestyle, environmental exposure (recently added)	Continuous survey using self-reported questionnaires, health tests/biomarkers, personal air sampling monitors.	Personal exposure to VOCs and air toxics; tobacco smoking; occupational exposure.	Only recently being expanded to include measurements of exposure to VOCs and air toxics; potentially limited by the inability to make person or site specific correlations data due to the strict subject confidentiality guideline of NCHS, such that specific linkages to the Houston area may be limited.
Housing characteristics	US Census Bureau: American Housing Survey (AHS) http://www.census.gov/hhes/www/ahs.html	Nationwide for 47 selected metropolitan areas, including Houston.	Household characteristics, income, housing and neighborhood quality, housing costs, equipment and fuels, size of housing unit, and recent movers.	Self-reported questionnaires. National sample covering an ~55,000 houses. Metropolitan area data collected every 4 years, each covering 4,800 or more housing units.	Equipment and fuel for heating, cooling, and cooking; other housing characteristics.	Metropolitan data are collected every 4 years from the same housing units; last Houston data in 2002 (next in 2006).

Table 8. Data Sources on Exposure and Lifestyle (18 of 18)

Data Type	Source	Geo. Scope	Parameters	Methodology	Potentially Useful Indices	Limitations
Time Activity Pattern (cohort)	US EPA: Consolidated Human Activity Database (CHAD) http://www.epa.gov/chadnet1/index.htm	Nationwide	Time activity pattern; demographics, housing characteristics, location, smoking and health status.	Self-reported questionnaire data obtained from pre-existing human activity studies that were collected at city, state, and national levels.	Time activity pattern.	Limited Houston data (approximately 80 datapoints); does not measure change in lifestyle pattern over time (need continuous tracking) does not account for rate of response.
Modes of transport	U.S. Department of Transportation: National Household Travel Survey (NHTS) http://www.bts.gov/nhts/	Nationwide	Data on daily trips including means of transportation, how long the trip took, day of week and month, number of people on trip, etc. Also data on long distance travel (i.e., over 50 miles) during a four-week period.	Telephone questionnaires. One-year data from 4/2001to 5/2002 involved 25,721 households from national sample and 40,000 from state and local jurisdictions	Houston-based data on trips made by bicycling, walking, public transportation, and personal vehicle.	
Behavioral risk factors	Behavioral Risk Factor Surveillance System (BRFSS) http://www.cdc.gov/brfss/	Nationwide	State-level data on health-risk behavior of adult U.S. population.	Telephone questionnaires.	Tobacco smoking; exercise; etc.	Useful only if data can be tracked back to the city; details likely to be insufficient for health effects research.