



Lipid Profiles in Different ABO Blood Groups in Owerri Metropolis, South East Nigeria- A Preliminary Study

Samuel O. Ureme¹, Innocent C. Anioke^{1*} and C. Igboerika²

¹Department of Medical Laboratory Sciences, Faculty of Health Science and Technology, College of Medicine, University of Nigeria Enugu Campus, Nigeria.

²Department of Medical Laboratory Sciences, Imo State University, Owerri, Nigeria.

Authors' contributions

This work was carried out in collaboration between all authors. Author SOU designed the study protocol and performed the statistical analysis. Author ICA wrote the first draft of the manuscript. Author CI carried out the study analyses. Author ICA managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/AJRB/2018/v2i2424

Editor(s):

(1) Mohamed Fawzy Ramadan Hassanien, Professor, Biochemistry Department, Faculty of Agriculture, Zagazig University, Zagazig, Egypt.

Reviewers:

(1) Ismail Kucukkurt, Afyon Kocatepe University, Turkey.

(2) Toba Abass Anifowose, University of Ilorin, Nigeria.

Complete Peer review History: <http://www.sciencedomain.org/review-history/24404>

Original Research Article

Received 2nd January 2018
Accepted 7th March 2018
Published 1st May 2018

ABSTRACT

Objective: The study investigated the possibility of using blood groups as predictive indices for diseases associated with lipid metabolism.

Methods: Lipid profiles were examined in 100 apparently healthy male (40) and female (60) subjects of different ABO blood groups aged between 18-30 years from Imo State University. Of these, 20 were blood group A, 30 were B blood type, 4 were AB blood type, and 46 were blood group O. Lipid profile parameters were determined according to enzymatic assay using a commercial kit from Randox Laboratories, United Kingdom and calculation using Friedewald's equation. Monoclonal ABO blood grouping reagent by CLAS Technology, United Kingdom was used to determine the blood group.

Results: Total Cholesterol (140.62 ± 21.66 mg/dl) and High-Density Lipoprotein (HDL) (96.20 ± 40.32 mg/dl) were highest in blood group B. Blood group A had the highest level of Triglyceride (80.84 ± 18.60 mg/dl) and Very Low-Density Lipoprotein (VLDL) (15.21 ± 6.24 mg/dl). Blood group O showed TC level of 130.60 ± 34.76 mg/dl with the highest level of LDL (70.74 ± 20.15 mg/dl).

*Corresponding author: E-mail: innocent.anioke@unn.edu.ng;

mg/dl) and the lowest level of HDL (51.68 ± 20.50 mg/dl) compared to non- O blood types ($P < 0.05$).

Conclusion: The study revealed that blood group O might have a higher propensity for dyslipidemia, suggesting an increased risk for disease associated with lipid metabolism.

Keywords: ABO blood group; lipid metabolism; lipid profile; dyslipidemia.

1. INTRODUCTION

ABO blood group constitute ABH-antigens which are complex carbohydrate molecules (glycoprotein and glycolipids) expressed on the extracellular surfaces of human cells and tissues, including red cell membrane, platelets, and vascular endothelial cell [1-3]. ABO blood group has been recognized as an essential system in clinical practice particularly in the field of transfusion and transplantation medicine [4]. Since its discovery, ABO group has been studied as etiological factors of many diseases [5,6,7]. The activity of glycosyltransferases encoded by the inheritance of gene on chromosome 9(9q34) determines the individual ABO phenotype depending on the specific oligosaccharides residues transfer to H antigen (Fig. 1); however, O individuals lack such activity [8,9].

Studies on phenotypic distribution of ABO blood group have reported a considerable variation in different geographical areas, which reflects the underlying genetic and ethnic diversity of human populations [9]. However, several studies [11-17] including a recent study done in Nigeria [9] have not only reported O blood type as the most prevalent but also have shown the frequencies in the order of $O > A > B > AB$. In South East Nigeria particularly in Enugu, Abia

and Ebonyi states, the average phenotypic distribution of ABO blood group revealed the frequencies in the order 57.69% , 23.83% , 16.14% , 2.33% ($O > A > B > AB$) respectively [9]. The clinical implication of ABO blood group system may well extend beyond immuno-haematology [18]. Studies in the recent past have reported a critical involvement of ABO blood group system in the development of cardiovascular diseases (CVDs) [3,19-23] certain malignancies such as gastric cancer [24-26] and malaria [7,27-29]. Considerable evidence underpins CVDs to be linked with ABO blood group system [3,4,23,45] although the mechanism linking ABO blood group with CVD, particularly atherosclerosis, remains unclear. However, previous studies suggest that ABH antigen act as the principal agent for endothelial cell proliferation [3], as such plays a role in the disease process by a modulation in the vascular endothelial haemostasis [1,30]. Besides, the theory that glycosyltransferases (enzymes involved in the modification of the A, B and H antigens) regulate circulation of soluble products of cellular adhesion molecules (CAMs) - biomarkers of inflammation-related diseases - may explain the relationship [4,31]. Previous evidence has reported increase in the biomarkers to be associated with cardiovascular diseases (CVDs) [32-36]. For instance, it was quantitatively demonstrated that individuals

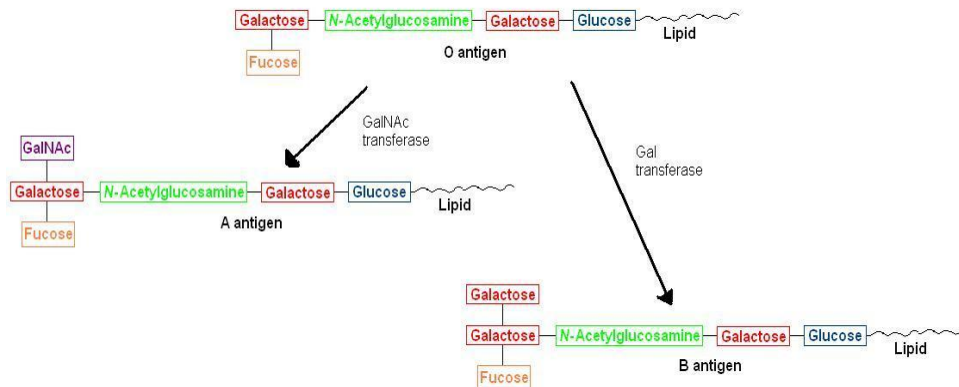


Fig. 1. Production of A, B, and H (O) antigens [10]

with A blood group had reduced level of some of these inflammation-related disease biomarkers compared to B or O blood groups [4]. A similar finding was reported in an individual with blood group A in another study, given a decreased cleavage of CAMs with A- antigen [37]. This may explain why individuals expressing the A-antigen have been reported to be less likely associated with CVD. In the contrary, documented evidence suggests a close relationship between non- O blood groups (A, B, AB) and CVD [23]. For instance, a study has shown that individuals with blood group A have a more frequent family history of CVD as opposed to other ABO blood groups [38]. Perhaps, due to an increased level of von Willebrand Factor (vWF) and factor VIII, which predispose non-O subjects to a higher risk of arterial and venous thrombo-embolism than individuals with O blood group [7,23]. Furthermore, the established evidence demonstrates a close association of ABO blood group with lipid metabolism [39] which may further explain the linkage between CVD and ABO blood group system. In the light of this, the study was set out to investigate the possibility of using ABO blood group system as predictive indices for disease associated with lipid metabolism by assessing the plasma lipid profile.

2. MATERIALS AND METHODS

This cross-sectional study utilized a convenience sampling procedure to recruit one hundred (100) students, comprising forty (40) males and sixty (60) females. Subjects recruited were students of Imo State University aged between 18 and 30 years. Ethical approval for the study was obtained from Department of Medical Laboratory Sciences on behalf of Students Affairs Department of Imo State University Owerri, Nigeria. Each study participants completed a well-structured questionnaire to help exclude participants with possible conditions and/or behavioural risk factors that may affect lipid metabolism. Among students recruited for the study, those who gave full consent were included whereas those with known cases of smoking, obesity, diabetes, hypertension and other related disorders or those on medication that could affect lipid metabolism were excluded from the study. Total cholesterol and triglycerides were measured by enzymatic colorimetric method [40,41], using a commercial kit by Randox laboratories, United Kingdom. HDL was measured enzymatically after all non HDL lipoproteins were removed. LDL-C was calculated using Friedewald's equation: $LDL =$

$\text{total cholesterol} - \{\text{HDL} \pm (\text{TG}/5)\}$. Blood grouping was done using monoclonal ABO blood grouping reagents by CLAS Technology, United Kingdom.

2.1 Data Analysis

Both the descriptive and inferential statistics was carried out using the SPSS version 22 .The results obtained were expressed in mean \pm standard deviation (SD). Analysis of variance (ANOVA) was used to calculate the difference in mean of each lipid profile fractions between different ABO blood groups. Within two blood groups comparison was determined using t-test. A statistically significant difference was set at $P < 0.05$.

3. RESULTS

Out of the a total of 100 students recruited for the study, 20% of the subjects were of blood group A, 30% were of blood group B, 4% were of blood group AB and 46% were of blood group O. Table 2 shows the blood groups of the subjects with their lipid profile levels. The mean result showed that total cholesterol(TC) (140.62 ± 21.66) and High-density cholesterol (HDL) (96.20 ± 40.32) were highest in blood group B while Triglyceride(TG) (80.84 ± 18.60) and very low-density lipoprotein (VLDL) (15.21 ± 6.24) was highest in group A. LDL level was lowest (29.71 ± 15.17) in AB blood group. Blood group O showed TC level of 130.60 ± 34.76 with highest level of LDL (70.74 ± 20.15) and lowest level of HDL (51.68 ± 20.50) compared to A, B ,and AB blood type respectively ($P < 0.05$).

4. DISCUSSION

The present study attempted the evaluation of the impact of ABO blood groups on the lipid profile of apparently healthy subjects. Subjects with O blood group have a higher level of LDL-C and lower HDL-C compared to non-O groups. This may suggests that subjects with O blood group may have higher propensity for diseases associated with lipid metabolism than non-O blood group types. However, this contradicts the study by Li et al. [39] which reported that non-O subjects (A, B, AB) had higher level of LDL-C compared with O subjects as such does not favour the report by previous researchers [3,22,23,42] who suggest that non-O blood type are at significantly greater risk for developing CVDs compared to O blood group. At this same time, the study does not underpin the report that only non-O subjects (A, B, AB) have a close

Table 1. Phenotypic distribution of ABO blood system of studied population

Blood group	A	B	AB	O
frequency	20%	30%	4%	46%

Table 2. Lipid profile in different ABO blood group systems among Students of Imo State University Owerri, Nigeria

	A	B	O	AB
TC (mg/dl)	129.89 \pm 34.83	140.62 \pm 21.66**	130.60 \pm 34.76	115.20 \pm 25.30
TG (mg/dl)	80.84 \pm 18.60	60.50 \pm 25.18	68.84 \pm 30.50	70.00 \pm 35.50
HDL (mg/dl)	74.61 \pm 37.62	96.20 \pm 40.32**	51.68 \pm 20.50*	55.41 \pm 18.20
LDL (mg/dl)	49.85 \pm 10.55	60.20 \pm 18.50	70.74 \pm 20.15**	29.71 \pm 15.17
VLDL (mg/dl)	15.21 \pm 6.24	12.64 \pm 5.21	14.43 \pm 4.57	13.50 \pm 4.10

(*) statistically significantly low, $p < 0.05$ across blood group types

(**) statistically significantly high, $p < 0.05$ across blood group types

Within two blood groups comparison:

- TG: A vs B; $p < 0.05$, B vs O; $P > 0.05$, O vs A; $p > 0.05$
- HDL: A vs B, B vs O, O vs A; $p < 0.05$
- LDL: A vs B, B vs O, O vs A; $p < 0.05$
- VLDL: A vs B, B vs O, O vs A; $p < 0.05$
- TC: A vs B, B vs O, O vs A; $p < 0.05$

relation with CVD as suggested by another researcher [7,23]. However, the current study seems to underpin the previous report by Anvari et al. [43] who demonstrated that the prevalence of coronary heart disease in blood group O is markedly higher than in all other ABO blood groups. Perhaps due to an elevated level of LDL-C in O blood type as shown in the present study. The mechanism underlining such blood-type differences in association with lipid profile levels is still unclear. However, it may be attributed to a distinct ABO genetic association pattern with plasma lipids [44]. The fact that transfer of a specific oligosaccharide residue to H antigen by glycosyl transferases does not occur in group O, leaving blood type O individuals with unconverted H-substance [8] may be a key role. Moreover, given that ABO glycotransferase is associated with cholesterol metabolism [39] and may have a broader impact on atherosclerotic-CVD [45] CVDs may not only be conferred on non-O blood types as suggested by previous studies. The fact that blood group B had highest TC and HDL while O has highest LDL indicates that both blood groups B and O might be predisposed to diseases associated with lipid disorders; however the consequence of increased level of TC in blood group B also could be ameliorated by elevated level of HDL present.

However, the findings should be considered in the context of the study limitations. First, selection bias may be likely; given that the sample was not randomly selected as such may

not be a representative sample for the population. Secondly, as a single centre study (among students) with relatively small size and convenience sampling technique, generalization of result is limited and should be undertaken with caution. Findings are only association, not causation as this study was a cross-sectional survey which cannot ascertain causal link between ABO blood group and dyslipidemia. The use of self-administered questionnaire as a method of excluding those with possible conditions/behavioral risk factors likely to impact lipid metabolism may be associate with a number of bias such as recall bias and social desirability bias [46] given that most participants may fail to fully remember all cases or decide to conceal some for fear of being stigmatized among their fellow students. Therefore, further study with large sample size, perhaps population-based survey across all age, may be necessary to validate the level of significant shown in this study with small sample size. However, taken together, the study had made important contribution in that, to the researchers' best knowledge, this is among the first study that shows the association of lipid profile parameters among ABO blood group system in adult Nigerian.

5. CONCLUSION

The study, therefore, demonstrates that among different ABO blood group studied, subjects with blood group O is associated with reduced level of

HDL-C and elevated level of LDL-C as opposed to non-O blood types. This suggests that individuals with blood group O may be most at risk for developing disease associated with lipid metabolism. However, given that ABO gene is located on chromosome 9(9q34) and encodes A and B alleles for specific glycosyltransferases of which O variant does not encode a functional one, further studies are needed on a large sample sizes to elucidate its mechanism.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCE

1. Zhou S, Welsby I. Is ABO blood group truly a risk factor for thrombosis and adverse outcomes? *World Journal of Cardiology*. 2014;6(9):985-992.
2. Karabuvu S, Carevic V, Radic M, Fabijaniic D. The association of ABO blood group with the extent of coronary atherosclerosis in Croatian patients suffering from chronic coronary disease. *Biochem Med Zagreb*. 2013;23(3):351-359.
3. Nafakhi H, Al-Nafakh H, Al-Mosawi A. ABO blood group differences relationship with coronary atherosclerotic markers. *Artery Research*. 2016;14:36-40.
4. Chen Y, Zhuo X, Lin Y, Huang W, Xiao J, Zeng J, Jiang L, Chen C, Lin H, Dettke M. Association of ABO blood group with P-selectin levels in Chinese Han healthy volunteers. *Transfusion*. 2015;55:2759-2765.
5. Garraty G, Dzik W, Issitt P, Lubin D. Terminology for blood group antigens and genes-historical origins and guideline in the new millennium. *Transfusion*. 2000;40:477-489.
6. Bloomfield P, Bradbury A, Grubb N, Newby D. Diseases of cardiovascular system. In: Boon N, Colledge N, Walker B. (eds). *Davidsons principles and practice of medicine*. 20th edition. Elsevier Edinburg: Churchill Livingstone. 2009;519-648.
7. Anstee D. The relationship between Blood groups and disease. *Blood*. 2010;115(23):4635-4643.
8. Yamamoto F, Clausen H, White T, Marken J, Hakomori S. Molecular genetic basis of the histoblood group ABO system. *Nature*. 1990;345(6272):229-33.
9. Anifowoshe AT, Owolodun OA, Akinseye KM, Iyiola OA, Oyeyemi BF. Gene frequencies of ABO and Rh blood groups in Nigeria: A review. *The Egyptian Journal of Medical Human Genetics*. 2017;18:205-210.
10. Nelson David L. *Principles of Biochemistry*. 4th ed. W. H. Freeman; 2004.
11. Garratty G, Glynn SA, McEntire R. ABO and Rh (D) phenotype frequencies of different racial/ethnic groups in the united states. *Transfusion*. 2004;44(5):703-6.
12. Hamed CT, Bollahi MA, Abdelhamid I, Mahmoud M, Ba B, Ghaber S, et al. Frequencies and ethnic distribution of ABO and Rh (D) blood groups in Mauritania: Results of first nationwide study. *Int J Immunogenet*. 2012;39(2):151-4.
13. Torabizade maatoghi J, Paridar M, Mahmodian Shoushtari M, Kiani B, Nori B, Shahjahani M, et al. Distribution of ABO blood groups and rhesus factor in a Large Scale Study of different cities and ethnicities in Khuzestan province, Iran. *Egypt J Med Hum Genet*. 2016;17(1):105-9.
14. Tesfaye K, Petros Y, Andargie M. Frequency distribution of ABO and Rh (D) blood group alleles in Silte Zone, Ethiopia. *Egypt J Med Hum Genet*. 2015;16(1):71-6.
15. Ndoula ST, Noubiap JJN, Nansseu JRN, Wonkam A. Phenotypic and allelic distribution of the ABO and Rhesus (D) blood groups in the Cameroon population. *Int J Immunogenet*. 2014;41(3):206-10.
16. Benahadi A, Alami R, Boulahdid S, Adouani B, Laouina A, Mokhtari A, et al. Distribution of ABO and rhesus D blood antigens in Morocco. *Int J Biol Anthropol*. 2013;6(1).
17. Said N, BenAhmed F, Doghri A, Ghazouani E, Layouni S, Gritli N, et al. The ABO system polymorphism in Tunisian blood. *Transfus Clin Biol*. 2003;10:331.
18. Liunbruno G, Franchini M. Beyond immunohaematology: The role of the ABO blood group in human disease. *Blood Transfusion*. 2013;11:491-499.
19. Dentali F, Sironi A, Ageno W. Non-O blood type is commonest genetic risk factor for VTE: Results from a meta-analysis of the literature. *Seminars in Thrombosis Hemostasis*. 2012;38:535-548.
20. Franchini M, Favaloro E, Targher G, Lippi G. ABO blood group, hypercoagulability,

- and cardiovascular and cancer risk. *Critical Revision Clinical Laboratory Science*. 2012;49:137-149.
21. Franchini M, Makris M. Non-O blood group: Important genetic risk factor for venous thromboembolism. *Blood Transfusion*. 2013;11:164-165.
 22. Spiezia L, Campello E, Bon M. ABO blood groups and the risk of venous thrombosis in patients with inherited thrombophilia. *Blood Transfusion*. 2013;11:250–253.
 23. Takagi H, Umemoto T. Meta-analysis of non-o blood group as an independent risk factor for coronary artery disease. *American Journal of Cardiology*. 2015;116: 699-704.
 24. Edgren G, Hjalgrim H, Rostgaard K. Risk of gastric cancer and peptic ulcer in relation to ABO blood type: A cohort study. *American Journal of Epidemiology*. 2010; 172:1280-1285.
 25. Wang Z, Liu L, Ji J. ABO blood group system and gastric cancer: A case-control study and meta-analysis. *International Journal of Molecular Sciences*. 2012;13: 13308-13321.
 26. Rizzato C, Kato I, Plummer M. Risk of advanced gastric precancerous lesion in helicobacter pylori infected subjects is influenced by ABO blood group and caga status. *International Journal of Cancer*. 2013;133:315-322.
 27. Cserti-Gazdewich C, Mayr W, Dzik W. Plasmodium falciparum malaria and the immunogenetics of ABO, HLA, and CD36 (platelet glycoprotein IV). *Vox Sanguinis*. 2011;100:99-111.
 28. Panda A, Panda S, Sahu A. Association of ABO blood group with severe falciparum malaria in adults: Case-control study and meta-analysis. *Malaria Journal*. 2011;10: 309.
 29. Timmann C, Thye T, Vens M. Genome-wide association study indicates two novel resistance loci for severe malaria. *Nature*. 2012;489:443-446.
 30. Lee H, Lin Y, Lin C, Wang C, Chang C, Hsu L. Association of blood group A with coronary artery disease in young adult in Taiwan. *Internal Medicine*. 2012;51:1815-1820.
 31. Larson N, Bell E, Decker P, Pike M, Wassel C, Tsai M, Pankow J, Tang W, Hanson N, Alexander K, Zakai N, Cushman M, Bielinski S. ABO blood group associations with markers of endothelial dysfunction in the multi-ethnic study of atherosclerosis. *Atherosclerosis*. 2016; 251:422-429.
 32. Barbaux S, Blankenberg S, Rupprecht H, Francomme C, Bickel C, Hafner G, Nicaud V, Meyer J, Cambien F, Tiret L. Association between pselectin gene polymorphisms and soluble p-selectin levels and their relation to coronary artery disease, Arteriosclerosis, Thrombosis and Vascular Biology. 2001;21:1668-1673.
 33. Ridker P, Hennekens C, Roitman-Johnson B, Stampfer M, Allen J. Plasma concentration of soluble intercellular adhesion molecule 1 and risks of future myocardial infarction in apparently healthy men. *Lancet*. 1998;351:88-92.
 34. Hwang S, Ballantyne C, Sharrett A, Smith L, Davis C, Gotto Jr. A, Boerwinkle E. Circulating adhesion molecules vcam-1, icam-1, and e-selectin in carotid atherosclerosis and incident coronary heart disease cases: The atherosclerosis risk in communities (aric) study. *Circulation*. 1997;96:4219-4225.
 35. Ridker P. Intercellular adhesion molecule (icam-1) and the risks of developing atherosclerotic disease. *European Heart Journal*. 1998;19:1119-1121.
 36. Polgar J, Matuskova J, Wagner D. The p-selectin, tissue factor, coagulation triad. *Journal of Thrombosis and Haemostasis*. 2005;3:1590-1596.
 37. Barbalic M, Dupuis J, Dehghan A, Bis J, Hoogeveen R, Schnabel R, Nambi V, Bretler M, Smith N, Peters A, Lu C, Tracy R, Aleksic N, Heeriga J, Keaney Jr. J, Rice K, Lip G, Vasani R, Glazer N, Larson M, Uitterlinden A, Yamamoto J, Durda P, Haritunians T, Psaty B, Boerwinkle E, Hofman A, Koenig W, Jenny N, Witteman J, Ballantyne C, Benjamin E. Large-scale genomic studies reveal central role of ABO in sselectin and sicam-1 levels. *Human Molecular Genetic*. 2010;19:1863-1872.
 38. Abdollahi A, Qorbani M, Asayesh H, Nouroozi M, Mansourian M. Association between ABO blood groups and cardiovascular risk factors in general population of Golestan province, Iran. *Scientific Journal of Iran Blood Transfusion Organisation*. 2012;8(4):293-297.
 39. Li S, Xu R, Guo Y, Zhang Y, Zhu C, Sun J, Li J. ABO blood group in relation to plasma lipids and proprotein convertase subtilisin/kexin type 9. *Nutrition, Metabolism & Cardiovascular Diseases*, 2014;25:411-417.

40. Trinder P. Annals of Clinical Biochemistry. 1969;6:24-27.
41. Fossati P, Prencipe L. Serum triglycerides determined colorimetrically with an enzyme that produces hydrogen peroxide. Clinical Chemistry. 1982;28(10):2077–2080.
42. Petrochko C. Type O blood carries lower CHD risk-patients with type A, B, or AB blood are at significantly greater risk for coronary heart disease than those with type O blood, researchers found. Cardiology [Online]; 2012. Available:<<http://www.medpagetoday.com/cardiology/myocardialinfarction/34205>> [Accessed 20 December 2016]
43. Anvari M, Boroumand M, Emami B, Karimi A, Soleymanzadeh M, Abbasi S, Saadat S. ABO blood group and coronary artery diseases in Iranian patients awaiting coronary artery bypass graft surgery: A review of 10,641 cases. Labmedicine. 2009;40(9):528-530.
44. Carpeggiani C, Coceani M, Landi P. ABO blood group alleles: A risk factor for coronary artery disease. An angiographic study. Atherosclerosis. 2010;211:461-466.
45. Zhang H, Mooney CJ, Reilly MP. ABO blood groups and cardiovascular diseases. International Journal of Vascular Medicine. 2012;11. Article ID 641917. DOI: 10.1155/2012/641917
46. Bowling A. Research methods in health. 3rd ed. New York: Open University Press; 2009.

© 2018 Ureme et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
<http://www.sciencedomain.org/review-history/24404>