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Triggering of acute myocardial infarction by different means of transportation

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Abstract

Background: Prior studies have reported an association between traffic-related air pollution in urban areas and exacerbation of cardiovascular disease. We assess here whether time spent in different modes of transportation can trigger the onset of acute myocardial infarction (AMI).

Design: We performed a case-crossover study. We interviewed consecutive cases of AMI in the KORA Myocardial Infarction Registry in Augsburg, Southern Germany between February 1999 and December 2003 eliciting data on potential triggers in the four days preceding myocardial infarction onset.

Results: A total of 1459 cases with known date and time of AMI symptom onset, who had survived 24 hours after the onset, completed the registry's standard interview on potential triggers of AMI. An association between exposure to traffic and AMI onset 1 hour later was observed (odds ratio: 3.2; 95% confidence interval [CI]: 2.7–3.9, $p < 0.001$). Using a car was the most common source of traffic exposure; nevertheless, times spent in public transport or on a bicycle were similarly associated with AMI onset 1 hour later. While the highest risk for AMI onset was within 1 hour of exposure to traffic, the elevated risk persisted for up to 6 hours. Women, patients aged 65 years or older, patients not part of the workforce, and those with a history of angina or diabetes exhibited the largest associations between times spent in traffic and AMI onset 1 hour later.

Conclusion: The data suggest that transient exposure to traffic regardless of the means of transportation may increase the risk of AMI transiently.

Keywords

Acute myocardial infarction, environment, epidemiology, registries, trigger

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Introduction

Acute myocardial infarction (AMI) is an important contributor to cardiovascular disease morbidity and mortality. AMI has a sudden onset and the timing of onset and often appears to occur by chance. In some cases, AMI can be attributed to lifestyle factors including strenuous exertion,[1–4] anger[5] or marijuana[6] or cocaine use.[7] Environmental factors such as elevated concentrations of ambient particulate matter has also been documented to trigger AMI in some,[8,9] but not all studies.[10]

We previously reported that time spent in traffic, travelling in a car, bus, tram, or riding a bicycle or a motorbike, was associated with the onset of myocardial infarctions 1 hour later applying the case-crossover

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design.[11] Traffic exposures were identified as posing the highest risks on a population level based on a review and a comparison of population attributable risks for triggers of myocardial infarctions.[12] This result is based on our previous study[11] only and there is the need to further substantiate these findings based on the fact that time spent in traffic is a necessity of modern lifestyles up to old age.[13]

In the present report, we have the following aims: (a) to extend the previous findings[11] including data on 769 additional cases of AMI, (b) to compare the effects sizes of different modes of transportation, and (c) to evaluate differences in risks for subgroups such as women and subjects with prior history of cardiovascular disease.

Methods

Augsburg Myocardial Infarction Registry

Hospitalized AMI survivors aged 25–74 years are routinely registered by the KORA-Myocardial Infarction Registry Augsburg (KORA: Cooperative health research in the study region of Augsburg) and details of the procedures can be found elsewhere.[14] Briefly, cases of AMI were identified in the Central Hospital on a daily basis, once a week in six hospitals within the study area and in four hospitals adjacent to the study area in hot pursuit based on a wide range of cardiovascular disease admission diagnoses. Informed consent for participation was received from all patients. Data on socio-demographic characteristics, medical history, and smoking behaviour was collected as part of the registry's standardized interview by trained research nurses. After the patients' discharge, clinical data was abstracted from medical records according to a standardized protocol.

Time of AMI onset was defined as time of onset of chest pain with at least 20 min of duration. In cases with atypical chest pain (53 of 1459 cases) or other symptoms (29 of 1459 cases), the time of the severest symptoms was considered. Supporting information was retrieved from the patient's medical records (e.g. history of symptoms recorded by the ambulance physician or the emergency room physician). If data conflicted, medical reports were considered as being more reliable than information provided by the patients.

Diary study

Activities of the patients on the day of the AMI and during the 3 days before the onset of AMI symptoms were recorded in a standardized, interview-based diary following the registry's routine interview.[15] The diary included sleeping periods, activity levels during the day, time spent outdoors, means of transportation used, location within the study area, angina pectoris symptoms, occurrence of extreme anger or joy, and exposure to dust or solvents. Activities taking place between 0 and 59 minutes after each hour were ascribed to that hour. The ability of the patients to remember activities grouped by categories were coded in the questionnaire administered to elicit the diary information. Adherence to standardized interview and coding procedures was ensured by repeated training and supervision of the three research nurses.

Data analyses

Conditional logistic regression models were used to assess the association between transient individual level traffic exposures and AMI onset.[16] We computed odds ratios as a measure of relative risk (RR) of having an AMI during periods of time spent in traffic compared with time not spent in traffic within an individual. One-hour periods in the 0 to 6 hours before AMI symptom onset were selected as the hazard periods. Control periods were defined as exposure of the same case 24 to 71 hours before AMI onset. By design, patient characteristics were controlled for, as each subject was their own control. In addition, time of day was controlled for using 23 dummy indicators in all analyses. Alternative approaches considering case and control periods matched by time of day were also computed. Multivariable adjustments considered additional information on triggers including standing up after sleeping, strenuous exertion and time spent outdoors. Stratified analyses assessed effect modifications by patient characteristics and p-values were computed using chi-squared tests.

Patients were required to be within the study area consisting of the city of Augsburg and the two adjacent counties during all times considered in the analyses. The rationale was to exclude extended travelling which is associated with additional unusual activities.

Role of the funding source

The Health Effects Institute reviewed the study design at the beginning of the study and an independent data integrity assessment was conducted for the data collected between 1999 and 2001 (for details, see Peters et al.[15]). The sponsor had no role in data collection, data analysis, data interpretation or writing the report.

Results

Between February 1999 and December 2003, 2059 consecutive cases of confirmed AMI were available for interview by the KORA Myocardial Infarction Registry. A total of 600 cases were unable to provide

Table 1. Description of the acute myocardial infarction survivors (aged 25–74) recruited between February 1999 and December 2003 from the KORA Myocardial Infarction Registry, Augsburg, Germany

		Feb 1999 to Dec 2003 $N = 1459$	Feb 1999 to July 2001 $N = 690$	Aug 2001 to Dec 2003 $N = 769$	Tests for differences p-value
Age [years]	Mean	60	60	60	0.98
	25–49	255 (17%)	126 (18%)	129 (17%)	0.49
	50–64	664 (46%)	303 (44%)	361 (47%)	
	65–74	540 (37%)	261 (38%)	279 (36%)	
Male		1119 (77%)	531 (77%)	588 (76%)	0.82
German		1384 (95%)	655 (95%)	729 (95%)	0.91
Employment status	Employed	589 (40%)	280 (41%)	300 (39%)	0.54
Education	low education*	1118 (77%)	531 (77%)	587 (76%)	0.84
First MI		1235 (85%)	595 (86%)	640 (83%)	0.06
28 day survival		1450 (99%)	686 (99%)	764 (99%)	1.00
STEMI‡	STEMI	725 (50%)	392 (57 %)	343 (45%)	0.001
	NSTEMI	625 (43%)	257 (37 %)	368 (48 %)	
	Bundle Branch Block	68 (5%)	30 (4 %)	38 (5 %)	
	Missing	31 (2%)	11 (2 %)	20 (3 %)	
Symptoms of MI	Typical	1377 (94%)	646 (94%)	731 (95%)	0.40
	Atypical	53 (4%)	27 (4%)	26 (3%)	
	Other	29 (2%)	17 (2%)	12 (2%)	
Cardiovascular disease before onset	Angina Pectoris	329 (23%)	162 (23%)	167 (22%)	0.42
	Hypertension	1048 (72%)	457 (66%)	591 (77%)	<0.0001
	Diabetes	397 (27%)	192 (28%)	205 (27%)	0.61
	Neither	295 (20%)	162 (23%)	133 (17%)	0.004
Smoking status	Smoker	531 (36%)	248 (36%)	283 (37%)	0.91
	Non-Smoker	489 (34%)	235 (34%)	254 (33%)	
	Ex-smoker	439 (30%)	207 (30%)	232 (30%)	
Hospitals	Central Hospital	1183 (81%)	558 (81%)	625 (82%)	0.84
Time [days]	Median (min, max)	8 (1,54)	9 (3,54)	6 (1,36)	<0.0001

*8–11 years; ‡STEMI: ST-elevation myocardial infarctions; NSTEMI: non-ST-elevation myocardial infarctions.

diary information and/or valid information on the time of AMI symptom onset. The 1459 AMI cases included in the diary study were predominantly male and with a mean age of 60 years (Table 1). The cases interviewed between February 1999 and July 2001 were comparable to those interviewed between August 2001 and December 2003, with the exception that the cases interviewed in the later period had a higher prevalence of non-ST elevation myocardial infarction, reported more frequently a history of hypertension, and were interviewed earlier after their myocardial infarction. A clear circadian pattern in AMI symptom onset was observed (Figure 1A).

Exposure to traffic was more frequent on the day of AMI onset (1265 hours of 18,458 person-hours equal to 6.9%) than on the previous 3 days (2008 hours of 35,441 person-hours equal to 5.7% [1 day before]; 1689 hours of 28,512 person-hours equal to 5.9% [2 days before]; 1314 hours of 22,665 person-hours equal to 5.8% [3 days before]) (Figure 1B). One hour before AMI onset, traffic exposure was more than twice as frequent (13.5%) than at other times (Figure 1C). A total of 74% of all hours spent in traffic were in a car, 10% on a bicycle, 14% in public transport (buses, trams and trains) and 2% on a motorbike. The frequency of public transport was slightly higher in the later period compared with the earlier period (14.3% versus 12.8%) and the frequency of bicycle use was slightly lower (9.9% versus 10.9%).

In the analysis adjusting only for time of day, exposure to traffic showed a 2.9- to 4.9-fold higher risk for AMI symptom onset 1 hour later for different means of transportation (Table 2). After additional adjustment for standing up after sleeping, strenuous exertion and

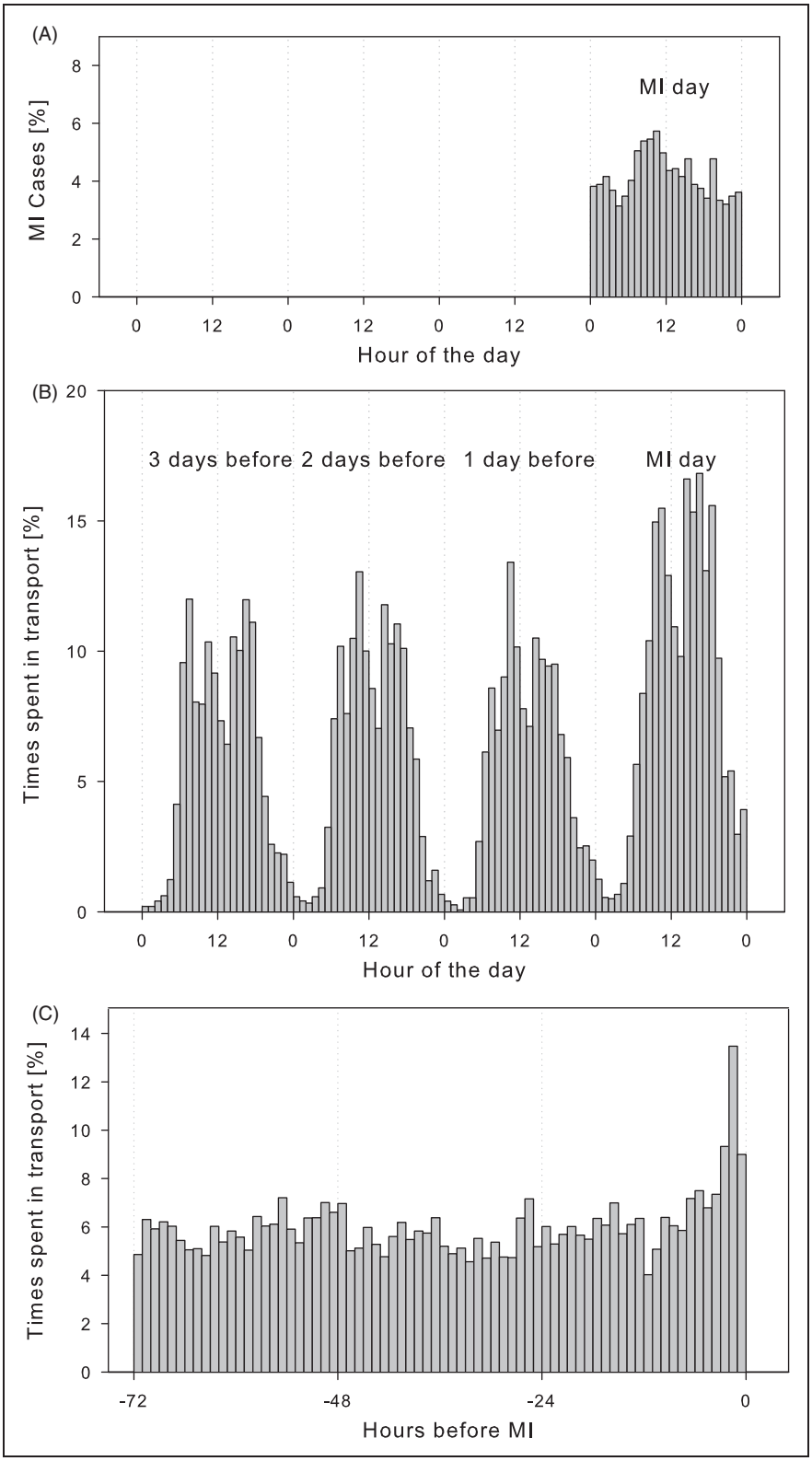

Figure 1. (A) Distribution of acute myocardial infarction (AMI) onset over the day in 1466 non-fatal myocardial infarction survivors, KORA Myocardial Infarction Registry, February 1999 to December 2003 in Augsburg, Germany. (B) Times spent in traffic on the day of myocardial infarction onset and 3 days before. (C) Times spent in traffic in the 72 hours preceding myocardial infarction onset.

Table 2. Relative risk for experiencing an acute myocardial infarction one hour after times spent in traffic by means of transportation adjusted for time of day. Vulnerable case period was 1 hour before MI onset, control periods were 24 to 71 hours before MI onset. KORA Myocardial Infarction Registry, February 1999 to December 2003

	Feb 1999 to Dec 2003		Feb 1999 to July 2001		Aug 2001 to Dec 2003	
	% exposed† (total *N*)	Relative Risk (95% CI)	% exposed† (total *N*)	Relative Risk (95% CI)	% exposed† (total *N*)	Relative Risk (95% CI)
Any means of transportation§						
Unadjusted for triggers*	12.8% (1339)	3.29 (2.74–3.95)	12.2% (624)	2.92 (2.22–3.83)	13.4% (715)	3.65 (2.85–4.67)
Adjusted‡	12.9% (1334)	3.20 (2.65–3.86)	12.2% (623)	2.74 (2.06–3.63)	13.5% (711)	3.66 (2.84–4.71)
Cars						
Unadjusted for triggers*	9.0% (1339)	2.89 (2.34–3.57)	8.3% (624)	2.60 (1.89–3.58)	9.5% (715)	3.14 (2.37–4.17)
Adjusted‡	9.0% (1334)	3.31 (2.67–4.10)	8.3% (623)	2.95 (2.13–4.08)	9.6% (711)	3.64 (2.73–4.86)
Bicycle						
Unadjusted for triggers*	2.4% (1339)	4.92 (3.22–7.52)	2.4% (624)	3.93 (2.14–7.23)	2.4% (715)	6.26 (3.45–11.4)
Adjusted‡	2.4% (1334)	2.56 (1.59–4.10)	2.4% (623)	1.85 (0.94–3.64)	2.4% (711)	3.58 (1.85–6.95)
Public transport						
Unadjusted for triggers*	1.2% (1339)	2.91 (1.68–5.04)	1.3% (624)	3.07 (1.40–6.70)	1.1% (715)	2.77 (1.28–6.01)
Adjusted‡	1.2% (1334)	2.90 (1.67–5.07)	1.3% (623)	3.12 (1.42–6.86)	1.1% (711)	2.70 (1.23–5.94)

*Analyses adjusted for the time of day to control for potential influences due to circadian variation by using 23 indicator variables; †One hour before the MI onset; ‡Adjusted in addition for strenuous exertion, being outdoors and standing up after sleeping concurrently; §Any means of transportation combines times spent in cars, in public transportation, on motorbikes or bicycles; MI, myocardial infarction; CI, confidence interval.

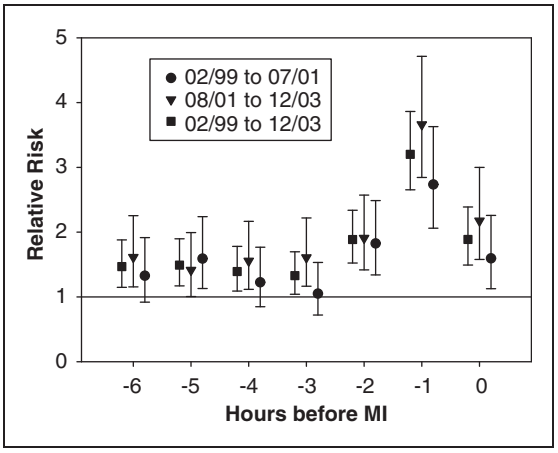

Figure 2. Relative risk for experiencing an acute myocardial infarction after times spent in traffic adjusted for time of day, strenuous exertion, being outdoors and standing up after sleeping concurrently. Vulnerable case periods were 0 to 6 hours before AMI onset, control periods were 24 to 71 hours before myocardial infarction (MI) onset. KORA Myocardial Infarction Registry, February 1999 to December 2003.

being outdoors, exposure to traffic showed a 2.6- to 3.3-fold higher risk for AMI onset 1 hour later. The adjusted odds ratios were similar for travel in cars and public transport, but halved for riding a bicycle compared with the unadjusted odds ratios. The RR for any traffic exposure 1 hour before AMI onset changed only slightly by adjustment for strenuous exertion (RR = 5.8, 95% CI: 4.0–8.5), being outside (RR = 2.1, 95% CI: 1.7–2.6) and standing up after sleeping (RR = 1.8, 95% CI: 1.5–2.3).

Effect estimates for time spent in traffic were slightly larger during the second period than in the first period. The peak of the estimated risk for traffic exposure was 1 hour before the onset of symptoms (Figure 2). However, a moderately elevated risk was observed for exposures 3–6 hours before the onset of symptoms. Supplemental Table 1 describes the induction times for the specific means of transportation indicating that potentially bicycling could have an immediate onset and less persistent elevated risk.

Information on traffic exposure was available for 99% (0–23 hours before AMI onset), 90% (24–47 hours before AMI onset), 71% (48–71 hours before AMI onset) and 63% of the hours (72–95 hours before AMI onset). A relative risk of 2.91 (95% CI: 2.07–4.08) was observed when considering only one control period matching on time of day (Supplemental Table 2). The equivalent of this model is the discordant pair analysis using McNemar's estimator. The odds ratio is derived by dividing 131 cases (exposed to traffic during case but not control period) by 45 cases (exposed to traffic during control but not case period) ($p < 0.001$). Estimates were slightly larger if the case-crossover analyses selected three controls matched on time of day (RR = 3.42, 95% CI: 2.59–4.53). Counterfactual analyses of traffic exposures during non-risk periods suggested no substantial recall

Table 3. Subgroup analyses for traffic exposures* 1 hour before AMI onset, case-crossover analyses restricted to activities within the study area adjusted for strenuous exertion, being outdoors and standing up after sleeping. KORA Myocardial Infarction Registry, February 1999 to December 2003 in Augsburg, Germany

	N	%	Relative risks†	95% confidence intervals	Test for heterogeneity of subgroups (*p*-value)
All	1334	100%	3.20	(2.65–3.86)	
Men	1009	76%	2.88	(2.33–3.56)	0.012
Women	325	24%	5.20	(2.46–7.80)	
Age 25–49	231	17%	2.76	(1.85–4.13)	0.066
Age 50–64	598	45%	2.89	(2.20–3.79)	
Age 65–74	505	38%	4.66	(3.29–6.61)	
Employed	516	39%	2.53	(1.93–3.31)	0.004
Not employed	818	61%	4.42	(3.38–5.77)	
First MI	1125	84%	3.04	(2.48–3.73)	0.22
Survival > 28 days	1326	99%	3.22	(2.67–3.89)	0.10
STEMI	486	36%	3.21	(2.49–4.12)	0.59
NSTEMI	402	30%	3.41	(2.57–4.53)	
Diabetes	370	28%	4.39	(3.05–6.31)	0.058‡
Hypertension	966	72%	3.48	(2.78–4.34)	0.20‡
Angina	303	23%	6.10	(4.07–9.13)	<0.001‡
Neither	265	20%	2.61	(1.73–3.92)	0.26§
Current Smokers	493	37%	2.73	(2.01–3.70)	0.24
Ex-Smokers	449	34%	4.03	(2.88–5.64)	
Non–Smokers	392	29%	3.31	(2.34–4.69)	
Intermittent AMI symptoms 4 days before AMI onset	346	26%	3.53	(2.45–5.10)	0.67
No symptoms	993	74%	3.22	(2.61–3.98)	
Flu-like symptoms during week before MI	108	8%	2.93	(1.43–6.03)	0.80
No flu-like symptoms	1225	92%	3.23	(2.66–3.93)	

*Traffic exposures combine times spent in cars, in public transportation, on motorbikes or bicycles; †Analyses adjusted for the time of day to control for potential influences due to circadian variation by using 23 indicator variables and for strenuous exertion, being outdoors and standing up after sleeping concurrently; ‡Compared with the neither group; MI, myocardial infarction; AMI, acute myocardial infarction; STEMI, ST–elevation myocardial infarctions; NSTEMI, non-ST-elevation myocardial infarctions.

bias considering the time 24–71 hours before AMI onset (Supplemental Table 3).

Exposure to traffic seemed to be associated with larger risks in women than in men and in patients aged 65 and older than in those younger (Table 3). Subjects not part of the workforce had larger effect estimates than those employed, which in part might be a function of age. Patients with a history of diabetes or angina appeared to have a higher risk of AMI onset following traffic exposures compared with those without these risk factors. We stratified patients for the occurrence of typical or atypical intermittent symptoms of AMI symptoms during the 72 hours before AMI onset. Comparable effect estimates were observed regardless of the presence of intermittent symptoms. In addition, the association between traffic exposure and AMI symptom onset was similar for patients with non-ST-elevation myocardial infarction and those with ST-elevation myocardial infarction.

Discussion

We found a higher risk of AMI symptom onset within 1 hour of being exposed to traffic (travelling in cars, busses, trams, riding a bicycle or a motorbike) applying the case-crossover study design. The study further suggested, while the highest risk for AMI onset was within 1 hour of exposure to traffic, the elevated risk persisted for up to 6 hours.

Using a car was the most common mode of transportation; nevertheless, time spent in public transport was also associated with AMI onset 1 hour later. While driving a car, symptoms of possible arrhythmia may be common in patients eligible for implanted defibrillator treatment.[17] There is no information in this study available as to whether subjects who used a car had been driving the car themselves or under which traffic conditions and where they travelled. However, because the association was observed for persons using public transportation, it is unlikely that the whole effect is attributable to the stress associated with driving a car.

While in the earlier analyses, estimates for using a bicycle were not statistically significant after adjustment for strenuous exertion and other transient factors, these associations with AMI onset remained statistically significant in the larger dataset presented here. The risk of AMI onset was highest immediately after riding a bicycle and returned to baseline after 2 hours. There is a clear need to further substantiate our findings, because signs of ischemia were induced upon dilute diesel exhaust exposures in a controlled setting, which mimic exposures in frequently travelled tunnels, in patients with stable coronary artery disease exercising on a bicycle ergometer.[18] Summarizing the results of different modes of transportation, we found comparable transient risks for AMI for all modes of transportation assessed.

Transient risk factors have only a short-term impact on AMI. Chronic risk factors such as smoking, dyslipidaemia, sedentary lifestyles promote atherosclerosis and prothrombotic states, and have a long-term impact by increasing vulnerability of a patient for an

acute coronary event.[19] Factors that increase plaque vulnerability were considered as potential effect modifiers of the association between times spent in traffic and AMI onset. The study suggested that women, patients age 65 years or older, patients with a history of diabetes and angina, and retired or currently non-employed persons have higher risks.

The result in women was unexpected, and given the relatively smaller sample size in women than in men, may be treated with caution. However, women suffering from myocardial infarctions may have more co-morbidities such as diabetes[20] which may render them more susceptible to triggering by traffic exposure. In addition, unemployment was an unexpected effect modifier. Based on the study design, diary data may mask exposure contrasts in employed subjects that spent more time and at regular hours of the day in traffic and therefore the effect of traffic-related exposures may be underestimated in employed subjects. A prior diagnosis of angina may be considered as a crude measure of progressed atherosclerosis. Consistently, the risk in subjects with re-infarction was slightly larger than in subjects with their first AMI without the difference achieving statistical significance. The fact that no difference was observed between subjects with ST-elevation myocardial infarction and non-ST-elevation myocardial infarction may indicate that multiple pathophysiologic mechanisms contribute to the onset of AMI in association with times spent in traffic.

Potentially, traffic exposure is a surrogate marker for a combination of different factors such as stress, noise and traffic-related air pollution might be responsible for the observed associations. Among the traffic-related pollution freshly generated exhaust particles may be of particular concern for being associated with cardiovascular disease exacerbation.[21] Studies on individual exposures to ambient particles indicated that passengers in cars and busses were more highly exposed than people at ambient background stations.[22,23] The concentrations varied by route and traffic density and might resemble those at kerbsides. Compared with people travelling by car or bus, particle exposure is about twice as high as for cyclists.[22,24–26] Although, higher ventilation rates increase the amount of particles deposited in the airways, cyclists might be able to leave congested situations, i.e. polluted microenvironments faster than people in cars or busses.[22] We used a combination of ambient soot concentration and the diary data to compute personal exposures to soot particle.[27] These analyses suggested an increased risk of AMI associated with personal estimated 24-hour average soot concentrations, while estimated ambient soot or measured fine particles showed no association. Noise from different sources has been associated with increases of ambulatory blood pressure transiently within 15 minutes[28] and thereby may contribute to the onset of myocardial infarctions. Also, acute exposure to stress is considered trigger myocardial infarctions.[29] Considering the short induction time, potentially the activation of the autonomous nervous system may be involved in triggering AMI during time spent in traffic.[21] In order to further disentangle the causal factors associated with times spent in traffic further studies are needed quantifying the health impact of exposures to stress, noise and traffic related air pollution jointly and elucidating the underlying pathomechanisms.

Strength and limitations

The study represents a unique collection of interviews in consecutive cases of AMI survivors. The interview provides information on triggers during the highly vulnerable time period before AMI onset. The diary was well accepted and the patients appreciated the interest in their individual circumstances. The interviewing nurses were unaware of the hypotheses tested here. Between 2001 and 2003, bedside interviews were conducted closer to the event than in the years 1999 and 2000 (difference in median: 3 days). This might have resulted in more accurate reports potentially providing a reason for the slightly higher effect estimates in the second time period compared with the first time period. Patient's differential recall of the activities before AMI onset is a major concern. We conducted two sets of additional analyses in order to address this concern. First, we conducted sensitivity analyses applying different control selection strategies and found consistent results. Second, we conducted counterfactual analyses selecting case and control periods in the a priori defined control periods and found no strong evidence for differential recall over time. Furthermore, car trips may have been made to seek help due to an evolving AMI. However, when considering only patients without typical or atypical symptoms during the 72 hours before AMI onset in the analyses, a comparable association was observed.

The case-crossover design matched on the individual so that confounding by time invariant conditions was eliminated. The results were robust when control periods matched on the time of day indicating that circadian variation was not confounding the results. Other transient risk factors such as strenuous exertion or stress might confound the observed associations. However, multivariable analyses using the available information on other triggers did not suggest evidence for strong within-person confounding for either taking a car or public transportation. Strenuous exertion was confirmed as a substantial risk factor for AMI onset in

this study and was previously reported.[1–4] Confounding by strenuous exertion is a particular concern when assessing bicycle use and indeed risk estimates for bicycle use halved after controlling for strenuous exertion. This indicated that the initially larger estimate presented a combination of the effect of traffic-related exposure and moderate exertion as shown previously.[4]

The results of the study may not be generalizable to other at-risk populations. In particular, the results cannot be extrapolated to a potential risk of fatal AMI due to traffic exposure. In fatal cases, interview data would need to be elicited from next of kin, which to the authors seems to be impossible with the instruments developed for the study presented here. Furthermore, the subgroup analyses suggested that the overall risk might be larger in all AMI survivors or even subjects who had a fatal AMI as the studied population is restricted to healthier and younger subpopulation of AMI survivors based on the ability to participate in a diary study. Traffic participation in other locations may be more or less hazardous than in Augsburg, which is a medium-sized German city without extended rush-hour strain.

Conclusion

The data presented here suggest that transient exposure to traffic might pose a risk in patients at higher risk of AMI. Relative risks of similar effect sizes were observed for using a car, public transport or riding a bicycle. The findings substantiate the conclusion of a recent review highlighting traffic exposure and ambient particles as important triggers for AMI,[12] especially when considering the population burden of AMI triggers.[13] With the updated estimates provided here, the population attributable fraction is estimated to be 11.1% (95% CI: 8.4–14.0%) based on the updated effect estimates and the 5.6% prevalence of traffic exposure in the population during the control periods 24–71 hours before AMI onset. Currently, it is impossible to apportion the relative contribution of risk factors such as stress or traffic-related air pollution. Nevertheless, patients vulnerable to acute coronary events are likely to profit from recent efforts to improve air quality in urban areas by low emission zones resulting in cleaner vehicle fleets and further improved city planning.

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Conflict of interest

The authors have no conflicts to disclose.

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