

Corrigendum

Corrigendum to "Association of Nuclear Factor-Erythroid 2-Related Factor 2, Thioredoxin Interacting Protein, and Heme Oxygenase-1 Gene Polymorphisms with Diabetes and Obesity in Mexican Patients"

Angélica Saraí Jiménez-Osorio,¹ Susana González-Reyes,¹ Wylly Ramsés García-Niño,¹ Hortensia Moreno-Macías,² Martha Eunice Rodríguez-Arellano,³ Gilberto Vargas-Alarcón,⁴ Joaquín Zúñiga,⁵ Rodrigo Barquera,⁶ José Pedraza-Chaverri,¹ Juan Pablo Meza-Espinoza,⁷ Evelia Leal-Ugarte,⁷ and Valeria Peralta-Leal⁷

¹Department of Biology, National Autonomous University of Mexico (UNAM), 04510 Mexico City, DF, Mexico

²Economy Department, Autonomous Metropolitan University-Iztapalapa, 09340 Mexico City, DF, Mexico

³Research Department, Regional Hospital "Lic. Adolfo López Mateos", ISSSTE, 01030 Mexico City, DF, Mexico

⁴Molecular Biology Department, National Institute of Cardiology "Ignacio Chávez", 14080 Mexico City, DF, Mexico

⁵Department of Immunology, National Institute of Respiratory Diseases "Ismael Cosío Villegas", 14080 Mexico City, DF, Mexico

⁶Molecular Genetics Laboratory, National School of Anthropology and History, 14030 Mexico City, DF, Mexico

⁷Departamento de Genética, Facultad de Medicina e Ingeniería en Sistemas Computacionales de Matamoros,

Universidad Autónoma de Tamaulipas, Sendero Nacional km 3, 87349 Matamoros, TAMPS, Mexico

Correspondence should be addressed to José Pedraza-Chaverri; pedraza@unam.mx

Received 20 February 2017; Accepted 28 February 2017; Published 30 May 2017

Copyright © 2017 Angélica Saraí Jiménez-Osorio et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

In the article titled "Association of Nuclear Factor-Erythroid 2-Related Factor 2, Thioredoxin Interacting Protein, and Heme Oxygenase-1 Gene Polymorphisms with Diabetes and Obesity in Mexican Patients," [1] typing errors were found after subsequent analyses of the entire dataset. These issues were raised to the attention of the original authors by Drs. Meza-Espinoza, Leal-Ugarte, and Peralta-Leal of the Universidad Autónoma de Tamaulipas. The allelic frequencies were added and the corrected data are shown in italics in Tables 2, 3, and 4.

It is important to remark that the model (recessive for allele T, Table 4) was calculated taking in count that CC + CT = 0. When CC is considered as the risk allele the OR increases to 2.4 (CI: 1.28–4.64, P = 0.006) under a dominant model (CC + CT = 1 versus TT = 0). This reveals that the

TT genotype in this study shows a protective factor against obesity and the genotype CC could be considered as the risk factor for obesity, as was stated in the original article.

The last paragraph of Section 3 should be "CC carriers had higher glucose levels in comparison with CA + AA carriers when genotype was compared as dominant model."

Finally, in Section 4 (Discussion) when Wang et al. [7] is cited, the correct statement is the following:

"Individuals with CC genotype had lower total antioxidant capacity, glutathione levels, superoxide dismutase, catalase, and glutathione peroxidase activities as well as lower homeostasis model assessment of β -cell function index (HOMA- β) in comparison with individuals with the AA genotype."

Gene/polymorphism	Genotypes	Diabetes	Controls	OR (95% CI)	Р	<i>P</i> of HWE
	Alleles	n (%)	n (%)			
	CC	345 (55.4)	528 (54.5)	Reference		
	СТ	239 (38.4)	376 (38.8)	0.97 (0.78–1.2)	0.798	
TXNIP rs7211	TT	39 (6.2)	65 (6.7)	0.92 (0.60–1.39)	0.690	0.880
	С	939 (74.6)	1432 (73.9)	Reference		
	Т	317 (25.4)	506 (26.1)	0.966 (1.82–1.37)	0.674	
NQO1 rs1800566	CC	216 (34.7)	327 (33)	Reference		
	CT	288 (46.2)	483 (48.6)	0.90 (0.7-1.1)	0.373	
	TT	119 (19.1)	183 (18.4)	0.98 (0.7–1.3)	0.915	0.406
	С	720 (57.8)	1137 (57.25)	Reference		
	Т	526 (42.2)	849 (42.75)	0.98 (0.85–1.13)	0.765	
HMOX-1 rs2071749	AA	269 (43.8)	413 (43.2)	Reference		
	AG	267 (43.5)	417 (43.6)	0.98 (0.79–1.2)	0.877	
	GG	78 (12.7)	126 (13.2)	0.95 (0.69–1.3)	0.757	0.625
	Α	805 (65.5)	1243 (65)	Reference		
	G	423 (34.5)	669 (35)	0.97 (0.84–1.13)	0.755	
NRF2 rs2364723	CC	210 (33.6)	301 (30.3)	Reference		
	CG	286 (45.7)	471 (47.5)	0.87 (0.7–1.1)	0.236	
	GG	129 (20.6)	220 (22.2)	0.84 (0.63–1.1)	0.223	0.092
	С	706 (56.5)	1073 (54)	Reference		
	G	544 (43.5)	911 (46)	0.91 (0.79–1.05)	0.182	
NRF2 rs6721961	CC	407 (65.3)	618 (62.5)	Reference		
	CA	189 (30.4)	317 (32)	0.90 (0.7–1.1)	0.374	
	AA	27 (4.3)	54 (5.5)	0.76 (0.5–1.2)	0.259	0.281
	С	1003 (80.5)	1553 (78.5)	Reference		
	A	243 (19.5)	425 (21.5)	0.88 (0.74–1.05)	0.176	

TABLE 2: Genotype and allele frequencies of the polymorphisms studied in diabetic patients and controls.

CI, confidence interval; HWE, Hardy-Weinberg equilibrium; HMOX-1, heme oxygenase 1; NQO1, NAD(P)H quinone oxidoreductase 1; NRF2, nuclear factorerythroid 2- (NF-E2-) related factor 2; OR, odds ratio; and TXNIP, thioredoxin-interacting protein.

Gene/polymorphism	Genotype	Obesity	No obesity	OR (95% CI)	Р
	Alleles	n (%)	n (%)		
	CC	350 (56.6)	523 (53.7)	Reference	
TRXNIP rs7211	СТ	239 (38.7)	376 (38.6)	0.95 (0.77–1.17)	0.633
	ТТ	29 (4.7)	75 (7.7)	0.57 (0.37–0.9)	0.017
	С	239 (76)	1422 (73)	Reference	
	T	297 (24)	526 (27)	0.85 (0.72–1)	0.061
	CC	212 (34.2)	331 (33.3)	Reference	
NQO1	CT	302 (48.8)	469 (47)	1 (0.8–1.25)	0.963
	TT	106 (17)	196 (19.7)	0.84 (0.6-1.1)	0.257
rs1800566	С	726 (58.5)	1131 (56.8)	Reference	
	Т	514 (41.5)	561 (43.2)	0.93 (0.8–1.07)	0.322
	AA	261 (40)	419 (45.7)	Reference	
	AG	305 (46.9)	378 (41.3)	1.3 (1–1.6)	0.019
HMOX-1	GG	85 (13.1)	119 (13)	1.1 (0.8–1.5)	0.399
rs2071749	G	827 (63.5)	1216 (66.4)	Reference	
	Α	475 (36.5)	616 (36.6)	1.13 (0.97–1.31)	0.097
	CC	194 (31)	317 (32)	Reference	
	CG	300 (48)	457 (46)	1.1 (0.85–1.35)	0.551
NRF2 rs2364723	GG	131 (21)	218 (22)	0.98 (0.7-1.3)	0.899
	С	688 (55)	1091 (55)	Reference	
	G	562 (45)	893 (45)	0.99 (0.86–1.15)	0.699
	CC	390 (63.1)	635 (63.9)	Reference	
	CA	195 (31.6)	311 (31.3)	1 (0.8–1.3)	0.853
NRF2 rs6721961	AA	33 (5.3)	48 (4.8)	1.1 (0.7–1.7)	0.631
	С	975 (78.9)	1581 (79.5)	Reference	
	Α	261 (21.1)	407 (20.5)	1.04 (0.87–1.23)	0.661

TABLE 3: Genotype and allele frequencies of the polymorphisms studied in obese and nonobese subjects.

TXNIP, thioredoxin-interacting protein; NQO1, NAD(P)H quinone oxidoreductase 1; HMOX-1, heme oxygenase 1; NRF2, nuclear factor-erythroid 2- (NF-E2-) related factor 2.

TABLE 4: Genotype frequency of the rs7211 polymorphism in subjects without diabetes and women.

	Obese	Nonobese	Crude OR (95% CI)	Р	Adjusted ^a OR (95% CI)	Р
Nondiabetic						
CC	189 (56.6)	339 (53.4)	Reference		Reference	
СТ	133 (39.8)	243 (38.3)	0.98 (0.75–1.3)	0.896	1 (0.77–1.4)	0.863
ТТ	12 (3.6)	53 (8.3)	0.4 (0.21–0.77)	0.007	0.3 (0.15-0.7)	0.003
CC + CT = 0 versus $TT = 1$	322 (96.4)	582 (91.5)	0.4 (0.21-0.76)	0.006	0.39 (0.18–0.8)	0.014
Women						
CC	197 (59)	259 (51)	Reference		Reference	
СТ	118 (36)	203 (40)	0.7 (0.6-1)	0.072	0.9 (0.6-1.2)	0.418
ТТ	17 (5)	47 (9)	0.5 (0.26-0.85)	0.013	0.5 (0.25-0.96)	0.04
CT + TT	135 (41)	250 (49)	0.70 (0.5-0.9)	0.016	0.7 (0.5-0.96)	0.028

CI, confidence interval; OR, odds ratio.

^aObesity in logistic regression was adjusted by age, gender (except in women model), glucose, triglycerides, LDL-C, and HDL-C levels.

References

 A. S. Jiménez-Osorio, S. González-Reyes, W. R. García-Niño et al., "Association of nuclear factor-erythroid 2-related factor 2, thioredoxin interacting protein, and heme oxygenase-1 gene polymorphisms with diabetes and obesity in Mexican patients," *Oxidative Medicine and Cellular Longevity*, vol. 2016, Article ID 7367641, 8 pages, 2016.





The Scientific World Journal



Research and Practice









Computational and Mathematical Methods in Medicine

Behavioural Neurology



Research and Treatment



Oxidative Medicine and Cellular Longevity