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Factors mediating demographic determinants of injury mortality

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ABSTRACT

Purpose: Elevated injury mortality rates persist for men and people of color despite attempts to standardize trauma care in the United States. This study investigates the role of injury characteristics and access to trauma care as mediators of the relationships between race, ethnicity, sex, and injury mortality.
Methods: Data on prehospital and trauma center care were examined for adult injured patients in Maryland who were transported by emergency medical services to designated trauma centers ($n = 15,355$) or who died while under emergency medical services care ($n = 727$). Potential mediators of the relationship between demographic characteristics and injury mortality were identified through exploratory analyses. Total, direct, and indirect effects of race, ethnicity, and sex were estimated using multivariable mediation models.
Results: Prehospital time, hospital distance, injury mechanism, and insurance status mediated the effect of African American race, resulting in a 5.7% total increase (95% CI: 1.6%, 9.9%) and 5.6% direct decrease (95% CI: 1.1%, 9.9%) in odds of death. Mechanism, insurance, and distance mediated the effect of Hispanic ethnicity, resulting in an 11.4% total decrease (95% CI: 6.4%, 16.2%) and 13.4% direct decrease (95% CI: 8.1%, 18.3%) in odds of death. Injury severity, mechanism, insurance, and time mediated the effect of male sex, resulting in a 27.3% total increase (95% CI: 21.6%, 10.9%) and a 6.2% direct increase (95% CI: 1.8%, 10.9%) in odds of death.
Conclusions: Distance, injury characteristics, and insurance mediate the effects of demographic characteristics on injury mortality and appear to contribute to disparities in injury mortality.

Introduction

Elevated mortality rates among African American [1–3], Hispanic [1,4], and male injured patients [5–7] are a troubling pattern in the United States health care system. Age-adjusted injury mortality rates are 1.5 times greater for men than for women and 6% higher for African Americans than for whites [8]. Overall mortality rates are lower for Hispanics than for non-Hispanic whites [8], but odds of death after injury may be higher for Hispanic patients [1,4]. Injury incidence partially explains overall mortality rates [8], but little is known about factors contributing to variation in outcomes after injury. Potential causes of this variation include differences in injury characteristics, access to trauma care, quality of care delivered, and premorbid health status. Improved understanding of the pathway between individual demographic characteristics and injury outcomes is essential for effective interventions to reduce mortality.

There is no evidence of racial or ethnic differences in initial management of injury at trauma centers [9], but there are differences in prehospital triage [10] and access to trauma center care [11]. Injury mortality rates are higher at hospitals with a large proportion of African American patients compared with hospitals with mostly white patients, suggesting hospital-level factors contribute to racial differences in outcomes [12,13]. Residential areas in the United States are often segregated by race and ethnicity [14], determining the geographic spaces where people spend their time and the care they receive after injury. Differences in injury severity [3,8], injury mechanism [8], and insurance status [2] may also contribute to differences in injury mortality.

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Men are more likely than women to experience and die from injury [5,6], although the relationship between sex and mortality varies by age and injury characteristics [7,15–17]. Anthropomorphic differences partially explain elevated injury mortality among male patients [16]. Occupation and recreation patterns [18] contribute to differences in the places where men and women spend time, and subsequently the trauma care they receive. As with race and ethnicity, differences in injury characteristics [8] and insurance status [19] may contribute to differences in mortality.

To identify clinical and location-specific factors contributing to variation in injury outcomes, we used injury incident data from the Maryland Institute for Emergency Medical Services Systems (MIEMSS) in mediation analyses of race, ethnicity, and sex as determinants of injury mortality.

Methods

Data sources, population, and setting

This study used data from the 2015 Maryland Adult Trauma Registry and associated emergency medical service (EMS) records from the MIEMSS eMEDS patient care reporting system. All data were collected according to published MIEMSS standards [20]. Records for adult patients (age $\geq$ 18) were included if the patient was injured in Maryland in 2015, transported by a Maryland-based EMS company (ground ambulance or helicopter), and treated at a designated trauma center. Patients who died while under EMS care were also included.

Variables and measures

The independent variables of interest were race (African American vs. non-Hispanic white), ethnicity (Hispanic vs. non-Hispanic white), and sex (male vs. female), all coded based on medical records. The dependent variable of interest was injury mortality (binary), including patients who died in EMS care or at the hospital. Potential mediating variables included insurance status, injury mechanism, injury severity, prehospital time, and distance from the injury scene to the nearest trauma center. Insurance status was coded as private, Medicare, Medicaid, or uninsured. Injury mechanism was coded as penetrating or not penetrating, based on external cause of injury codes. Using Injury Severity Score (ISS), severity was categorized as mild ($\leq$9), moderate (10–15), severe (16–24), or critical ($\geq$25). In the absence of ISS, unweighted revised trauma scores were coded as mild (12), moderate (11), severe (4–10), or critical ($\leq$3). Prehospital time was measured in minutes from the time of 911 call to trauma center arrival. For patients who died in EMS care, prehospital time was measured from the 911 call to time of death. Distance was measured in miles as the Euclidean distance from the injury incident scene to the nearest trauma center.

Analytic approach

All analyses were conducted using Stata 13 (StataCorp, College Station, Texas). Data appeared to be missing at random and were imputed with predicted mean matching, as described in previously published work [21] and in Supplementary Appendix A. Potential mediators were identified based on previously published analyses [21] and assessed using Baron and Kenny's process for identifying mediation [22], as described in Supplementary Appendix B. Bivariate relationships between independent and mediating variables were estimated with simple linear and logistic regression, as appropriate. Relationships between mediating variables and mortality were estimated using logistic regression, controlling for the relevant independent variable. Mediating variables were included in multivariable models if the relationships with the independent and dependent variables were both statistically significant at $\alpha = 0.05$. Multivariable logistic regression models were used to estimate the total and direct effects of the independent variables and the decomposition of indirect effects across multiple mediators. Total effect is the relationship between the independent and dependent variables, without adjustment for other factors. Direct effect is the relationship between the independent and dependent variables after controlling for mediating variables. Indirect effects (a*b) indicate the component of the total effect attributable to each mediating variable, calculated as the relationship between independent and mediating variables (a path), multiplied by the mediator-outcome relationship (b path). The relative scales of coefficients from multivariable mediation models with binary outcomes are different for each predictor-mediator-outcome pathway. To support comparison of the magnitude and direction of multiple relationships within a single model, we calculated standardized effects using the method proposed by MacKinnon and Dwyer [23] and used bootstrapping to estimate variances. Semivariograms of standardized residuals were examined to assess each mediation model for residual spatial dependence (Supplementary Appendix C) [24].

Results

Distributions of potential mediating variables by race, ethnicity, and sex are presented in Table 1. The study sample included 16,082 individual patient-injury records. Mean prehospital time for white patients was 72.1 minutes, mean trauma center distance was 12.3 miles, 12.4% were uninsured, and 7.9% had penetrating injury. Mean prehospital time for African American patients was 56.0 minutes, mean trauma center distance was 6.6 miles, 26.3% were uninsured, and 26.9% had penetrating injury. Mean trauma center distance for Hispanic patients was 8.5 miles, 45.3% were uninsured, and 13.4% had penetrating injury. Mean prehospital time for female patients was 70.4 minutes, 13.5% were uninsured, and 5.6% had penetrating injury. Mean prehospital time for male patients was 62.4 minutes, 23.5% were uninsured, and 20.2% had penetrating injury.

Mediation of race

Compared with white patients, prehospital intervals for African American patients were 18.3 minutes shorter ($P < .01$, Table 2) and trauma center distances were 5.7 miles shorter ($P < .01$). African American patients were 4.9 times more likely to have a penetrating injury ($P < .01$), 2.4 times more likely to have Medicaid ($P < .01$), 2.6 times more likely to be uninsured ($P < .01$), and 73% less likely to have Medicare ($P < .01$). Controlling for race, there was a 2% increase in odds of death for every 5-minute increase in prehospital time ($P = .01$), and a 1.0% increase in odds of death for every 1-mile increase in distance to the nearest trauma center ($P < .01$). Patients without insurance were 2.3 times more likely to die than those with private insurance ($P < .01$).

Standardized coefficients from the multivariable mediation model for the relationship between race and injury mortality are illustrated in Figure 1 and standardized indirect effects of mediating variables are presented in Table 3. The total effect of African American race demonstrated a slight increase in odds of death, relative to white patients (OR = 1.06, 95% CI: 1.02, 1.10). The effect of race was fully mediated by the combined effects of time (a*b = −0.015, 95% CI: −0.025, −0.005), distance (a*b = −0.024, 95% CI: −0.036, −0.013), penetrating injury (a*b = 0.104, 95% CI: 0.092, 0.115), Medicaid enrollment (a*b = 0.028, 95% CI: 0.016, 0.041),

Table 1
Estimated distribution of patient characteristics by race, ethnicity, and sex

Covariate	White (non-Hispanic)		African American (non-Hispanic)		Hispanic		Female		Male	
	%	95% CI	%	95% CI	%	95% CI	%	95% CI	%	95% CI
Mortality rate	8.1	7.5, 8.8	9.7	8.9, 10.6	4.9	3.3, 6.4	5.1	4.6, 5.7	10.1	9.5, 10.6
Insurance										
Private	47.2	46.1, 48.5	29.6	28.4, 30.8	31.7	28.6, 34.8	43.7	42.4, 45.0	37.4	36.5, 38.4
Medicaid	20.6	19.6, 21.5	37.7	36.4, 39.0	19.7	17.0, 22.4	21.5	20.4, 22.6	29.7	28.7, 30.7
Medicare	19.7	19.0, 20.6	6.4	5.7, 7.0	3.3	2.2, 4.4	21.3	20.3, 22.4	9.3	8.8, 9.9
None	12.4	11.6, 13.3	26.3	25.1, 27.6	45.3	42.1, 48.6	13.5	12.6, 14.4	23.5	22.5, 24.5
Penetrating										
No	92.1	91.5, 92.7	73.1	72.0, 74.2	86.6	84.6, 88.7	94.3	93.7, 94.8	79.8	79.0, 80.5
Yes	7.9	7.3, 8.4	26.9	25.8, 28.0	13.4	11.3, 15.4	5.7	5.2, 6.3	20.2	19.5, 21.0
Severity										
Mild	86.0	85.0, 86.9	85.8	84.8, 86.8	88.2	86.2, 90.3	89.4	88.4, 90.3	84.7	83.9, 85.5
Moderate	3.7	3.3, 4.1	3.7	3.3, 4.2	3.4	2.3, 4.6	3.4	2.9, 3.9	3.8	3.4, 4.2
Severe	4.6	4.0, 5.3	4.2	3.7, 4.8	4.3	3.0, 5.6	4.0	3.4, 4.6	4.6	4.1, 5.2
Critical	5.7	5.1, 6.2	6.3	5.5, 7.0	4.0	2.7, 5.4	3.3	2.8, 3.8	6.9	6.4, 7.4
Trauma level										
Level I/II	79.7	78.6, 80.8	94.0	93.3, 94.7	90.3	88.3, 92.3	84.9	83.7, 86.2	86.6	84.2, 85.7
Level III	20.3	19.2, 21.4	6.0	5.3, 6.7	9.7	7.7, 11.7	15.1	13.8, 16.3	13.4	14.3, 15.8
	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
Time	72.1	22.5, 121.8	56.0	54.7, 57.3	54.8	51.9, 57.8	70.4	68.4, 72.4	62.4	61.0, 63.7
Distance	12.3	12.1, 12.3	6.6	6.3, 6.8	8.5	7.8, 9.1	10.5	10.2, 10.8	9.5	9.3, 9.8

Medicare enrollment (a*b = −0.046, 95% CI: −0.062, −0.030), and lack of insurance (a*b = 0.067, 95% CI: 0.003, 0.010), resulting in a modestly protective direct effect of African American race (OR = 0.94, 95% CI: 0.90, 0.99). The semivariogram of the standardized residuals from the multivariable mediation model for race did not exhibit residual spatial dependence (Appendix C).

Table 2
Estimated associations between independent and mediating variables predicting injury mortality

Covariate	Independent variables					
	African American		Hispanic		Male	
	b	95% CI	B	95% CI	b	95% CI
IV→ MV (a path)*						
Time (min)†	−18.28	−18.70, −17.85	−18.64	−19.44, −17.85	−9.70	−11.85, −7.55
Distance (miles)‡	−5.69	−5.76, −5.61	−3.74	−3.90, −3.58	−1.06	−1.43, −0.70
Moderate§	1.01	0.84, 1.20	0.90	0.62, 1.31	1.16	0.96, 1.40
Severe§	0.91	0.75, 1.10	0.92	0.63, 1.35	1.21	1.01, 1.45
Critical§	2.02	1.41, 2.90	0.67	0.19, 2.30	2.13	1.32, 3.43
Penetrating§	4.85	4.36, 5.40	1.98	1.60, 2.45	4.28	3.76, 4.88
Trauma level§	3.92	3.39, 4.53	2.31	1.80, 2.97	1.19	1.06, 1.32
Medicaid§	2.44	2.25, 2.64	0.96	0.79, 1.17	1.55	1.43, 1.69
Medicare§	0.27	0.24, 0.31	0.13	0.09, 0.19	0.37	0.35, 0.41
No Insurance§	2.63	2.39, 2.90	6.37	5.42, 7.49	1.97	1.79, 2.18
MV → DV (b path)†						
Time (per 5-min)§	1.02	1.01, 1.03	1.01	0.99, 1.03	1.02	1.01, 1.03
Distance (per mile)§	1.01	1.00, 1.01	1.02	1.01, 1.03	1.01	0.99, 1.01
Severity§						
Mild	Ref.	—	Ref.	—	Ref.	—
Moderate	0.86	0.52, 1.44	0.85	0.36, 1.98	0.83	0.50, 1.38
Severe	5.79	4.53, 4.36	6.38	4.57, 8.89	5.38	4.25, 6.81
Critical	41.12	28.22, 59.90	26.67	14.55, 48.86	43.45	29.32, 64.40
Penetrating§						
No	Ref.	—	Ref.	—	Ref.	—
Yes	4.94	4.02, 5.82	2.29	1.63, 3.23	4.69	3.95, 5.57
Trauma level§						
Level I/II	Ref.	—	Ref.	—	Ref.	—
Level III	1.22	0.89, 1.66	1.22	0.86, 1.73	1.36	1.00, 1.86
Insurance§						
Private	Ref.	—	Ref.	—	Ref.	—
Medicaid	0.79	0.64, 0.97	0.76	0.54, 1.08	0.87	0.71, 1.05
Medicare	1.90	1.53, 2.36	2.61	2.03, 3.35	1.97	1.60, 2.43
No insurance	2.30	1.91, 2.79	1.47	1.05, 2.06	2.03	1.70, 2.43

* Bivariate regression of independent variable on mediating variables. Reference group was non-Hispanic white for African American and Hispanic patients, and female for male patients.
† Multivariable regression of mediating variable on dependent variable (mortality), while controlling for independent variable.
‡ Linear coefficient.
§ Odds ratio.

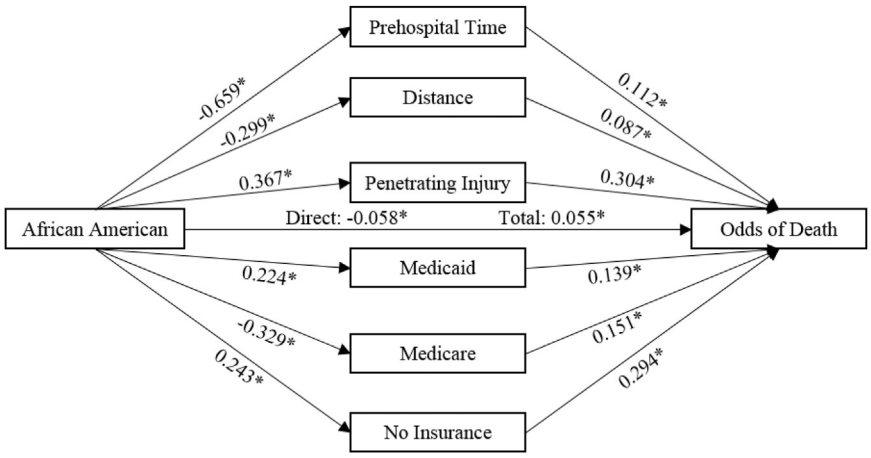

Fig. 1. Multivariate mediation model for injury mortality, comparing African American and white patients in Maryland, 2015. * Indicates statistical significance based on 95% confidence interval.

Mediation of ethnicity

Trauma center distances for Hispanic patients were 3.7 miles shorter ($P < .01$) than those for white patients (Table 2). Hispanic patients were 98% more likely to have penetrating injuries ($P < .01$), 87% less likely to have Medicare coverage ($P < .01$), and 6.4 times more likely to be uninsured ($P < .01$). Controlling for ethnicity, patients with penetrating injuries were 2.3 times more likely to die than those without penetrating injuries ($P < .01$). Compared with patients with private health insurance, odds of death increased by 2.6 times for Medicare patients ($P = .04$) and by 47% for patients without insurance ($P < .01$). Odds of death increased by 2% for every 1-mile increase in distance to the nearest trauma center ($P < .01$).

Standardized coefficients from the multivariable mediation model for the relationship between Hispanic ethnicity and injury mortality are illustrated in Figure 2 and standardized indirect effects of mediating variables are presented in Table 3. The total effect of Hispanic ethnicity was an 11.4% decrease in odds of death, compared with non-Hispanic white patients (95% CI: 6.4%, 16.2%). The effect of ethnicity on mortality was partially mediated by penetrating injury (a*b = 0.020, 95% CI: 0.012, 0.028) and lack of insurance (a*b = 0.073, 95% CI: 0.050, 0.095) and inconsistently mediated by trauma center distance (a*b = −0.014, 95% CI: −0.019, −0.009) and Medicare enrollment (a*b = −0.055, 95% CI: −0.075, −0.036). As described in Appendix B, inconsistent mediation occurs when mediating variables suppress the effect of the independent variable on the dependent variable. The direct effect of ethnicity was a 13.4% decrease in odds of death for Hispanic patients (95% CI: 8.1%, 18.3%), relative to non-Hispanic white patients. The semivariogram of the standardized residuals for the multivariable mediation model of ethnicity did not exhibit residual spatial dependence (Appendix C).

Mediation of sex

Prehospital times for male patients were 9.7 minutes shorter than those for female patients ($P < .01$, Table 2). Males were 2.1 times more likely to have critical injuries ($P < .01$), 4.3 times more likely to have penetrating injuries ($P < .01$), 63% less likely to have Medicare ($P < .01$), 55% more likely to have Medicaid ($P < .01$), and 97% more likely to be uninsured ($P < .01$). Controlling for sex, odds of death increased by 2% for every 5-minute increase in prehospital time ($P < .01$). Compared to patients with mild injury, those with severe injuries were 5.4 times more likely to die ($P < .01$), and those with critical injuries were 43.5 times more likely to die ($P < .01$). Patients with penetrating injuries were 4.7 times more likely to die than those without penetrating injury ($P < .01$). Odds of death were 2.0 times greater for those without insurance ($P < .01$) compared with private insurance.

Standardized coefficients from the multivariable mediation model for the relationship between sex and injury mortality are illustrated in Figure 3 and standardized indirect effects of mediating variables are presented in Table 3. The total effect of male sex was a 27.3% increase in odds of death compared with female patients (95% CI: 21.6%, 33.2%). The effect of sex was partially mediated by critical injury (a*b = 0.114, 95% CI: 0.089, 0.138), penetrating

Table 3
Indirect effects* of variables mediating the relationship between individual demographics and injury mortality

Covariate	African American			Hispanic			Male		
	a*b	95% CI	% Effect mediated	a*b	95% CI	% Effect mediated	a*b	95% CI	% Effect mediated
Total	0.113	0.092, 0.134	−204.6%	0.022	−0.001, 0.044	−18.3%	0.181	0.016, 0.150	82.6%
Time	−0.015	−0.025, −0.005	−27.3%	—	—	—	−0.005	−0.009, −0.001	9.5%
Distance	−0.024	−0.036, −0.013	−44.3%	−0.014	−0.019, −0.009	11.5%	—	—	—
Penetrating	0.104	0.092, 0.115	187.4%	0.020	0.012, 0.028	−16.7%	0.069	0.057, 0.080	28.4%
Moderate Injury	—	—	—	—	—	—	0.001	−0.001, 0.003	0.3%
Severe Injury	—	—	—	—	—	—	0.007	−0.001, 0.015	2.9%
Critical Injury	—	—	—	—	—	—	0.114	0.089, 0.138	47.2%
Medicaid	0.028	0.016, 0.041	51.5%	−0.001	−0.006, 0.003	1.2%	0.003	−0.003, 0.008	1.1%
Medicare	−0.046	−0.062, −0.030	−83.8%	−0.055	−0.075, −0.036	45.7%	−0.040	−0.051, −0.029	−16.6%
No Insurance	0.067	0.003, 0.010	121.0%	0.073	0.050, 0.095	−59.9%	0.024	0.017, 0.031	9.9%

* Indirect effects (a*b) indicate direction and magnitude of mediation. Exponentiated indirect effects cannot be interpreted as odds ratios.

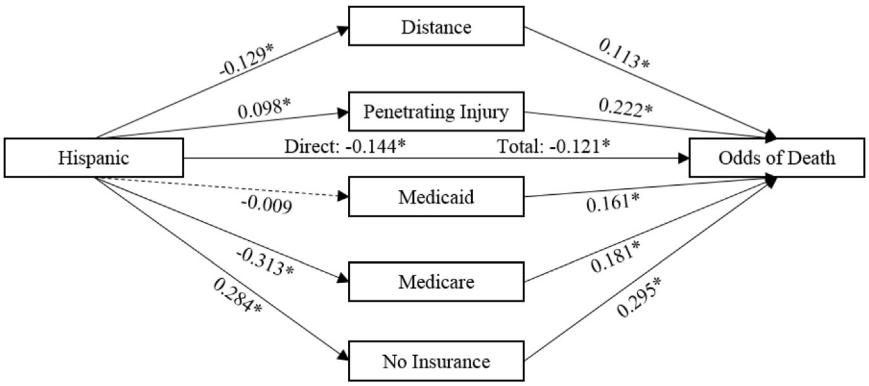

Fig. 2. Multivariate mediation model for injury mortality, comparing Hispanic and non-Hispanic white patients in Maryland, 2015. * Indicates statistical significance based on 95% confidence interval.

injury (a*b = 0.069, 95% CI: 0.057, 0.080), and lack of insurance (a*b = 0.024, 95% CI: 0.017, 0.031). Medicare enrollment (a*b = −0.040, 95% CI: −0.051, −0.029) and prehospital time (a*b = −0.005, 95% CI: −0.009, −0.001) inconsistently mediated the effect of sex, reducing the total effect of sex and masking the direct effect. After adjustment for mediating variables, the direct effect of male sex was a 6.2% increase in odds of death, compared with female patients (95% CI: 1.8%, 10.9%). The empirical semivariogram of the standardized residuals from the multivariable mediation model of sex did not exhibit residual spatial dependence (Appendix C).

Discussion

This study demonstrates the role of trauma center distance, prehospital time, injury characteristics, and insurance coverage as mediators of the relationships between individual demographic characteristics and injury mortality. Although prior studies controlled for these factors when examining injury disparities, this is the first study to place these factors in the causal pathway between individual demographic characteristics and mortality. These findings provide valuable insight into causes of injury disparities, which can inform efforts to improve injury outcomes.

Prior studies of racial differences in injury mortality controlled for individual health and injury characteristics but did not control for prehospital time or trauma center distance [1]. Prehospital times and trauma center distances observed in Maryland were shorter for African American patients, but travel times are longer for African Americans in the United States as a whole [25–27]. Shorter prehospital times and trauma center distances reduced the effect of race on mortality in this study; however, the role of prehospital time and trauma center distance as mediators suggests that differences in access to care in other parts of the United States may contribute to mortality differences observed in national samples. Injury mechanism appears to mediate the effect of race on mortality, consistent with the known mortality effect of penetrating injury [28] and differences in the incidence of intentional penetrating injury by race [8]. The role of insurance type in the pathway between race and mortality may indicate differences in quality of care by payment type. Although the Emergency Medical Treatment and Active Labor Act and other regulations prohibit denial of trauma care based on insurance status [29], there is evidence that the trauma care experience varies by insurance status,

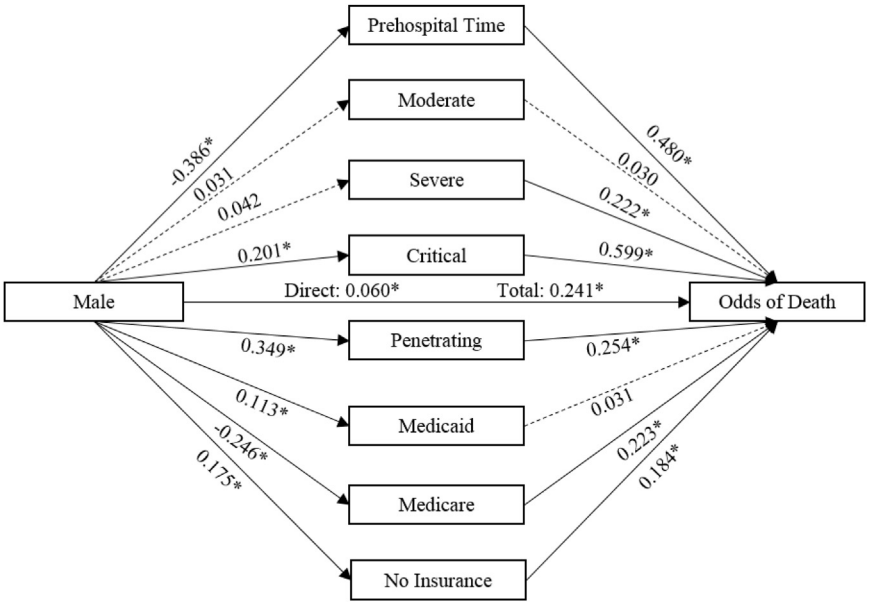

Fig. 3. Multivariate mediation model for injury mortality, comparing male and female patients in Maryland, 2015. * Indicates statistical significance based on 95% confidence interval.

including longer wait times [10] and fewer diagnostic tests [30] for uninsured patients. Insurance status may act as a proxy for factors that could influence injury outcomes, including undiagnosed comorbidities [31].

Trauma center distance appears to mediate the relationship between ethnicity and mortality. Although prehospital time varied by ethnicity, time was not associated with injury mortality when controlling for ethnicity, suggesting that ethnicity confounds the relationship between prehospital time and mortality. No prior studies examined differences in prehospital experience by ethnicity. The patterns observed in this study indicate a need for additional research as variation in prehospital care may explain the apparent protective effect of Hispanic ethnicity in Maryland. Penetrating injuries appear to mediate the relationship between ethnicity and mortality, consistent with known patterns in injury mechanism [8]. Insurance status mediates the relationship between ethnicity and mortality. As with African American populations, this may suggest differential treatment because of insurance status, as well as unmeasured differences in health status. Given the magnitude of the effect of uninsured as a mediator, and the large proportion of Hispanic injured patients without insurance, this is an important area for intervention to improve injury outcomes. This mediation analysis does not explain the observed protective effect of Hispanic ethnicity. It is possible that the sickest or most severely injured Hispanic patients were excluded from this analysis because of undertriage to nontrauma centers or high incidence of death before EMS arrival or that other unmeasured factors mediate the relationship between ethnicity and mortality, including community-level social factors.

The effect of sex was mediated by prehospital time, with male patients benefiting from shorter prehospital intervals. Trauma center distance did not mediate the relationship between sex and mortality, suggesting that the effect of sex confounds the relationship between distance and mortality. This may indicate differences in the prehospital experience for men and women. There is evidence of differences in prehospital triage by sex [32], with male patients receiving higher priority assessments, which may explain the effects observed in this study. Injury mechanism and severity also mediate the effect of sex. Physiologic differences in injury recovery for male and female patients [33,34] may contribute to the indirect effects of both mechanism and severity as determinants of elevated mortality among men. Insurance status mediates the relationship between sex and injury mortality. The indirect effect of Medicare appears to reduce mortality because of the small proportion of men enrolled in Medicare; however, this effect is offset by the increase in odds of death for men because of a higher proportion of patients without health insurance. As with race and ethnicity, this may indicate differences in the trauma care experience, as well as unmeasured differences in health status and warrants further investigation.

Limitations

This study used data from a single state trauma registry. The Maryland trauma care system is uniquely structured and standardized [35], potentially limiting generalizability to other settings. The variables examined as mediators were limited to measures available in trauma registry and EMS records. There are likely unmeasured variables in the causal pathways between patient demographics and injury mortality. The Maryland Trauma Registry includes a limited set of codes for comorbid conditions, based on the best available information at the time of trauma center treatment. These data likely underrepresent incidence and severity of comorbidities. Although incidence of comorbidities is higher in African American and Hispanic populations [36,37], comorbidities did not vary by race, ethnicity, or sex in our analyses nor were they associated with injury mortality. Owing to the structure of the MIEMSS data, treating hospitals were not identifiable in individual patient records, and actual distance traveled was not measured. Based on the geographic distribution of trauma centers in Maryland and the structure of trauma triage protocols, it is unlikely that injured patients would bypass the nearest trauma center.

Conclusion

Identification of factors in the causal pathway between individual characteristics and injury mortality is an important step toward improved injury outcomes through public health and clinical interventions. Insurance status appears to contribute to disparities in injury mortality. Implementation of the Affordable Care Act reduced the proportion of U.S. citizens and permanent residents without insurance, but it did not fully address disparities in insurance coverage for African American and Hispanic populations [38]. Policymakers should consider alternative approaches for trauma care payment to address cost concerns that may contribute to differences in treatment for uninsured patients. Trauma care providers should be mindful of other health factors associated with insurance status, including undiagnosed comorbidities, as these factors may change the needs of individual patients and contribute to injury disparities. Injury mechanism and severity mediate the effects of demographic characteristics on injury mortality, indicating a critical need for primary prevention to reduce disparities in injury mortality. Access to trauma care can improve outcomes for populations with a high incidence of penetrating and critical injuries, but some injuries are not survivable, regardless of the quality and timeliness of care. Primary prevention strategies, including gun violence prevention, are essential to eliminate disparities in injury mortality.

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Appendix A. Methods for multiple imputation

Multiple imputation (MI) with predictive mean matching was used to estimate variance for measures with missing data included in this analysis. Exploratory data analyses did not indicate any patterns in missing data by county, zip code, or calendar month; therefore, missing data were treated as missing at random. A total of ten imputations were used, with a burn in period of twenty iterations per imputation and the ten nearest neighbors included in the random selection for each iteration. Imputed variables included trauma center distance (for patients without a zip code starting point), prehospital time (for patients with missing of implausible times), and Injury Severity Score ([ISS] for patients with insufficient diagnostic information to calculate ISS). All variables were imputed as continuous measures, and ISS was categorized after imputation. Auxiliary variables in the imputation model included injury mechanism, sex, race and/or ethnicity, patient origin (scene/transfer), and mortality outcome. Distribution of ISS and mean time and distance were assessed for observed and MI estimates (Table A). Regression model parameters and standard errors were estimated separately for each imputed data set and then pooled using Rubin's methods for MI [1].

Appendix B. Methods for mediation analysis

Mediation analyses were used in this project to identify factors in the causal pathway between demographic characteristics and injury mortality. We used Baron and Kenny's process for identifying mediation [1] to assess potential mediators and select variables for inclusion in each mediation model (Fig. B). The “c path” or total effect of independent variable is estimated as the unadjusted effect of the independent variable (X) on the dependent variable (Y). The “c' path” or direct effect is the residual effect of the independent variable on the dependent variable after accounting the contributions of mediators (M). Coefficients from bivariable regressions of independent variables on potential mediating variables (“a path”) were estimated with simple linear regression for continuous mediators, and with simple logistic regression for binary mediators and binary dummy variables based on categorical mediators. Coefficients for the relationships between mediating variables and mortality (“b path”) were estimated using logistic regression while controlling for the relevant independent variable. Mediating variables were selected for inclusion in multivariate mediation models if the relationships with both the independent and dependent variables (a and b paths) were statistically significant at $\alpha = 0.05$. Fully adjusted models were then estimated, and likelihood ratio tests were used to assess model fit. Indirect effects, or the effect of the independent variable on the dependent variable via the mediating variable, are calculated as the product of the a and b path effects.

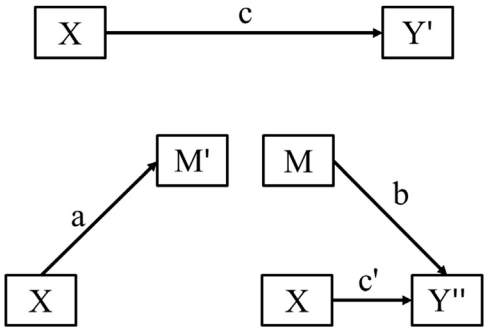

Fig. B. Mediation model for binary outcomes.

In the case of mediation for binary outcomes, such as the analysis we conducted, indirect effects are standardized to account for the difference in scale between the a and b pathways using methods proposed by MacKinnon and Dwyer [2]. Indirect effects are standardized by multiplying each coefficient by the standard deviation of the predictor variable (X or M) and dividing by the standard deviation of the outcome variable (M or Y).

Inconsistent mediation occurs when the mediating variable suppresses the effect of the independent variable on the dependent variable. For example, our findings indicate that prehospital time inconsistently mediated the relationship between sex and injury mortality, such that shorter prehospital times for male patients partially reduce risk of death, offsetting increased risk of death for male patients from other factors associated with sex. Inconsistent mediation is indicated when the direction of the direct effect (c') is different than that of the total effect (c) or when the direct effect is larger than the total. Inconsistent mediation with multiple mediators can result in some mediators “canceling out” the effect of other mediators. Inconsistent mediation can also result in a proportion of effect mediated that is negative or greater than 100%.

Table A
Observed and multiple imputation (MI) estimated trauma center distance and ISS

Covariate	Missing (%)	Observed (%/mean)	MI estimated (%/mean)	95% CI for MI estimates
Injury severity	5.22			
Mild		91.84	89.76	89.11, 90.41
Moderate		3.22	3.65	3.32, 3.97
Severe		3.03	4.41	3.94, 4.88
Critical		1.91	2.18	1.95, 2.41
Miles to trauma center (mean)	31.13	9.12	9.90	9.65, 10.15
Prehospital time (mean min)	27.09	51.17	64.55	63.16, 65.95

Table D
Coefficients (log odds) from bivariable logistic regression on mortality

Covariate	β	95% CI	*P*
Injury severity			
Mild	Ref	—	—
Moderate	−0.650	−1.156, −0.143	.002
Severe	1.039	0.811, 1.266	<.001
Critical	4.048	3.714, 4.381	<.001
Injury mechanisms			
Blunt	Ref	—	—
Penetrating	1.843	1.716, 1.971	<.001
Blunt and penetrating	0.700	0.352, 1.048	<.001
Other	2.232	2.021, 2.444	<.001
Insurance status			
Private	Ref	—	—
Public	0.838	0.644, 1.033	<.001
None	1.492	1.208, 1.777	<.001
Miles to trauma center	0.010	0.005, 0.016	<.001
Prehospital time (min)	0.003	0.001, 0.006	.002

Mediating variables in this analysis were selected based on bivariable analysis of the association between potential mediators and injury mortality. The results of these analyses were previously published [3]. Coefficients from the bivariable analyses for selected mediating variables are presented in Table D.

Appendix C. Assessment of residual spatial dependence

In geospatial statistics, events occurring at the same or similar location are inherently related to each other because of similarity of exposures for spatially proximal events. If not addressed properly, this spatial autocorrelation violates the assumption of independence in regression modeling, thus residuals from regression models with spatially indexed events must be assessed to ensure that there is no autocorrelation of residuals. One method for assessing spatial autocorrelation is visual examination of the empirical semivariogram of the residuals or the spatial distribution of semivariance ($\widehat{\gamma}(h)$) derived from the observed data. Semivariograms are plotted by pairing each observation ($z_i, \ldots, z_k$) with all other observations and then calculating the spatial distance between each pair (h). Each pair is then placed in a bin based on the distance between observations ($h \pm \delta$), and the semivariance is calculated for each bin as one-half of the mean squared difference between the studentized residuals for each pair of observations:

$$\widehat{\gamma}(h \pm \delta) = \frac{\sum_{(i,j) \in N(h \pm \delta)} |Z_i - Z_j|^2}{2|N(h \pm \delta)|}$$

Average semivariance for each bin (Y axis) is then plotted against average distance within each bin (X axis). If the plot indicates that semivariance is associated with distance, then the model should be adjusted to either include additional variables that account for spatial dependence or the model should be specified to account for autocorrelation. If the semivariogram does not indicate residual spatial correlation, then it can be inferred that the variables in the model account for all spatial dependence between observations and the residuals are spatially independent.

For our analyses, we visually examined semivariograms for each of the three multivariable mediation models we estimated. The semivariograms of the studentized residuals are presented in Figure C.

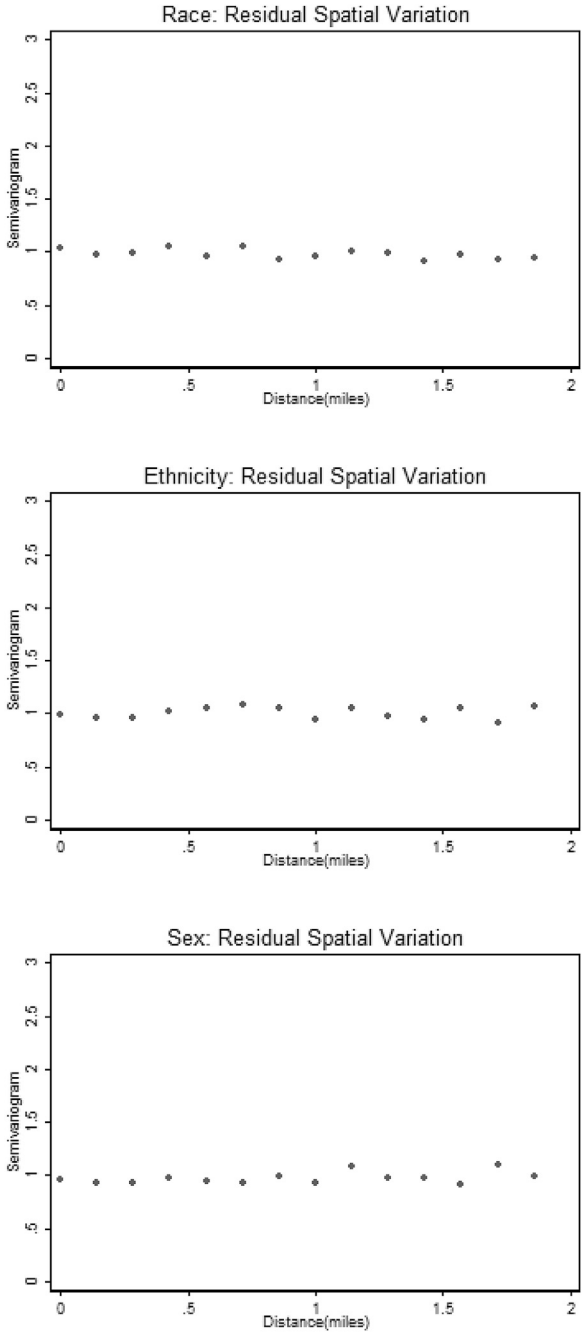

Fig. C. Semivariograms of studentized residuals for multivariable mediation models.

Further readings

Appendix A

[1] Rubin DB. Multiple imputation for nonresponse in surveys. New York: Wiley; 1987.

Appendix B

[1] Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. J Pers Soc Psychol 1986;51(6):1173–82.
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