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ORIGINAL RESEARCH

The Influence of Smoking on Renal Functions Among Apparently Healthy Smokers

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Background: Cigarette smoking is an important modifiable risk factor in kidney disease progression. Although long-term smoking has been associated with chronic kidney disease (CKD), its effect on kidney function in early stages has not been clarified.

Objective: To detect the early effects of smoking either active or passive on kidney functions.

Methodology: The current study was comparative cross sectional study conducted on 280 participants, 140 were non-smokers and 140 were smokers (70 passive smokers and 70 active smokers). The two groups were comparable in terms of all parameters. We investigated the possible effects of smoking on kidney functions using both serum kidney function tests especially; serum urea, serum creatinine, serum cotinine levels and detection of albumin in urine. Smoking history, full Laboratory investigations, Ventilatory function test including (FEV1/FVC, FEV1, FEF 25–75%, VC and FVC) were done.

Results: Serum urea, serum creatinine, serum cotinine levels and urinary albumin were statistically significant higher in smokers group in comparison to nonsmokers, also the serum cotinine levels and urinary albumin were statistically significant in active smokers in comparison to passive smokers. There were positive correlations between the level of urinary albumin and pack/year ($r = 0.9$, $p<0.05$), smoking index ($r = 0.9$, $p<0.05$), smoking duration ($r = 0.4$, $p<0.05$), and serum cotinine ($r = 0.6$, $p<0.050$) with good statistical significance. The most significant predictive risk factors of microalbuminuria among smokers group in descending orders were active smoking, passive smoking, age and serum cotinine level.

Conclusion: Both active and passive smoking, especially among heavy smokers, is a significant risk factor for microalbuminuria. This finding increase the importance of early cessation of smoking in order to minimize early renal affection among healthy smokers that may not be discovered by routine renal function tests.

Keywords: active smokers, passive smokers, microalbuminuria, renal function

Introduction

The World Health Organization reported that tobacco use leads to catastrophic effects on public health and that it is a direct cause of death for more than 7 million people and an indirect cause of death for 1.2 million people per year, worldwide.[1] Many studies reported the adverse effects of smoking on renal functions. Albuminuria, glomerulonephritis and nephrosclerosis were reported more commonly in smokers.[2] Epidemiological studies postulated that smoking should be considered as one of the most significant risk factors for nephropathies. This is especially true for those with pre-existing kidney disease and elderly men with chronic hypertension, but recently many studies suggest that smoking can also affect renal functions in patients with known normal renal functions. So; an early detection of renal affection is important, allowing early interventions while the renal functions still not yet been exhausted and the excretory capacity has not been markedly affected.[3] Smoking can induce renal damage by different potential mechanisms. It include acute effects like sympathetic stimulation (with elevation in blood pressure and heart rate, Increasing the renal vascular resistance leading to a reduction in GFR and renal plasma flow), and chronic effects, especially

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impaired endothelial cells functions.[4] Exposure to passive smoking has been reported to be associated with significantly increases lipid peroxidation in liver, increased catalase activity in the kidney, cardiac and blood vessels diseases, leading to abnormal renal blood flow and causing renal affections over time.[5] Nicotine is the main component of tobacco smoking. Nicotine is mainly transformed to cotinine (active metabolite). Cotinine is usually used as a biomarker for tobacco exposure due to its higher blood concentration and longer half life time than nicotine.[6] It is a good quantitative test for detection of smoking exposure that can be measured in different body fluids, including saliva, plasma, and urine.[7] A negative correlation between serum cotinine and the eGFR has been concluded, indicating that as the smoking increases, It leads to a decrease in renal function.[8] Recent studies have shown a strong association between second-hand smoke exposure and impaired renal function.[9]

Aim of the Work

To detect early effects of smoking (both active and passive) on kidney function tests in terms of serum urea, serum creatinine, and microalbuminuria.

Subjects and Methods

The current study was conducted at AL-Azhar University Hospitals, Cairo, Egypt, in the period from 1\62021 to 1\8\2022.

Study Participants

An analytic cross sectional comparative study was implemented on 280 individuals. The subjects included in this study were divided into two groups as follows. The 1st group was healthy smokers (n = 140), including active tobacco smokers (group n = 70), and passive smokers (n = 70), the 2nd group was healthy nonsmokers (n = 140) as a comparative (control group). The control group was age and sex matched with smoker group. The included participants were chosen by systematic random technique from relatives of the patients who came to our hospital due to several diseases, hospital workers and the volunteers frequented public places such as cafes where cigarette smoke was dense. Volunteers who had no previous medical complaints or symptoms and were not exposed to cigarette smoke were selected as control subjects.

Active smokers are defined based on self-reported smoking status as those who are currently smoke at least one cigarette per day, while passive smokers for at least 5–6 hours during the day. The nonsmokers were defined as those who never smoke cigarette or shisha in their life. Both groups were asymptomatic and have not any history of renal disease.

National Center for Health Statistics 2017 defined current smoker, passive smoker and non-smoker:

- Current smoker: Person who has smoked 100 cigarettes or more in his or her lifetime and who is still smoking cigarettes. Since 1991 this group has been divided into “everyday” smokers and “some days” smokers.
- Environmental Tobacco Smoke: (second-hand smoker or passive smoker). Refers to cigarette smoke and exposure from the nearby environment of a nonsmoker.
- Never smoker: person who did not smoke at all, or who smoked less than 100 cigarettes in his or her life.[10]

Exclusion Criteria

- Smokers above 55 years old.
- Any individual with known chest disease.
- Patient known to have any liver, kidney, malignancy or autoimmune diseases.
- Patients with chronic diseases such as diabetes mellitus, hypertension, Hypo/hyperthyroidism, metabolic syndrome and hematological abnormalities.
- Patients receive any medical treatment or receiving long term analgesics, Routine alcohol intake and ex-smokers.

Methods

All participants were subjected to full clinical examination; thorough history was taken with special emphasis on age, sex, age of starting smoking, number of cigarette smoked daily and duration of smoking. The smoking index (pack/year) was calculated as a number of packs smoked daily multiplied by number of years of smoking. According to smoking index the active smokers group divided into:

(a) Mild active cigarette smoker (n = 33) (SI< 400 cigarettes/year).
(b) Moderate active cigarette smoker (n = 11) (SI = 400–800 cigarettes/year).
(c) Heavy active cigarette smoker (n = 26) (SI = > 800 cigarettes/year).[11]

Spirometry was done using SPIROSIFT SP5000, (Japan). The following measurements were recorded; Vital Capacity (VC), Forced Vital Capacity (FVC), Forced Expiratory Volume in the First Second (FEV1), FEV1\FVC ratio and Forced Expiratory Flow rate 25–75 (FEF25-75). Spirometric indices were calculated in accordance with the recommendations of the ERS by using the best out of three technically acceptable trials.[12,13]

Only subjects with normal pulmonary function test were included in this study.

The blood pressure was measured by auscultatory method using mercury sphygmomanometer. Two readings were taken (at least 1–2min. interval) and the mean value of the two measurements was used, High BP was defined as a systolic blood pressure more than or equal 130 mmHg or a diastolic blood pressure more than or equal to 85mmHg, any individual with high blood pressure was excluded.

Routine laboratory investigations: Complete blood count (CBC) was performed using the automated haematology analyser Sysmex KX 21N (Kobe, Japan), and biochemical analyses using the Cobas c311 system (Roche Diagnostics, Mannheim, Germany) for kidney, liver function parameters and microalbumin in urine (using 24 hours urine).

Serum cotinine level was measured by ELISA using an ELISA kit supplied by antibodies-online GmbH (Cat. No ABIN2683886, Germany) according to the manufacturer's instructions. The minimum detection limit of the assay was 5 ng/mL.

We defined normal creatinine levels range from 0.5 to 1.5 mg/dL, urea concentration with reference values 10–50mg/dl. The Microalbuminuria was defined by a rise in urinary albumin excretion between 30 to 300 ug/day.

Sample Size

Sample size was calculated using formula, N = Z2pq/e2,[14] with assumption of prevalence of CKD among daily smokers was 10%.[5] Accordingly, the minimum acquired sample size was 140 healthy smokers. We added another 140 as a healthy comparative nonsmoker group.

Ethical Considerations

The study was conducted in accordance with the Declaration of Helsinki. The study objectives, design and all details were fully explained to participants. The study was conducted after its approval by the institutional review board (IRB), faculty of medicine for girls, Al-Azhar University, Cairo, Egypt. Participation was voluntary; an informed consent was taken before enrolment into the study. Each participant had the right to refuse participation or withdraw from the study at any time without giving any reasons and without any interference with their rights of medical care. Also, data were anonymous and coded to assure confidentiality of participants.

Statistical Analysis

Data were analyzed using the statistical package for social sciences, version 21.0 (SPSS Inc., Chicago, Illinois, USA). Tests of normality were done. Quantitative normally distributed data presented using mean and standard deviation, while non-normally distributed one were presented using median and inter-quartile range (IQR). Independent-samples *t*-test was used for comparing between two means (parametric data) and Mann Whitney *U*-test in non-parametric one. We used ANOVA or Kruskal–Wallis tests for comparing between more than 2 means. Qualitative variables were presented as frequency and percentages and Chi-square (x2) test was used to compare between qualitative parameters. Pearson's or Spearman's rho correlation tests were used to evaluate the relationships between quantitative variables. Logistic regression analysis was used to identify the independent determinants of microalbuminuria. Statistical significance was considered at a p-value less than 0.05.

Results

The current study was conducted on 280 participants, 140 were non-smokers and 140 were smokers (70 passive smokers and 70 active smokers). There was no statistical significant difference between smoker and nonsmoker

groups regarding age, CBC parameters, liver functions, serum electrolytes level and blood sugar. Serum urea, serum creatinine, serum cotinine levels and urinary albumin were statistically significantly more in smokers group compered to nonsmokers group (P = 0.001) (Table 1). The serum cotinine levels and urinary albumin were significantly increased in active smokers in comparison to passive smokers (P = 0.001), while there was no significant difference in serum urea and creatinine levels between the two groups (Table 2). Among the active smokers group the serum cotinine and urinary albumin levels were significantly increased in both moderate and

Table 1 Baseline Characteristics Among Smokers and Non-Smokers

Groups / items	Non-smokers N = 140	Smokers N = 140	*P*-value
Age (yrs)			
Mean± SD	42±12.7	43.5±10.9	0.3
PLT			
Mean± SD	276.9±76.2	265.6±83.8	0.2
WBCs			
Mean± SD	7.3±1.9	7.2±2.2	0.7
RBCs			
Mean± SD	4.5±0.7	4.4±1	0.9
Urea (mg/dL)			
Mean± SD	26.5±7	31.1±6.9	0.000*
Creatinine (mg/dL)			
Mean± SD	0.7±0.2	0.9±0.3	0.000*
Urinary albumin (mg/day)			
Mean± SD	15.9±6.5	67.1±62.3	0.000*
Median	16.9	32	
IQR	10.4	63.2	
Serum cotinine (ng/mL)			
Mean± SD	6.02±2.3	297.7±288.7	0.000*
Median	6	160	
IQR	4	533.7	
AST (mg/dL)			
Mean± SD	22.2±5.6	22. 9±8.2	0.8
ALT (mg/dL)			
Mean± SD	22±7.8	22.2±10.5	0.8
Na (mmol/l)			
Mean± SD	139.2±3.6	139.5±3.9	0.5
K (mmol/l)			
Mean± SD	4.1±0.4	4.2±0.4	0.3
HBA1C (mg/dL)			
Mean± SD	4.3±0.7	4.5±0.9	0.06
Random blood sugar (mg/dL)			
Mean± SD	130.3±12.4	132.7±9.2	0.07

Notes: *Indicates statistically significant difference, *P*-value <0.05.

Table 2 Kidney and Liver Function Parameters Among Active and Passive Smokers

Groups / items	Active smokers N = 70	Passive smokers N = 70	*P*-value
Urea (mg/dL)			
Mean± SD	31.5±6.9	30.6±7	0.5
Creatinine (mg/dL)			
Mean± SD	0.9±0.3	0.9±0.2	0.7
Urinary albumin (mg/day)			
Mean±SD	91.5±77.3	42.7±24.9	0.000*
Median	33	28.5	
IQR	159.4	37	
Serum cotinine (ng/mL)			
Mean±SD	421.4±354	173.9±107.3	0.000*
Median	400	150	
IQR	713	162.5	
AST (mg/dL)			
Mean± SD	30.3±9.3	28.2±6.9	0.1
ALT (mg/dL)			
Mean± SD	23.1±12.2	21.3±8.4	0.3
Na (mmol/l)			
Mean± SD	139.6±4.2	139.5±3.6	0.8
K (mmol/l)			
Mean± SD	4.2±0.5	4.1±0.4	0.2

Notes: *Indicates statistically significant difference, *P*-value <0.05.

heavy smoker's groups relative to light smokers group (P = 0.00) (Table 3). There was a statistically significant positive correlations between the level of urinary albumin and pack/year (r = 0.9, p<0.05), smoking index (r = 0.9, p<0.05), smoking duration (r = 0.4, p<0.05), and serum cotinine (r = 0.6, p<0.05) while there was weak statistically negative correlation with FVC (r = −0.3, p<0.05) among studied active smokers, and there were statistically significant correlations between the level of urinary albumin and smoking duration (r = 0.4, p<0.05), serum cotinine (r = 0.4, p<0.05), and FVC (r = 0.3, p<0.05) among studied passive smokers. Additionally, there was no statistically significant correlation between creatinine and urinary albumin either among active or passive smokers (Table 4). Multiple comparisons of spirometric indices between active smokers, passive smokers and control group showed significant reduction in FVC, FEV1 and FEV1\FVC in active and passive smokers groups in compassion to control group (p<0.05) (Table 5). Table 6 demonstrates the most significant predictive risk factors of microalbuminuria

Table 3 Distribution of Urinary Albumin and Serum Cotinine Among Active Smokers

Groups / items	Light smokers N = 33	Moderate smokers N = 11	Heavy smokers N = 26	*P*-value
Urinary albumin (mg/day)	24.9±6.2	78.6±66.9	181.4±17.9	0.000*
Mean±SD	P1 = 0.000*, P2 = 0.000*, P3 = 0.000*			
Serum cotinine (ng/mL)	207.7±316.1	341±336.8	726.5±113	0.000*
Mean±SD	P1 = 0.1, P2 = 0.000*, P3 = 0.000*			

Notes: P_1 (difference between light and moderate smokers), P_2 (difference between light and heavy smokers), P_3 (difference between moderate smokers and heavy smokers). * indicates statistically significant difference, *P*-value <0.05.

Table 4 Correlation Between Urinary Albumin and Some Parameters Among Active Smokers and Passive Smokers

Different parameters	Active smokers		Passive smokers	
	r	*p*-value	r	*P*-value
Age (years)	0.02	0.8	0.2	0.1
Pack/year	0.9	0.000*	———	———
Smoking index	0.9	0.000*	———	———
Smoking duration	0.4	0.000*	0.4	0.001*
Serum cotinine (ng/mL)	0.6	0.000*	0.4	0.000*
Serum creatinine (mg/dL)	0.04	0.7	0.1	0.1
FVC	−0.2	0.03*	0.3	0.005*
FEV1	−0.3	0.008*	0.1	0.2
FEV1\FVC	0.1	0.2	0.2	0.06

Note: *Indicates statistically significant difference, *P*-value <0.05.

Table 5 Multiple Comparisons of Spirometric – Indices Between Active Smokers, Passive Smokers and Control Groups

Spirometric indices		Non-smokers	Active smokers	Passive smokers
FVC	Mean±SD	86.8±3.1	82.1±6.4	83.3±4.8
	P-value	P1 = 0.000*, P2 = 0.000*, P3 = 0.09		
FEV1	Mean±SD	85.8±3.9	80.5±7. 2	82.9±5.5
	P-value	P1 = 0.000*, P2 = 0.000*, P3 = 0.007*		
FEV1\FVC	Mean±SD	85.4±3.6	78.8.9±6.2	82.4±6.6
	P-value	P1 = 0.000*, P2 = 0.000*, P3 = 0.000*		

Notes: P_1 (difference between non-smokers and passive smokers), P_2 (difference between non-smokers and active smokers), P_3 (difference between active smokers and passive smokers). *Indicates statistically significant difference, *P*-value <0.05.

Table 6 Determinants of Microalbuminuria Among Studied Group

Risk factors	OR (95% CI)	*P*-value
Smoking status (ref non-smoker)		
-Passive smoker	23.5 (6.4–86)	0.000*
-Active smoker	60.5 (15–243)	0.000*
Age	1.04 (1.01–1.1)	0.001*
Serum cotinine (ng/mL)	1.003 (1.001–1.005	0.01*

Note: *Indicates statistically significant difference, *P*-value <0.05.

among smokers group in descending orders were active smoking, passive smoking, age and serum cotinine level. There was a statistically significant moderate positive correlations between the pack/year and, level of urinary albumin (r = 0.9 p<0.05) among studied participants (Figure 1).

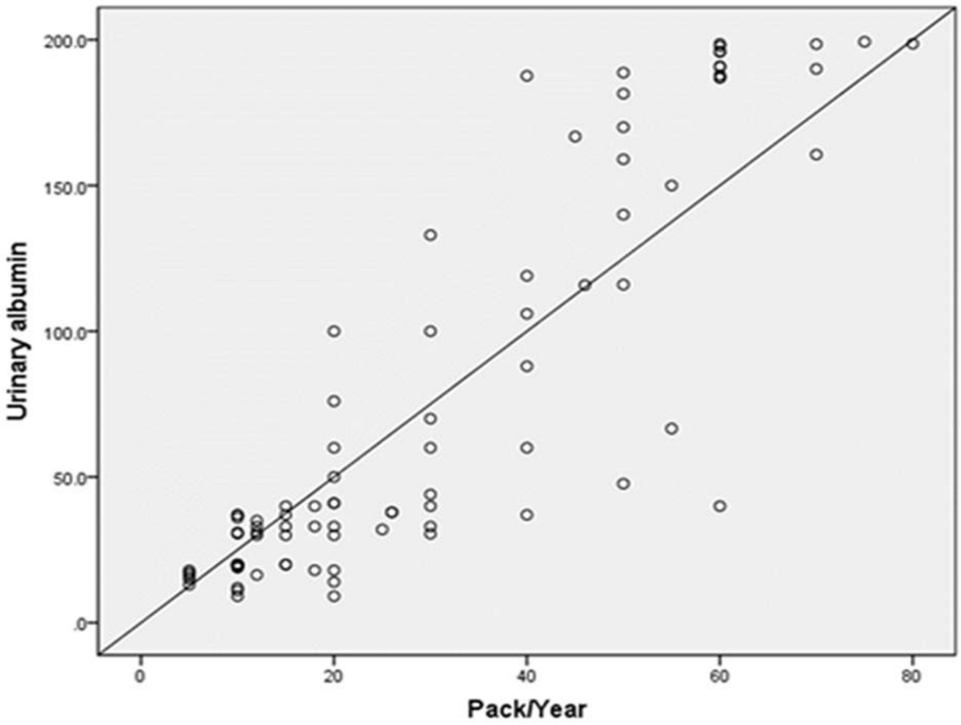

Figure 1 Correlation between urinary albumin (mg/day) and pack/year among active smokers. There was a statistically significant moderate positive correlations between the pack/year and, level of urinary albumin ($r = 0.9$ $p<0.05$) among studied participants.

Discussion

Smoking is a well known risk factor for many critical diseases, including respiratory, cardiovascular and nervous system diseases, and there is more studies showing that cigarette smoking can cause renal diseases and damage.[15] Smoking increases the renal diseases severity in patients with chronic diseases as diabetes, hypertension, polycystic kidney diseases, and post-kidney transplant disease.[16] Moreover, smoking can cause de novo renal disease and damage even in a healthy person without any history of chronic kidney disease (CKD).[17]

Studies that analyze the relationship between exposure to cigarette smoke and early kidney diseases and pathologies are still limited in the literature. Many previous studies have investigated the conditions of renal functions in existing diseases. However, this study was done on persons who had no known diseases with aiming to detect the early effects of active and passive smoking on kidney functions in terms of serum urea, serum creatinine, and microalbuminuria. Actually risk factors for renal disease and rising kidney functions are numerous. In the current study we excluded any patients with other risk factors for nephropathy as D.M, Hypertension, Auto immune diseases, metabolic diseases, Alcoholics, chronic medications and analgesics medications. In the current study both smoker groups and controls were matched regarding age, sex, CBC parameters, liver function, serum electrolyte and serum blood sugar (HBA1C and random blood sugar) (Table 1). Passive smoking can negatively affect renal morphology and glomerular filtration rate, with effects more or less similar to that described in the literature regarding active smoking.[18]

The main findings of the current study were that statistically significant increase of serum urea, creatinine, urinary albumin and serum cotinine in smoker group in comparison to nonsmoker group. Additionally, urinary albumin and serum cotinine were significantly higher in active smoker relative to passive smoker group, without significant difference in serum urea and serum creatinine between the two groups (Table 2).

Tascón et al's (2022) study confirmed the effect of smoking on urinary albumin/protein excretion in persons from the general population with known normal renal functions also concluded that the albuminuria excretion rate was in correlation with the number of daily cigarettes smoking (measured as cotinine excretion).[19]

Mimran et al (1994) reported that smoking was associated with excessive urinary albumin excretion in hypertensive patients. They reported that the prevelance of microalbuminuria was twofold in lean never treated hypertensive smokers than non-smokers.[17]

Similar findings were reported by Dülger et al (2011), as the levels of urine microalbumin in active smokers, increased relative to passive smokers and controls.[20] This difference was statistically significant as compared with the control group ($p < 0.01$). The urine microalbumin/creatinine ratio was significantly higher in both passive and active smokers relative to the control group ($p < 0.01$). Yacoub et al (2011) reported that, urinary albumin is well known to be a reliable indicator of glomerular affection, and the fact that smoking is linked to albuminuria indicates direct or indirect smoking-induced renal damage.[21] Peraza et al reported that, smoking is also responsible for the worsening of lupus nephritis renal function, in a retrospective study of 160 adults, smoking was found to be an independent risk factor for the faster development of CKD during nephritis.[22]

In addition, our study concluded that cigarette smoking was associated with an increased risk of albuminuria, dose dependently as Prevalence of urinary albuminuria was higher in active smokers (75.7%) compared to passive smokers (45.7%) and control group (2.1%). Among the active smokers group, we demonstrated that severity of albumin urea increase significantly in heavy smokers when compared with moderate and light smokes groups, and it was correlated significantly with serum cotinine levels, pack/year smoking index and smoking duration.

These observation were in agreement with the study done by Bleyer et al (2000), in which analyzing data obtained in 4142 nondiabetic subjects with ages more than 64 years who had two measurements of serum creatinine performed at least 3 years apart.[23] The number of cigarettes smoked was highly associated with an increase in serum creatinine >27 umol/L (>0.3mg\dl. Similar finding supported our current study were reported by García et al (2013) as eGFR was highly significant decrease in heavy smokers; relative to other groups; with highly substantial statistical difference ($p < 0.01$), which came in agreement with our results and with results reported by Obert et al (2011).[24,25]

Exposure to passive smoking has been reported to be associated with a higher risk of several types of malignancies and cardiovascular diseases. In addition, recent studies have shown that side-stream smoke, which is the main component of passive smoke, contains more toxic substances than those found in mainstream smoke, suggesting that passive smoking exposure can also cause serious effects.[26]

The current study reported high serum cotinine and significant high level of urinary micro-albumin in passive smokers compared to control group.

These findings suggest that the association of CKD with passive smoke exposure could be at least similar to active smoking.[9]

Lowering the risk of passive smoke exposure by improving public smoking restriction policies and educating the population about the potential harmful effects of passive smoking could reduce the risk of CKD development in non-smokers with normal kidney function.[27]

Limitations

Our study was cross-sectional study, and its participants were of a single ethnic origin, which might limit the generalization of our results. The present data do not allow for the drawing of a definite conclusion about the magnitude of the renal benefit derived from smoking cessation. Further studies including large numbers of participants with different ethnic groups are recommended. Further studies on the suggested beneficial effect of smoking cessation on renal functions are recommended.

Conclusion

Smoking is an important risk factor for nephropathy especially with active heavy smokers. It has a valuable negative impact on renal function even in persons without apparent renal disease. Both active and passive smoking, especially among heavy smokers, is a significant risk factor for microalbuminuria. Smoking cessation may be helpful in improving kidney functions.

Data Sharing Statement

The datasets generated and/or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request.

Disclosure

The authors declare that they have no competing interests.

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