

Evaluating Chicago's Success in Reaching the Healthy People 2000 Goal of Reducing Health Disparities

ABIGAIL SILVA, MPH^a
STEVEN WHITMAN, PhD^a
HELEN MARGELLOS, MPH^a
DAVID ANSELL, MD, MPH^b

SYNOPSIS

Objective. This study was designed to assess Chicago's progress from 1980 to 1998 in addressing the Healthy People 2000 goal of reducing health disparities.

Methods. Chicago vital statistics and surveillance data were used to calculate black:white rate ratios of mortality and morbidity for 1980–1998. Mortality and morbidity rate ratios were also used to compare people living in areas with the lowest median household income with those living in the highest for 1979–1981, 1991–1993, and 1996–1998. The health measures included mortality associated with leading causes of death; all-cause mortality, incidence rates for two communicable diseases; and two birth outcomes.

Results. Both black:white and low-income:high-income rate ratios monotonically increased for virtually all measures of mortality and morbidity. Almost all of the rate ratios and linear trends were statistically significant. From 1980 to 1998, the black:white rate ratio for all-cause mortality increased by 57% to 2.03. From 1979–1981 to 1996–1998, the low-income:high-income rate ratio for all-cause mortality increased by 56% to 2.68.

Conclusions. These findings provide clear evidence that disparities in health did not decrease in Chicago. Instead, racial and economic disparities increased for almost all measures of mortality and morbidity used in this study. The fact that the Healthy People 2000 campaign to reduce and then eliminate health disparities was not effective must serve as a stimulus for improved strategies.

^aSinai Urban Health Institute, Mt. Sinai Hospital, Sinai Health System, Chicago, IL

^bDepartment of Medicine, Mt. Sinai Hospital, Sinai Health System, Chicago, IL

Address correspondence to: Abigail Silva, MPH, Kurtzon 439, Mt. Sinai Hospital, California Ave. at 15th St., Chicago, IL 60608; tel. 773-257-5785; fax 773-257-5680; e-mail <silva@sinai.org>.

© 2001 Association of Schools of Public Health

One of the overarching goals set forth in 1990 by the Healthy People 2000 initiative was to reduce health disparities. Since then, there has been increasing attention to and interest in reducing health disparities in the form of national initiatives and statewide efforts.¹⁻⁶ This enthusiasm inspired President Clinton to commit the nation to the ambitious goal of eliminating racial disparities by 2010 in six key areas: infant mortality, cancer screening and management, cardiovascular disease, diabetes, HIV/AIDS, and immunizations.⁷ This goal parallels the Healthy People 2010 call for the elimination of all disparities in health.⁸

Part of the process of achieving any goal is measuring the progress made toward reaching it. National data show that racial disparities in mortality are not decreasing. While in recent years death rates have declined overall, black people have consistently experienced higher rates of mortality than whites.⁹⁻¹² From 1990 to 1996, the black:white mortality rate ratio for heart disease, cancer, diabetes, and all causes essentially remained the same (declining an average of less than 2%), with each rate ratio ranging from 1.34 to 1.60.⁹

Much has been published regarding the excess mortality that black and poor people suffer in comparison to their white or wealthier counterparts.¹⁰⁻¹² Researchers such as McNamara,¹³ Williams,¹⁴⁻¹⁶ Pappas,¹⁷ and their colleagues have demonstrated racial disparities in mortality at the national level, while others such as Geronimus and colleagues,^{18,19} Polednak,^{20,21} and McCord and Freeman²² have explored racial differentials in mortality either across selected urban areas or at the local level. Despite the vast array of research in this field, we have been unable to locate any study that analyzes disparities across racial or income groups at the local level using multiple measures of mortality and morbidity. In addition, no studies were found that evaluated the progress toward achieving the 2000 goals at the local level.

This study was designed to assess Chicago's progress in achieving the goals of reducing and ultimately eliminating health disparities. We used rate ratios for 1980-1998 to examine differences in mortality and morbidity for black and white people and for low-income and high-income community areas. Selected mortality and morbidity measures were utilized for this analysis. Among these measures were three that address focus areas defined by President Clinton: infant mortality, cardiovascular disease, and diabetes. These measures allowed us to determine Chicago's success in achieving the 2000 goal of reducing racial/ethnic health disparities and thus gain some insight into the national pathway toward eliminating these disparities.

METHODS

Population

According to 2000 US Census data obtained from the Chicago Department of Public Health, Chicago is the third largest city in the country, with a racial/ethnic breakdown as follows: 31% non-Hispanic white, 36% non-Hispanic black, 26% Hispanic, 4% Asian, and 3% "other races."

More than 50 years ago, a research team at the University of Chicago divided the city into 75 "community areas" based on social, cultural, and geographic factors.²³ Since then, two community areas have been added, for a total of 77.

Measures

We looked at leading causes of death, all-cause mortality, communicable diseases, and birth outcomes to track disparities in health.

First, we explored the 10 leading causes of death in 1998 for black people and white people in the United States.²⁴ All-cause mortality and four site-specific cancers (lung, female breast, colorectal, and prostate) were also considered. These 17 causes of death and their respective International Classification of Diseases, Ninth Revision (ICD-9) codes are listed in Figure 1. In addition, we examined the incidence of two communicable diseases, AIDS and tuberculosis, and two birth outcome measures, infant mortality and low birth-weight (<2500 grams) births. In all, 22 measures of mortality (including all-cause mortality) and morbidity were analyzed for 1980-1998.

These measures of mortality and morbidity were examined for disparities in two ways. First, we used 1980-1998 data to compare rates for black residents of Chicago ($n = 1,065,009$ per 2000 Census data obtained from the Chicago Department of Public Health) with rates for white residents of Chicago ($n = 1,215,315$ per the 2000 Census). Because Hispanic ethnicity information was not collected until 1989, the Hispanic population was not analyzed as a separate group. Instead, data were analyzed by race with no regard for ethnicity.

Second, we compared mortality and morbidity rates for the 10 community areas with the lowest median household income (with a combined population of 233,707 per the 1990 Census) to the 10 with the highest (with a combined population of 298,797 per the 1990 Census).²⁵

Data sources

Vital statistics data were obtained from Illinois Department of Public Health Vital Records tapes (death and birth files). Surveillance data were acquired through

Figure 1. Measures used in analyses and associated ICD-9 codes

Mortality due to leading cause of death	ICD-9 code
Heart disease	390–398, 402, 404–429
Stroke	430–438
All cancer	140–208
Lung cancer	162.2–162.9
Female breast cancer	174
Liver disease	571
Homicide/legal intervention	E960–E978
Unintentional injuries	E800–E949
HIV/AIDS	136.3, 279.1 (before 1987) 042–044 (after 1986)
Diabetes	250
Pneumonia/influenza	480–487
Colorectal cancer ^a	153–154
Prostate cancer ^a	185
Chronic obstructive pulmonary disease ^a	490–496
Suicide ^a	E950–E959
Nephritis, nephrotic syndrome, nephrosis ^a	580–589
Perinatal conditions ^a	760–779
All-cause mortality	
Communicable diseases	
AIDS	
Tuberculosis	
Birth outcomes	
Infant mortality	
Low birthweight	

^aCauses of death not included in the low-income/high-income disparities analysis

the Office of HIV/AIDS Surveillance and the Tuberculosis Control Program of the Chicago Department of Public Health. All data were provided to us without any personal identifying information.

To study low-income/high-income disparities, we used existing data available through published Chicago Department of Public Health reports,^{23,25} which included three-year average annual age-adjusted mortality and morbidity rates as well as 1990 US Census demographic information on all 77 community areas of Chicago. For each community area, the following measures were readily available for analysis: mortality associated with 12 leading causes of death (including all causes), incidence rates for two communicable diseases, and two birth outcomes (Figure 1). Therefore,

a total of 16 measures were investigated for the following years: 1979–1981, 1991–1993, and 1996–1998.

Exponential interpolation between the 1980, 1990, and 2000 Census figures was used to estimate population denominators for intercensal years. Because the age distribution of the 2000 Census was not yet available, the age distribution of the 1998 estimated figures (obtained from Claritas, 5375 Mira Sorrento Place, San Diego, CA 92121) was applied to 2000 US Census estimates prior to interpolation. Age distributions were used in order to age-adjust the mortality rates.

Statistical analysis

Mortality rates were directly age-adjusted to the 1940 US population and expressed as the number of deaths per 100,000 population. Rates of communicable diseases (AIDS and tuberculosis) were calculated as the number of cases per 100,000 population. The infant mortality rate is expressed as the number of deaths of infants younger than one year of age per 1,000 live births. Percent of low birthweight births is presented as the number of newborns weighing less than 2500 grams per 100 live births. Black:white rate ratios (calculated as the black rate divided by the white rate) were used to examine the racial disparity in mortality and morbidity rates for 1980–1998.

Three-year average annual rates for selected health measures for community areas were used in our analysis of income disparity. Three time periods, 1979–1981, 1991–1993, and 1996–1998, were selected in order to investigate time trends. Rates were averaged and weighted to account for the population sizes of the selected community areas. The low income:high income community area rate ratio for each measure was then calculated for the selected time intervals.

Data analysis involved three steps. First, in order to evaluate racial health disparities, we calculated the black:white rate ratios for each year from 1980 through 1998. We then tested the 1980, 1990, and 1998 rate ratios for significance using a Taylor Series expansion to generate 95% confidence intervals (CIs).²⁶ Second, linear regression was employed to test the significance of these 19-year time trends in the black:white rate ratios. Beta coefficients and *p*-values were used to assess significance. Finally, low-income:high-income rate ratios were also calculated and tested for significance for the three sets of years noted above.

RESULTS

Black-white disparities: 1980, 1990, and 1998

Table 1 shows the black:white rate ratios for the selected causes of death, communicable diseases, and

birth outcomes for the specified years. Three summary observations can be made from these data. First, the rate ratios for all but one measure (liver disease mortality) increased from 1980 to 1998. For 19 of the 22 measures, the rate ratios increased significantly. Second, of the 63 available rate ratios in Table 1, only 7 were not statistically significant. Finally, all 22 of the slopes assessing the time trends in these black:white rate ratios were positive.

Almost all of the 1980 mortality and morbidity rate ratios were >1 and statistically significant. The largest mortality rate ratios were for homicide (RR = 3.10; 95% CI 2.69, 3.57), prostate cancer (RR = 2.33; 95% CI 1.84, 2.96) and nephritis/nephrotic syndrome/nephrosis (RR = 2.27; 95% CI 1.77, 2.90). The rate ratios were not statistically significant for female breast cancer, colorectal cancer, chronic obstructive pulmonary disease, and unintentional injuries. The suicide rate ratio was the only measure that was <1 and statistically significant (RR = 0.45; 95% CI 0.33, 0.61). That is, in 1980, black people were about half as likely to die from suicide as white people.

By 1990, all the rate ratios had increased by at least 11% (lung cancer) and as much as 82% (perinatal conditions). In addition, all of the rate ratios, except the one for HIV/AIDS mortality, were statistically significant. In 1980, four measures of mortality and morbidity had rate ratios >2 , but by 1990, ratios for seven of the measures were at least that high. The greatest increases ($>50\%$) occurred for suicide, homicide, unintentional injuries, and perinatal conditions. Moreover, the infant mortality rate ratio rose 44% to 2.6 (95% CI 2.26, 3.00).

Eight years later, the rate ratios had continued to increase to the point where, in most cases, the black rate was at least twice the white rate. From 1980 to 1998, rate ratios (excluding the ratio for liver disease mortality) increased by at least 12% (perinatal conditions) and as much as 112% (colorectal cancer). The liver disease mortality rate ratio actually decreased and became statistically insignificant during this time. In addition, the suicide rate ratio increased to the point of no significance (RR = 0.85; 95% CI 0.64, 1.15) by 1998; that is, blacks and whites were at equal risk of dying from suicide. From 1990 to 1998, the rate ratios for HIV mortality increased by 251%, while the rate ratio for AIDS incidence increased by 158%. By 1998, the HIV epidemic had dramatically impacted black people in Chicago, who were three times (RR = 3.14; 95% CI 2.74, 3.61) as likely as whites to be diagnosed with AIDS and almost four times (RR = 3.90; 95% CI 3.10, 4.92) as likely to die from HIV.

Figures 2–5 display the consistent trends for selected

causes of mortality and morbidity. The graphs illustrate how increases in disparity may be produced in different ways. For instance, Figure 2 shows that the all-cause mortality rate for whites decreased across the interval from 1980 to 1998, while the rate for black people remained virtually unchanged in the decade 1988–1998. This was reflected in a 1980 rate ratio of 1.30, a 1998 rate ratio of 2.03, and a statistically significant positive trend in the rate ratios over the interval. In another scenario, the increasing disparity in mortality due to heart disease (Figure 3) resulted from differential improvements for both groups in which the white rate decreased at a much faster pace than the black rate. Figure 4 demonstrates growing disparity resulting from a dramatic decline in the white female breast cancer rate coupled with a black rate that increased considerably over the same time period. Finally, Figure 5 illustrates that a consistently low diabetes mortality rate for whites paired with an increasing rate for blacks produced an increasing disparity.

Low- and high-income community areas: 1979–1981, 1991–1993, and 1996–1998

Table 2 illustrates the striking differences between the low- and high-income community areas. For instance, the average median household income (\$42,612) of the lowest income community areas in 1990 was almost five times as high as the average median household income (\$8,911) of the highest income areas. In addition, the low-income areas were predominantly non-Hispanic black whereas the high-income areas were primarily non-Hispanic white. Finally, the lowest income areas had a lower percentage of high school graduates and a higher percentage of people living below the poverty level. By comparing the health measures of these two sets of community areas, we can assess economic disparities in health.

Table 3 displays the low-income:high-income rate ratios for all deaths, 11 leading causes of death, two communicable diseases, and two birth outcomes for the selected years. Two overarching observations may be made. First, for 12 of the 16 measures, the rate ratios increased every subsequent period. In fact, the rate ratios for all but two measures (liver disease mortality and low birthweight births) increased from 1979–1981 to 1996–1998. Second, 46 of the 48 available rate ratios shown in Table 3 were >1 and statistically significant.

The rate ratios for 1979–1981, with the exception of the ratio for female breast cancer mortality, show that people living in the lowest median income community areas were at least 1.5 times as likely as those in the highest median income areas to have a given out-

Table 1. Age-adjusted black:white rate ratios with 95% confidence intervals and regression parameters for selected measures of mortality and morbidity, Chicago, 1980, 1990, 1998

Measure	1980		1990		1998		β
	RR	95% CI	RR	95% CI	RR	95% CI	
Mortality due to leading cause of death ^a							
Heart disease	1.13	1.09, 1.18	1.33	1.27, 1.39	1.81	1.72, 1.90	0.052
Stroke	1.41	1.28, 1.56	1.74	1.55, 1.95	2.30	2.04, 2.60	0.047
All cancer	1.30	1.23, 1.37	1.50	1.42, 1.58	2.02	1.91, 2.14	0.051
Lung cancer	1.41	1.27, 1.57	1.57	1.41, 1.74	2.19	1.96, 2.45	0.061
Female breast cancer	1.02	0.84, 1.23	1.45	1.20, 1.75	2.07	1.69, 2.53	0.060
Colorectal cancer	0.96	0.84, 1.13	1.33	1.14, 1.56	2.03	1.70, 2.44	0.064
Prostate cancer	2.33	1.84, 2.96	2.64	2.14, 3.26	3.30	2.64, 4.13	0.078
Chronic obstructive pulmonary disease	1.06	0.88, 1.27	1.45	1.24, 1.69	1.93	1.65, 2.25	0.045
Liver disease	1.25	1.07, 1.46	1.58	1.33, 1.87	1.09	0.88, 1.35	0.010
Suicide	0.45	0.33, 0.61	0.68	0.53, 0.88	0.85	0.64, 1.15	0.024
Homicide/legal intervention	3.10	2.69, 3.57	4.73	4.03, 5.55	3.93	3.31, 4.65	0.108
Unintentional injuries	1.03	0.90, 1.17	1.70	1.50, 1.93	1.86	1.63, 2.11	0.052
HIV/AIDS ^b	—	—	1.11	0.94, 1.31	3.90	3.10, 4.92	0.160
Nephritis, nephrotic syndrome, nephrosis	2.27	1.77, 2.90	2.61	2.10, 3.26	3.02	2.44, 3.72	0.064
Perinatal conditions	1.64	1.39, 1.94	3.00	2.45, 3.66	1.85	1.45, 2.35	0.044
Diabetes	1.48	1.23, 1.77	1.95	1.65, 2.30	2.23	1.90, 2.61	0.046
Pneumonia/influenza	1.57	1.33, 1.85	1.94	1.69, 2.22	2.27	1.95, 2.65	0.056
All-cause mortality	1.30	1.27, 1.33	1.60	1.56, 1.64	2.03	1.98, 2.09	0.054
Communicable diseases							
AIDS ^b	—	—	1.22	1.08, 1.38	3.14	2.74, 3.61	0.198
Tuberculosis	—	—	3.20	2.72, 3.78	3.37	2.74, 4.13	0.119
Birth outcomes							
Infant mortality	1.81	1.61, 2.04	2.60	2.26, 3.00	2.31	1.94, 2.75	0.031
Low birthweight	2.12	2.01, 2.24	2.52	2.40, 2.66	2.25	2.13, 2.37	0.003

NOTE: Shading indicates RRs or β values that are not statistically significant ($p > 0.05$).

^a β values based on linear regression of year vs RR. Except where indicated, all trends were statistically significant ($p < 0.005$).

^bThere were < 5 AIDS cases or AIDS-related deaths in 1980.

RR = risk ratio

CI = confidence interval

come (Table 3). For some measures, people in the lowest income community areas experienced a four- or five-fold higher risk compared to their wealthier counterparts. For instance, the rate ratio was 4.04 (95% CI 3.56, 4.58) for low birthweight births, 4.24 (95% CI 3.02, 5.96) for tuberculosis, and 5.52 (95% CI 4.03, 7.57) for homicide. In addition, Chicago's lowest income areas experienced an infant mortality rate almost four times as high (RR = 3.87; 95% CI 2.87, 5.21) as that of the highest income areas. The only measure that failed to be statistically significant in 1979–1981 was the rate ratio for female breast cancer (RR = 1.14; 95% CI 0.77, 1.68).

Just over a decade later, in 1991–1993, the disparities in health had increased to the point that, for almost all measures, the lowest income community areas suffered from rates at least twice those of their highest income neighbors. Again, one of the exceptions was female breast cancer (RR = 1.08; 95% CI 0.70, 1.65). The all-cause mortality rate ratio increased by 26% from 1979–1981 to 1991–1993. Increases in cause-specific mortality rate ratios ranged from 2% (liver disease) to 110% (unintentional injury). The largest increases (>30%) occurred for stroke mortality, unintentional injury mortality, infant mortality, and low birthweight births. The greatest disparities in 1991–

Figure 2. All-cause mortality, Chicago, 1990 to 1998: black and white rates and black:white rate ratios

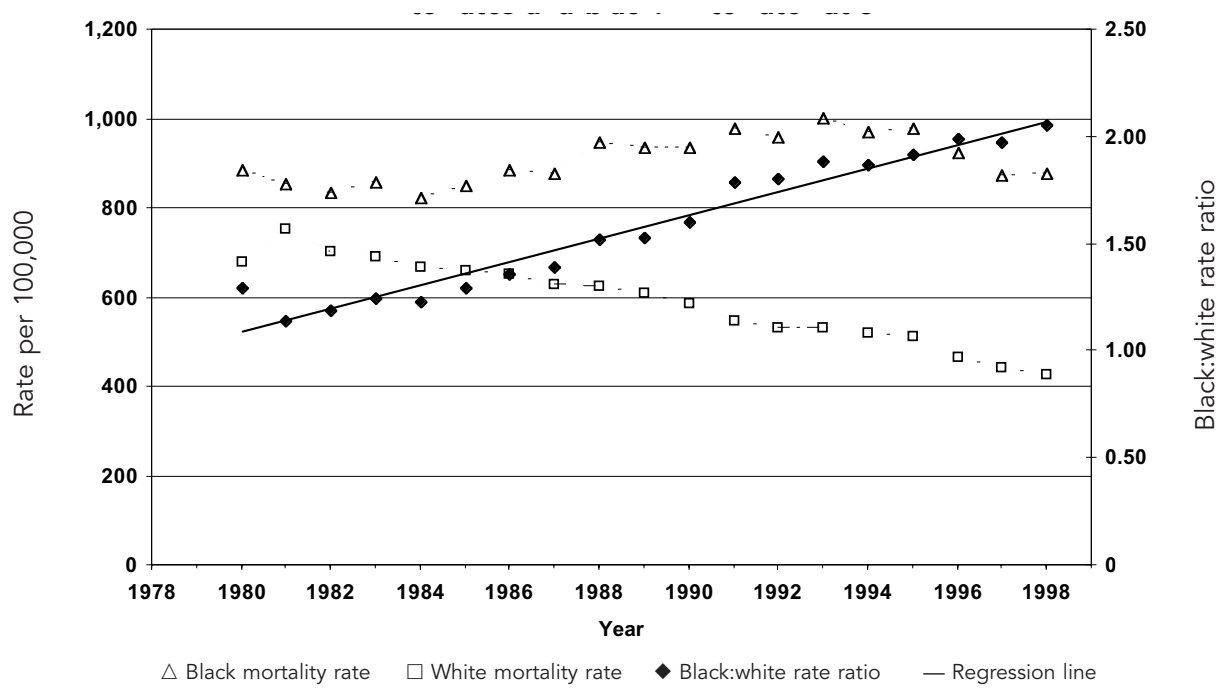


Figure 3. Heart disease mortality, Chicago, 1980 to 1998: black and white rates and black:white rate ratios

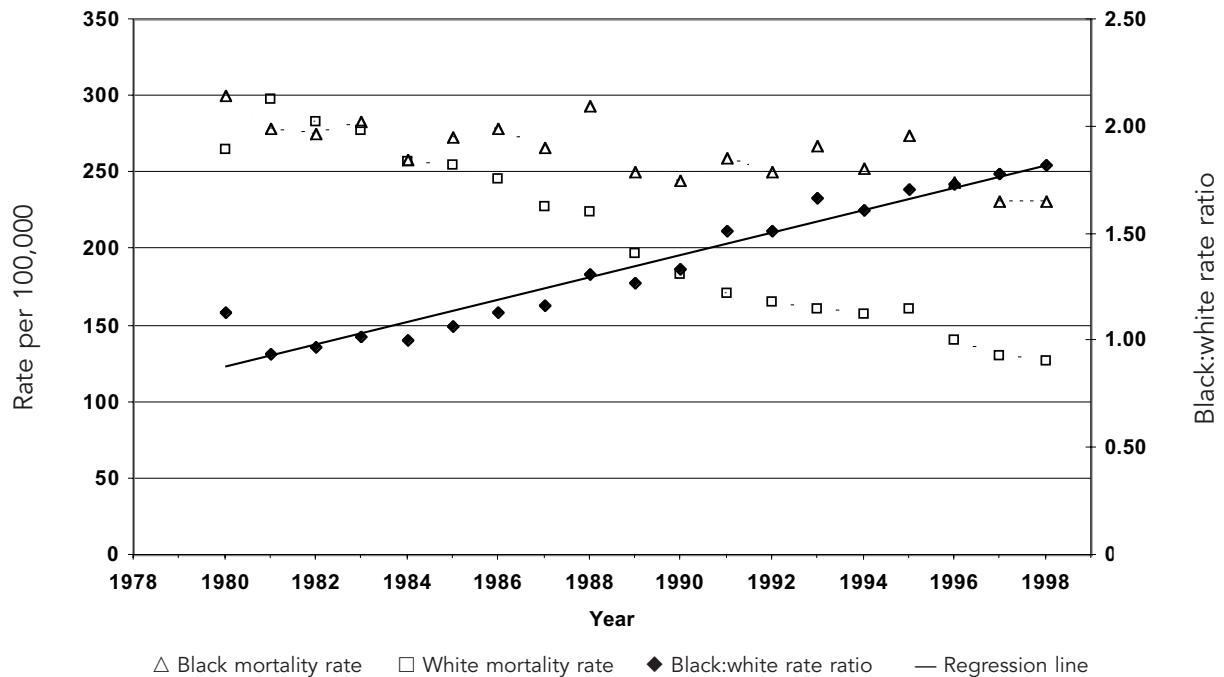


Figure 4. Female breast cancer mortality, Chicago, 1980 to 1998: black and white rates and black:white rate ratios

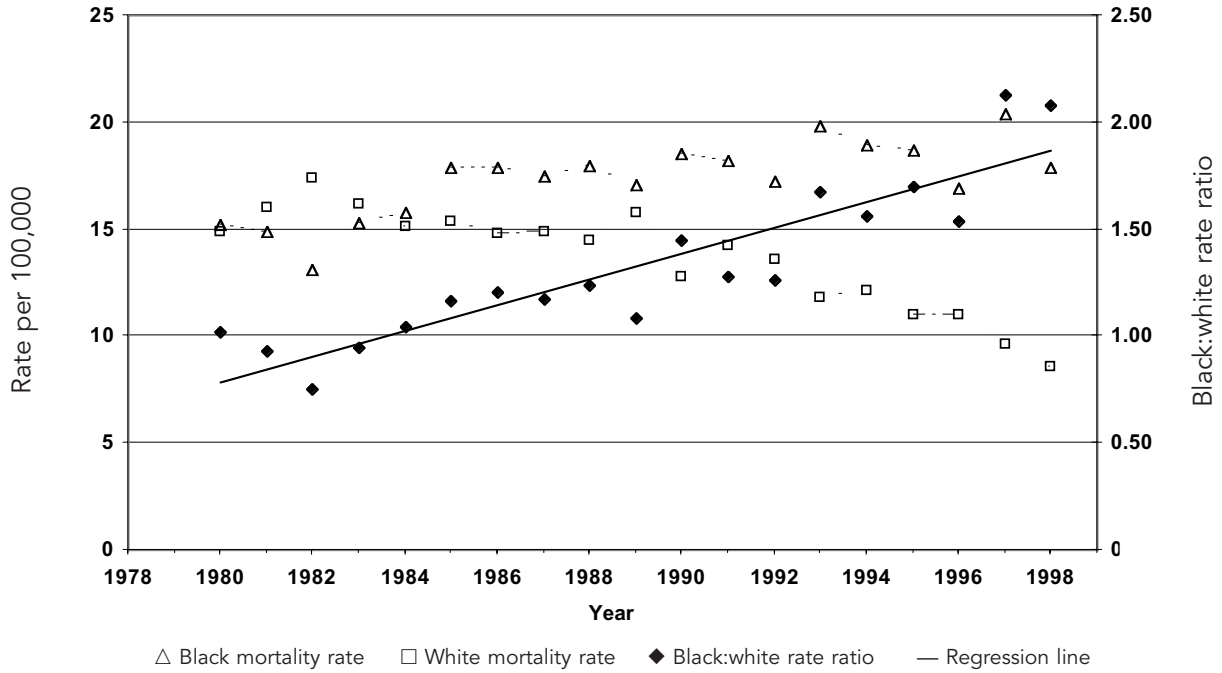


Figure 5. Diabetes mortality, Chicago, 1990 to 1998: black and white rates and black:white rate ratios

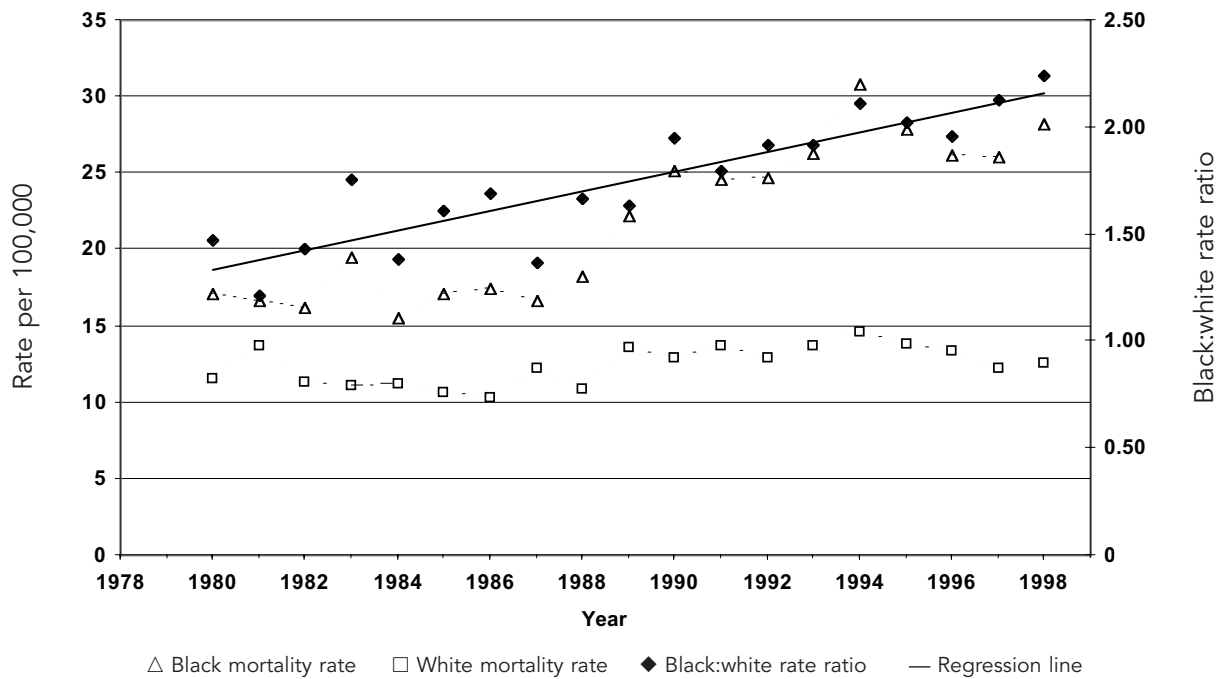


Table 2. Characteristics of 10 community areas with the lowest median household income compared to those of the 10 areas with the highest median household income, Chicago, 1990

Characteristic	Lowest	Highest
Population	233,707	298,797
Percent of population that was non-Hispanic black	90.6	15.3
Percent of population that was non-Hispanic white	4.9	78.4
Percent of population that was Hispanic	2.9	4.1
Percent of high school graduates among adults ages > 25 years	52.6	84.8
Percent of population below poverty line	55.0	8.5
Mean of median household income	\$8,911	\$42,612

1993 were found for homicide (RR = 6.54; 95% CI 4.61, 9.28), unintentional injury (RR = 3.27; 95% CI 2.43, 4.40), infant mortality (RR = 5.61; 95% CI 3.91, 8.05), low birthweight births (RR = 5.42; 95% CI 4.78, 6.14), and tuberculosis (RR = 5.38; 95% CI 3.77, 7.67).

By 1996–1998, with the exception of the rate ratio for female breast cancer mortality, all rate ratios were >2. Further, the female breast cancer mortality rate ratio was now statistically significant at 1.66 (95% CI

1.02, 2.70). Although most of the rate ratios had increased, some had decreased. For instance, from 1991–1993 to 1996–1998, rate ratios dropped by 11% for liver disease mortality, 19% for infant mortality, and 37% for low birthweight births. Even so, the rate ratios for these measures remained statistically significant. One exceptional increase that occurred during this time was the 110% increase in the HIV mortality rate ratio. By 1996–1998, people living in the lowest in-

Table 3. Age-adjusted low-income:high-income rate ratios with 95% confidence intervals for selected measures of mortality and morbidity, Chicago

Measure	1979–1981		1991–1993		1996–1998	
	RR	95% CI	RR	95% CI	RR	95% CI
Mortality due to leading cause of death						
Heart disease	1.45	1.32, 1.59	1.86	1.65, 2.09	2.56	2.24, 2.92
Stroke	1.45	1.15, 1.56	1.93	1.55, 1.95	2.76	2.04, 2.60
All cancer	1.50	1.34, 1.69	1.63	1.43, 1.85	2.14	1.86, 2.47
Lung cancer	1.55	1.22, 1.97	1.89	1.46, 2.44	2.39	1.83, 3.13
Female breast cancer	1.14	0.77, 1.68	1.08	0.70, 1.65	1.66	1.02, 2.70
Liver disease	2.84	2.06, 3.90	2.90	1.97, 4.25	2.59	1.56, 4.29
Homicide/legal intervention	5.52	4.03, 7.57	6.54	4.61, 9.28	7.28	4.81, 11.02
Unintentional injuries	1.56	1.19, 2.04	3.27	2.43, 4.40	3.41	2.48, 4.67
HIV/AIDS ^a	—	—	2.58	1.80, 3.71	5.41	3.34, 8.76
Diabetes	2.03	1.31, 3.16	2.49	1.62, 3.82	2.56	1.68, 3.91
Pneumonia/influenza	2.17	1.49, 3.17	2.76	1.90, 4.00	3.04	2.05, 4.53
All-cause mortality	1.72	1.62, 1.82	2.16	2.03, 2.30	2.68	1.98, 2.09
Communicable diseases						
AIDS ^a	—	—	2.60	2.01, 3.36	3.65	2.67, 4.98
Tuberculosis	4.24	3.02, 5.96	5.38	3.77, 7.67	5.57	3.67, 8.44
Birth outcomes						
Infant mortality	3.87	2.87, 5.21	5.61	3.91, 8.05	4.52	2.91, 7.02
Low birthweight	4.04	3.56, 4.58	5.42	4.78, 6.14	3.44	3.02, 3.92

NOTE: Shading indicates RRs that are not statistically significant ($p > 0.05$). RRs are based on 3-year averages of rates, and are significant ($p < 0.05$) except where indicated.

^aThere were <10 HIV-related deaths or cases in 1979–1980.

RR = risk ratio

CI = confidence interval

come community areas were 5.41 times as likely as those living in the highest income areas to die of HIV-related causes.

DISCUSSION

Our findings paint a stark picture of Chicago's persistent racial inequalities in health. Despite an overall decline in the city's mortality rates from 1980 to 1998, black and white people still died at increasingly disparate rates. This is demonstrated by the large and increasing rate ratios. Statistically significant black:white rate ratios were observed for almost every measure and time period analyzed (Table 1), as were statistically significant low-income:high-income rate ratios (Table 3). The findings are even more credible given the wide choice of measures. These ranged from all-cause mortality, to mortality associated with various cancers, homicide, diabetes, pneumonia and influenza, to infant mortality, to the incidence of AIDS and tuberculosis. The only measure in which blacks fared better than whites was the suicide rate. Even here, however, the situation grew comparatively worse for black people, as the rate ratio gradually increased from 1980 to the point where by 1998 the white rate was no longer significantly larger than the black rate. The widening of differences in mortality and morbidity was also evident in the increasing trend in rate ratios. For all but three of the 22 measures, the rate ratios significantly increased over the 19-year period.

We also compared experiences for people who lived in the 10 community areas with the lowest median household income with those of people who lived in the 10 highest median income areas. Although these areas were almost entirely segregated and thus generally corresponded to racial groupings (Table 2), we were interested to see how those at the extremes of the income distribution compared. The results were in a similar direction to those in the black:white analyses but revealed even greater disparities (Table 3). For example, the black:white rate ratio for infant mortality for the last time period studied was 2.31, while it was 4.52 for the low-income:high-income comparison. Similarly, the black:white ratio for HIV-related mortality was 3.90 and the low-income:high-income ratio was 5.41. For virtually all the measures in any given time period, the low-income:high-income rate ratios were statistically significant. Female breast cancer mortality was the only measure that did not achieve statistical significance until 1996–1998. Likewise, for almost every measure, the low-income:high-income rate ratios increased over time.

The results of both analyses demonstrate that con-

trary to the Healthy People 2000 goals, health disparities in Chicago had increased so significantly by 1998 that for most of the measured indicators of mortality and morbidity, black and low-income people experienced at least twice the risk of their white or high-income counterparts. The trend data provide absolutely no evidence to suggest that excess mortality and morbidity among black and low-income people will not continue to increase.

Previous studies have examined racial differentials in certain measures at select points in time.^{11–13,27} Some other studies have cited trends in all-cause mortality.^{14,16–19} While the results of these studies lend support to our findings, none considered as many time periods for as many measures of health. The consistent racial disparities we observed across virtually every one of the 22 health measures we analyzed suggest that our findings are strong and reliable. An additional contribution of this study was the uncovering of the magnitude and trend of these disparities at the local level. Given the large population of the city of Chicago, the findings are suggestive for other large urban areas. The fact that they are consistent with the findings of Williams,^{14–16} Geronimus,^{18,19} and Pappas¹⁷ for selected measures and time periods further strengthens their generalizability. For instance, Williams documents that although the overall US black mortality rate has declined over time, the mortality rate for several causes of death such as cancer and diabetes was higher in 1995 than in 1950. In fact, he notes that the black:white rate ratio for all-cause mortality remained unchanged over this 45-year period.¹⁴

It is difficult to imagine weaknesses of the data being responsible for any of these findings. Numerators were generally in the range of hundreds and thousands and denominators were in the hundred thousands and even millions, thus lending reliability and stability to the calculations. Although some miscodings of race, address, and cause of death likely occurred, they would not have generated such a large number of consistent findings across measures.

One methodological area of concern is the allocation of Hispanic people. Since Hispanic coding of both population and cause of death was not available until 1989 and not reliable in Illinois until the early 1990s, it was not possible to compute comparisons between non-Hispanic whites and non-Hispanic blacks and still consider time trends going back to 1980. The question is: To what extent does the inability to separate Hispanic whites from non-Hispanic whites alter our findings? We have done considerable analyses of these groups of rates. When we calculated the age-adjusted all-cause mortality rate for whites and com-

pared it with that for non-Hispanic whites, the rates for 1989 through 1998 differed by no more than 5%. Thus, had we been able to also analyze long-term trends in rate ratios for non-Hispanic blacks compared to non-Hispanic whites, we would most likely have generated similar findings.

As Cooper and his colleagues,^{28,29} Krieger and hers,^{30,31} and others have shown over and over again, race is not a biological category but a socially constructed one. Several historical studies have also documented this fact.³²⁻³⁴ Thus, we are not measuring the effects of biology but of those characteristics associated with the social construction of race. Indeed, there are fundamental causes that place some of the most disadvantaged populations at risk of suffering from higher rates of illness and mortality. Much has been written about social determinants of health^{35,36} such as environmental stresses, inequalities in access to education and health care, income inequality, and especially racism^{37,38} and segregation,³⁹⁻⁴¹ which work together in a complex way to impede improvements in health and quality of life for some of the most impoverished communities in our city and nation.⁴²

One absolutely essential question that must be posed is why existing disparities, still as formidable as they were in 1980, continue to widen and do so in the face of a prominent national campaign against them and in an era that experienced almost unparalleled economic growth. Furthermore, in the past two decades there have been major advancements in the prevention, detection, and treatment of chronic diseases in the United States. These advancements have been seen for a number of diseases including hypertension, stroke, heart disease, female breast cancer, and AIDS among others.⁴³⁻⁴⁹ Our analysis indicates that despite public health campaigns, general economic prosperity, and health advances, the health status of significant portions of the population, the black and poor, in Chicago has gotten relatively worse compared to that of the white and wealthier populations. One thing is virtually certain: if we keep doing the same things we have been doing, we will be sitting here in 2010 wondering why the racial disparities in health are still increasing.

A multifaceted problem such as this begs for an equally comprehensive solution that entails broad economic, social, and political changes that allow everyone equal access to the opportunities that will lead to healthy lives.⁵⁰ Geronimus warns that anything short of understanding and changing the socioeconomic, historical, and structural factors that help create and maintain populations that live in poverty, segregation, and ill health inhibits our goal of eliminating health disparities.⁵¹ As Cohen and Northridge have written, "It is

impossible to have a frank discussion of inequality . . . without confronting the continuing blight of racism head on" (p. 841); only in truly committing ourselves to solving inequities in access and opportunity can we begin to make strides toward achieving equality.⁵² To do otherwise is to allow disparities to exist and almost certainly grow. This cannot be an option.

REFERENCES

1. Ren XS, Amick BC. Racial and ethnic disparities in self-assessed health status: evidence from the National Survey of Families and Households. *Ethn Health* 1996;1:293-303.
2. Mason JO. Understanding the disparities in morbidity and mortality among racial and ethnic groups in the United States. *Ann Epidemiol* 1993;3:120-4.
3. Moss N. Socioeconomic disparities in health in the US: an agenda for action. *Soc Sci Med* 2000;51:1627-38.
4. Effective interventions for reducing racial and ethnic disparities in health. *Am J Public Health* 2001;91:485-6.
5. Mansfield CJ, Kirk D, Curry MD, Bobbitt-Cooke M. The challenge of eliminating health disparities in North Carolina. *N C Med J* 2001;62:19-25.
6. Bruechner JS. Health disparities among racial and ethnic groups in Rhode Island. *Med Health R I* 2000;83:257-8.
7. Department of Health and Human Services (US). Eliminating racial and ethnic disparities in health: response to the Presidential Initiative on Race [reprint]. *Public Health Rep* 1998;113:372-5.
8. Department of Health and Human Services (US). Healthy people 2010: understanding and improving health. Washington: DHHS; 2000.
9. National Center for Health Statistics (US). Health, United States, 1998 with socioeconomic status and health chartbook. Hyattsville (MD): NCHS; 1998.
10. Walker B, Figgs LW, Zahm SH. Differences in cancer incidence, mortality, and survival between African Americans and whites. *Environ Health Perspect* 1995;103:275-81.
11. Ng-Mak DS, Dohrenwend BP, Abraido-Lanza AF, Turner JB. A further analysis of race differences in the National Longitudinal Mortality Study. *Am J Public Health* 1999; 89:1748-51.
12. Smith GD, Neaton JD, Wentworth D, Stamler R, Stamler J. Mortality differences between black and white men in the USA: contribution of income and other risk factors among men screened for the MRFIT. *Lancet* 1998;351:934-9.
13. McNamara KM. Racial disparities in health: a sociological analysis. *J S C Med Assoc* 1999;95:95-8.
14. Williams DR. Race, socioeconomic status, and health: the added effects of racism and discrimination. *Ann NY Acad Sci* 1999;896:173-88.
15. Williams DR. Race and health: trends and policy implications. In: Auerbach JA, Krimgold BK, editors. *Income,*

- status, socioeconomic status, and health. Washington: National Policy Association; 2001. p. 67-85.
16. Williams DR, Collins C. US socioeconomic and racial differences in health. *Annu Rev Sociol* 1995;21:349-86.
 17. Pappas G, Queen S, Wilbur H, Fisher G. The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *N Engl J Med* 1993;329:103-9.
 18. Geronimus AT, Bound J, Waidmann TA, Hillemeier MM, Burns PB. Excess mortality among blacks and whites in the United States. *N Engl J Med* 1996;355:1552-8.
 19. Geronimus AT. Poverty, time, and place: variation in excess mortality across selected US populations, 1980-1990. *J Epidemiol Community Health* 1999;53:325-34.
 20. Polednak AP. Black-white differences in sentinel causes of death: counties in large metropolitan areas. *J Urban Health* 2000;77:501-7.
 21. Polednak AP. Mortality in Hartford, Connecticut: a comparison with the South Bronx, New York. *J Urban Health* 1998;75:550-7.
 22. McCord C, Freeman HP. Excess mortality in Harlem. *N Engl J Med* 1990;322:173-7.
 23. Chicago Department of Public Health. Community area health inventory. Vol. 1: Demographic and health profiles. Chicago: The Department; 1994 Oct.
 24. National Center for Health Statistics (US). Health, United States, 1996. National Vital Statistics Report Vol. 48, No. 11. Hyattsville (MD): NCHS; 1996.
 25. Chicago Department of Public Health. Community area health inventory, 1996-1998. Vol. 1: Demographic and health profiles. Chicago: The Department; 2000 May.
 26. Kleinbaum DG, Kupper LL, Morgenstern H. Epidemiologic research: principles and quantitative methods. Belmont (CA): Lifetime Learning Publications; 1982.
 27. Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality. *JAMA* 1998;279:1703-8.
 28. Cooper R, David R. The biological concept of race and its application in public health and epidemiology. *J Health Polit Policy Law* 1986;11:97-116.
 29. Cooper RS. Health and social status of blacks in the United States. *Ann Epidemiol* 1993;3:137-44.
 30. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research. *Annu Rev Public Health* 1997;18:341-78.
 31. Krieger N, Fee E. Social class: the missing link in U.S. health data. *Int J Health Serv* 1994; 24:25-44.
 32. Allen TW. The invention of the white race. New York: Verso Press; 1994.
 33. Ignatiev N. How the Irish became white. New York: Rotheledge Press; 1995.
 34. Bennett L. Before the Mayflower: a history of black America. New York: Penguin USA; 1993.
 35. Link BG, Phelan JC. Social conditions as fundamental causes of disease. *J Health Soc Behav* 1995;80-94.
 36. Sorlie PD, Backlund E, and Keller JB. US mortality by economic, demographic, and social characteristics: the national longitudinal study. *Am J Public Health* 1995; 85:949-56.
 37. Krieger N, Rowley DL, Herman AA, Avery B, Phillips MT. Racism, sexism, and social class: implications for studies of health, disease, and well being. *Am J Prev Med* 1993; 9:82-122.
 38. Ren XS, Amick BC, Williams DR. Racial/ethnic disparities in health: the interplay between discrimination and socioeconomic status. *Ethn Dis* 1999;9:151-65.
 39. Collins CA. Racism and health: segregation and causes of death amenable to medical intervention in major U.S. cities. *Ann N Y Acad Sci* 1999;896:396-8.
 40. Williams DR. Racism and health: a research agenda. *Ethn Dis* 1996;6:1-6.
 41. Polednak AP. Segregation, discrimination, and mortality in U.S. blacks. *Ethn Dis* 1996;6:99-108.
 42. Susser M. The logic in ecological: I: the logic of analysis. *Am J Public Health* 1994;84:825-9.
 43. Gueyffier F, Froment A, Gouton M. New meta-analysis of treatment trials of hypertension: improving the estimate of therapeutic benefit. *J Hum Hypertens* 1996;10: 1-8.
 44. Antiplatelet Trialists' Collaboration. Collaborative overview of randomised trials of antiplatelet therapy I: prevention of death, myocardial infarction, and stroke by prolonged antiplatelet therapy in various categories of people. *BMJ* 1994;305:81-106.
 45. Early Breast Cancer Trialists' Collaborative Group. Polychemotherapy for early breast cancer: an overview of the randomised trials. *Lancet* 1998;351:30-42.
 46. Early Breast Cancer Trialists' Collaborative Group. Tamoxifen for early breast cancer: an overview of the randomized trials. *Lancet* 1998;351:1451-67.
 47. Cohen OJ, Fauci AS. Current strategies in the treatment of HIV infection. *Adv Intern Med* 2001;46:207-46.
 48. Dutcher JP, Novik Y, O'Boyle K, Marcoullis G, Secco C, Wiernik PH. 20th-century advances in drug therapy in oncology. Part I. *J Clin Pharmacol* 2000;40:1007-24.
 49. Sakamoto S, Takeda Y, Nakabayashi M. Advances in perinatal medical care—from our experience. *Int J Gynaecol Obstet* 1998;63:S107-14.
 50. Kaplan GA, Lynch JW. Is economic policy health policy? *Am J Public Health* 2001;91:351-3.
 51. Geronimus AT. To mitigate, resist, or undo: addressing structural influences on the health of urban populations. *Am J Public Health* 2000;90:867-72.
 52. Cohen HW, Northridge ME. Getting political: racism and urban health. *Am J Public Health* 2000;90:841-2.