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Drugs and Firearm Deaths in New York City, 1990–1998

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ABSTRACT *Firearm deaths remain among the leading causes of mortality in the United States. Changing law enforcement activities, incarceration, drug use, and socioeconomic conditions may have played roles in the declining rates of firearm deaths during the 1990s. Using records from the Office of the Chief Medical Examiner, we analyzed the role of drugs in firearm deaths in New York City between 1990 and 1998. Positive drug toxicology was present in over half of all firearm death victims during this time. Cocaine, cannabis, opiates, and alcohol accounted for almost all of these deaths with drug-positive toxicology. There were decreases in cocaine- and alcohol-positive toxicology for firearm deaths in New York City starting in the early 1990s; there was a more gradual decrease in heroin-positive toxicology for firearm deaths. Cannabis-positive toxicology for firearm deaths increased in the early part of the 1990s and then decreased starting in the mid-1990s. Although the disparities between minority and white firearm death rates narrowed during this time, minorities remained about three times more likely to be victims of fatal firearm violence than whites in 1998. The highest firearm death rates were among African American and Latino male decedents, with a larger proportion of Latinos testing cocaine or opiate positive, while a larger proportion of African Americans tested cannabis positive. These results suggest a complex role of drugs in firearm-related deaths.*

KEYWORDS *Drugs, Firearms, Law Enforcement.*

INTRODUCTION

In 1998, deaths due to firearms, including homicides, suicides, and accidents, claimed the lives of approximately 30,000 Americans.[1] Firearms are currently the leading means of homicide, accounting for about 65% of all homicides in the United States and are the leading means of suicide, accounting for 57% of all suicides in the United States.[1,2] In 1997, firearm homicide was the second leading cause of death among all persons aged 15 to 24 years and firearm suicide the third leading cause of death among all persons aged 15 to 24 years.[3] During the last decade, firearm mortality has changed markedly throughout much of the United States; a steep increase in firearm deaths in the late 1980s was followed by a decline in deaths starting around 1993–1994 in most US major metropolitan areas.[4]

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A pattern of declining firearm incidents has been observed in New York City. The New York City Mayor's Management Report indicates that shooting incidents in New York City dropped 62.5%, from 5,269 in 1993 to 1,977 in 1997; the number of shooting victims dropped 61.4%, from 5,862 in 1993 to 2,262 in 1997. Arrests for gun possession dropped 43% during the same period.[5]

A number of factors have been proposed as reasons for the decline in firearm incidents during the 1990s; these include changing patterns of alcohol and illicit drug use, law enforcement activities, changing demographics, escalating incarceration rates, and improving overall socioeconomic conditions.[6–9] Unfortunately, the relative contributions of these factors to the changing rates of firearm deaths in New York City remain poorly understood.[10] For example, the 1990s were a period of unprecedented, sustained prosperity in much of the United States, including 8 consecutive years of economic growth and record low unemployment rates; the likely relation among increasing legitimate job opportunities, the attendant lower need for persons to become involved in higher-risk, illegal activities (e.g., drug trade), and the decline in firearm deaths remains unclear.

Changing patterns of illicit drug use are frequently considered one of the primary causes of changing patterns of firearm deaths.[11] The increase in homicides in the late 1980s, driven primarily by a rise in firearm homicides, coincided with the introduction of crack cocaine to big American cities such as New York City and Los Angeles, California.[10,12] In some cities, homicide rates started rising within a year of the introduction of crack.[10] The high profits possible in the crack trade, a depressed overall economy, and limited opportunities for legitimate employment contributed to the involvement of greater numbers of inner-city youths in drug dealing.[13] The rapid increase in illegal firearms used to protect crack businesses was likely a primary factor in the rise in firearm murder rates observed in the late 1980s.

Changes in policing aimed at decreasing drug possession have also been cited as a principal "cause" of the decrease in firearm-related violence in New York City.[8] In March 1994, the New York City Police Department (NYPD) introduced its "Police Strategy No. 1: Getting Guns Off the Streets of New York."[14] The gun strategy primarily involved using technologically advanced equipment, expanding patrols and training, and increasing public support to reduce firearm crimes. This strategy coincided with the introduction of the CompStat program in New York City, which allowed more direct precinct-level decision making, and was subsequently bolstered by an expansion of the NYPD's Street Crime Unit by 300 officers in February 1997 and a citywide reorganization of borough units to fight firearm offenses.[15,16] Changes in law enforcement activity and in patterns of drug use both then probably played a role in the decline in firearm deaths in New York City during the 1990s.

We were interested in clarifying the involvement of drugs in firearm mortality in New York City. This study examined all firearm deaths in New York City between 1990 and 1998. New York City was under the jurisdiction of one chief medical examiner at this time, thus providing an opportunity for consistent reporting of causes of death and determination of drug toxicology. We were interested in determining (1) the changing prevalence of drugs involved in firearm deaths between 1990 and 1998, (2) the prevalence of drug involvement in firearm deaths in persons of different racial/ethnic groups between 1990 and 1998, and (3) the relative proportion of drug involvement in firearm deaths in different racial/ethnic groups between 1990 and 1998.

METHODS

All cases of fatal firearm deaths, including homicides, suicides, accidents, and deaths from undetermined causes, in New York City from 1990 through 1998 were identified through manual review of all medical files at the Office of the Chief Medical Examiner of New York (OCME). The OCME is responsible for assessing all deaths of persons believed to have died in an unnatural manner. Thus, all firearm deaths in New York City would have been reviewed by the OCME and included in this chart extraction. Data regarding demographics, cause of death, race/ethnicity, circumstance of death, and toxicology were collected from the OCME files. The OCME investigators use the decedent's medical history, the circumstances and environment of the fatality, autopsy findings, and laboratory data in attributing cause of death and other criteria to each case reviewed.

Rates of autopsy vary by manner of death. During 1990–1998, complete autopsy was performed for 99.8% of homicides, 85.6% of suicides, 79.4% of accidents, and 88.2% of deaths by undetermined manner. All autopsied cases undergo toxicological screening. In some cases when an autopsy was not performed, specimens were still submitted for toxicologic analysis.

Blood and urine samples were obtained at autopsy and stored at 4°C until they were assayed. Benzoylecgonine, the principal metabolite of cocaine, was screened in urine samples using a homogenous enzyme immunoassay. Positive results (≥0.3 mg/L) from urine samples were substantiated in testing of blood samples by radioimmunoassay. If urine samples were not readily available at autopsy, benzoylecgonine was screened in blood by radioimmunoassay; the screen was considered positive if the concentration of benzoylecgonine was equal to or greater than 0.1 mg/L and was substantiated by radioimmunoassay in another tissue. Specimens positive for benzoylecgonine suggest that cocaine could have been used up to 2 days before death. Blood was screened for cocaine by gas chromatography involving a nitrogen-phosphorus detector. A specimen was considered positive if the concentration of cocaine was equal to or greater than 0.1 mg/L; all samples considered positive were analyzed by gas chromatography. For the purposes of this study, specimens were considered positive for cocaine if they were positive for cocaine or benzoylecgonine.

Initial alcohol screening was done on blood, and alcohol analysis was performed by headspace gas chromatography using a flame ionization detector. A specimen was considered positive for alcohol if the concentration was equal to or greater than 0.01 gram percent. Opiates were screened by enzyme immunoassay of urine or, if urine was not available, by radioimmunoassay of blood; specimens that contained a concentration of 0.3 mg/L or greater in urine or 0.1 mg/L or greater in blood were considered positive, and results were substantiated by radioimmunoassay in another tissue. Other drug screens were performed; their presence was confirmed by a variety of analytic techniques, including enzyme immunoassay, thin-layer and high-performance liquid chromatography, and gas chromatography–mass spectrometry. The reader is referred to previous studies for further details on this data collection methodology and toxicological measurement of drugs involved in these cases.[17–20]

Statistical analysis was carried out on all firearm deaths. We first described relevant demographic characteristics of all firearm deaths in New York City between 1990 and 1998; demographic characteristics considered included age, race, gender, manner of death, borough of death, place of death, and drugs detected.

Manner of death was derived from the OCME files; the medical examiner attribution of death is based on both clinical evidence and the circumstances of death as noted above. We focused our observations on the principal drugs that appear in positive drug toxicology: cocaine, opiates, cannabis, and alcohol. The four groups of drugs categorized in these analyses are not mutually exclusive; that is, a decedent can have positive toxicology for several of these drugs at the same time. We also created an "any drug" category to include all persons who screened positive toxicologically for any drug.

We calculated the total firearm death rate in New York City for each year between 1990 and 1998. We also calculated the rate of firearm deaths with positive drug toxicology; the rate of firearm deaths with any combination of cocaine-, opiates-, cannabis-, or alcohol-positive toxicology, and the separate rates of firearm deaths with positive toxicology for cocaine, opiates, cannabis, and alcohol. The numbers of cases used to determine rates were obtained by manual extraction of firearm deaths from the OCME files as described here. All population denominators for rate determination were obtained from US census data.[21] We obtained census population counts for New York City for 1990 and 2000 and carried out a linear interpolation of the census population counts to obtain population denominators for the intervening years. All rates were expressed per 100,000 person-years. We subsequently directly standardized overall rates for New York City by age, sex, and race to the 1990 census population for New York City to increase comparability between years relevant to this analysis.

We calculated firearm death rates in New York City between 1990 and 1998 stratified by race and sex. We limited our analysis to the three largest racial/ethnic groups in New York City: white, African American, and Latino. Thus, we calculated the rate of firearm deaths and the rate of firearm deaths with positive toxicology for cocaine, opiates, cannabis, or alcohol within each of six racial/gender strata: white male, white female, African American male, African-American female, Latino male, Latina female. We did not standardize these rates since all rates were calculated within race and sex strata.

We calculated the proportion of firearm deaths with positive cocaine, opiate, cannabis, or alcohol toxicology among firearm deaths for the strata with the highest overall firearm death rates—African American men and Latino men—to compare the relative prevalence of drugs involved in firearm deaths. Rates for all of New York City, rates by sex and race, and proportions of drugs detected in decedents were explored descriptively.

RESULTS

There were 11,133 firearm deaths extracted from OCME files in New York City between 1990 and 1998. More than a third of these decedents (36.8%) were between 15 and 24 years old; a third (33.7%) were between 25 and 34 years old (Table 1). A plurality of decedents was African American (48.1%); 35.9% were Latino, and 12.0% were white. The majority of decedents were male (91.4%), and a majority of firearm deaths were classified as homicides by the OCME (87.5%). Deaths were distributed throughout four New York City boroughs, with 20.0% in Manhattan, 26.0% in the Bronx, 35.1% in Brooklyn, and 16.7% in Queens; only a small proportion of deaths were in Staten Island (2.3%). Over half of all firearm

TABLE 1. Demographics of firearm deaths, New York City, 1990–1998

Demographic characteristics	Number	Percentage
Total	11,133	100.0
Age, years		
Under 14	131	1.2
15–24	4,100	36.8
25–34	3,747	33.7
35–44	1,766	15.9
45–54	796	7.1
55–64	350	3.1
65–74	160	1.4
Over 74	83	0.7
Race		
White	1,337	12.0
African American	5,358	48.1
Hispanic	4,002	35.9
Other	436	3.9
Gender		
Male	10,176	91.4
Female	957	8.6
Manner of death		
Homicide	9,745	87.5
Suicide	1,074	9.6
Accident	69	0.6
Murder-suicide	214	1.9
Undetermined	31	0.3
Borough of death		
Manhattan	2,227	20.0
Bronx	2,981	26.0
Brooklyn	3,906	35.1
Queens	1,858	16.7
Staten Island	251	2.3
Place of death		
Residence	3,245	29.1
Other inside	2,218	19.9
Outside	5,429	48.8
Not recorded	241	2.2
Drugs detected		
Any drug	6,153	55.3
Any cocaine, opiates, cannabis, or alcohol	6,126	55.0
Cocaine	2,696	24.2
Opiates	1,067	9.6
Cannabis	2,168	19.5
Alcohol	2,995	26.9

deaths (55.3%) had positive drug toxicology; the most prevalent four drugs were cocaine (24.2%), opiates (9.6%), cannabis (19.5%), and alcohol (26.9%). Detailed demographic characteristics of the overall sample are presented in Table 1.

The total number of firearm deaths decreased from 1,720 in 1990 to 526 in 1998. The firearm death rate in 1990 was 23.49 deaths per 100,000 person-years, and the comparable age-, race-, and sex-adjusted firearm death rate in 1998 was 6.30 deaths per 100,000 person-years (Table 2). The proportion of firearm deaths with positive drug toxicology did not change markedly between 1990 and 1998; in 1990, there were 917/1720 (53.3%) of firearm deaths with positive drug toxicology, and in 1998, this number was 284/526 (54.0%). Almost all the positive drug toxicology during this period of analysis was accounted for by cocaine, opiates, cannabis, or alcohol; 913/917 (99.6%) of all cases with positive drug toxicology in 1990 were positive for at least one of these four drugs, with the same true for 284/284 (100%) of cases in 1998. Firearm deaths with cocaine-positive toxicology were the most common among those involving illicit drugs in 1990 (6.81 persons per 100,000 person-years), but had declined to 0.97 persons per 100,000 person-years in 1998; in 1998, deaths with cannabis-positive toxicology were more common than those with cocaine-positive toxicology, with an age-, sex-, and race-adjusted rate of 1.47 persons per 100,000 person-years. Firearm deaths for which the individuals were positive for opiates were consistently lower than those deaths with cocaine-positive toxicology throughout the period studied; deaths with opiate-positive toxicology occurred at a rate of 2.51 per 100,000 person-years in 1990 and at an adjusted rate of 0.58 per 100,000 person-years in 1998. Alcohol was the most common of all drugs in both 1990 and 1998 (6.98 per 100,000 person-years and 1.79 per 100,000 person-years, respectively), although rates for cocaine were higher in 1991 and 1992. Detailed counts and crude and adjusted rates for firearm deaths between 1990 and 1998 are presented in Table 2.

Overall trends in firearm death rates and firearm death rates with positive drug toxicology are presented in Fig. 1. The relative importance of cocaine among the illicit drugs throughout the time interval is highlighted in the graphic trend. The graphs also illustrate the decline in overall firearm deaths that started in 1991. Cocaine-positive toxicology in firearm deaths declined between 1990 and 1998; opiate deaths also declined, albeit more gradually, between 1990 and 1995 and plateaued between 1996 and 1998. Deaths with cannabis-positive toxicology, however, climbed between 1990 and 1993 and subsequently declined between 1993 and 1998. The rate of firearm deaths with cannabis-positive toxicology overtook the rate of firearm deaths with cocaine-positive toxicology in 1994, while the rate of deaths with cocaine-positive toxicology remained higher than that of deaths with opiate-positive toxicology throughout the time interval. Deaths with alcohol-positive toxicology declined steadily over the 9-year period.

In considering gender and racial/ethnic strata, firearm death rates were higher among African American men than among any other stratum throughout the time period studied; firearm death rates among Latino men were second highest throughout the same period. In 1990, there were 88.67 firearm deaths per 100,000 person-years among African American men and 72.45 firearm deaths per 100,000 person-years among Latino men (compared to 11.32 deaths per 100,000 person-years among white men); these rates had declined to 28.56 deaths per 100,000 person-years among African American men and 13.76 deaths per 100,000 person-years among Latino men (compared to 6.06 deaths per 100,000 person-years among

TABLE 2. Total firearm deaths and positive drug toxicology, New York City, 1990–1998

	1990		1991			1992			1993			1994		
	N	Crude rate	N	Crude rate	Adjusted rate*	N	Crude rate	Adjusted rate*	N	Crude rate	Adjusted rate*	N	Crude rate	Adjusted rate*
Total firearm deaths	1,720	23.49	1,828	24.73	24.14	1,729	23.18	22.16	1,669	22.17	20.85	1,331	17.52	16.22
Any drug	917	12.52	996	13.48	13.17	972	13.03	12.43	931	12.37	11.60	765	10.07	9.25
Any cocaine, opiates, cannabis, or alcohol	913	12.47	994	13.45	13.15	967	12.96	12.37	927	12.31	11.55	759	9.99	9.18
Cocaine	499	6.81	516	6.98	6.83	478	6.41	6.14	404	5.37	5.12	292	3.84	3.59
Opiates	184	2.51	179	2.42	2.41	171	2.29	2.20	173	2.30	2.18	135	1.78	1.67
Cannabis	164	2.24	286	3.87	3.78	327	4.38	4.15	371	4.93	4.54	310	4.08	3.66
Alcohol	511	6.98	487	6.59	6.47	457	6.13	5.87	402	5.34	5.09	356	4.69	4.37

	1995			1996			1997			1998		
	N	Crude rate	Adjusted rate*	N	Crude rate	Adjusted rate*	N	Crude rate	Adjusted rate*	N	Crude rate	Adjusted rate*
Total firearm deaths	956	12.47	11.74	785	10.15	9.47	589	7.55	7.10	526	6.68	6.30
Any drug	521	6.80	6.38	424	5.48	5.14	343	4.40	4.07	284	3.61	3.37
Any cocaine, opiates, cannabis, or alcohol	520	6.78	6.37	421	5.44	5.11	341	4.37	4.05	284	3.61	3.37
Cocaine	183	2.39	2.33	151	1.95	1.93	94	1.2	1.19	79	1.00	0.97
Opiates	82	1.07	1.06	58	0.75	0.74	44	0.56	0.58	41	0.52	0.58
Cannabis	234	3.05	2.72	174	2.25	2.03	169	2.17	1.91	133	1.69	1.47
Alcohol	255	3.33	3.19	214	2.77	2.64	166	2.13	2.04	147	1.87	1.79

*Adjusted rate directly standardized by age, sex, and race to 1990 census population for New York City.

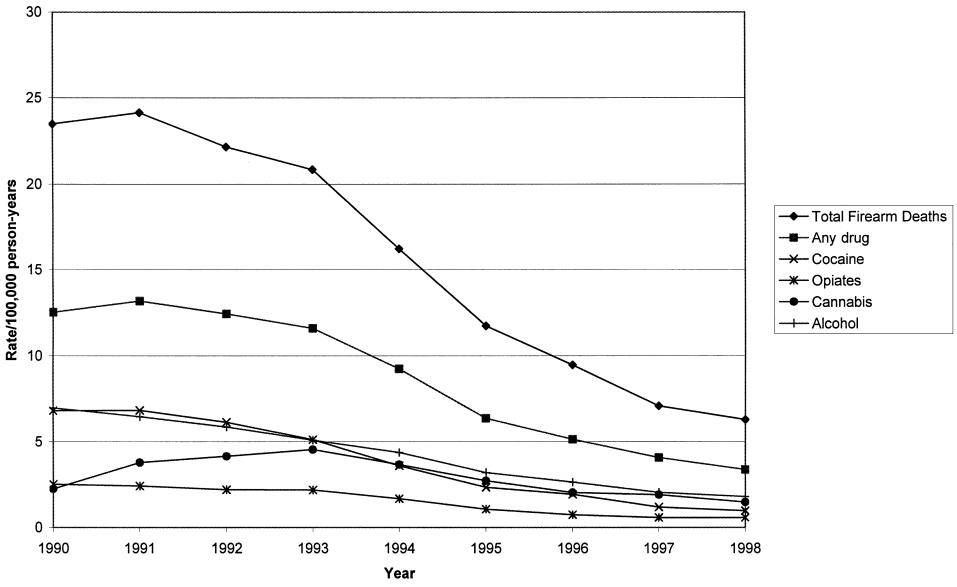


FIGURE 1. Standardized firearm death rates with drugs detected, New York City, 1990–1998.

white men) in 1998. African American and Latina female firearm deaths were comparable and were consistently higher than rates among white females throughout the period; the firearm death rate among African American women was 5.88 per 100,000 person-years; among Latino women, it was 5.57 per 100,000 person-years; and among white women, it was 1.20 per 100,000 person-years in 1990. This compared to 1.95 per 100,000 person-years among African American women, 0.92 per 100,000 person-years among Latino women, and 0.53 per 100,000 person-years among white women in 1998. Trends in firearm death rates by sex and race between 1990 and 1998 are presented in Fig. 2.

Among African American men, firearm death rates with positive cocaine toxicology were consistently higher than deaths with opiate-positive toxicology throughout the period studied; as observed in the overall firearm death trends, rates with cannabis-positive toxicology exceeded rates with cocaine-positive toxicology in 1993. Death rates with alcohol-positive toxicology were similar to and consistently higher than rates with cocaine-positive toxicology. Among Latino men, firearm death rates with cocaine-positive toxicology were higher than rates for all other drugs until 1995, when death rates with alcohol-positive toxicology exceeded death rates with cocaine-positive toxicology. Among white men, deaths with alcohol-positive toxicology were consistently highest. Detailed rates of drugs involved in the six major gender and racial/ethnic groups studied are presented in Table 3.

We also calculated the proportion of drugs involved in firearm death rates among African American and Latino men as these two groups had the highest firearm death rates. Among Latino male firearm deaths, 33% had cocaine-positive toxicology in 1990, and 18% were positive toxicologically for cocaine in 1998. These proportions were higher than proportions among African Americans; 30% of African American male firearm deaths were positive toxicologically for cocaine in 1990, and 15% were positive toxicologically for cocaine in 1998 (Fig. 3a). Similarly, the proportions of Latino male firearm deaths with opiate-positive toxicology

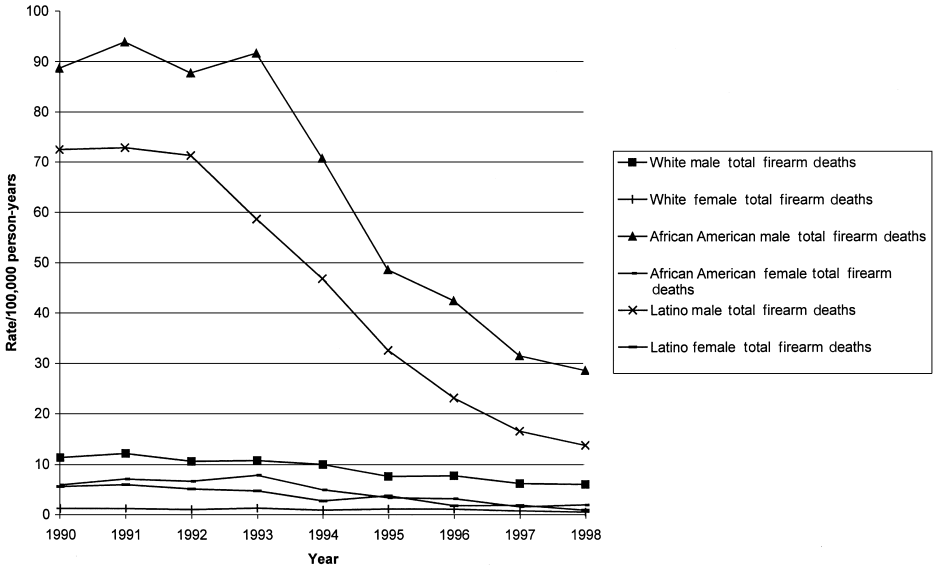


FIGURE 2. Firearm death rates by sex and race, 1990–1998, New York City.

in 1990 (12%) and 1998 (8%) were higher than the proportions of African American male firearm deaths with opiate-positive toxicology during the same years (11% and 7% in 1990 and 1998, respectively) (Fig. 3b). Conversely, the proportion of African American male firearm deaths with cannabis-positive toxicology was 12% in 1990 and 34% in 1998, which was consistently higher than rates for Latinos (9% and 23% in 1990 and 1998, respectively) (Fig. 3c). Overall proportions of firearm deaths with cocaine- and opiate-positive toxicology thus declined among African American and Latino men between 1990 and 1998; the proportion of deaths positive toxicologically for cannabis increased between 1990 and 1998 in these same groups. The proportions of African American and Latino male decedents with toxicology positive for alcohol was consistent between 1990 and 1998 at between 25% and 30% (Fig. 3d).

DISCUSSION

There are several observations arising from this study. First, over half of all firearm deaths had positive drug toxicology throughout the study period. This lends further support to the role of alcohol and illicit drugs in the study of firearm mortality. Second, overall firearm deaths with positive drug toxicology and firearm deaths with positive cocaine toxicology declined between 1991 and 1998; cocaine was positive toxicologically in a decreasing proportion of firearm deaths in African American and Latino males (the two largest groups of decedents) during the same period. Third, the rates of firearm deaths with opiate-positive toxicology have been consistently lower than firearm deaths with cocaine-positive toxicology, and the decline has been slower for opiates than it was for cocaine. Fourth, firearm deaths with cannabis-positive toxicology rose between 1990 and 1993 and declined subsequently, but at a slower rate than did firearm deaths positive toxicologically for cocaine; as such, cannabis overtook cocaine as the most common illicit drug associ-

TABLE 3. Firearm deaths and positive drug toxicology by race and sex, New York City, 1990–1998

Race and sex	Drug detected	1990		1991		1992		1993		1994		1995		1996		1997		1998	
		N	Crude rate	N	Crude rate	N	Crude rate	N	Crude rate	N	Crude rate	N	Crude rate	N	Crude rate	N	Crude rate	N	Crude rate
White male	Total firearm deaths	169	11.32	180	12.18	155	10.60	156	10.78	143	9.99	108	7.63	109	7.78	86	6.21	83	6.06
	Cocaine	39	2.61	29	1.96	28	1.91	30	2.07	23	1.61	16	1.13	18	1.28	6	0.43	10	0.73
	Opiates	18	1.21	20	1.35	14	0.96	18	1.24	8	0.56	8	0.56	9	0.64	7	0.51	12	0.88
	Cannabis	11	0.74	12	0.81	16	1.09	16	1.11	14	0.98	9	0.64	8	0.57	12	0.87	11	0.80
	Alcohol	49	3.28	61	4.13	48	3.28	47	3.25	46	3.21	34	2.40	32	2.28	29	2.09	23	1.68
White female	Total firearm deaths	20	1.20	20	1.21	17	1.04	21	1.31	15	0.95	18	1.15	17	1.10	12	0.79	8	0.53
	Cocaine	4	0.24	4	0.24	2	0.12	7	0.44	1	0.06	3	0.19	0	0	2	0.20	0	0
	Opiates	2	0.12	1	0.06	2	0.12	3	0.19	4	0.25	3	0.19	1	0.06	1	0.07	0	0
	Cannabis	0	0	2	0.12	1	0.06	0	0	3	0.19	1	0.06	1	0.06	2	0.13	0	0
	Alcohol	5	0.30	3	0.18	1	0.06	8	0.50	1	0.06	8	0.51	4	0.26	3	0.20	2	0.13
African American male	Total firearm deaths	733	88.67	781	93.93	734	87.78	771	91.68	598	70.71	413	48.6	363	42.44	271	31.51	247	28.56
	Cocaine	219	26.49	217	26.10	197	23.56	166	19.74	122	14.43	80	9.41	61	7.13	37	4.30	36	4.16
	Opiates	79	9.56	64	7.70	69	8.25	61	7.25	51	6.03	30	3.53	21	2.46	16	1.86	16	1.85
	Cannabis	89	10.77	169	20.33	167	19.97	212	25.21	183	21.64	141	16.58	120	14.03	98	11.40	82	9.48
	Alcohol	238	28.79	219	26.34	201	24.04	201	23.90	158	18.68	105	12.35	93	10.87	78	9.07	73	8.44

TABLE 3. *Continued*

Race and sex	Drug detected	1990 N	1990 Crude rate	1991 N	1991 Crude rate	1992 N	1992 Crude rate	1993 N	1993 Crude rate	1994 N	1994 Crude rate	1995 N	1995 Crude rate	1996 N	1996 Crude rate	1997 N	1997 Crude rate	1998 N	1998 Crude rate
African American female	Total firearm deaths	60	5.88	73	7.11	69	6.67	82	7.88	52	4.96	39	3.40	34	3.20	17	1.59	21	1.95
	Cocaine	18	1.76	26	2.53	26	2.51	24	2.31	13	1.24	7	0.66	11	1.04	1	0.09	4	0.37
	Opiates	8	0.78	3	0.29	5	0.48	5	0.48	4	0.38	3	0.28	2	0.19	3	0.28	0	0
	Cannabis	6	0.59	7	0.68	8	0.77	10	0.96	7	0.67	6	0.57	3	0.28	7	0.66	4	0.56
	Alcohol	13	1.27	12	1.17	8	0.77	14	1.35	10	0.95	12	1.14	8	0.75	3	0.28	6	0.56
Latino male	Total firearm deaths	616	72.45	633	72.82	633	71.26	532	58.63	434	46.85	308	32.58	223	23.12	163	16.57	138	13.76
	Cocaine	203	23.88	222	25.54	211	23.75	168	18.51	127	13.71	69	7.30	58	6.01	43	4.37	25	2.49
	Opiates	74	8.70	81	9.32	72	8.10	74	8.16	60	6.48	31	3.28	23	2.38	13	1.32	11	1.10
	Cannabis	54	6.35	91	10.47	128	14.41	122	13.45	98	10.58	67	7.09	37	3.84	43	4.37	32	3.19
	Alcohol	187	21.99	176	20.25	174	19.59	121	13.33	126	13.60	88	9.31	70	7.26	47	4.78	40	3.99
Latino female	Total firearm deaths	52	5.57	57	5.99	50	5.15	47	4.75	28	2.78	39	3.80	19	1.82	20	1.88	10	0.92
	Cocaine	13	1.39	14	1.47	13	1.34	6	0.61	4	0.40	8	0.78	3	0.29	3	0.28	1	0.09
	Opiates	2	0.21	9	0.95	6	0.62	9	0.91	5	0.50	4	0.39	2	0.19	3	0.28	1	0.09
	Cannabis	3	0.32	1	0.11	5	0.52	7	0.71	2	0.20	4	0.39	5	0.48	3	0.28	1	0.09
	Alcohol	9	0.96	9	0.95	12	1.24	4	0.40	4	0.40	3	0.29	4	0.38	3	0.28	0	0

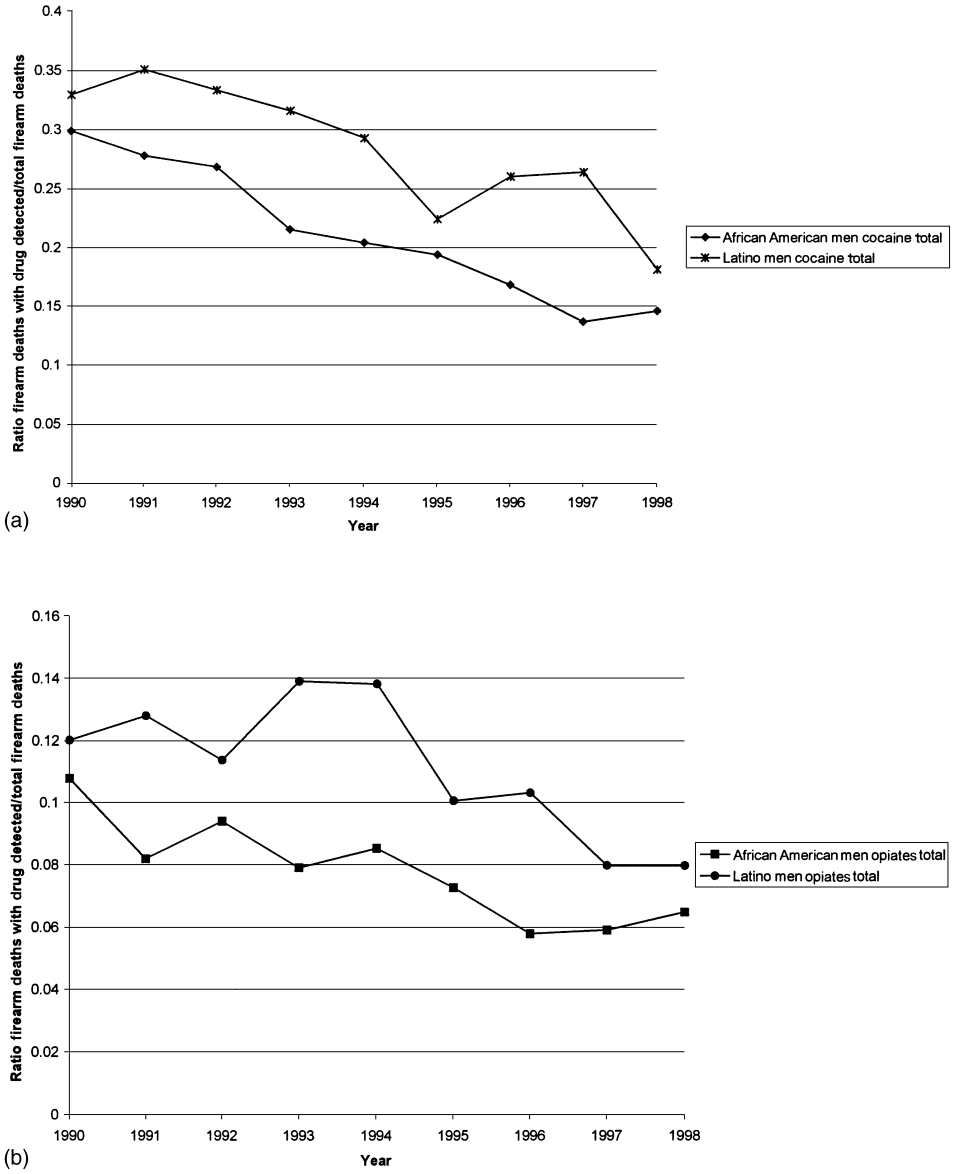
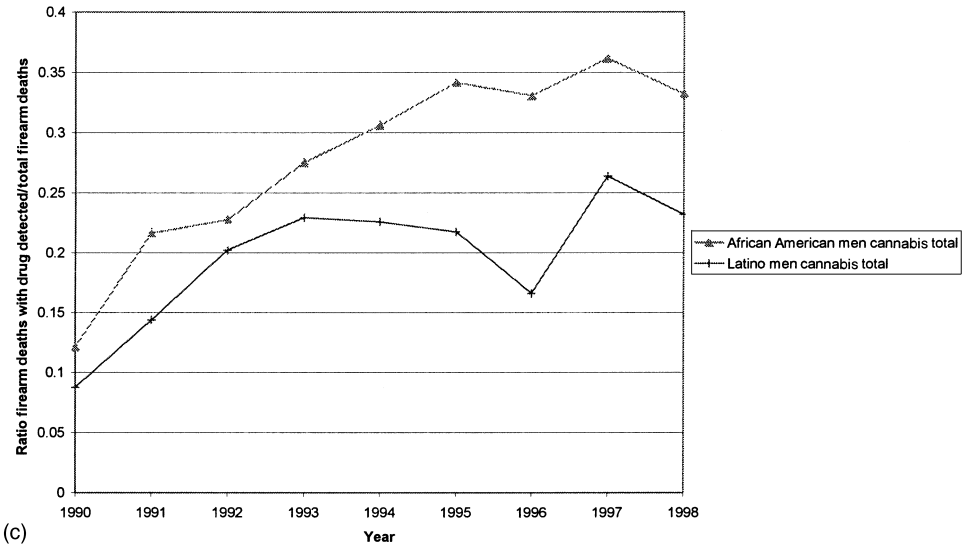
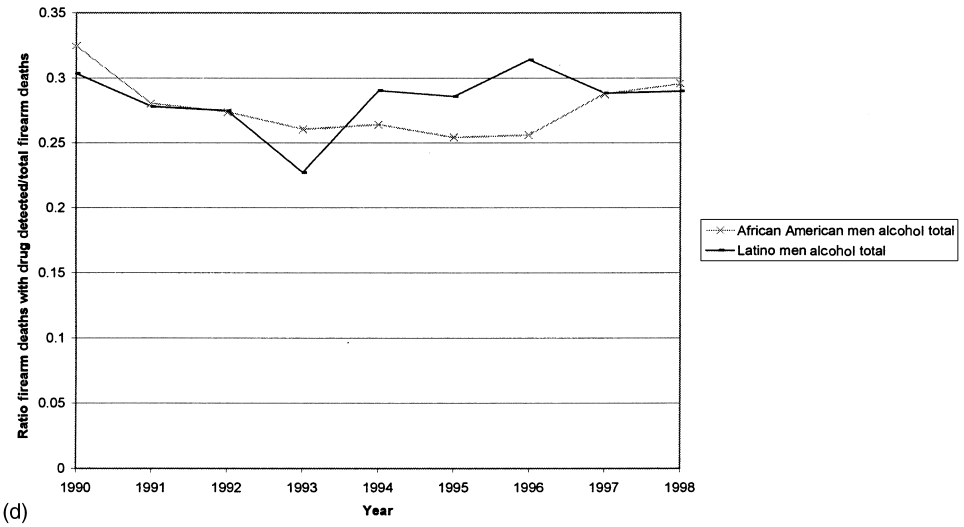


FIGURE 3. Ratio of firearm death rates with a drug detected to total firearm death rate for African American and Latino men.

ated with firearm deaths during 1994–1998. Fifth, firearm deaths with positive alcohol toxicology were consistently high, but declined throughout the 9 years of the study. Sixth, there was a tremendous disparity in firearm mortality among racial/ethnic groups in 1990, with firearm death rates among African Americans more than eight times that among whites, and among Latinos, it was more than six times that among whites. A steep decline in firearm deaths among minority groups has



(c)



(d)

FIGURE 3. Continued.

narrowed, but has not eliminated, these disparities; death rates among African Americans were four times that among whites, and among Latinos, it was two times that among whites in 1998. These disparities were more pronounced among men than women. Seventh, the proportion of firearm deaths positive toxicologically for either cocaine or opiates was consistently higher among Latino men than among African American men throughout the period studied; the proportion of firearm deaths positive toxicologically for cocaine declined two-fold between 1990 and 1998, while the proportion of firearm deaths with opiate-positive toxicology declined gradually between 1996 and 1998. Eighth, the proportion of firearm deaths positive toxicologically for cannabis was consistently higher among African Ameri-

can men than Latino men over the study period; the proportion of deaths with positive toxicology for cannabis increased three-fold between 1990 and 1998.

Rates of overall firearm deaths and firearm deaths with cocaine-positive toxicology declined in parallel throughout most of the 1990s. The role of cocaine in homicide fatalities in New York City during the past decade has been reported previously by several other authors.[13,22] Surveys of male suspects taken into custody have demonstrated that, during the 1990s, cocaine was the most commonly used illicit drug among lawbreakers.[13] It is worth noting that the first gradual decline in firearm deaths with cocaine-positive toxicology started at the beginning of the 1990s, pre-dating the major NYPD initiatives to decrease illegal firearms in New York City and the introduction of CompStat and the increasing NYPD focus on quality-of-life policing.[23] A steeper decline in firearm deaths toxicologically positive for cocaine started around 1993. The initial decline in firearm fatalities with cocaine-positive toxicology may have been linked to a waning of the crack epidemic in the late 1980s; further research is needed to determine if the subsequent steeper decline in firearm deaths with cocaine-positive toxicology was associated with changes in law enforcement activity. Although the decline in firearm death rates with cocaine-positive toxicology between 1993 and 1995 was steep, the subsequent decline between 1995 and 1998 was more gradual; this may suggest that levels of cocaine use are stabilizing.

Firearm deaths positive toxicologically for opiates were consistently lower than deaths with positive toxicology for cocaine throughout the decade. However, the more gradual decrease in deaths with opiate-positive toxicology resulted in the overall rates of firearm deaths with opiate-positive toxicology approaching the rates for firearm deaths with cocaine-positive toxicology in 1998. Research has suggested that availability of treatment (reducing dependence on heroin) and vigorous law enforcement activities are both related to decreases in opiate addictions.[24] In the New York City context, there was no appreciable increase in availability of drug treatment during the 1990s. At the same time, the decrease in deaths with opiate-positive toxicology was gradual during the decade and pre-dated most significant changes in law enforcement activities. These trends in firearm deaths with positive toxicology for both cocaine and opiates suggest that complex factors were involved in these changes, and that a simple causal model attributing the changes to police efforts is likely insufficient.

Trends in firearm fatalities positive toxicologically for cannabis were distinct from trends of cocaine and heroin, the other two illicit drugs discussed here. Firearm deaths with cocaine-positive toxicology rose in the earlier part of the decade and started a decline in the mid-1990s. In explaining the waning of the crack epidemic, it has been suggested that persons born in the 1970s (hence young adults in the 1990s), having witnessed the high personal and social toll associated with crack cocaine, did not initiate cocaine use at previous rates, resulting in much of the waning of the cocaine epidemic of the late 1980s.[13] Given that opiates did not rise either in the early 1990s, it is plausible that cannabis use may have replaced use of cocaine or opiates among certain segments of the population. The increase in proportion of deaths toxicologically positive for cannabis during the decade supports this hypothesis. Availability of marijuana has been associated with increasing weapon carrying among youths,[25,26] particularly among youths who live in risky environments and who are less likely to have coping resources to deal with social stressors.[27] Although longitudinal studies of the relation between drugs and violence among youths have not established a causal link,[28] it is feasible that the role of

cannabis in firearm-related death is following a different pattern from that of other illicit drugs. The decline of firearm deaths positive toxicologically for cannabis starting in the mid-1990s appears more closely related temporarily to increasing police efforts in New York City than is the decline in either cocaine or opiates.

It has been suggested that an overall decrease in alcohol use played a role during the 1990s in the decrease in homicide in the United States.[9] In our data, although firearm deaths with alcohol-positive toxicology declined, alcohol remained highly prevalent in all firearm deaths and was responsible for a consistent proportion of deaths in African American and Latino men (the two largest groups of decedents). This suggests that alcohol has had a role in firearm deaths between 1990 and 1998 that has not changed during these years. As with opiates, there was no specific change in law enforcement policy with regard to alcohol during the study period except for overall increased police enforcement of quality-of-life crimes that could have included decreasing alcohol consumed in public places.

Racial/ethnic disparities in morbidity and mortality have been documented in most of the leading causes of death in the United States.[29,30] There was a marked decrease in firearm death racial/ethnic disparities in New York City between 1990 and 1998. This observation is tempered, however, by the fact that, in 1998, rates of firearm deaths among minority men remained three times higher than firearm deaths among white men. Differential circumstances of drug use are likely associated with some of the disparities in firearm-related death. Although the involvement of cocaine in firearm deaths has been decreasing, the decrease in opiates has been gradual and appears to have plateaued between 1996 and 1998. The relatively higher proportions of firearm-related deaths among Latino men that are associated with cocaine and opiate use compared other ethnic groups is cause for concern. National surveys do not suggest disproportionately high cocaine, opiate, or cannabis use rates among Latinos[31]; our findings suggest the need for further study of this group. The larger proportion of firearm deaths with cannabis-positive toxicology among African American males is also worth further exploration.

There are a number of considerations relevant to the interpretation of results in this study. During the period of this study, New York City had the same chief medical examiner and endeavored to apply uniform guidelines to its reporting about all cases. This suggests that data over the period of investigation were comparable, enabling analysis of temporal trends. The drugs considered in this study were detected by various toxicologic testing carried out by the OCME laboratories; the sensitivity of detection of different drugs was then subject to both sensitivity of the particular test involved as well as the period of time within which a particular drug is detectable before it is metabolized. Also, our data relate only to the toxicology of victims of firearm violence. As such, few conclusions can be drawn about the role of drugs in the circumstances of the firearm-related deaths; thus, for example, no conclusions can be drawn about the changing role of cocaine as relates to perpetrators of firearm violence. To better understand trends in drugs associated with firearm deaths throughout the decade, we included in our analysis homicide, suicide, and accidental victims of firearm violence. Although most firearm-related deaths are homicides, the specific roles of drugs and other factors in each of these three manners of death need to be definitively considered in separate analyses that are beyond the scope of this article.

In summary, analysis of trends of firearm deaths and associated toxicology in New York City between 1990 and 1998 demonstrated the decreasing role of cocaine and heroin, with the former declining at a faster rate. Declines in firearm

deaths that were both cocaine and opiate positive toxicologically started in 1990, before targeted police efforts to limit illegal firearms and enforce quality-of-life crimes that started in New York City in the mid-1990s. Firearm deaths toxicologically positive for cannabis, on the other hand, increased in the early 1990s and started to decline in the mid-1990s. Firearm deaths that were toxicologically alcohol positive declined, but remained a consistent proportion of all firearm deaths, suggesting no changing role of this substance in firearm deaths. Although racial/ethnic disparities in firearm deaths decreased during the past decade, they persist, with minority firearm death rates remaining three times higher than that among whites in 1998. The observations that different drugs followed different patterns of increase and decrease (both as absolute drug-positive firearm death rates and as proportions of firearm deaths) suggest that each drug studied played a different role in firearm deaths in New York City during the 1990s; further research is needed to untangle what is likely the complex relation among changing drug use patterns, policing, socioeconomic conditions, and firearm deaths.

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REFERENCES

1. Firearm Mortality. National Center for Health Statistics. 2000. Available at: http://www.cdc.gov/nchs/fastats/firearms.htm. Accessed October 6, 2001.
2. Murphy SL. Deaths: final data for 1998. *Natl Vital Stat Rep*. 48(11):1–105. Available at: http://www.cdc.gov/nchs/data/nvsr/nvsr48/nvs48_11.pdf. Accessed October 6, 2001.
3. National Center for Injury Prevention and Control 1997. Injury: leading cause of death. Available at: http://www.tf.org/tf/injuries/97death.html. Accessed October 6, 2001.
4. Cook PJ, Moore MH. Guns, gun control, and homicide. In: Smith DM, Zahn MA, eds. *Studying and Preventing Homicide : Issues and Challenges*. Thousand Oaks, CA: Sage Publications; 1998:246–271.
5. *The Mayor's Management Report, Preliminary Fiscal 1998*, Vol. 1. Agency Narratives, City of New York, p. 13.
6. Klebba JA. Homicide trends in the United States, 1900–74. *Public Health Rep*. 1975; 90(3):195–204.
7. McBride DC, Burgman-Abermehl C, Alpert J, Chitwood DD. Drugs and homicide. *Bull NY Acad Med*. 1986;62(5):497–508.
8. Blumstein A, Rivara FP, Rosenfeld R. The rise and decline of homicide—and why. *Annu Rev Public Health*. 2000;201:505–541.
9. Parker RN, Carmill SI. Alcohol and homicides in the United States 1934–1995—Or one reason why US rates of violence may be going down. *J Crim Law Criminol*. 1998; 88(4):1369–1399.
10. Helmuth L. Has America's tide of violence receded for good? *Science*. 2000;289:582–585.
11. Riley KJ. Homicide and drugs: a tale of six cities. *Homicide Stud*. 1998;2(2):176–205.
12. Goldstein PJ. Homicide related to drug traffic. *Bull NY Acad Med*. 1986;62:509–516.
13. Karmen A. *New York Murder Mystery: The True Story Behind the Crime Crash of the 1990s*. New York, NY: New York University Press; 2000:160–193.
14. New York City Police Department. *Police Strategy No. 1: Getting Guns Off the Streets of New York*. New York, NY: New York City Police Department; March 7, 1994:8–9.
15. CompStat Web site. Available at: http://www.ditpublishing.com/justice/frames/Pt63.html. Accessed October 6, 2001.

16. *The Mayor's Management Report, Fiscal 1997*, City of New York, p. 11.
17. Tardiff K, Marzuk PM, Leon AC, et al. A profile of homicides on the streets and in the homes of New York City. *Public Health Rep*. 1995;110(1):13–17.
18. Tardiff K, Gross EM, Messner SF. A study of homicides in Manhattan, 1981. *Am J Public Health*. 1986;76:139–143.
19. Tardiff K, Marzuk PM, Leon AC, et al. Homicide in New York City: cocaine use and firearms. *JAMA*. 1994;272(1):43–46.
20. Tardiff K, Marzuk PM, Leon AC, et al. Accidental fatal drug overdoses in New York City: 1990–1992. *Am J Drug Alcohol Abuse*. 1996;22(2):135–146.
21. Bureau of the Census. *Census Summary Tape, File 3A (STF3A)*. Washington, DC: US Dept of Commerce; 1990. Available at: http://www.census.gov/ftp/pub/population/www/estimates/co_crh.html. Accessed October 6, 2001.
22. Bowling B. The rise and fall of New York murder: zero tolerance or crack's decline? *Br J Criminol*. 1999;39(4):531–554.
23. Kelling GL, Coles CM. *Fixing Broken Windows: Restoring Order and Reducing Crime in Our Communities*. New York: Touchstone; 1996.
24. DuPont RL, Greene MH. The dynamics of a heroin addiction epidemic. *Science*. 1973; 181:716–722.
25. Dahlberg LL. Youth violence in the United States. Major trends, risk factors, and prevention approaches. *Am J Prev Med*. 1998;14(4):259–272.
26. Lowry R, Cohen LR, Modzeleski W, Kann L, Collins JL, Kolbe LJ. School violence, substance use, and availability of illegal drugs on school property among US high school students. *J School Health*. 1999;69(9):347–355.
27. Kulig J, Valentine J, Griffith J, Ruthazer R. Predictive model of weapon carrying among urban high school students: results and validation. *J Adolesc Health*. 1998;22(4):312–319.
28. McNabb SJ, Farley TA, Powell KE, Rolka HR, Horan JM. Correlates of gun-carrying among adolescents in south Louisiana. *Am J Prev Med*. 1996;12(2):96–-102.
29. Thomas SB. The color line: race matters in the elimination of health disparities. *Am J Public Health*. 2001;91(7):1046–1048.
30. Williams DR. Race, socioeconomic status, and health. The added effects of racism and discrimination. *Ann NY Acad Sci*. 1999;896:173–188.
31. *National Household Survey on Drug Abuse Main Findings 1998*. Department of Health and Human Services. Substance Abuse and Mental Health Services Administration; 2000. Available at: http://www.samhsa.gov. Accessed October 6, 2001.