ORIGINAL ARTICLE

Long driving time is associated with haematological markers of increased cardiovascular risk in taxi drivers

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Aims: To examine the association between driving time and changes in haematological markers of increased risks for cardiovascular diseases (CVD).

Methods: The authors conducted a cross sectional analysis of baseline data from the Taxi Drivers' Health Study cohort in Taipei, Taiwan. They retrieved information on comorbidity, laboratory tests, age, and anthropometric measures from medical records of 1157 subjects (mean age 44.6 (SD 8.6) years). Whole blood cell (WBC) count was used as the primary haematological marker for increased CVD risk, and platelet count and haematocrit as the secondary markers. Standardised questionnaires were implemented to collect information on demographics, lifestyle, work related physical and psychosocial factors, and driving time profiles. Multiple regression was used to estimate the adjusted effects of driving time on three haematological markers.

Results: The mean measured hematological marker was 6656 (SD 1656) cells $\times 10^6/l$ for WBC, 47.2 (SD 3.5) % for hematocrit, and 243 (SD 52) cells $\times 10^9/l$ for platelets. The driving time was 264 (SD 76) hours/month. Compared with drivers who drove ≤ 208 hours/month (1st quartile cut off), drivers who drove > 208 hours/month had a higher WBC count (by $317 \times 10^6/l$; 95% CI 99 to 535), haematocrit (by 0.8%; 95% CI 0.3 to 1.2), and platelets ($7.9 \times 10^9/l$; 95% CI 1.0 to 14.8). After adjusting for conventional CVD risk factors (age, sex, smoking, hypertension, diabetes, and hypercholesterolaemia), obesity, alcohol drinking, regular exercise, and sociodemographics (education, marital status, income, and so on), long driving time was still associated with significant increases in WBC and platelets, whereas the effect on haematocrit was diminished and became statistically non-significant. Additional controls for physical workload, self-perceived job stress, and job dissatisfaction did not alter the associations with increased WBC and platelets.

Conclusions: Longitudinal studies are needed to confirm the observed cross sectional association and to further examine the specific occupational exposures accountable for the association between driving time and haematological markers of systemic inflammation and haemostatic alteration.

Cardiovascular diseases (CVD) of professional drivers remain an important issue in occupational health research and clinical practice. For decades, occupational epidemiological studies have provided a large body of consistent evidence showing that professional drivers are at high risk for CVD. High morbidity and mortality related to coronary artery diseases^{1–4} and cerebrovascular events⁵ have been found among professional drivers. Using a population based hospital discharge registry of myocardial infarction (MI), Bigert *et al*⁶ compared the relative risk (RR) of MI among professional drivers in Stockholm County, Sweden, during both 1977–84 and 1985–96. They found that, compared with manual workers, taxi drivers and lorry operators had an increased MI risk which persisted throughout the entire study period (with corresponding RR of 1.38 and 1.35 among taxi drivers and both of 1.14 among lorry drivers during each period). Their data suggested that, although CVD incidence in professional drivers has been decreasing since the 1970s, existing preventive measures seemed to have only little effect on the excessive CVD risk directly imparted by occupational factors involved in driving. This disturbing time trend also underscores the needs for a better understanding of work related CVD among professional drivers.

In spite of the convincing epidemiological data, the underlying mechanisms linking professional driving to increased CVD risks remain unclear. Earlier studies in the 1980s suggested that some conventional risk factors, such as

smoking and hypercholesterolaemia, were more prevalent among professional drivers and accounted for their increased risk for CVD.^{7–8} Later studies have mostly refuted this speculation and revealed an increased CVD risk independent of conventional risk factors among drivers. In their prospective study in Gothenburg, Rosengren *et al*⁹ demonstrated that the excess risk of coronary heart disease among middle aged bus and tram drivers was independent of conventional CVD risk factors, as the RR was only diminished slightly (from 3.3 to 3.1) after controlling for major cardiac risk factors. Similar findings were also reported among bus and taxi drivers.⁴

Researchers have begun to examine more specific pathophysiological changes related to CVD among professional drivers,¹⁰ and a few neuroendocrine and neuroelectrophysiological pathways have been investigated. For example, studies have demonstrated that various driving related activities, such as physical loading, mechanised work, traffic congestion, long distance driving, might result in increased urinary excretion of catecholamine,^{11–12} increased cortisol level,^{13–14} and decreased heart rate variability.^{15–16} These neurocardiological responses are all plausible mediators as they may exert a transient effect on the cardiovascular system or amount to significant cardiac risk over a long period. However, the sample size of most of these studies was small,

Abbreviations: BMI, body mass index; CBC, complete blood count; CVD, cardiovascular diseases; TDHS, Taxi Drivers' Health Study; WBC, white blood cell

and potential confounding by other concurrent CVD risk factors was not adequately addressed.

Enormous progress made during the past few decades has dramatically enhanced our understanding of the pathophysiology responsible for CVD. Investigations in vascular biology have pointed to a central role of inflammation in the initiation and progression of atherosclerosis.^{17–19} Whether inflammation and haemostatic alteration are also involved in the increased CVD of professional drivers has not been very well studied. In the current study, we aimed to examine the association between driving time and haematological markers of increased CVD risks. We hypothesised that the levels of haematological markers of CVD risks increased with driving time.

METHODS

Study population

The study population was the baseline cohort of the Taxi Drivers' Health Study (TDHS) in Taipei City, Taiwan. Details about the design of TDHS have been described elsewhere.^{20–21} In brief, in 2000 the Taipei City Government designated five hospitals to provide taxi drivers with free physical examinations. Between January 31 and May 31, 3295 taxi drivers participated in this medical monitoring programme, and the TDHS subjects came from the hospital with the largest assigned service volume. We previously reported that TDHS participants were a representative sample of taxi drivers in Taipei City and their baseline characteristics of TDHS participants were not systemically different from those receiving medical examinations in other hospitals.²⁰ The main outcomes of research interest at the design phase of TDHS included cardiovascular disease risks, job stress, and musculoskeletal disorders, among others. For drivers to be eligible for enrollment in the TDHS, they had to have been: (a) registered taxi drivers in Taipei City for at least one year, (b) willing to participate, and (c) able to read. The research protocols and consent forms were approved by the institutional review board of the Taipei Veterans General Hospital, Taipei, Taiwan. Informed consent was obtained from every TDHS participant in the selected hospital.

Outcome measures

Data on the haematological markers were retrieved from the laboratory blood routines which included complete blood count (CBC), fasting blood sugar, and other clinical biochemistries. Although previous studies have linked CVD risks to increased white blood cells (WBC) count,^{22–24} haematocrit,²⁵ and platelets count,²⁶ findings on the increased CVD risks and adverse CVD outcomes associated with high WBC counts were more consistent than those studies on haematocrit and platelet counts.²⁷ Therefore, we used WBC as the primary haematological marker of increased CVD risks, and the other two as the secondary marker. The CBC test was performed in the clinical laboratory at the selected hospital by an Automated Hematology Analyzer (SE-9000, Sysmex Corp, Kobe, Japan) following conventional validated methods.

Measurement of covariates

We developed a standardised self-administered instrument to measure personal characteristics and occupational factors in this study. The feasibility of this instrument was tested in a convenience sample of drivers before the study began. In addition to data on demographics and lifestyle factors (smoking, alcohol drinking, exercise), the questionnaires consisted of items for professional seniority in years, monthly driving duration in hours, and average frequency of physical workload (lifting task and bending/twisting) at work and during leisure time. Previous studies have found that self-reporting is a relatively reliable and valid method to assess

the time spent in driving.^{28–29} In a small subset of baseline data from drivers who also participated in an exposure assessment study,³⁰ we also found that self-reported monthly driving time was fairly reliable (Pearson's correlation coefficient = 0.79), in comparison with data from driving diaries records and structured interviews. Age, sex, anthropometric measures (body weight and height), measures of blood pressure, and results of other relevant laboratory tests were also retrieved from the medical examination files. Drivers were classified as diabetics if they had fasting sugar >126 mg/dl or physician diagnosed diabetes with subsequent treatment. Hypertension was defined as either measured systolic/diastolic blood pressures $\geq 140/90$ mm Hg or physician diagnosed hypertension with subsequent treatment. Drivers were also classified as having normal (<200 mg/dl), borderline high (200–239 mg/dl), or high (≥ 240 mg/dl) total cholesterol. LDL and HDL cholesterol levels were not measured. Body mass index (BMI) was used to evaluate whether drivers were normal (BMI <24 kg/m²), overweight (BMI 24–26.9 kg/m²), or obese (BMI ≥ 27 kg/m²).³¹

Statistical analysis

We used multiple regression to estimate the effect of monthly driving time on haematological markers of CVD risk. We grouped drivers into four categories according to the quartile distribution of total hours of taxi driving per month. For a covariate to be considered in the final model, it must have resulted in at least a 10% change in the regression coefficient representing effect of driving on haematological markers or each covariate should be statistically significant at the 0.2 level in the univariable analysis. In order to make statistical inference conditional on all established CVD risk factors, we included age, sex, smoking (non-smoker *v* ex-smoker *v* current smoker), hypertension, diabetes, and hypercholesterolaemia (normal *v* borderline high *v* high) in the final model, regardless of their levels of statistical significance in the univariable analyses. We assumed no interaction terms among potential predictors, and only included cases with complete data information in the final analyses. All of these statistical analyses were carried out by STATA 7.0 statistical software (STATA Corporation, College Station, TX, USA). The distribution of residuals calculated for presented final models conformed to the normality assumption for each of three dependent variables.

RESULTS

During the study period for collecting baseline TDHS data, of the 1355 drivers receiving medical examinations in the selected hospital, 1242 (92%) participated in the questionnaire survey, and 1192 (89%) had complete information on both driving time and routine CBC data. Thirty five subjects with WBC >12 000 or <3500 cells $\times 10^6/l$, which likely reflected some acute pathological processes or underlying diseases, were excluded, thus leaving 1157 (mean age 44.6 (SD 8.6) years) eligible for the analyses. The measured haematological marker was 6656 (SD 1656) cells $\times 10^6/l$ for WBC, 47.2 (SD 3.5) % for haematocrit, and 264 (SD 76) cells $\times 10^9/l$ for platelets. The majority (52%) of drivers had been professional taxi drivers for more than 10 years. The mean driving time per month was 264 (SD 76) hours, corresponding to approximately 10 (SD 2) hours per day and 26 (SD 2) days per month. Forty two per cent (42%) were current smokers, 35% were overweight, and 24% were obese. The prevalence of hypertension, diabetes, and hypercholesterolaemia (total cholesterol >240 mg/dl) was 15%, 12%, and 32% respectively.

In the crude analyses, monthly driving time was associated with all three haematological markers of increased CVD risks (table 1). Compared with drivers who drove ≤ 208 hours/month (1st quartile cut off), drivers who drove >208 hours/

Table 1 The average white blood cells (WBC) count, haematocrit, and platelet count, and their associations* with driving time in Taxi Drivers' Health Study, Taipei, Taiwan

Monthly driving time (hours) ×10 ⁶ /l†	WBC count (cells ×10 ⁶ /l)†	Adjusted‡ regression coefficients	Haematocrit (%)†	Adjusted§ regression coefficients	Platelets count (cells ×10 ⁹ /l)†	Adjusted¶ regression coefficients
1st quartile (≤208)	6420 (1599)	Reference	46.6 (3.6)	Reference	236.9 (50.6)	reference
2nd quartile (210–260)	6680 (1686)	264 (–2 to 529)	47.2 (3.7)	0.3 (–0.3 to 0.8)	241.9 (53.9)	2.7 (–6.4 to 11.8)
3rd quartile (261–312)	6733 (1671)	278 (20 to 539)**	47.3 (3.1)	0.1 (–0.5 to 0.6)	245.5 (51.7)	7.5 (–1.4 to 16.4)
4th quartile (318–450)	6799 (1652)	285 (16 to 555)**	47.6 (3.3)	0.4 (–0.1 to 1.0)	246.9 (53.4)	9.6 (0.3 to 18.9)**
(p value for trend test)	0.006	0.04	0.001	0.24	0.01	0.02

*All regression coefficients obtained from multiple regression were expressed as the difference in average levels of haematological markers between the reference and the indicated categories.

†Results shown as mean (standard deviation).

‡Adjusted for age, sex, marital status, education, registration type, exercise frequency, alcohol drinking, BMI, smoking, hypertension, diabetes, and hypercholesterolaemia.

§Adjusted for age, sex, professional seniority, marital status, income, registration type, BMI, smoking, hypertension, diabetes, and hypercholesterolaemia.

¶Adjusted for age, gender, marital status, education, income, registration type, BMI, smoking, hypertension, diabetes, and hypercholesterolaemia.

**p<0.05.

month had a higher WBC count (by 317 cells ×10⁶/l; 95% CI 99 to 535), haematocrit (by 0.8%; 95% CI 0.3 to 1.2), and platelet count (7.9 ×10⁹/l; 95% CI 1.0 to 14.8). We noted that such an increase in WBC count was not predominated by any specific cell type, since the proportions of increased neutrophils (60%, p = 0.02), monocytes (7%, p = 0.05), and lymphocytes (30%, p = 0.05) were comparable with the usual haemogram. The univariable regression also revealed that, for drivers who were unmarried, beyond high school education, less engaged in strenuous exercise, or affiliated with taxi cab companies, the WBC counts were higher than those of their counterparts (all p values<0.07). The average WBC count of drivers with normal BMI (6531×10⁶) was lower than average WBC count of overweight (6703×10⁶) or obese drivers (6802×10⁶). The WBC count did not differ significantly by professional seniority.

The presence of conventional CVD risk factors except age was associated with high WBC counts of taxi drivers in the univariable analyses (table 2). Active smokers had statistically significant higher WBC counts than past smokers and those who never smoked. Female drivers had lower WBC count than male drivers. Hypertension, diabetes, and hypercholesterolaemia were all associated with increased WBC counts in taxi drivers. In the multiple regression model which included the main effect of monthly driving time adjusting for age, marital status, education, registration type, regular exercise, BMI, and all the other conventional CVD risk factors, higher WBC count was consistently associated with smoking, hypertension, diabetes, and hypercholesterolaemia.

Additional analyses were conducted to evaluate residual confounding and to examine whether the observed long driving time/WBC count association could be explained by work related physical or psychosocial factors. Firstly, the modest but positive correlation between driving time and number of cigarettes used (Spearman's $r = 0.14$, $p = 0.01$) prompted us to evaluate the residual confounding by the amount of cigarette smoking. We found that WBC count increased with the amount of cigarette smoking: each 10 cigarettes smoked per day was associated with an increase in WBC count by 423 cells ×10⁶/l among active smokers. Even after adjusting for the amount of active cigarette smoking, those who drove more than 208 hours per month had significantly higher WBC count (increased by 458 cells ×10⁶/l) than drivers who drove 208 hours or less. Secondly, we additionally included variables representing the frequency of physical workload (lifting task and bending/twisting) at work, and self-reported job stress perception and job dissatisfaction index, which were derived from the validated Chinese version of Job Content Questionnaire.³² None of the

physical workload was associated with WBC count. In the multiple regression, job dissatisfaction index did not affect WBC count, although a marginal increase in WBC count was noted among those in the highest quartile ($p = 0.09$). In contrast, self-perceived job stress was associated with WBC count ($p = 0.02$). Compared with drivers reportedly with no perceived stress, the WBC count increased by 198 (95% CI –46 to 442) cells ×10⁶/l for drivers with mild job stress and by 333 (95% CI 53 to 612) cells ×10⁶/l for drivers perceiving moderate to severe job stress. Additional adjusting for self-reported physical workload and work related psychosocial factors did not alter the association between long driving time and high WBC count. Compared with the average WBC count of drivers who drove 208 hours/month or less, drivers

Table 2 The average white blood cells (WBC) count in relation to conventional risk factors for cardiovascular diseases (CVD)

CVD risk factors	WBC count (cells ×10 ⁶ /l)*	Adjusted regression coefficients*†
Smoking		
Never smokers	6122 (1360)	Reference
Ex-smokers	6469 (1498)	412 (123 to 700)‡
Current smokers	7259 (1776)	1204 (999 to 1409)‡
Sex		
Female	6132 (1642)	Reference
Male	6678 (1654)	41 (–443 to 525)
Hypertension		
No	6605 (1637)	Reference
Yes	6937 (1734)	439 (175 to 703)‡
Diabetes		
No	6610 (1666)	Reference
Yes	6991 (1547)	346 (65 to 627)§
Total cholesterol		
Normal	6475 (1567)	Reference
Borderline high	6641 (1683)	161 (–61 to 384)
High	6854 (1693)	372 (137 to 606)‡

*All the differences in average WBC counts across categories of each risk factor were statistically significant ($p < 0.05$). All regression coefficients obtained from multiple regression were expressed as the difference in average levels of haematological markers between the reference and the indicated categories.

†Adjusted for age, sex, marital status, education, registration type, exercise frequency, alcohol drinking, body mass index, monthly driving duration, and all the other CVD risk factors in the table.

‡ $p < 0.01$.

§ $p < 0.05$.

Diabetes: fasting sugar >126 mg/dl or physician diagnosed diabetes with subsequent treatment; hypertension: systolic/diastolic blood pressures ≥140/90 mm Hg or physician diagnosed hypertension with subsequent treatment; total cholesterol: classified as normal (<200 mg/dl), borderline high (200–239 mg/dl), or high (≥240 mg/dl).

with driving time longer than 208 hours/month had a statistically significant ($p = 0.03$) increase in the WBC count by 241 (95% CI 23 to 459) cells $\times 10^6/L$.

DISCUSSION

Our cross sectional study provided the first epidemiological data suggesting the involvement of increased inflammation and altered haemostasis in CVD of taxi drivers. We demonstrated that haematological markers of increased cardiovascular risks were associated with long driving time, and such association could not be attributable completely to the conventional CVD risk factors. The consistent driving related increase in WBC count highlighted an associated inflammatory responses involved either in the development or progression of CVD in taxi drivers. Adhesion of circulating leukocytes has been known to be one of the earliest morphological events in the development of atherosclerotic plaque. Indeed, large population based cohort studies, such as the Framingham Heart Study²² and Atherosclerosis Risk in Communities (ARIC) Study,²³ have reported an increase in long term CVD risk associated with high WBC count. Recruitment, activation, and infiltration of inflammatory leukocytes have significant contributions to plaque rupture. Also, clinical investigations have indicated that patients with an increased WBC count have an increased early (30 day) mortality following MI³³ or acute coronary syndrome.³⁴

Which specific occupational exposures might account for the observed association between long driving time and the presumed inflammatory marker of increased CVD risk in taxi drivers? Urban taxi drivers are regularly exposed to a variety of physical (for example, physical workload, traffic noise, whole body vibration) and chemical hazards (for example, carbon monoxide, vehicle exhaust) and work related psychosocial factors (for example, job stress, night-time driving), which have all been considered detrimental for cardiovascular health. The existing evidence on physical workload/CVD relation is mixed;^{35–36} our analyses did not reveal any association of WBC count with work related physical load (lifting, bending, and/or twisting) in Taipei taxi drivers. The observed association between self perceived job stress and WBC count conforms to the notion that work related psychosocial stressors may promote inflammatory responses and culminate in atherosclerosis.^{37–38} In spite of the likelihood of residual confounding by other psychosocial domains of job stress, given the statistical significance which remained after adjusting for the work related psychosocial factors in the multiple regression, our data might imply that the observed driving time/WBC count association perhaps involved other important occupational factors. Nevertheless, we did not measure drivers' exposures to vehicle exhaust, although convincing data from environmental studies have shown that exposures to particulate air pollution in urban environment are associated with increased WBC count.^{39–40} Further studies are needed to identify the specific occupational exposures with the most significant contribution to systemic inflammation and altered haemostasis, and differentiate their relative and joint effects on the CVD of professional drivers.

Among the TDHS participants, conventional CVD risk factors constituted an important portion of the inflammation associated increase in cardiac risk, as smoking, hypertension, diabetes, and hypercholesterolaemia were all positively associated with WBC count (table 2). This finding was consistent with other clinical investigations and supported the central role of inflammation underlying the process of atherosclerosis contributed by each conventional CVD risk factor.^{41–42} Many previous studies have found that professional drivers had a high prevalence of conventional CVD risk factors, which had been implicated in the intermediates

between driving and CVD.^{7–8} Compared with the national estimates for Taiwanese men aged 45–64 years,⁴³ male taxi drivers of similar age did have significantly higher diabetes prevalence (15.6% *v* 7%, $p < 0.001$) and total cholesterol level (225.3 (SD 41.4) *v* 198.2 (SD 38.3) mg/dl; $p < 0.001$), although the difference in diastolic blood pressure (85.2 (SD 12.8) *v* 84.4 (SD 12.7) mm Hg; $p = 0.14$) and active smoking (43% *v* 44%; $p = 0.52$) were not statistically distinguishable.⁴⁴ In the final multiple regression model, adjusting for these conventional CVD risk factors did downsize the effect of driving time (comparing the 4th and 1st driving time quartile) on WBC count (285 *v* 379 $\times 10^6$). However, it is noteworthy that the driving time/WBC count association remained statistically significant in the adjusted analysis. Our study results thus reinforced the previous notion that increased frequency of CVD among professional drivers could not be solely explained by the high prevalence of conventional CVD risk factors.

We recognise several limitations to our study. Firstly, because of its cross sectional design, the observed association between long driving time and increased WBC and platelet counts cannot definitely be interpreted as causal. Although we could not provide any good reasons that drivers with high WBC count might have preferred or chosen long driving time, it is statistically arguable that these drivers might happen to have high WBC counts at baseline. Secondly, WBC count is a sensitive but non-specific haematological marker for various physical and psychosocial stressors. Although we have restricted our analyses to those with presumably normal WBC count and also adjusted for many demographical, socioeconomic, lifestyle, and clinical variables, we could not rule out the possibility of unmeasured confounding by non-occupational sources of mental stress (for example, stressful life events with increased financial needs) which may translate to both long driving time and increased WBC count. Thirdly, high WBC and platelets counts assessed in this study may simply reflect surrogate measures of acute phase reactants (for example, C-reactive protein, fibrinogen) involved in the pathogenesis of CVD; they do not necessarily reflect functional abnormalities with direct involvement in atherosclerosis, such as leukocyte activation, overexpression of leukocyte adhesion molecules, or leukocyte-platelet interaction. Future studies investigating the speculated vascular inflammation and haemostatic alteration in the development and progress of CVD in professional drivers should look into more specific mechanistic measures.

CONCLUSION

Our study demonstrates that haematological markers of increased cardiovascular risks are associated with long driving time independent of conventional risk factors in taxi drivers. Longitudinal studies are needed to confirm the observed cross sectional association and to further investigate the associations between specific occupational exposures and biomarkers of systemic inflammation and haemostatic alteration.

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Main messages

- The mechanisms underlying high frequency of cardiovascular disease in professional drivers remain unclear.
- The study provided the first epidemiological data suggesting the involvement of inflammation and haemostatic alteration.
- The consistent association between driving time and high WBC count was independent of conventional CVD risk factors.

Policy implications

- Longitudinal studies are needed to confirm the observed cross sectional association and to further examine the associations between specific occupational exposures and biomarkers of systemic inflammation and haemostatic alteration.

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