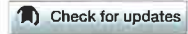


*C'est la population à risque qui doit être visée... et pas "tout le monde"...*

COMMENT OPEN



Epidemiology and Population Health

# Correlation between body mass index and COVID-19 transmission risk

Daniela de la Rosa-Zamboni<sup>1</sup>, Fernando Ortega-Riosvelasco<sup>2</sup>, Nadia González-García<sup>3</sup>, Sergio Saldívar-Salazar<sup>2</sup> and Ana Carmen Guerrero-Díaz<sup>2</sup>

© The Author(s) 2022

International Journal of Obesity; <https://doi.org/10.1038/s41366-022-01215-y>

We write in response to the article by Aghili et al. [1] "Obesity in COVID-19 era, implications for mechanisms, comorbidities, and prognosis: a review and meta-analysis". Although plenty has been written about the increased risk of obesity for COVID-19 morbidity and mortality [2–4], this paper is one of the few that addresses obesity as a risk of COVID-19 contagion.

As part of an ongoing COVID-19 contact tracing study among hospital workers in our institution, we have individually traced all contacts of 218 COVID-19 cases to determine the most likely source of infection. We found that obesity (Body Mass Index [BMI] > 30 kg/m<sup>2</sup>) was associated with spread of the infection to 2 or more coworkers: 3.47% (7 of 202) of workers who did not exhibit obesity infected 2 or more coworkers, while 25% (4 of 16) of workers with obesity infected 2 or more coworkers. A positive association was found between obesity and the spread of infection (OR 9.29, CI<sub>95%</sub> 2.38–36.17, *p* = 0.001). Once the risk was adjusted to confounders such as age, gender, comorbidities, and symptoms the risk was even higher (AOR 10.89, CI<sub>95%</sub> 2.67–44.33, *p* = 0.001). The duration of workers' symptoms in the moment of measuring was similar in all study groups.

In addition, a stepwise binomial logistic regression was calculated to determine the risk of BMI for infecting 0–1 coworker (low spreaders) against the risk of infecting ≥2 people (high spreaders); results are displayed in Table 1. Figure 1 shows the probability (odds/1 + odds) of falling into the "high spreading" category per each unit of BMI in the study subjects:

The addition of other variables, such as age, gender, and BMI-years, as was described by Edwards et al. [5] did not improve the predictive power of the model. This may obey to small age

differences in our group, composed mainly of young to middle age hospital workers.

These findings indicate that the increased BMI and obesity convey an increased risk of infection for their contacts, although confirmation of this will certainly require additional studies. It is known that patients with obesity and influenza shed the virus for a significantly longer period of time than people who are lean [6], and that obesity creates a state of chronic inflammation which impairs the immune response and favors the emergence of new, more virulent influenza strains [7, 8]. We agree with Aghili et al. [1] that relations between influenza and obesity can certainly be extrapolated to the current COVID-19 pandemic [9], which undoubtedly embodies a worrisome synergy with the concurrent obesity pandemic [10].

Probability of high spreading

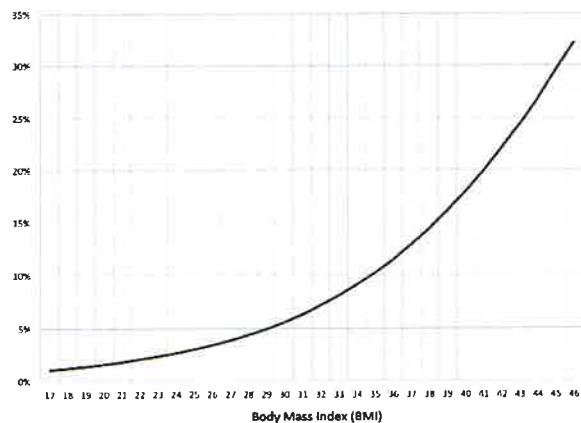


Fig. 1 Probability of high spreading. Probability of falling into the "High Spreading" category per unit of BMI.

Table 1. BMI as a predictive factor for low vs. high spreading.

	B	S.E.	Wald	df	Sig.	Exp(B)
BMI	0.13	0.056	5.486	1	0.019	1.139
Constant	-6.741	1.737	15.061	1	0	0.001

BMI body mass index, S.E. standard error, df degrees of freedom, Sig significance.

<sup>1</sup>Comprehensive Patient Care Department, Hospital Infantil de México Federico Gómez, Mexico City, Mexico. <sup>2</sup>Epidemiology Department, Hospital Infantil de México Federico Gómez, Mexico City, Mexico. <sup>3</sup>Research Department, Hospital Infantil de México Federico Gómez, Mexico City, Mexico. <sup>✉</sup>email: rzdaniela@hotmail.com

Received: 7 April 2022 Revised: 5 August 2022 Accepted: 10 August 2022  
Published online: 24 August 2022

**DATA AVAILABILITY**

Data are available upon request from the corresponding author.

**REFERENCES**

1. Aghili SMM, Ebrahimpur M, Arjmand B, Shadman Z, Pejman Sani M, Qorbani M, et al. Obesity in COVID-19 era, implications for mechanisms, comorbidities, and prognosis: a review and meta-analysis. *Int J Obes.* 2021;45:998–1016.
2. Lighter J, Philips M, Hochman S, Sterling S, Johnson D, Francois F, et al. Obesity in patients younger than 60 years is a risk factor for COVID-19 hospital admission. *Clin Infect Dis.* 2020;71:896–7.
3. Popkin BM, Du S, Green WD, Beck MA, Algaith T, Herbst CH, et al. Individuals with obesity and COVID-19: a global perspective on the epidemiology and biological relationships. *Obes Rev.* 2020;21:e13128.
4. Bello-Chavolla OY, Bahena-López JP, Antonio-Villa NE, Vargas-Vázquez A, González-Díaz A, Márquez-Salinas A, et al. Predicting mortality due to SARS-CoV-2: a mechanistic score relating obesity and diabetes to COVID-19 outcomes in Mexico. *The J of Clin Endocrinol Metab.* 2020;105:2752–61.
5. Edwards DA, Ausiello D, Salzman J, Devlin T, Langer R, Beddingfield BJ, et al. Exhaled aerosol increases with COVID-19 infection, age, and obesity. *Proc Natl Acad Sci USA.* 2021;118:e2021830118.
6. Maier HE, Lopez R, Sanchez N, Ng S, Gresh L, Ojeda S, et al. Obesity increases the duration of influenza A virus shedding in adults. *J Infect Dis.* 2018;218:1378–82.
7. Honce R, Schultz-Cherry S. Impact of obesity on influenza A virus pathogenesis, immune response, and evolution. *Front Immunol.* 2019;10:1071.
8. Honce R, Karlsson EA, Wohlgemuth N, Estrada LD, Meliopoulos VA, Yao J, et al. Obesity-related microenvironment promotes emergence of virulent influenza virus strains. *mBio.* 2020;11:e03341–19.
9. Luzi L, Radaelli MG. Influenza and obesity: its odd relationship and the lessons for COVID-19 pandemic. *Acta Diabetol.* 2020;57:759–64.
10. Zakka K, Chidambaram S, Mansour S, Mahawar K, Salminen P, Almino R, et al. SARS-CoV-2 and obesity: “CoVesity”—a pandemic within a pandemic. *Obes Surg.* 2021;31:1745–54.

**AUTHOR CONTRIBUTIONS**

D de la R-Z Planning, statistical analysis, and manuscript. FO-R Collection and classification of data, creation of the database and analysis of contacts. NG-G

Collection and classification of data, creation of the database, and analysis of contacts. SS-S Search of references, statistical analysis, and writing of the manuscript. ACG-D Search of references and writing of the manuscript.

**COMPETING INTERESTS**

The authors declare no competing interests.

**ADDITIONAL INFORMATION**

**Correspondence** and requests for materials should be addressed to Danieladela Rosa-Zamboni.

**Reprints and permission information** is available at <http://www.nature.com/reprints>

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.



**Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons license, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons license and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this license, visit <http://creativecommons.org/licenses/by/4.0/>.

© The Author(s) 2022