Regional Epidemiology and the Medical Environment as it Pertains to Healthcare Quality and Outcomes Evaluation and Applicable Health Policy

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REGIONAL EPIDEMIOLOGY AND THE MEDICAL ENVIRONMENT AS IT PERTAINS TO HEALTHCARE QUALITY AND OUTCOMES EVALUATION AND APPLICABLE HEALTH POLICY

by

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SUMMARY

REGIONAL EPIDEMIOLOGY AND THE MEDICAL ENVIRONMENT AS IT PERTAINS TO HEALTHCARE QUALITY AND OUTCOMES EVALUATION AND APPLICABLE HEALTH POLICY

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While still evolving, healthcare evaluation in the United States is predominately analyzed via a model that examines the structures, processes, and outcomes of medical care. In order to provide a conceptual framework that better predicts health outcomes and more accurately reflects local healthcare, vested healthcare professionals can incorporate more relevant patient characteristics and the medical environment – which is here defined as the context in which the patient and healthcare system reside that has any health-related influences.

To better appreciate why medical evaluation should include local environmental factors, this thesis demonstrates how common disease outcomes are regionally different across populous counties in Texas. Epidemiologic evidence from the Texas Department of State Health Services indicates that Alzheimer's deaths, suicide rates, percent of preterm births, and rate of infant mortality significantly vary from county to county. Three medical environmental factors are then presented and discussed as examples typifying influences that warrant objective quantification because of their potential effect on health outcomes. Also presented is an illustrative case of how a specific county in Texas combined epidemiology, patient characteristics and the medical environment to evaluate healthcare and make appropriate health policy proposals for improvement.

This thesis concludes with a summary of findings, suggested policy proposals, limitations and future questions.

Disclosure: I have no financial or other conflicts of interest to make known.

Mistakes are my own.

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INTRODUCTION

Despite how current vernacular paints healthcare in the United States as a homogenous mixture of doctor and disease it is more accurately a vast collection of unique, localized concoctions - each representing a dynamic yet local healthcare phenomenon. While many of the critical elements that comprise any particular healthcare system are preserved, the quantity and quality of each component is variable. In other words, healthcare is a reflection of a distinct but regional milieu – principally characterized by patients, the setting and manner in which care is delivered, and the surrounding environment that may shape or at least influence the practice of medicine.

This treatise will therefore address how healthcare has been evaluated in the past and then introduce the concept of incorporating patient characteristics and the medical environment into the framework of healthcare evaluation. Additionally, in order to grasp why the provision of health services is necessarily variable from one place to another, we will define epidemiology and examine three different health outcomes across counties in Texas. Next, this treatise will illustrate the interplay between medicine and the local environment by acquainting the reader with three potential examples of health-related environmental factors. Then we'll provide a real world example of how epidemiology, patient characteristics and the medical environment can be used to evaluate and improve healthcare. Finally, this thesis will conclude with a brief summary of our findings, a few policy proposals, the limitations of this treatise and future questions to address.

EVALUATING HEALTHCARE

BRIEF HISTORY

It is reasonable to assume from the primordial Hippocratic Oath, that most physicians, whether bound to Greek deity[1] or not[2], have long intended to provide the most effective, desirable form of care to their patients. Translated directly from its original text, the Oath explicitly voices the following physician's vow: "I will use those [therapeutic] regimens which will benefit my patients according to my greatest ability and judgment, and I will do no harm or injustice to them.[1]" For millennia, physicians had treated patients in the best way they knew how, but the methodology and especially the outcomes of care remained a practice of obscurantism.

It was not until the 1900s – when Dr. Ernest Codman devoted himself to the "end result idea," a notion that would inveterately consume the remainder of his career[3] – that the evaluation of medical care outcomes became conceptualized. Viewed retrospectively in a world now swirling with multiple-criteria decision analysis and Lean Six Sigma, Codman's idea could perhaps sound simplistic (see note below), but in his own words it was "merely the common-sense notion that every hospital should follow *every* patient it treats, long enough to determine whether or not the treatment has been successful, and then to inquire 'if not, why not?' with a view to preventing a similar failure in the future."

Note: Don Berwick has written that, "It is, of course, a special form of arrogance to imply that people of an earlier time were somehow less complex than we of today. Multi-attribute utility did not arrive with the term; it was here all along. Cavemen had it." (Berwick, DM. E.A. Codman and the Rhetoric of Battle: A Commentary. The Milbank Quarterly, Vol. 67, No.2 (1989), pp. 262-267.)

He further posited of patients in his day that "[t]hey suppose that of course somebody is looking into this important matter. They do not realize that the responsibility is not fixed upon any person or department.[3]" Sadly it would be nearly a century before "Chief Quality Officer" would be introduced into clinical practice. Like medical reformers before him, Codman's legacy would only be appreciated in the aftermath[9].

FRAMEWORK OF EVALUATION

Since Codman, several academic prominenti have laboriously advanced the evaluation of the quality of health care. Hooker[4], Kohl[5], Lembcke[6], Lehman[7], Dubois and Brook[8], Wennberg[9-10], Berwick[11-12], and countless others have legitimized and refined healthcare evaluation, but none come to mind who have done more than Avedis Donabedian. In 1966, he impactfully introduced a model that would become the dominant paradigm for quality evaluation[13-14] and coined the popular concepts of "structure", "process", and "outcome" that have allowed us to codify healthcare in a consistent, understandable manner (see Figure 1 below). "Structure" is commonly accepted to be the context in which medical care is delivered and all the factors that affect those circumstances. "Process" refers to all the actions of health care while "outcome" refers to its consequences, both objective and subjective[15-16]. While other frameworks representing healthcare exist, none is more prevalent than this three-part depiction.

Figure 1. Donabedian's Quality Framework.[17]

Although Donabedian's life-long contributions to the field of public health and health policy are more appropriately captured in the finest academic tomes and corpora[18] rather than paragraphs, I introduce a facet of evaluating the quality of medical care that is not explicitly captured in his model, but is central to this treatise – what Coyle and Battles referred to as "antecedents of care.[19]" The term denotes any pertinent influences on medical care before a patient enters the "structure", endures the "process" or has their "outcomes" appraised. These "involve the environmental context of an individual and an individual's personal characteristics (i.e. genetics, socio-demographics, health habits, beliefs, and attitudes, and preferences). Health related environmental factors may be cultural, social, political, personal, physical, or related to the health professions.[19] " Contrary to "antecedent" however, patients are *continuously* environed by a multitude of factors influencing their health status: before, during, and after care is provided. Recent literature in the area of social determinants of care confirm this idea. Environment factors and a patient's personal or predisposing characteristics affect their health and the corresponding quality of medical care received. Thus it behooves the medical profession and those responsible for its evaluation to elucidate all medically meaningful influences via close inspection, appropriate risk adjustment[19-20], and objective quantification. To facilitate how one may go about elucidating said influences, we look to the science of epidemiology.

EPIDEMIOLOGY

Epidemiology investigates the incidence, source, patterns, transmission, distribution and potential management of disease or health related events. It evaluates the summation of elements controlling the presence or absence of a disease or pathogen.[21-22] Truly, epidemiology has become the foundation for public health and integral to evidence based medicine. The fruits of its abundant labor empower us to formulate a more accurate and comprehensive depiction of disease and thus design more informed policies. It is thus revealing and applicable that the modern origins of epidemiology are in fact archetypal for healthcare as a distinguishable ecosystem.

In a tale that is both classic and now popular for its retrospective elegance, Dr. John Snow is commonly introduced as the son of a coal yard laborer who was both studious and methodically inquisitive. He became well acquainted, despite still being a physician in training, to the nature of "Asiatic Cholera" as he called it then[23], during the epidemic of 1831 that suddenly and calamitously ended, having claimed over fifty thousand citizens.[24-25] The magnitude, rapidity and mystery only compounded his curiosity for what he already knew as a devastating disease.

Beginning in 1848 and continuing until mid-1853, Snow eagerly gathered detailed evidence that suggested cholera was being transmitted by "a morbid matter" or "poison" which had the capacity to "multiply" within the intestinal tract before contaminating local sewers and nearby water supplies. (It seems now that Snow deliberately subdued his

nomenclature by refraining from calling the seed of contagion anything that might resemble confirmation of Girolamo Fracastoro's germ theory. Such ideas were highly unfashionable compared to the miasma theory of transmission of disease via polluted air[25-26].) His ideas explicating the means of cholera transmission were unceremoniously tolerated amongst his peers but it was his fastidious, even unprecedented collection of precise locations and survey information regarding symptomatic patients and their water supplies that could not be ignored. Snow's dogged methodology prepared him for the transformative, late summer epidemic of 1853.

Towards the end of August 1853, infectious death by volume depletion struck Soho – a district located less than a 5-minute walk away[25] from Snow's home in western London. On September 3, Dr. Snow learned of the outbreak and quickly identified a nearby, public water pump as the likely source of infection. Within hours and then again on the following day, he took water samples from said pump for microscopic analysis and recorded, as he had done with the prior outbreaks, the exact date, location, and distance from pump to domicile of each identified victim. Despite the fact that he was unable to identify a physical source from the aqueous samples in question, Snow persisted in suggesting swift action to the Board of Guardians to place the water pump on Broad Street out of commission to prevent further dissemination. He demonstrated a map depicting his dutiful results as rational evidence, but the Board remained unconvinced. Owing to ethical pressures of protecting the citizens of Soho, the Board resolved to principally spread lime on all the nearby streets, in order to eliminate the percolating, suspect miasma. As a precautious afterthought, they also indulged Snow by removing the handle from the Broad Street pump. The epidemic killing of 197

individuals ended abruptly and later investigations of the interior lining of the well beneath the pump by the Board of Health and Paving Board both failed to reveal any structural findings suggestive of contamination.

It wasn't until March 1855, that Snow, with the help of Reverend Henry Whitehead, identified a five-month-old child as the first cholera victim in Soho. Sarah Lewis, the mourning mother, detailed how she had emptied the water used to clean the suffering infant's soiled diapers into a little-known cesspool in front of her home at #40 Broad Street (see Figure 1). A month later, excavation of the pump revealed a trickling but steady flow from the cesspool to the externally decaying bricks of the well. Authorities failed to concede the validity of Snow and Whitehead's final claims but this could not prevent the investigators' ultimate vindication.

Granted, Snow wasn't the sole historic figure to have devoted considerable efforts to epidemiologic studies (as an aside, he was also not alone in his predilection for the germ theory). While recognizing Semmelweis[29-30], and more recently, Wynder, Graham[31], Doll, Hill[32] and many others, Dr. John Snow's experience instructively corroborates the viewpoint that health-related issues/events/diseases must be critically evaluated utilizing contextual information. While collaborative, national and global studies facilitate sufficient sample sizes, power analysis, and generalizability, applicability is diluted. In looking so broadly, dissenters to Snow's conclusions[33] were unable to appreciate how local details unveiled the

critical truths essential for understanding the etiology of cholera but more importantly the potential ways in which they could have improved their community healthcare.

Figure 1. Portion of Snow's original map with inset picture of the contaminated Broad Street pump, squared in green, in front of #40, circa 1850. Scale is 30 inches to a mile.[27-28]

MEDICAL EPIDEMIOLOGY IN TEXAS

According to their explanatory webpage[34], the Texas Department of State Health Services (TDSHS) has compiled forty eight unique indicators in order "to provide descriptive information that serves as a starting point for discussions to begin the process of achieving greater understanding of health and health risks in Texas" because "a review of the state's health status is extremely complex and ultimately requires complex analyses to fully understand the interplay between multiple interrelated health factors." Findings from these indicator maps[35] go beyond the simple transmission of infectious disease in a confined area; they call into question how counties may vary in a whole host of implicated factors, such as office hours available for primary care, average distance to the emergency room, the number of nursing or retirement homes in operation, local employers introducing occupational hazards, etc.

In the name of concision, this treatise will touch on a few illustrative indicators from the TDSHS to provide a means by which we can examine regional discrepancies and ask the type of questions that might enable providers, policy makers, and systems managers to make appropriate value decisions and adaptations. Additionally, because geographic profile maps are often and legitimately criticized for being simply re-imaged population maps, I include below a census depiction of Texas from the United States Census Bureau[36-37] to act as a negative control (see Figure 2).

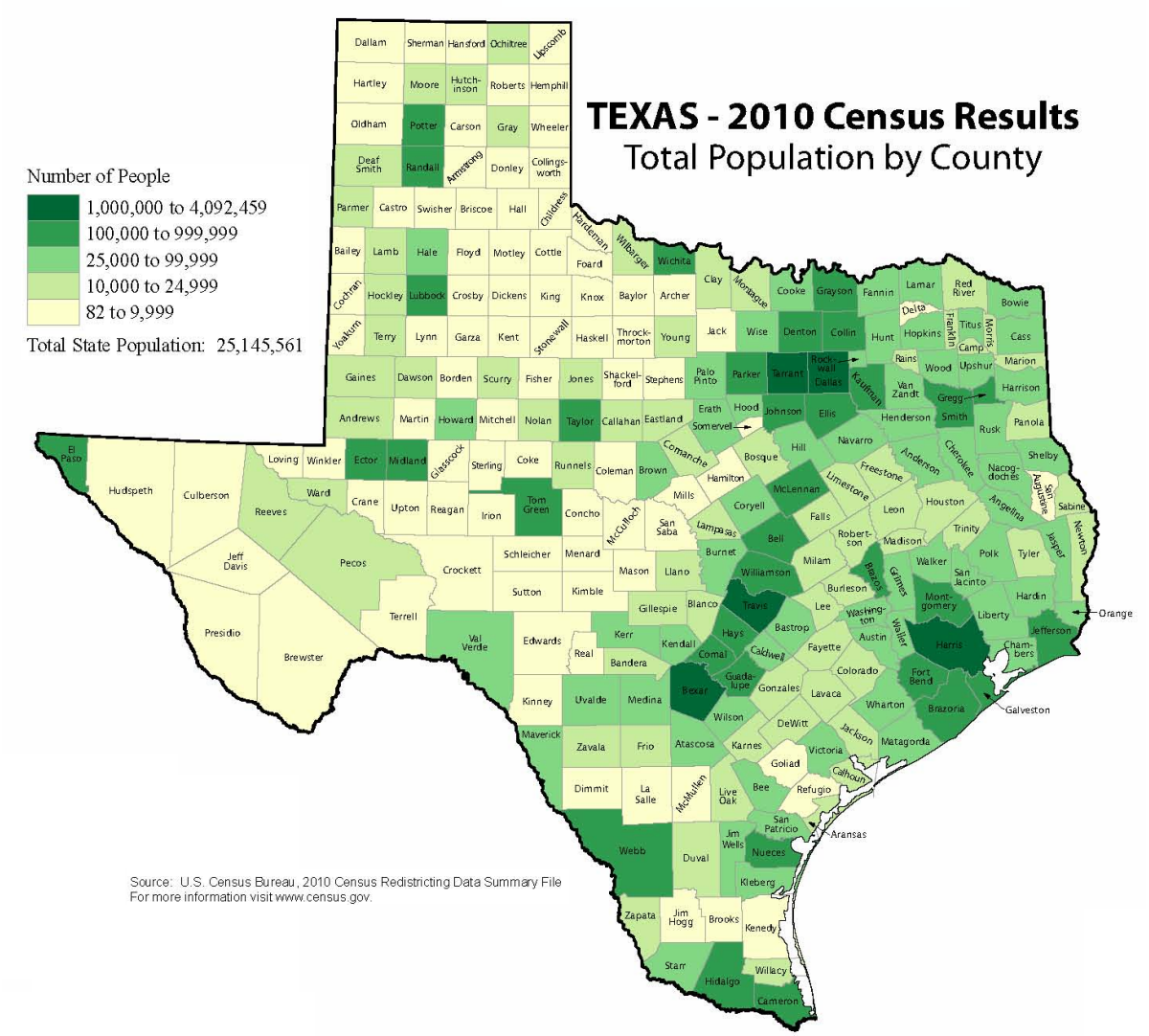

Figure 2

ALZHEIMER'S DEATHS

Alzheimer disease (AD), a progressive dementia that primarily affects individuals over 65 years old[38], is now the 6th leading cause of death in America[39]. Figures 3 and 4 map out annual Alzheimer's deaths in Texas by county in the years 2000 and 2009. By comparing each year, side by side, the number of counties with an increase in pertinent deaths is

visually detectable – reflecting perhaps how the number of individuals over the age of 65 (as a percentage of the total population) in Texas increased from 9.9 - 10.9 percent from the

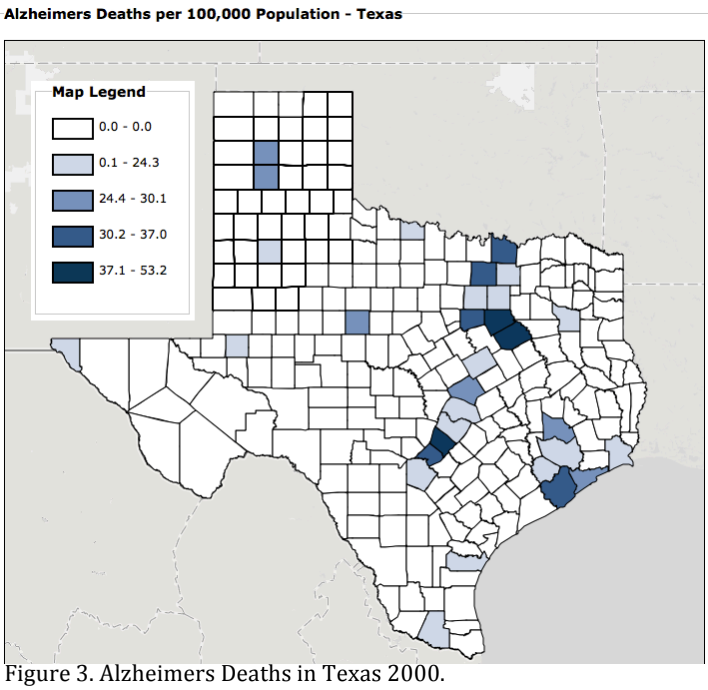

Figure 3. Alzheimers Deaths in Texas 2000.

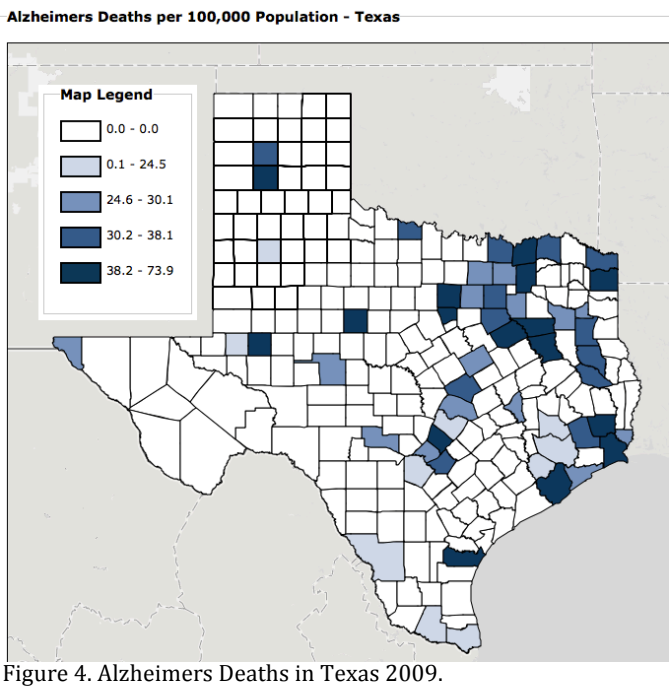

Figure 4. Alzheimers Deaths in Texas 2009.

year 2000 to 2010 [40]. Specific data confirms the visual impression by informing us that Alzheimer's deaths in Texas have steadily increased from 20.4 to 26.9[38]. Interestingly, when we look closely at the individual county numbers we begin to see significant differences. Figure 5 represents the four most populace counties in Texas, chosen for their presumably smaller margins of error for mean number of deaths per year (secondary to larger sample sizes), and one additional county, chosen for consistently reporting statistically stable Alzheimer's deaths during the selected 2000-2009 window.

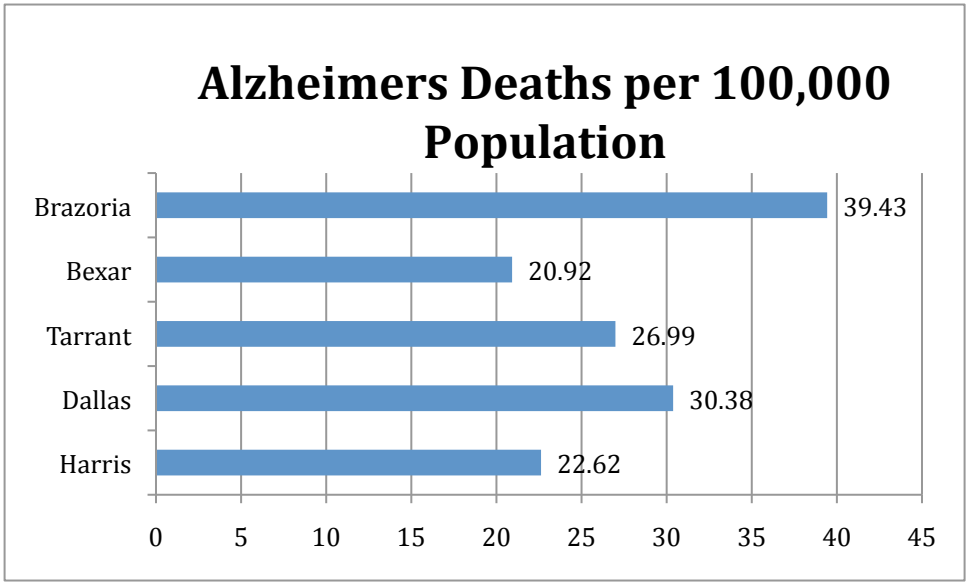

Figure 5. Data used to make chart from TDSHS.

County	Bexar	Harris	Tarrant	Dallas	Brazoria
99% CI	18.98 – 22.86	21.16 – 24.08	25.76 – 28.22	26.95 – 33.81	34.01 – 44.85

Table 1. Displaying confidence intervals for the number of Alzheimers deaths/100k averaged from 2000- 2009. Data used to make chart from TDSHS.

As you see from Table 1, Dallas and Tarrant Counties have significantly higher rates of Alzheimer's deaths compared to Bexar and Harris counties, while Brazoria County uniquely persists as having one of the highest rates of Alzheimer's deaths in Texas. Why is that? Were their differences in how local practitioners applied the diagnostic criteria that has since been revised[41-42]? If so, was the primary management of dementia equitably different between counties? Or were their systematic discrepancies in the number of or ability to perform autopsies for pathologic confirmation[43]? Was there any difference of age in the constituency of each county? Consider the high rates of Alzheimer's deaths in Brazoria County despite having a lower percentage of its population above 65 compared to the rest of the state (10.2 versus 10.9 percent). Is there something, unidentified to date, locally

inducing the pathogenesis of hyperphosphorylated tau[44]? Do they have a higher regional prevalence of the ε4 allele of the apolipoprotein E (APOE) gene[45-46]? Is there something unique about the genotype of HSV1 in certain counties that makes it more likely to play a causal role in the development of AD[47-49]? Or is there an excessive burden of copper in the nearby soils or diet that could be contributing[50]? These questions and disparities warrant further investigation.

SUICIDE FATALITIES

Suicide is a complex behavior that leads to intentional, lethal self-harm. According to the Centers for Disease Control and Prevention (CDC), it is the 10th leading cause of death in the United States and one that may be preventable with early recognition of relevant risk factors and prompt intervention. The American Foundation for Suicide Prevention teaches that while mental disorders, previous attempts, a positive family history, or a serious medical condition increase the risk for suicidal behavior, its important to remember that warning signs, especially when individuals communicate their terminative intentions, nearly always precede the actual attempt[51].

According to the CDC, suicide results in $34.6 *billion* of combined work loss and medical costs.[52] Contemplate the following Table 2 that details six counties selected at random from the statistically stable counties on the TDSHS website[35] that reported annual suicide deaths per one hundred thousand people from 2000 to 2009.

County	El Paso	Denton	Dallas	Nueces	Lubbock	Galveston
Mean	8.17	9.05	9.9	10.5	12.09	14.1
99% CI	7.464 – 8.876	8.190 – 9.910	9.2518 – 10.548	8.880 – 12.120	10.575 – 13.605	12.131 – 16.069

Table 2. Ten year averages for annual county suicide deaths per 100,000 in Texas. Data used to make table from TDSHS.

Essentially, El Paso County has significantly lower suicide rates than all except Denton, while Galveston County has statistically higher rates than all except Lubbock. Additionally, suicide rates in Dallas County are significantly different from El Paso, Lubbock and Galveston.

These observations engender inquisition: Has managed care in these areas offered dissimilar reimbursement levels for mental healthcare? What instructions do local providers give regarding individuals at risk for suicide? What are the regional rates of depression, psychosis, or substance abuse? What is the culture and history of suicide in these areas? Are certain antidepressants used more effectively in one area compared to another? Are there more mental health outpatient clinics in El Paso?

A study published in Nov. 2010 from the journal of *Administration and Policy in Mental Health and Mental Health Services Research*[53] evaluated community-based outpatient mental health services in Seattle for 493 young adults and found unsettling evidence: "typical" outpatient mental health treatments over a 9 year period did not reduce mental disorders, including depression, dysthymia, anxiety, social phobia, post-traumatic stress, or their symptoms. Could this be what is occurring in Galveston, for example, as well? Is typical therapy simply inadequate or not sufficiently evidenced based as suggested by some[53-54].

Or is there something about the healthcare provided during the immediate period preceding suicide that could be optimized for better outcomes? A retrospective review by the Veterans Health Administration (VHA) investigated 1843 VHA patients with diagnosed depressive disorders who died from suicide from April 1999 to September 2004. Over half (n=938) of patients had a VHA visit within 30 days of suicide. Of those patients, 57 percent (n=533) were seen in non-mental health settings for their final visit. Among those 533 patients, 65.9 percent *did not* have a mental health condition coded at their final visit, and only 58.5 percent were receiving *inadequate* dosages of antidepressant (versus 44.7 percent last seen by mental health services) ($p<0.0005$)[55]. These results are consistent with previous investigation[56] but remain unsatisfactory. What policies could be implemented to provide greater assurances that patients of any service receive appropriate dosages or effective therapy[57]? How could a reimbursement system be designed to incentivize appropriate treatment of patients at risk for suicide or rapid referral to specialty mental health services?

PRETERM BIRTHS

Babies born before term (<37 to 41 weeks of gestation) carry risks for long-term morbidity, neurologic impairment, disability and often require intensive care after birth[58]. According to TDSHS, "average hospital stays for preterm infants without complications are three times longer than a term infant, and for a preterm birth with complications, hospital stays are over eight times longer"[59]. Importantly, roughly three quarters of perinatal deaths occur among premature or preterm infants[60]. According to Mathews, the length of gestation

is predictive of survival rates; approximately 20 percent of babies born before 32 weeks of gestation do not survive the first year of life, compared to the 1 percent of infants born between 32 and 36 weeks that do not survive the first year of life. Furthermore, the "infant mortality rate (IMR) per 1,000 live births for infants born at less than 32 weeks of gestation was 180.9, nearly 70 times the rate for infants born" between 37 and 41 weeks of gestation[61].

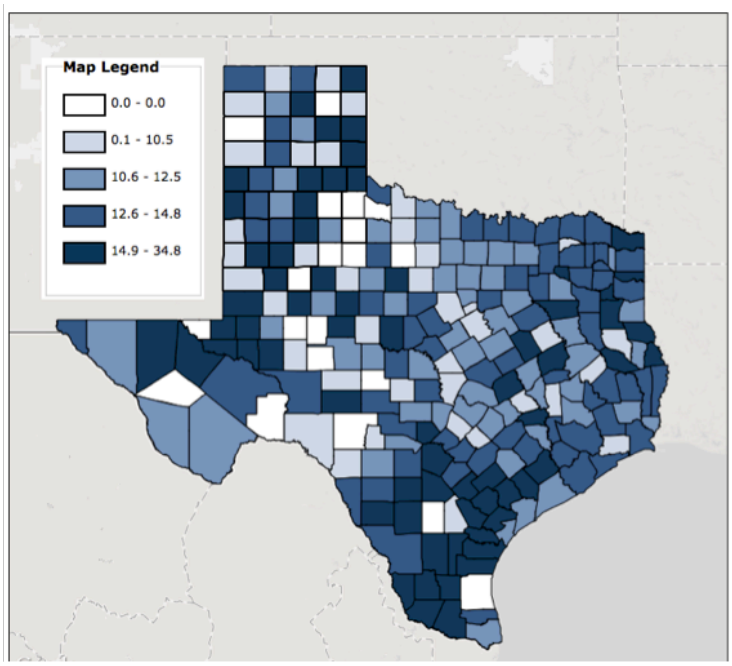

Figure 6. Map of Texas Counties color coded by percent of preterm births in 2009.

The emotional and economic repercussions of years of potential life lost, ICU expenses, and the cost of extended hospital stays are just a few of the reasons to elucidate the patterns and etiology of preterm births in Texas. As you can see from Figure 6, the distribution of preterm births as a percentage of live births in Texas is a far cry from the statistically sparse geographical spread of Alzheimers deaths.

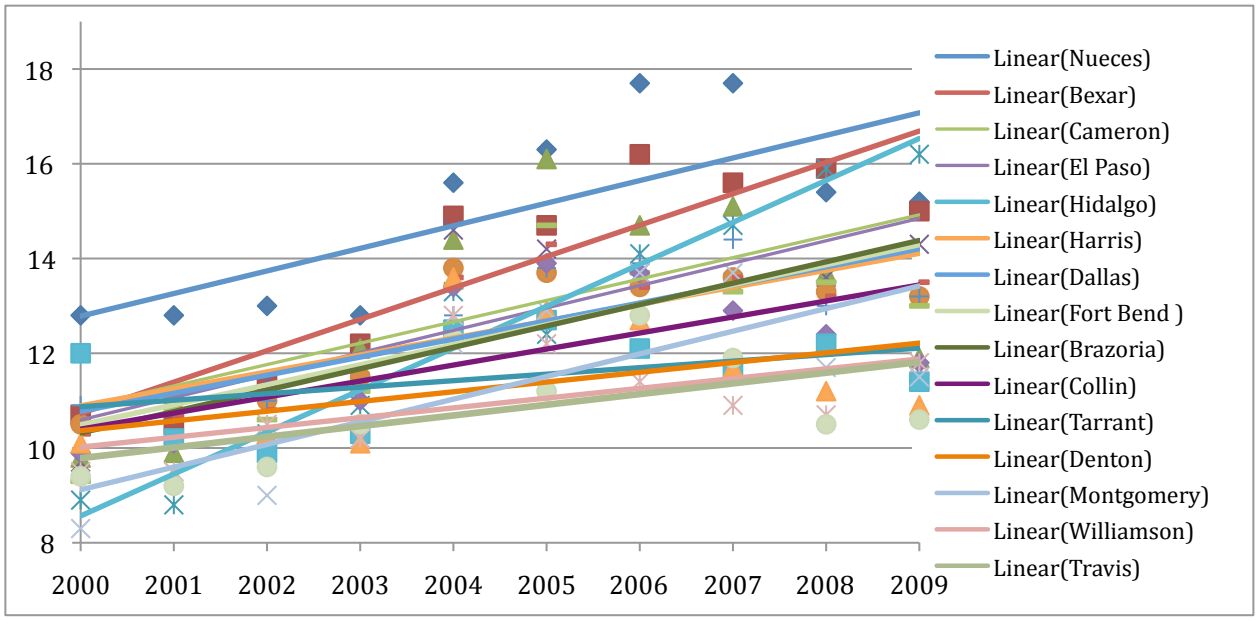

Figure 7. Preterm births as a percentage of live births; Best fit line applied to annual data points in each county.

Figure 7 represents the percentage of preterm births in the fifteen most populous counties of Texas from 2000 to 2009, with a best fit line applied to each counties' data points to enable the reader to distinguish significant trend differences. Table 3, on the other hand, is a sampling of the fifteen counties of Figure 7 charted with their respective mean percent of live births born prematurely as well as a corresponding confidence interval. This permits us to see that Nueces County has the highest rates of preterm birth amongst the most populous counties in Texas while Travis County (along with Williamson, Denton, and Tarrant; which are not significantly different) has the lowest rate of preterm births within the same large cohort. Alternatively, Dallas County has nearly identical preterm rates to Harris County but both are still higher than Travis.

County	Travis	Williamson	Denton	Tarrant	Dallas	Harris	El Paso	Nueces
Mean	10.8	10.95	11.29	11.49	12.49	12.5	12.72	14.93
Std Dev.	1.24	1.1	1.4	1.01	1.41	1.32	1.73	1.98
95% CI	10.03-11.57	10.27-11.63	10.42 - 12.16	10.86 – 12.11	11.62 – 13.36	11.68-13.32	11.65 - 13.79	13.70 – 16.16

Table 3. Percent of Live births that are born preterm in Texas. Data used to make table from TDSHS.

One other observation of note from Figure 7 is the general upward trend of preterm births in Texas; such births have conspicuously risen by 23 percent from 1990 to 2003 compared to the national average of 16 percent[62] over the same time frame. Of interest, 2003 also brought in a single year national increase in the rate of premature births of 2 percent[42] that can be seen in Figure 8.

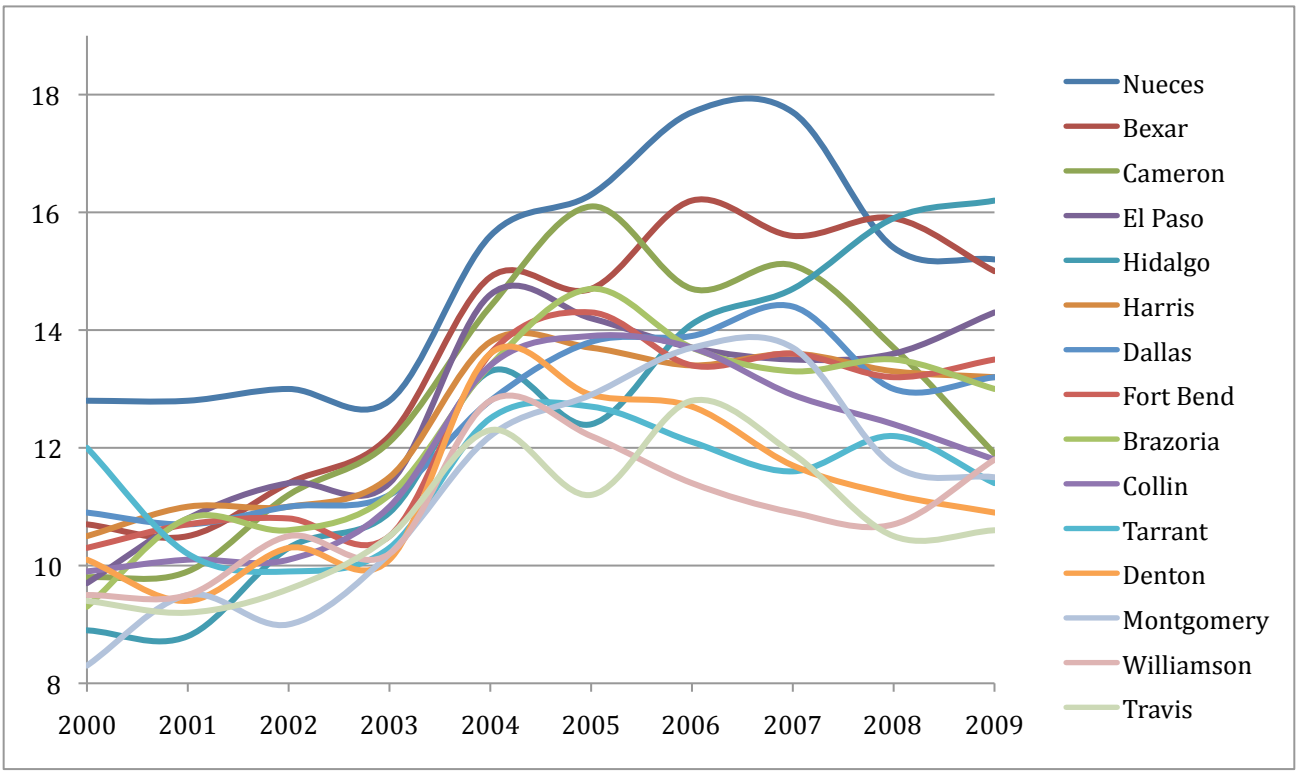

Figure 8. Data used to make chart from TDSHS

By simply smooth graphing each county's annual rate of preterm birth rather than fitting a trend line to a scatter plot we're able to see the dramatic and unified slope change

(22 percent increase) starting in 2003 and rounding off erratically after 2004. So what happened preceding and during 2003 that facilitated such a dramatic increase in the rate of preterm births in the counties that account for nearly 70 percent of the state's population?

A UK study published in 2007 reviewed over five hundred thousand births from 1994 to the end of 2003 and found that singleton children born to the most socioeconomically deprived decile of the population were "at nearly twice the risk of very preterm birth (22-32 weeks) compared with those from the least deprived decile, with 16.4 per 1000 births in the most deprived decile compared with 8.5 per 1000 births in the least deprived decile (incidence rate ratio 1.94; 95% CI (1.73 to 2.17)). This deprivation gap remained unchanged throughout the 10-year period"[63].

Could a sudden increase in state poverty or unemployment explain this? While a comparison of the ten-year rates of preterm births in the aforementioned counties in Figures 7 and 8 with the percentage of homes below the poverty line for each respective county shows a correlation of 0.40, the coefficient of determination (R^2) is 0.16. Analogize that with the correlation between preterm birth rates and the percentage of the population that is Black or African American alone or Hispanic or Latino (r=0.63), and its corresponding coefficient of determination (R^2=0.39) (see Table 4). These finding suggest that maternal race may also play a significant role amongst the host of converging factors that contribute to births before 37 weeks. But what else is percolating underneath the surface of healthcare that is affecting preterm births?

County	Percent of persons below poverty level	Mean rate of **Preterm Births** (2000-'09)	Percent of population that is Black or African American + Hispanic or Latino
Travis	17.4	10.8	42
Williamson	6.8	10.95	30.4
Montgomery	12.3	11.27	26.3
Denton	8.5	11.29	27.9
Tarrant	14.7	11.49	43
Collin	7.5	11.92	24.2
Brazoria	10.9	12.35	41.5
Fort Bend	8.3	12.39	45.4
Dallas	18.8	12.49	61.8
Harris	17.9	12.5	61
Hidalgo	35	12.55	91.7
El Paso	24	12.72	85.1
Cameron	34.9	12.89	89.2
Bexar	17.8	13.71	67.2
Nueces	18.4	14.93	65.8
Correlation=r	0.40		
Coefficient of Determination (R^2)	0.16		
Correlation=r		0.63	
Coefficient of Determination (R^2)		0.39	

Table 4. Calculations based on data from US Census Bureau[64]and TDSHS[35].

In this section discussing regional epidemiology, we have delved into three of the forty-eight indicators concerning priority health issues in Texas. From 2000-2009, there was significant variation between counties in the Alzheimers deaths, suicides, and preterm births suggesting a geographic regionality to the presentation of disease and possibly the nearby healthcare system responsible for its prevention, reduction, treatment, and control.

HEALTHCARE ENVIRONMENT

INTRODUCTION

Because of variable disease outcomes evident in the regional epidemiology of Texas and the claim that the local medical environment affects the quality of care, we briefly appreciate here a few illustrations of the complexity that surrounds the health of individuals and populations. Expertise across a variety of related fields, including genetics, food science, law, economics, sociology, anthropology, and others, are more suited to recapitulate the vast details underlying the nature and mechanisms of influence, thus we simply suggest how three components of the healthcare environment may have interplay with the healthcare system and its patients. These elements include medical ecology, food deserts, and occupational risk factors. With more time, we would examine city design, social determinants of care, maternal or paternal education level, cultural perceptions of substance abuse, air quality control, and a myriad of other fascinating environmental influences.

MEDICAL ECOLOGY

Ecology, broadly defined, is the branch of science that devotes itself to understanding the relations of organisms one to another and to their respective environments[20,65-66]. *Medical* ecology is an emerging scientific approach to try and understand the "community of human-associated microbes …… [and] their influence upon human development, physiology, immunity, and nutrition[67-69]". For example, in 2008, Lundin et al[69], showed

through the expression of nuclear receptors and toll-like receptors that intestinal microbiota contribute to the development of innate immunity and epithelial barrier function along the intestinal tract. Then in 2011, researchers from Germany demonstrated how normal gut flora modulates behavior and brain development in a mammalian[70] (see also Appendix Figure B). We are just beginning to glimpse the profound repercussions of our relationship with our personal microbiome, from developmental programming to the structure and function of human physiology.

FOOD DESERTS AND DISPARITIES

Pockets and restricted regions of America have limited access to affordable and nutritious food, thus the term food desert[71]. Furthermore, food discrepancies persist throughout the United States based on racial, geographic, socioeconomic, and ethnic characteristics. As mandated by the Food, Conservation, and Energy Act of 2008[72-73], the U.S. Department of Agriculture investigated this topic and reported that,

> [C]onsumers are constrained in their ability to access affordable nutritious food because they live far from a supermarket or large grocery store and do not have easy access to transportation. Urban core areas with limited food access are characterized by higher levels of racial segregation and greater income inequality. In small-town and rural areas with limited food access, the lack of transportation infrastructure is the most defining characteristic.[77]

A more recent study of variation in low food access areas encouraged "field work … for local community efforts aimed at identifying and improving food access,"[75] thus reinforcing the philosophy that local inspection must follow large-scale surveillance. But regardless of the scale of application, knowledge concerning the distribution and allocation of food resources

has the potential to influence on a variety of chronic health issues such as obesity, diabetes, cancer[76], seizures[77], and many more.

OCCUPATIONAL RISK FACTORS

Health specific hazards can be encountered in any setting, but few are as predictably present as those in the work place. According to the World Health Organization, back pain, hearing loss, asthma, unintentional injuries, chronic obstructive pulmonary disease (COPD), respiratory cancer, pneumoconiosis, dermatitis, musculoskeletal disorders of the upper extremities, infectious diseases, leukemia, infection, and work-related deaths are all significantly influenced by occupational risks[78]. Recent data has also shown how inappropriate disease-specific-accommodations in the work place put patient at risk for loss of employment[79], not to mention disease exacerbations, underperformance, and subsequent dissatisfaction.

Radiation exposure to workers during the United States' nuclear weapons development (1945 -1962) is an excellent example of an occupational hazard shaping healthcare. Uranium mining, processing, and transportation were fundamental to the testing, advancement and expansion of our nation's nuclear armory in the 20th century. According to the Department of Justice, nearly two hundred atmospheric nuclear weapons development tests were conducted via the manpower of tens of thousands of workers[80]. As a result of the extraordinary levels of radiation exposure, individuals contracted more than twenty different medical conditions for which the national government would later

compensate. Figure 13 is a diagrammatic representation of the states involved with uranium mining and nuclear testing, as identified by the 1990 Radiation Exposure Compensation Act (RECA).

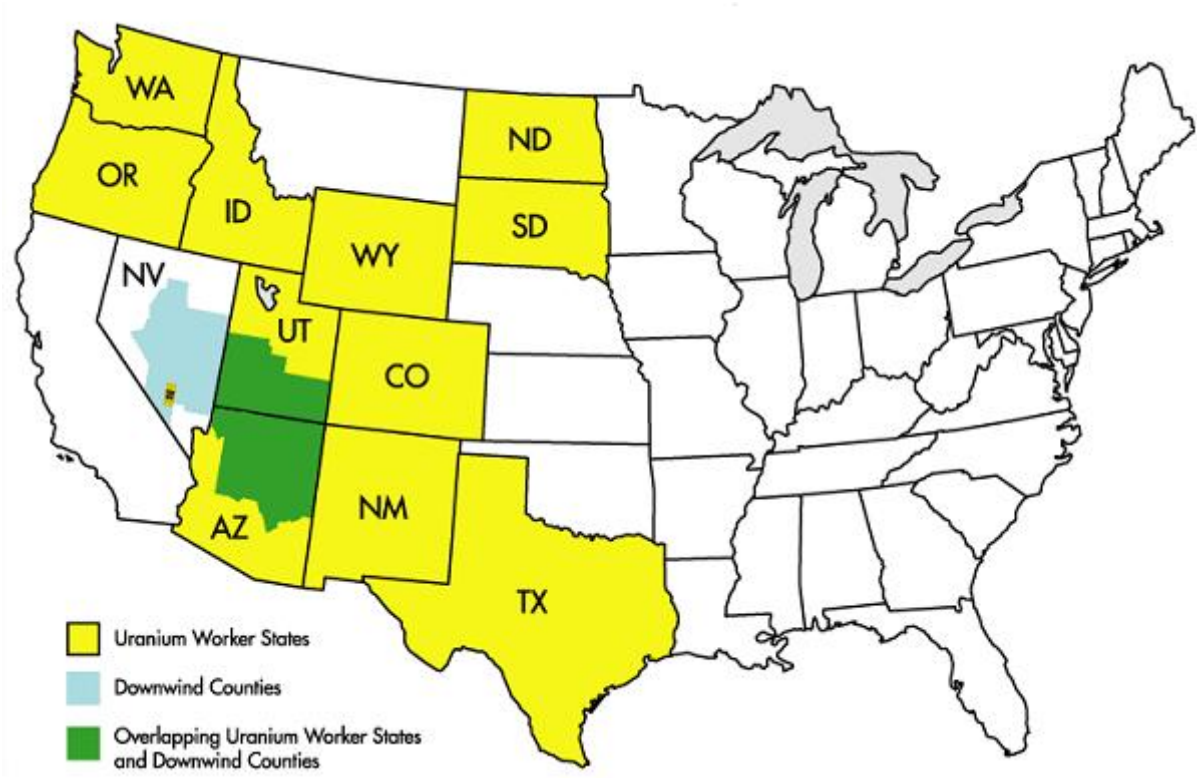

Figure 13. Map courtesy of the United States Department of Justice[80]

Literally, tens of thousands of American citizens developed malignancies and other radiation induced maladies – most of them seeking compensation and care. Is it not reasonable that the density of radiation exposed workers correlated well with diagnostic accuracy of neoplasms while electromagnetic dosing burden inversely correlated with local medical care outcomes?

EXAMPLE OF EPIDEMIOLOGIC APPROACH TO EVALUATE THE MEDICAL ENVIRONMENT: INFANT MORTALITY

Infant mortality is defined as the death of child before the age of one year. According to TDSHS, the infant death rate serves as a "measure of a nation's health and a worldwide indicator of health status and social well-being… a proxy indicator of the quality of, and access to, medical care for pregnant women and infants"[81]. Figure 9 shows an analysis of the number of infant deaths per thousand live births in the fourteen most populous counties of Texas.

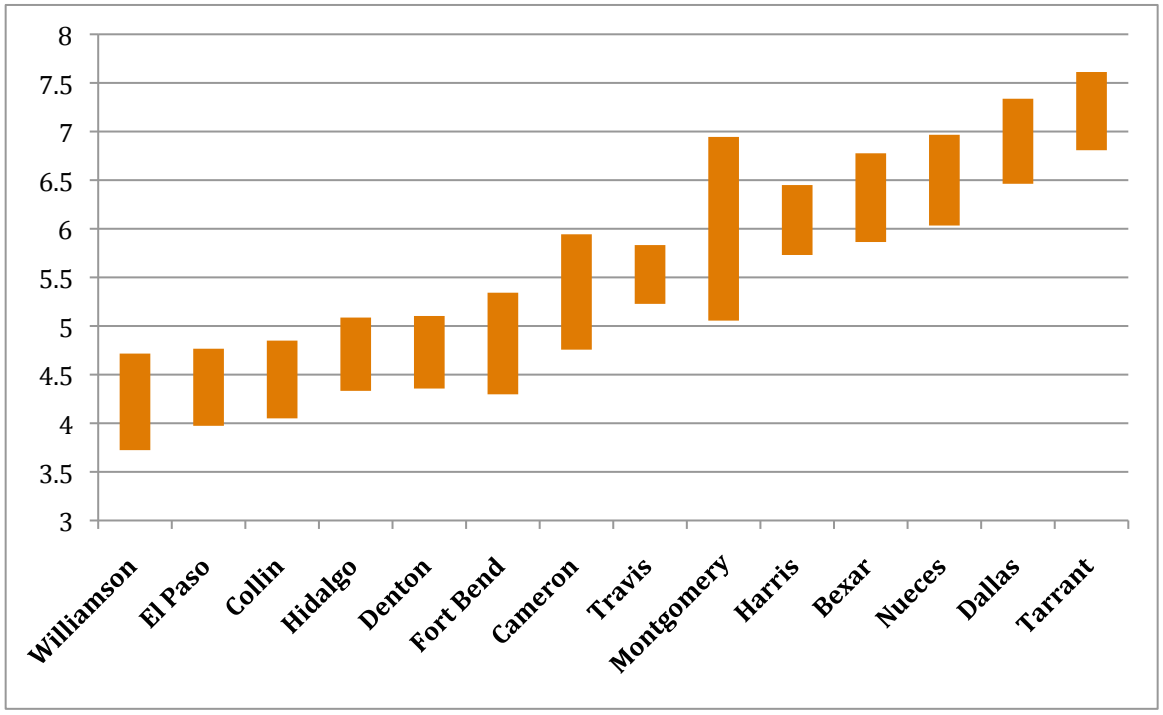

Figure 9. Infant deaths/1,000 Live Births – 14 Most Populous Counties; each orange bar represents the mean rate for the 10 year period between 2000 and 2009, with a 95% Confidence Interval included. Data from TDSHS Infant deaths map[81].

Several observations can be made from the information above. There is a lower cohort of counties whose rate is below 5.5 deaths/thousand live births (Williamson to Fort Bend), a

middle cohort that straddles that cutoff (Cameron, Travis, Montgomery), and a higher cohort whose infant death rate is above 5.5. There are no significant differences between the counties in the lower cohort. Tarrant County, on the other hand, has significantly higher infant death rates than all except Dallas, Nueces, and Montgomery counties. Do we know why? Figure 10 certainly alludes to a partial explanation but thanks to a proactive county health department we know considerably more about the etiology of preterm births in at least one county of Texas.

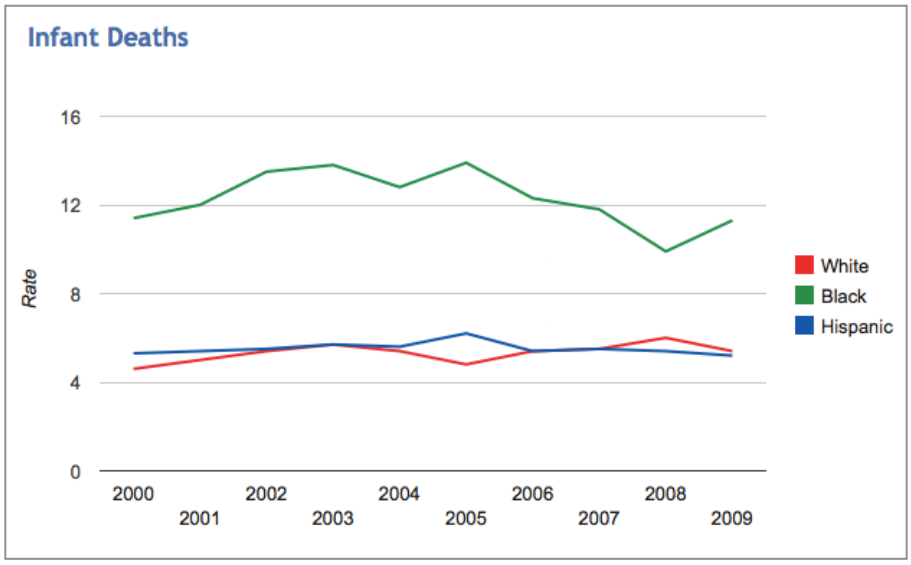

Figure 10. Showing infant death rate data by race demographics[45].

Motivated by the high infant death rate seen in Figure 9, Tarrant County Public Health (TCPH) recently published an investigation into their fetal-infant mortality for the period from 2007 to 2009[82]. Researchers first identified an external Texas reference group with which to compare their fetal-infant death rate and then calculated the number of deaths that were in excess of expectations. They dutifully reported that their reference group was "noted to generally have better birth outcomes [and include] non-Hispanic white mothers aged 20+ years with a high school diploma or higher education level.[82]" Their findings are

subdivided by subpopulation, presented in their original form in Figure 11, and are consistent with the racial trends of preterm and infant deaths in the remainder of Texas. Excess death rates were highest amongst Non-Hispanic Blacks and Teens, and accounted for over 40% of all deaths – signifying that nearly half of all fetal-infant deaths in Tarrant County were potentially preventable had the modifiable risk factors been reduced or ablated.

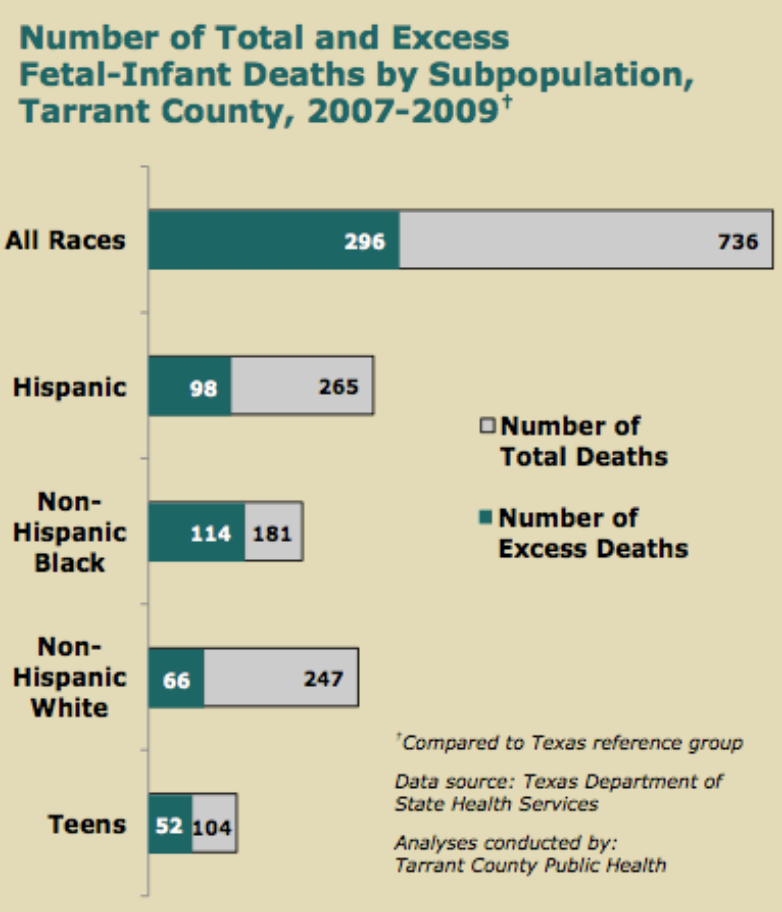

Figure 11. Analysis done by Tarrant County Public Health

To further elucidate which factors potentially contributed to the cause and timing of fetal-infant death, TCPH categorized the deaths into four periods of perinatal risk based on birth weight and age of the child at the time of demise: Maternal Health/Prematurity, Maternal Care, Newborn Care, and Infant Health (see Appendix Figure A). Excess fetal-infant

death rates were then stratified by subpopulation and these four periods of risk (sometimes referred to as 'intervention areas' as in Figure 12). Forty four percent of all excess mortality, regardless of race, occurred during the Maternal Health/Prematurity risk period. In addition, Non-Hispanic Blacks in Tarrant County had the highest rates of excess mortality rates in Maternal Health/Prematurity and Infant Health. This alludes to the suggestion that Non-Hispanic Black mothers and Teens in Tarrant County need more attention and education regarding general healthy behaviors, particularly during period of time preceding conception, and perinatal care[83-84].

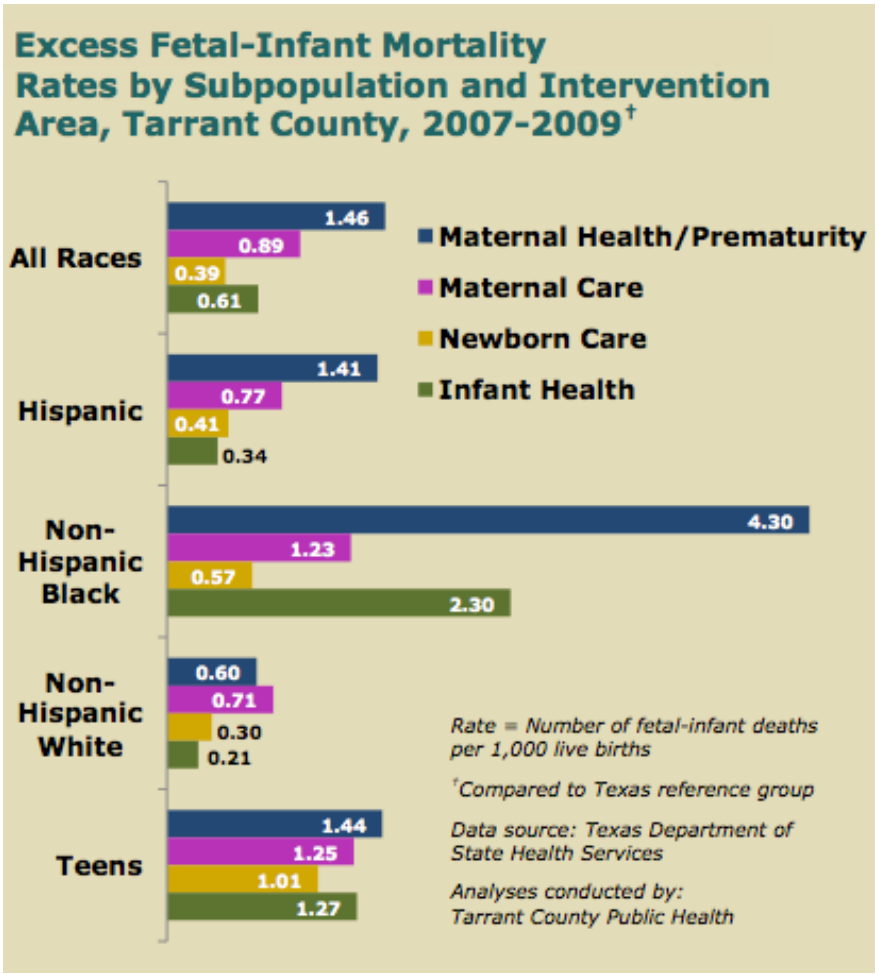

Figure 12. Analysis and figure provided by Tarrant County Public Health

TCPH also found three characteristics shared by the Hispanic and Non-Hispanic Black mothers whose children died during their first year of life. They were more likely to be teen mothers, be overweight and obese, and not attend an adequate number of prenatal care visits. Non-Hispanic Whites, on the other hand were more likely to smoke. Collectively, these findings enabled TCPH to identify and target interventions to curtail maternal smoking, increase the use (and if necessary the access) of prenatal care, lower the rate of teenage pregnancy, and reduce the percent of women who may be overweight or obese before pregnancy[82]. While this analysis is pragmatically applicable to Tarrant County and, ceteris paribus, may be indicative of the etiology of fetal-infant across Texas, it is not sufficient to definitively explain the disparities *between* counties or permit local health officials or policy makers to forego their own in-depth analysis and evaluation. Nevertheless, it is an excellent application of epidemiological data that reveals factors contributing to an elevated rate of infant mortality and utilizes this information to mold healthy policies applicable to Tarrant County's medical environment.

CONCLUSION

INITIAL FINDINGS

Disease outcome discrepancies currently exist between populous counties in Texas; this is true for Alzheimer's deaths, suicide rates, percent of preterm births, and infant

mortality. Adequate literature exists to suggest possible patient characteristics and medical environmental targets for evaluation into their role in altering pertinent healthcare processes. Investigators have already demonstrated that quantification of patient characteristics and medical environmental factors influencing a single disease process more accurately correlates with disease outcomes than an evaluation of relevant processes or structure. Individual counties in Texas can and have adopted health policies to address medical environmental factors to improve health outcomes.

POLICY PROPOSALS

Initial findings from this thesis suggest that we shift the paradigm in which we evaluate our healthcare system to incorporate more patient characteristics and the medical environment; by doing so, a more comprehensive and realistic analysis would lead to more accurate evaluations for healthcare quality improvement. Once the paradigm is thus altered, several steps would need to be taken. These include the following: 1) identify the most detectable healthcare related discrepancies in Texas, 2) identify the most quantifiable, influential factors from the local medical environment, starting with priority diseases or health processes, 3) encourage greater investigation and oversight of our healthcare systems by local & regional administrators, experts, and authorities, 4) provide greater education to the public concerning published quality measures (such as hospital compare or physician compare) and how they are affected by patient characteristics and the local

medical environment, and lastly 5), suggest or require that generalized quality metrics be published in the context of the local medical environment.

LIMITATIONS AND FUTURE QUESTIONS

Counties may be arbitrary divisions. Is there a better designation system to detect and evaluate unique medical environments? Would such a system have the data as readily available as they are from the Texas Department of Health Services? Presumably, there multiple, geographically overlapping medical environments for different diseases. But is there evidence for this in such cases as air quality for asthma, water quality for gastroenteritis, food desserts for endocrinopathies, factory districts for occupational hazards, etc? Which metrics will best quantify the relative contribution of each environmental factor? Could we reproducibly measure and quantify cultural influences on health-related habits? These questions, and many others, warrant our attention as the science and practice of healthcare evaluation and improvement progresses.

Ultimately, healthcare is distinctly characterized by regional manifestations of disease, special population subsets, unmatched environmental influences, and idiosyncratic combinations of structure, process, and outcomes. Healthcare evaluation should include these influential factors. Additionally, healthcare policies regarding medical outcomes will need to be adjusted to facilitate and incorporate such evaluations.

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APPENDIX

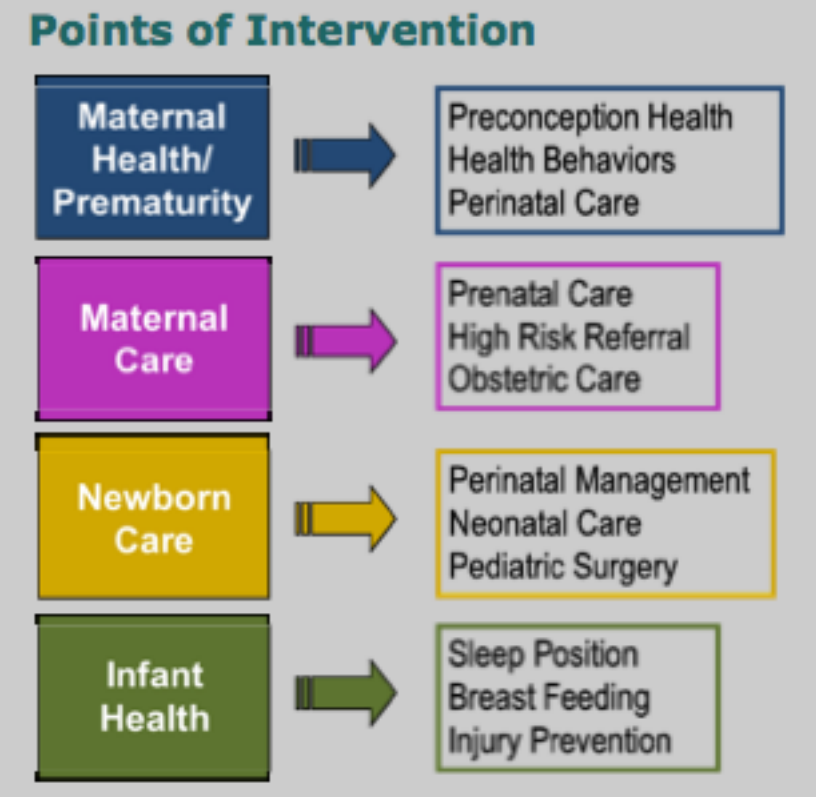
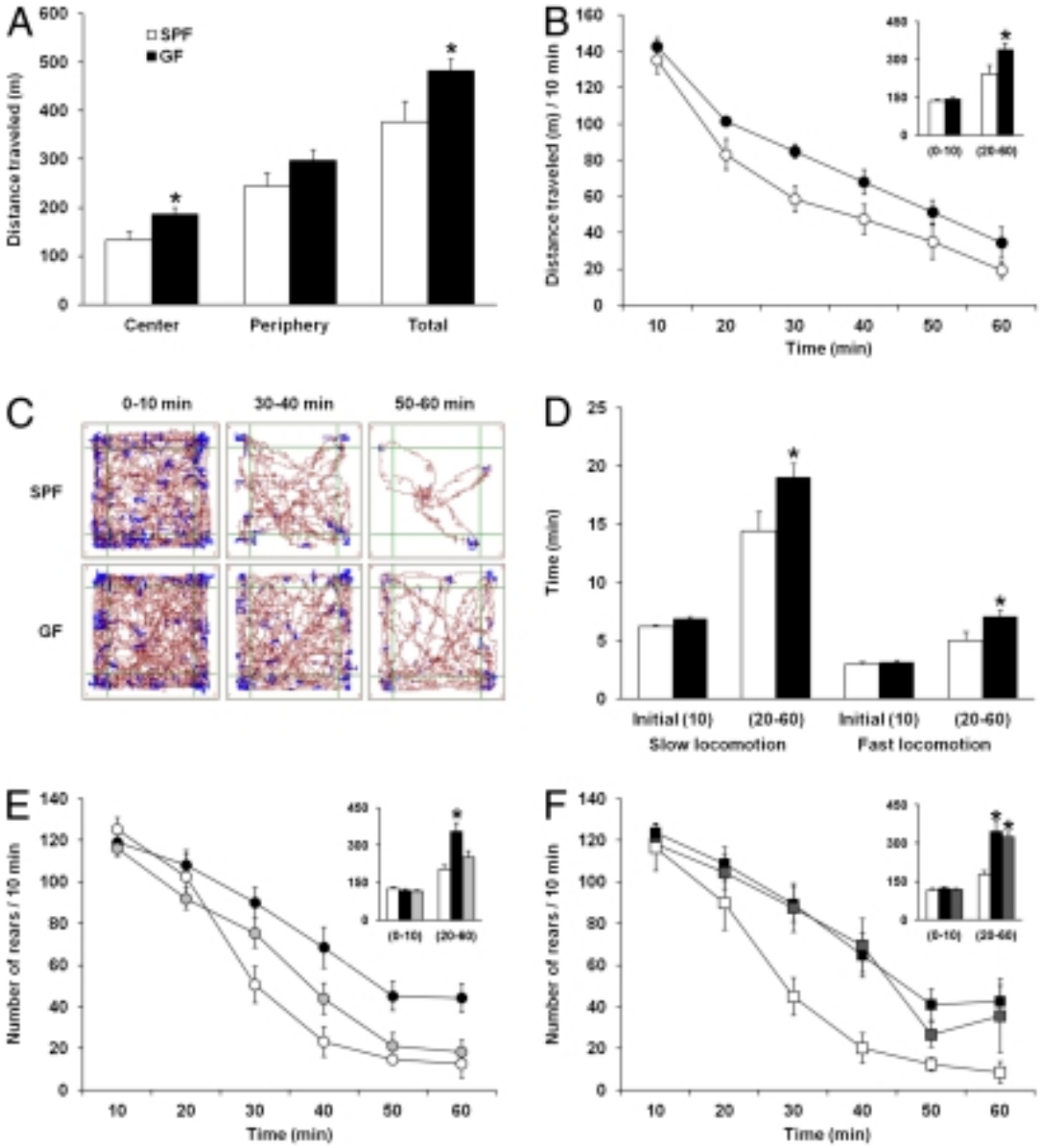


Figure A. Perinatal Periods of Risk as identified by Tarrant County paired with points of intervention[81]



Germ Free (GF) mice display increased spontaneous motor activity. (*A*) Bars show cumulative distance traveled (meters) per zone and in the entire box (total) during the 60-min open field test session by Specific Pathogen Free (SPF) (open bars) and GF (filled bars) mice. (*B*) Average distance traveled (meters) measured in 10-min time bins across a 60-min session in an open field box. (*Inset*) Bars show cumulative distance traveled (meters) during the initial 10 min and the 20- to 60-min time interval of open field testing. (*C*) Representative tracks of movement patterns of SPF and GF mice at the 0–10, 30–40, and 50–60 min time intervals of the 60-min open field test session; distance traveled and rearing activity is shown in dark red and blue colors, respectively. (*D*) The time that SPF and GF mice spent in slow (>5 cm/s) or fast (>20 cm/s) locomotion during the initial 10 min of testing and the 20–60 min time interval. (*E*) Rearing activity of SPF (white), GF (black), and conventionalized (CON; light gray) mice. Circles show the average number of rears measured in 10-min time bins across a 60-min session in an open field box. (*F*) Rearing activity of SPF, GF, and adult CON mice (dark gray); lines connecting cumulative data in *B*, *E*, and *F* were drawn for clarity only. All data (*A*, *B*, and *D*–*F*) are presented as means (± SEM; n = 7–14 per group). *$P < 0.05$ compared with SPF mice.

Figure B. Excerpt from Pettersson[70]