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Incidence and Causes of Sudden Death in U.S. College Athletes

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Objectives The goal of this study was to reliably define the incidence and causes of sudden death in college student-athletes.

Background The frequency with which cardiovascular-related sudden death occurs in competitive athletes importantly influences considerations for pre-participation screening strategies.

Methods We assessed databases (including autopsy reports) from both the U.S. National Registry of Sudden Death in Athletes and the National Collegiate Athletic Association (2002 to 2011).

Results Over the 10-year study period, 182 sudden deaths occurred (age 20 ± 1.7 years; 85% male; 64% white), 52 resulting from suicide (n = 31) or drug abuse (n = 21) and 64 probably or likely attributable to cardiovascular causes (6/year). Of these 64 athletes, 47 had a confirmed post-mortem diagnosis; the most common were hypertrophic cardiomyopathy in 21 and congenital coronary anomalies in 8. The 4,052,369 athlete participations (in 30 sports over 10 years) incurred mortality risks as follows: suicide and drugs combined, 1.3/100,000 athlete participation-years (5 deaths/year); and documented cardiovascular disease, 1.2/100,000 athlete participation-years (4 deaths/year). Notably, cardiovascular deaths were 5-fold more common in African-American athletes than in white athletes (3.8 vs. 0.7/100,000 athlete participation-years; $p < 0.01$) but did not differ from the general population of the same age and race ($p = 0.6$).

Conclusions In college student-athletes, risk of sudden death due to cardiovascular disease is relatively low, with mortality rates similar to suicide and drug abuse, but less than expected in the general population, although highest in African-American athletes. A substantial minority of confirmed cardiovascular deaths would not likely have been reliably detected by pre-participation screening with 12-lead electrocardiograms. (J Am Coll Cardiol 2014;63:1636–43) © 2014 by the American College of Cardiology Foundation

Sudden death in young competitive athletes has become a highly visible issue, attracting substantial public interest within communities and in cardiovascular medicine (1–10). Much of the current debate regarding pre-participation screening concerns selection of the most effective and practical strategy for identifying (or raising suspicion of) a diverse array of cardiovascular diseases known to be responsible for sudden death in this population (2–16). A particularly relevant consideration is the magnitude of this public health issue, that is, the incidence of sudden deaths due to unsuspected cardiovascular disease relative to other mortality risks.

Most mortality data in young athletes suggest that these catastrophes occur with relatively low event rates (1,3,6,9,11,15). However, a recent nonforensic-based analysis in college student-athletes reported a relatively high frequency of 2.3/100,000 athlete-years for deaths believed to be of cardiovascular origin (7). Such data have heightened concern in the college sports community that intercollegiate student-athletes have uniquely high (even excessive) risks; this concern has contributed to a promotion of cardiovascular screening with electrocardiograms (ECGs) (2,5–7,10,12–14). We have therefore taken this opportunity to analyze our forensic database for both the causes and incidence of sudden deaths in college student-athletes over the recent decade.

Methods

Athlete deaths. The U.S. National Registry of Sudden Death in Athletes was instituted at the Minneapolis Heart Institute Foundation in 1992 to systematically assemble (prospectively and retrospectively) data on young athletes participating in organized competitive sports. Subjects are considered for inclusion when they are a participant in an organized team or individual sport requiring regular training and competition (15). Deaths occurring in club or intramural

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sports or from automobile accidents, homicides, cancer, or systemic diseases were not tabulated. This project was approved by the Allina Health System Institutional Review Board.

For the years 2002 to 2011, we used a variety of sources to identify deaths in intercollegiate athletes active in competitive organized programs (but not necessarily occurring on the athletic field) (Table 1). For each case, systematic tracking was established to obtain available clinical data and/or the circumstances of death, as well as complete autopsy reports with gross, histopathological, and toxicological data from the medical examiner of record. Post-mortem findings were adjudicated by 1 senior investigator (B.J.M.) with >30 years experience and expertise in this discipline (1,8,15–17). When necessary, autopsy findings were verified by communication with medical examiners. Detailed information on demographic characteristics, circumstances of death, and pertinent clinical data was assembled, sometimes from written accounts or telephone interviews with family members, witnesses, or coaches.

National Collegiate Athletic Association participation data. The National Collegiate Athletic Association (NCAA) is a governing body for colleges and universities with organized competitive athletic programs, comprising 1,273 member institutions and representing 85% of students competing in college. For 2001 through 2011, participation data were tabulated from the comprehensive NCAA Sports Sponsorship and Participation Rates Report (18); these findings, however, do not take into account the possibility of multisport participation. Demographic, sex, and race/ethnic information were obtained from the NCAA Student-Athlete Ethnicity Report (19).

Comparison of data sources. The U.S. National Registry of Sudden Death in Athletes (which primarily accesses public domain sources) and the internal NCAA Memorial Resolutions List were compared with respect to identification of sudden cardiovascular deaths in NCAA athletes. Of the 64 total sudden death cases on initial interrogation, the registry method recognized 56 sudden deaths (88%), including 14 not represented on the NCAA list. The NCAA included 50 (78%) of 64 cases, including 8 not recognized by initial registry searches, due to absence within the media reports of key words prospectively established for tracking these events (e.g., "athlete" or "sudden death").

Table 1 Sources Used to Identify Sudden Deaths in NCAA Athletes

1.	NCAA Memorial Resolutions List (compiled annually to recognize student-athletes who have died of any cause)
2.	LexisNexis archival informational database*
3.	News media accounts†
4.	Internet search engines (e.g., Google, Yahoo)
5.	Reports from U.S. Consumer Product Safety Commission (Washington, DC)
6.	Records of National Center for Catastrophic Sports Injury Research (University of North Carolina, Chapel Hill, North Carolina)
7.	Reports directly to U.S. National Registry of Sudden Death in Athletes‡

*Access to authoritative news, legal, and public records (n = 5 billion searchable documents from thousands of sources). †Assembled through Burrelle's Informative Services (Livingston, New Jersey) with access to 18,000 U.S. newspapers and international media sources on a daily basis. ‡From physicians, attorneys, coroners/medical examiners, colleges, and patient advocacy/support organizations.

NCAA = National Collegiate Athletic Association.

Abbreviations and Acronyms

CI = confidence interval
ECG = electrocardiogram
LQTS = long QT syndrome
NCAA = National Collegiate Athletic Association

Statistics. Mortality rates in NCAA athletes were compared with available data for suicide, drugs, drowning, and cardiovascular causes in the general population: 1) U.S. National Vital Statistics Reports (20) with all causes of death for ages 15 to 24 years during 2006, the most recent year in which causes of mortality were tabulated according to age and race; and 2) data reported for suicide in U.S. college students, ages 18 to 24 years, for 2009 through 2010 (21). The extent of 1 athlete-year was defined as participating in a sport from August to July.

Mortality rates were estimated by using Poisson regression and compared across populations and subpopulations. Mortality rates, incidence, and 95% confidence intervals (CIs) were reported as deaths/100,000 athlete participation-years (or person-years). All analyses were performed using SAS version 9.3 (SAS Institute, Inc., Cary, North Carolina). Statistical significance was defined as $p < 0.05$.

Results

NCAA student-athletes. Over the 10-year study period, the total number of student-athlete participations in 30 intercollegiate sports was 4,052,369, ranging from 367,653 (2001 to 2002) to 449,576 (2010 to 2011) for a mean of 405,237 per year. Age at death ranged from 17 to 26 years (20 ± 1.7 years); 154 (85%) were male; and 116 (64%) were white and 60 (33%) were African American, with 6 (3%) subjects of other races.

Epidemiology of sudden deaths. There were a total of 182 sudden deaths recorded during the 10-year study period (0.004% of 4,052,369) or 18/year (Table 2, Fig. 1). Events occurred at a similar rate over time ($p = 0.61$) but were highest in 2004 and 2007 (n = 22 each) and lowest in 2006 (n = 11).

Causes of death. A variety of diseases or circumstances were responsible for mortality in this athlete population (Table 2, Fig. 2). In 16 of 182 sudden deaths, cause was unresolved because post-mortem examinations were not performed (n = 2), it was not possible to obtain autopsy findings due to state confidentiality restrictions (n = 6), or the report was ambiguous without sufficient detail to reliably assign the cause of death (n = 8). These 16 deaths occurred while sedentary (or during sleep) and were unrelated to sports or physical exertion.

NONCARDIOVASCULAR DISEASE–RELATED SUDDEN DEATH. Of the 182 sudden deaths, 118 (65%) were due to causes other than cardiovascular disease (Table 2, Fig. 2). The most

Table 2 Demographic Characteristics and Causes of Death in 182 NCAA Student-Athletes

					Race				NCAA Division		
	n	Age (yrs)	Male	Female	White	AA	Other	Sports*	I	II	III
Suicide	31	19.9 ± 1.5	25	6	24	6	1	Football (10); track/CC (7); baseball (5); swimming (3)	14	8	9
Drugs	21	20.1 ± 1.3	21	0	16	4	1	Football (7); basketball (4); soccer (4); baseball (2)	10	5	6
Confirmed/presumed CV deaths	64	20.2 ± 1.8	54	10	37	26	1	Basketball (23); football (16); soccer (8); track/CC (6)	33	14	17
Confirmed CV deaths	47	20.3 ± 1.9	41	6	22	24	1	Basketball (19); football (11); soccer (5); swimming (4)	25	12	10
Trauma	15	19.1 ± 1.4	13	2	13	0	2	Track/CC (5); football (3); lacrosse (2); swimming (2)	5	5	5
Accidental drowning	11	19.8 ± 1.5	11	0	5	5	1	Track/CC (4); soccer (3); baseball (2); basketball (1); football (1)	4	3	4
Sickle cell trait	11	19.7 ± 1.3	11	0	0	11	0	Football (8); basketball (2); track (1)	8	2	1
Heat stroke	5	19.8 ± 1.6	5	0	3	2	0	Football (3); baseball (1); basketball (1)	3	1	1
Brain aneurysm	3	20.0 ± 2.0	3	0	2	1	0	Football (2); basketball (1)	0	2	1
Pulmonary	3	19.7 ± 2.1	1	2	1	2	0	Basketball (2); football (1)	2	1	0
Sepsis	2	20.5 ± 0.7	1	1	2	0	0	Baseball (1); field hockey (1)	0	1	1
Unresolved	16	20.3 ± 2.4	9	7	13	3	0	Football (5); track/CC (2); tennis (2); swimming (2)	9	2	5

*Four most common sports shown.
AA = African-American; CC = cross-country; CV = cardiovascular; NCAA = National Collegiate Athletic Association.

common of these was suicide (n = 31 [17%]), including gunshot trauma or hanging (n = 8, each), or were drug related as confirmed by toxicology (n = 21 [12%]); together, drugs and suicide were thus responsible for 29% of all sudden deaths (n = 52). Other noncardiovascular causes of sudden death were blunt trauma (n = 15; including 2 with commotio cordis); sickle cell trait (n = 11) (17); drowning (n = 11); and heat stroke (n = 5).

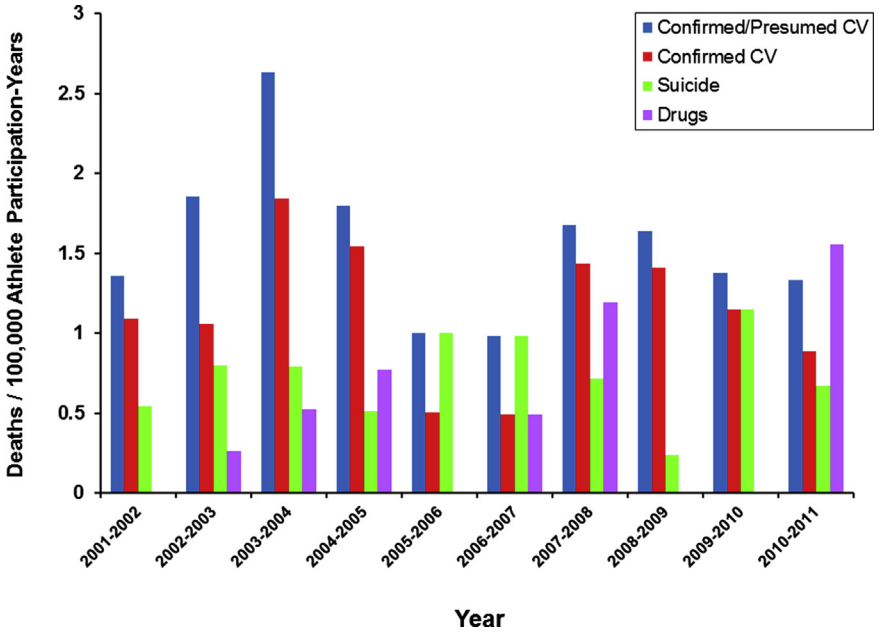

Figure 1 Comparative Incidence of Sudden Death

Comparative incidence of sudden death due to cardiovascular (CV) disease, suicide, or drugs in National Collegiate Athletic Association athletes from 2001 to 2011, per 100,000 athlete participation-years.

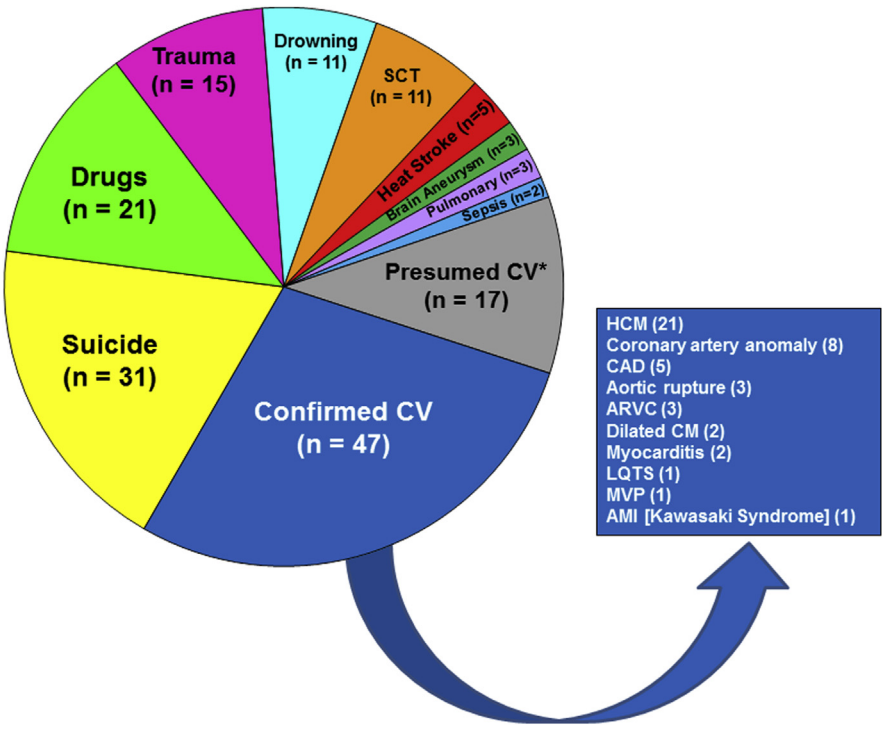

Figure 2 **Causes of Sudden Death in NCAA Athletes**

Left: All causes. **Right:** Confirmed CV causes in 47 athletes. *Collapse was virtually instantaneous during physical activity, suggesting underlying CV disease was responsible, but in the absence of a firm post-mortem diagnosis. Not included here are 16 athletes in whom the cause of death remains unresolved. AMI = acute myocardial infarction; ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; CM = cardiomyopathy; CV = cardiovascular; HCM = hypertrophic cardiomyopathy; LQTS = long QT syndrome; MVP = (myxomatous) mitral valve prolapse; NCAA = National Collegiate Athletic Association; SCT = sickle cell trait.

CARDIOVASCULAR DISEASE–RELATED SUDDEN DEATH. In 64 athletes, a cardiovascular abnormality was judged the likely or probable cause of death. Most common sports were basketball (n = 23) and football (n = 16) (Table 2); 29 collapsed with physical exertion during competition (n = 6) or practice (n = 23), 14 others during recreational sports, and 21 while sedentary. Of these, 58% were white and 41% were African American.

In 47 of 64 athletes, a post-mortem examination documented a cardiovascular abnormality considered responsible for the sudden death. Most common were: hypertrophic cardiomyopathy (n = 21 [12 African American, 9 white]); mean heart weight 563 ± 70 g; mean maximum left ventricular wall thickness 22 ± 4 mm (range 17 to 31 mm); myocyte disarray (n = 10); anomalous coronary artery of wrong sinus origin (n = 8); atherosclerotic coronary artery disease (n = 5); aortic dissection and rupture (n = 3 [including 2 positive for a fibrillin-1 gene mutation]); arrhythmogenic right ventricular cardiomyopathy with right ventricular enlargement and extensive fatty infiltration (n = 3); healed myocarditis with patchy myocardial fibrosis (n = 2); dilated cardiomyopathy with 4-chamber enlargement (n = 2); myxomatous mitral valve (n = 1); myocardial infarction (n = 1 [Kawasaki syndrome]); and 1 athlete with a structurally normal heart and family history of sudden death due to long QT syndrome (LQTS), which strongly suggested LQTS as the cause of death (Table 2, Fig. 2).

In 17 of 64 athletes, collapse occurred virtually instantaneously after physical activity during competition or practice, suggesting that underlying cardiovascular disease was responsible ("presumed cardiovascular") (Table 2, Fig. 2). However, the precise cause of death could not be established, either because a post-mortem examination was not performed (n = 3), the autopsy report was inconclusive (i.e., judged inadequate to reliably assign the specific cause of SD [n = 12]), or because confidentiality considerations restricted access to the report (n = 2). Also included in this group were 3 athletes in whom the sole pathologic abnormality was myocardial bridging of left anterior descending coronary artery (22).

Sudden death incidence. Given that there were 4,052,369 NCAA athlete participations/year over the 10-year study period, calculated mortality rates were: 1) suicide, 3/year (0.8/100,000 athlete participation-years) and drug-related, 2/year (0.5/100,000 athlete participation-years); 2) suicide and drugs combined, 5/year (1.3/100,000 athlete participation-years); 3) confirmed cardiovascular disease (n = 47), 4/year (1.2/100,000 athlete participation-years); and 4) combined

confirmed or presumed cardiovascular disease (n = 64), 6/year (1.6/100,000 athlete participation-years).

Mortality from suicide and drugs combined did not differ from the 47 confirmed cardiovascular causes (p = 0.39) but was less than confirmed/presumed cardiovascular disease (p < 0.0006). Deaths due to confirmed cardiovascular disease were 2.5-fold more common in athletes competing in Division I compared with Division III sports (1.5 vs. 0.6/100,000 participation-years; p = 0.012). Mortality from any cause did not increase significantly over the 10-year observation period (p = 0.06 to 0.9) (Fig. 1).

NCAA and general population mortality. NCAA athletes had significantly lower cardiovascular mortality rates than expected in the general population of similar age, either for the 47 athletes with confirmed diagnoses (p < 0.0001) or the 64 athletes with combined confirmed/presumed cardiovascular diseases (p < 0.0007) (Tables 3 and 4).

Suicide rates in the general population were significantly higher than in NCAA athletes (p < 0.0001); however, suicide was less common in college student-athletes than in other college students (p < 0.0001). Drug-related deaths occurred with similar frequency in NCAA athletes and in the general population of comparable age and sex (p = 0.32).

Impact of race and sex. RACE. Cardiovascular deaths were 5-fold more common in African-American subjects than in white subjects (3.8 vs. 0.7/100,000 athlete participation-years; p < 0.01) but were similar to that in a general black population of the same age (4.3/100,000 athlete participation-years; p = 0.6) (Table 4).

In contrast, the suicide rate was similar in African-American and white athletes (1.0 vs. 0.8/100,000 athlete participation-years; p = 0.7), but in both instances, it was significantly less common than in the general population (p < 0.0001). Drug-related death rates also were similar in African-American athletes (0.6/100,000 athlete participation-years) and white athletes (0.5/100,000 athlete participation-years; p = 0.75) but were not significantly different from the general population (p > 0.2).

SEX. Cardiovascular deaths were 6-fold more common in male athletes than in female athletes (1.8/100,000 athlete participation-years [95% CI: 0.3 to 9.3/100,000 athlete participation-years] vs. 0.3/100,000 athlete participation-years [95% CI: 0.2 to 0.8/100,000 athlete participation-years]; p < 0.0001) but lower than the general male population of the same age (2.8/100,000 athlete participation-years [95% CI: 0.6 to 14/100,000 athlete participation-years]; p < 0.001).

The suicide rate was 3-fold higher in male subjects than in female subjects (1.1/100,000 athlete participation-years [95% CI: 0.2 to 5.8/100,000 athlete participation-years] vs. 0.3/100,000 athlete participation-years [95% CI: 0.2 to 0.8/100,000 athlete participation-years]; p < 0.01) but was much lower than in the general male population of the same age (16/100,000 athlete participation-years; p < 0.0001).

Drug deaths were more common in male subjects than in female subjects (0.9/100,000 athlete participation-years [95% CI: 0.6 to 1.4/100,000 athlete participation-years] vs. zero; p < 0.0001) but were similar to the general male population of the same age (1.0/100,000 athlete participation-years; p = 0.7).

Reliability of screening for cardiovascular causes of sudden death. We estimated reliability of the 12-lead ECG for diagnosis of specific cardiac causes of sudden death. Of 47 diagnoses confirmed at autopsy, 28 (60%) would most likely have been identified or suspected of being cardiac related according to the ECG findings: hypertrophic cardiomyopathy (n = 19 of 21) (30); arrhythmogenic right ventricular cardiomyopathy (n = 3); aortic rupture (n = 2 with Marfan syndrome); dilated cardiomyopathy (n = 2); LQTS (n = 1); and mitral valve prolapse (n = 1).

Discussion

Considerable discussion has emerged over the last several years regarding the impact on the general public health of sudden deaths tragically occurring in young competitive athletes (1–16) and the most effective screening strategy to detect the responsible causes in this population (2–7,9–14). Indeed, the justification for broad-based (even national) screening initiatives would seem to rest importantly on the frequency with which cardiovascular catastrophes do occur

Table 3 Mortality Rates in NCAA Athletes Compared With the General Population

	n	NCAA Athletes 2002–2011: Deaths/100,000 Athlete Participation-Years* (95% CI)	General Population: Deaths/100,000 Person-Years* (95% CI)
Confirmed CV deaths	47	1.2 (0.8–1.5)†	2.4 (2.2–2.5)
Confirmed/presumed CV deaths	64	1.6 (1.2–2.0)†	2.4 (2.2–2.5)
Suicide	31	0.8 (0.5–1.1)†‡	9.8 (4.8–19.8)
Drugs	21	0.5 (0.3–0.8)	0.6 (0.3–1.5)
Suicide/drugs combined	52	1.3 (1.0–1.7)†	10.4 (6.0–18.0)
Accidental drowning§	11	0.3 (0.2–0.5)†	1.5 (0.5–5.0)
Unresolved	16	0.4 (0.2–0.6)†	1.1 (0.4–3.0)

*Person-years in the general population (20) are equivalent to athlete participation-years. †p < 0.01 to < 0.0001 compared with the general population age 15 to 24 years. ‡p < 0.0001 compared with 2009 to 2010 suicide rate reported for college students 18 to 24 years of age (i.e., 6.2/100,000 person-years [95% CI: 2.9 to 13.2/100,000 person-years]) (21). §Events occurring in a body of water, with or without a nonmotorized vessel.
CI = confidence interval; CV = cardiovascular.

Table 4 Mortality Rates in NCAA Athletes Compared With the General Population Stratified According to Race/Ethnicity

	African-American		White	
	NCAA Athletes: Deaths/100,000 Athlete Participation-Years (95% CI)	General Population Age 15 to 24 Years: Deaths/100,000 Person-Years* (95% CI)	NCAA Athletes: Deaths/100,000 Athlete Participation-Years (95% CI)	General Population Age 15 to 24 Years: Deaths/100,000 Person-Years* (95% CI)
Confirmed CV death	3.8 (1.7–8.8)	4.3 (3.4–5.3)†	0.7 (0.3–1.7)‡	2.0 (1.9–2.2)†
Confirmed/presumed CV death	4.2 (1.8–9.5)	4.3 (3.4–5.3)†	1.2 (0.9–1.7)‡	2.0 (1.9–2.2)†
Suicide	1.0 (0.8–3.5)‡	6.2 (2.7–13.9)†	0.8 (0.5–1.2)‡	10.4 (4.7–23.2)†
Drugs	0.6 (0.1–3.1)	0.3 (0.1–0.9)†	0.5 (0.3–0.9)	0.7 (0.3–1.9)†
Suicide and drugs combined	1.6 (0.6–4.4)‡	6.5 (3.4–12.2)†	1.3 (1.0–1.8)‡	11.1 (6.0–20.6)†

*Person-years in the general population (28) are equivalent to athlete participation-years. †$p < 0.0001$ between general population strata. ‡$p < 0.01$: NCAA athletes versus the general population, 15 to 24 years of age within race/ethnic category.
Abbreviations as in Tables 2 and 3.

in trained athletes (3,9,11,15,16,23). For example, in Denmark, systematic screening of athletes (and others) is not performed by any method because the sudden death rate is considered particularly low (11).

In a previous survey of NCAA athletes without forensic (autopsy) confirmation of causes of death, Harmon et al. (7) reported 9 athlete deaths/year (over a 5-year period) among 400,000 participations, which these investigators believed were cardiovascular based, but without diagnostic anatomic substantiation. The present data essentially target the same population (albeit with a 2-fold longer study period) and have identified a significantly lower event rate of 4 to 6 deaths/year, aided by access to documented causes of death by virtue of post-mortem examinations. Perhaps not unexpectedly, the 2 studies in the literature that report the highest cardiovascular sudden death rate in athletes of >2 per 100,000 do not include diagnostic autopsy or clinical data (i.e., Steinvil et al. [6] from Israel and the Harmon et al. [7] study of U.S. college athletes).

However, we wish to underscore that the differences in incidence of athletic field deaths between Harmon et al. (7) and the present study are nevertheless relatively small when compared with the more compelling rates of sudden death due to the many other risks to living in the general population of the same age group (Fig. 3). Indeed, an important aspect of the present investigation is the placing of cardiovascular sudden deaths in college athletes within a broader context.

Our analysis underscores the principle that NCAA athlete sudden deaths represent a numerically low event rate phenomenon (1,3,6,9,11,15,23); for example, by comparison, annual automobile fatalities in the same age group are >2,500-fold more common. Furthermore, cardiovascular deaths in NCAA athletes are significantly less common than those in the general population, but similar to the unexpectedly high combined suicide and drug-related mortality rates identified in these college athletes.

Notably, the cardiovascular sudden death rate in our college athletes is higher than that previously reported in a cohort of high school athletes (1.2 vs. 0.7/100,000) (9). Although the determinants are uncertain, these differences are potentially attributable to the longer exposure by college student-athletes to rigid and rigorous training regimens extended over more substantial periods of life. Sudden deaths were, in fact, most common in Division I (scholarship) athletes for whom training and competition are most demanding. In addition, susceptibility to cardiovascular events could conceivably be influenced by risk factors associated with the initial experience of independent living at college (e.g., failure to adhere to healthy lifestyle practices with proper rest and nutrition, increased exposure to alcohol and drugs).

In a sizeable minority of athlete sudden deaths, it was not possible to conclusively determine the cause of death, even after considering the submitted autopsy findings. This circumstance largely reflects the current state-of-the-art in the medical examiner community regarding post-mortem diagnoses in sudden deaths due to genetic heart diseases (24,25). Indeed, a minority of our sudden death events (n = 17) were considered likely of cardiovascular origin, given the instantaneous collapse linked to physical exertion, even though a precise cause of death could not be assigned due to ambiguous (or absent) autopsy findings. Nevertheless, some of these latter deaths could have been due to noncardiac causes, a potential source for overestimating our sudden death rates. Alternatively, the small subset of athletes for whom diagnosis remained unresolved (n = 16) could possibly have included cardiovascular disease–related deaths and in this way underestimated the incidence of sudden deaths.

Available estimates of the at-risk athlete population (i.e., the denominator) have limitations, given that we and other investigators (7) are obligated to use NCAA data expressed as participations, rather than for individual participants. With the possibility of multisport involvement by a small minority of athletes, there is the potential for overestimating the at-risk population and thereby underestimating the incidence of sudden death. In addition, ion channelopathies (e.g., LQTS, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia), which are important but uncommon causes of sudden death, are unlikely to be identified within a forensic-based registry such as ours due to the characteristic absence of structural cardiac abnormalities. This issue underscores the advantage of expert

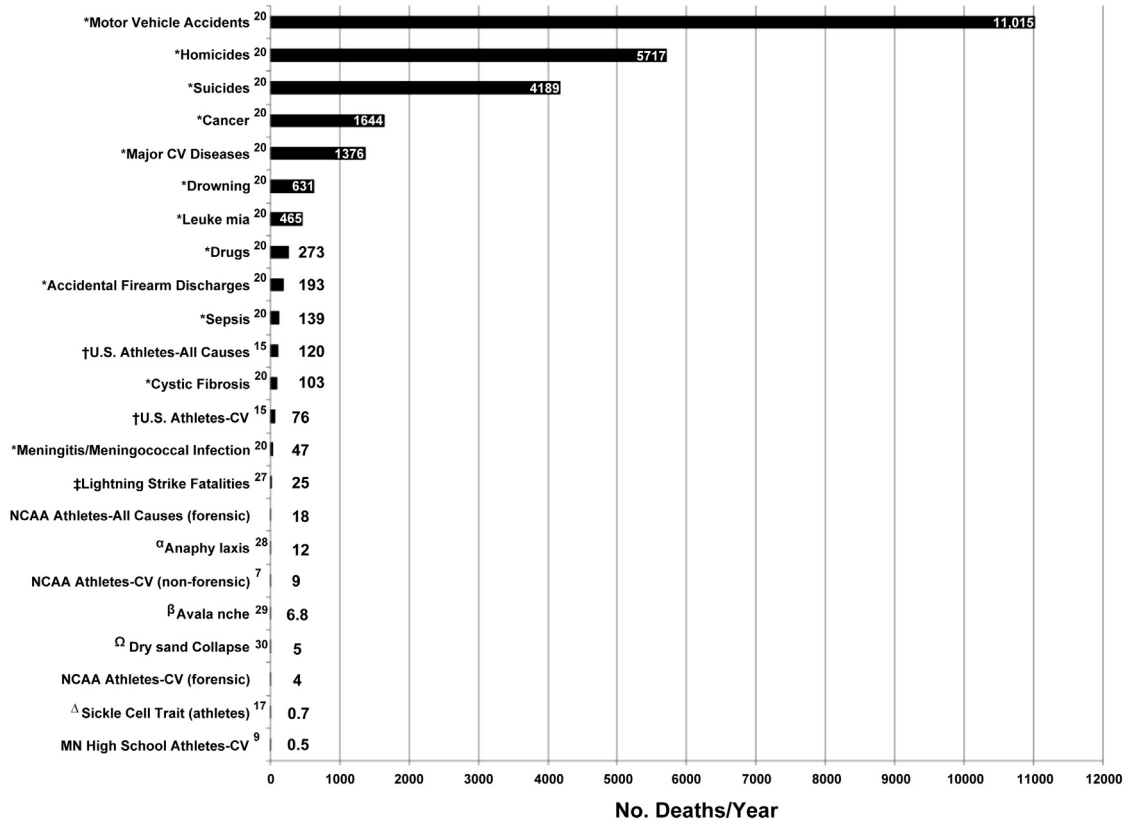

Figure 3 Causes of Death in Young People Annually in the United States, Including in NCAA Athletes

Reference citations are given in each line. *Data for 15- to 24-year-olds (20). †Data available only for 8- to 39-year-olds (15). ‡Estimated deaths in this age group, from the National Weather Service (27). α Average annual event rate for 15- to 24-year-olds (28). β Average annual event rate for 16- to 25-year-olds (29). Ω Average annual event rate for survivors and nonsurvivors of all ages (30). Δ Age 12 to 22 years (17). MN = Minnesota; other abbreviations as in Figures 1 and 2.

cardiac pathologist assessment of autopsy findings in sudden deaths of young athletes (25). The general level of expertise for cardiovascular pathology in the U.S. medical examiner community has been questioned (24).

African-American college athletes were at 5-fold greater risk for cardiovascular sudden death than white athletes (16). However, this sudden death rate in African-American athletes did not differ from the general population of the same age and race. For male athletes, mortality rates for cardiovascular disease, as well as suicide and drugs, exceeded that in female athletes (3- to 6-fold), consistent with reports in other athlete populations (1,3,8,9,15,16). Determinants of such racial and sex differences remain unresolved.

Our data do not focus primarily on the current controversy regarding the most effective pre-participation screening strategy for detection of cardiovascular disease (2–7,10–15). However, certain inferences are unavoidable. In particular, our findings underscore the frequency and importance of false-negative results, whether screening is limited to history taking and physical examination or includes a 12-lead ECG (i.e., the principle that not all at-risk athletes are detectable by screening) (1,3,4,9,26). Based on autopsy confirmation of cause of death, approximately 60% of the athletes in our study cohort would probably have been identified by a screening 12-lead ECG. However, we also estimate that at least 40% would likely have been "false-negative" findings, not reliably suspected by 12-lead ECGs (26).

Conclusions

NCAA student-athletes do not seem to be at unique or particularly high risk for sudden death due to unsuspected cardiovascular disease. Furthermore, the relative importance of suicide and drug deaths reported here with respect to the health and safety of college athletes suggests that there may be a disproportionate focus on cardiovascular disease in this population. These observations support the principle of enhanced resources and energy for suicide prevention and control of drug use, as well as wider dissemination of automatic external defibrillators for secondary prevention of cardiovascular sudden death. Such initiatives may best serve the overall public health of college student-athletes.

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REFERENCES

1. Maron BJ. Sudden death in young athletes. N Engl J Med 2003;349:1064–75.
2. Corrado D, Basso C, Pavei A, et al. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. JAMA 2006;296:1593–601.
3. Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: update 2007. Circulation 2007;115:1643–55.
4. Chaitman BR. An electrocardiogram should not be included in routine preparticipation screening of young athletes. Circulation 2007;116:2610–4.
5. Myerburg RJ, Vetter VL. Electrocardiograms should be included in preparticipation screening of athletes. Circulation 2007;116:2616–26.
6. Steinvil A, Chundadze T, Zeltser D, et al. Mandatory electrocardiographic screening of athletes to reduce their risk for sudden death proven fact or wishful thinking? J Am Coll Cardiol 2011;57:1291–6.
7. Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in National Collegiate Athletic Association Athletes. Circulation 2011;123:1594–600.
8. Maron BJ, Shirani J, Poliac LC, et al. Sudden death in young competitive athletes: Clinical, demographic and pathological profiles. JAMA 1996;276:199–204.
9. Maron BJ, Haas TS, Ahluwalia A, Rutten-Ramos SC. Incidence of cardiovascular sudden deaths in Minnesota high school athletes. Heart Rhythm 2013;10:374–7.
10. Corrado D, Pelliccia A, Bjørnstad HH, et al. Cardiovascular preparticipation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Eur Heart J 2005;26:516–24.
11. Holst AG, Winkel BG, Theilade J, et al. Incidence and etiology of sports-related sudden cardiac death in Denmark—implications for preparticipation screening. Heart Rhythm 2010;10:365–71.
12. Thiene G, Corrado D, Schiavon M, Basso C. Screening of competitive athletes to prevent sudden death: implement programmes now. Heart 2013;99:304–6.
13. Drezner JA. ECG screening in athletes: time to develop infrastructure. Heart Rhythm 2011;8:1560–1.
14. Corrado D, Pelliccia A, Heidbuchel H, et al. Recommendations for interpretation of 12-lead electrocardiogram in the athlete. Eur Heart J 2010;31:243–59.
15. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation 2009;119:1085–92.
16. Maron BJ, Carney KP, Lever HM, et al. Relationship of race to sudden cardiac death in competitive athletes with hypertrophic cardiomyopathy. J Am Coll Cardiol 2003;41:974–80.
17. Harris KM, Haas TS, Eichner ER, Maron BJ. Sickle cell trait associated with sudden death in competitive athletes. Am J Cardiol 2012;110:1185–8.
18. DeHass DM. 1981-82-2007-2008 NCAA Sports Sponsorship and Participation Rates Report. Indianapolis, IN: NCAA, 2009.
19. Zgonc E. 1999-2000/2009-2010 NCAA Student-Athlete Ethnicity Report. Indianapolis, IN: NCAA; 2010.
20. Heron M, Hoyert DL, Murphy SL, et al. Deaths: final data for 2006. Natl Vital Stat Rep 2009;57:1–134.
21. Turner JC, Leno EC, Keller A. Causes or mortality among American college students: a pilot study. J Coll Student Psychother 2013;27:31–42.
22. Basso C, Thiene G, Mackey-Bojack S, et al. Myocardial bridging: a frequent component of the hypertrophic cardiomyopathy phenotype lacks systematic association with sudden cardiac death. Eur Heart J 2009;30:1627–34.
23. Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. J Am Coll Cardiol 1998;32:1881–4.
24. Friedewald VE, Maron BJ, Roberts WC. The editor's roundtable: sudden death in athletes. Am J Cardiol 2007;100:1451–9.
25. Papadakis M, Ragu H, Behr ER, et al. Sudden cardiac death with autopsy findings of uncertain significance. Circ Arrhythm Electrophysiol 2013;6:588–96.
26. Rowin EJ, Maron BJ, Appelbaum E, et al. Significance of false negative electrocardiograms in preparticipation screening of athletes for hypertrophic cardiomyopathy. Am J Cardiol 2012;110:1027–32.
27. National Weather Service. Lightning fatalities by state, 2002-2011. Available at: http://www.lightningsafety.noaa.gov/stats/02-11_deaths_by_state.pdf. Accessed May 6, 2013.
28. Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple cause of death 2005-2006 on CDC WONDER on-line database, released February 2010. Data are from the Multiple Cause of Death Files, 2005-2006, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistic Cooperative Program. Available at: http://wonder.cdc.gov/mcd-icd10-archive2006.html. Accessed January 20, 2014.
29. Colorado Avalanche Information Center. Fatalities by age, 1996-2006. Available at: http://avalanche.state.co.us/wp-content/uploads/2013/09/Slide10.jpg. Accessed January 9, 2014.
30. Maron BA, Haas TS, Maron BJ. Sudden death from collapsing sand holes. N Engl J Med 2007;356:2655–6.

Key Words: athletes ■ ECGs ■ sudden death ■ suicide.