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### Commentary

## Understanding the link between COVID-19, blood pressure and obesity: Perspectives from the New Orleans experience

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In New Orleans, Louisiana (NOLA), the population's very high social vulnerability led to the establishment of an early epicenter for severe acute COVID-19. To assess possible explanatory factors, we conducted a longitudinal observational cohort study (ClinSeqSer) of 89 patients hospitalized with COVID-19 in New Orleans during March–August 2020 [1]. Over that time a single strain of SARS CoV-2 circulated in LA through a superspreader event during the Mardi Gras festivities at the end of February 2020 [2]. The cohort's diverse population, ~70 % Black, 53 % female and 55 % obese, is representative of greater urban NOLA. Cohort pre-COVID prevalence of hypertension (HTN) is 83 %, much higher than state's prevalence (43 % among White, 56 % among Black residents) and that of an acute COVID cohort recruited in the Ochsner Health system (uptown NOLA) at the same period (24 % in White, 34 % in Black patients), but matches the 73 % prevalence reported among Black community churches in urban LA [3]. The very high proportion of pre-COVID HTN in this acute COVID cohort correlates with high social vulnerability, with 30 % enrollment in Medicaid vs. 10 % in the uptown Ochsner system. In our cohort, Black patients are younger than White (~50 % vs ≤30 % in 45–64 years age bracket). Outcomes were 47 % severe, including 17 % fatal, and 30 % non-fatal (required high flow oxygen supplement or intubation), and identical by race and age. Obesity, BMI, admit systolic blood pressure (SBP), pulse

BP, and C-reactive protein level correlated with fatal and severe outcomes. Patients with admission SBP ≥140 mmHg had a 2.25-fold increased risk factor of severe outcome; 80 % reached severe clinical state within 5 days of admission vs. 50 % with admit SBP < 140 mmHg by Kaplan-Meier curve ( $p = 0.0093$  by log-rank analysis). The correlation between high admit SBP with severe outcomes may be the result of the cumulative effects of hypertension and subtle changes in myocardial function associated with SARS CoV-2 infection, as suggested by Park et al. [4]. These authors find that abnormal left ventricular global longitudinal strain predicted worse outcomes in patients hospitalized with COVID, and suggest mechanisms of myocardial damage, either directly caused by virus or as a consequence of antiviral cytokines released by the host. In COVID the "degradation of lung function that could associate with rise in BP" was suggested by Vicenzi et al. [5]. A pulse blood pressure over 60 mmHg is a risk factor for heart disease, and correlates with stiffness of the body's largest arteries in older adults [6]. Further studies and hypotheses are needed to investigate why SBP is associated with worse COVID outcomes and may include: 1) pre-COVID comorbidities, such as poorly controlled HTN, CKD, obesity, polypharmacy, advanced age, or, 2) a specific effect of SARS CoV-2 on the renin angiotensin axis, [7,8], or unique strain of SARS CoV-2 interaction with specific variants of the ACE2 receptor or, 3) specific polymorphisms,

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frequently encountered in LA populations, of sodium channel (Liddle phenotype) or 4) hypersensitivity to bradykinin (ACEi induced angioedema) [9,10]. High admit SBP may also be a risk factor for developing long-COVID [11] and contributing to recently reported damage to the blood brain barrier [12].

### Ethics approval

Study was conducted with approval from Tulane University School of Medicine Institutional Review Board (IRB) under protocol “Collection of SARS CoV-2 Serum and Secretions for Countermeasure Development” [IRB 2020-396, approved 3/20/2020]. All procedures were performed in accordance with ethical standards of Tulane IRB and Helsinki Declaration of 1975 (revised 1983).

### Clinical trial registration number

NCT04956445.

### CRediT authorship contribution statement

**Dahlene Fusco:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Methodology, Investigation, Funding acquisition, Data curation. **Sharon Liu:** Writing – review & editing, Resources, Investigation, Data curation. **Marc Theberge:** Writing – review & editing, Resources, Investigation, Data curation. **Anuhya S. Pulapaka:** Writing – review & editing, Visualization, Software. **William Rittmeyer:** Software. **Yitian Zha:** Writing – review & editing, Visualization, Software. **Marlowe Maylin:** Writing – review & editing, Resources, Investigation. **W. Ben Rothwell:** Writing – review & editing, Resources, Investigation. **Prateek Adhikari:** Writing – review & editing, Resources, Investigation. **Peter Raynaud:** Writing – review & editing, Resources, Investigation. **Keith Ferdinand:** Writing – review & editing, Validation, Supervision, Methodology. **Arnaud Drouin:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

### Declaration of competing interest

Keith Ferdinand is an Editorial Board Member/Associate Editor for American Heart Journal plus and was not involved in the editorial review or the decision to publish this article.

Arnaud Drouin and Dahlene Fusco declare the following financial interests/personal relationships which may be considered as potential competing interests:

AD and DF have received investigator-initiated study awards from Gilead Sciences, have served as site investigators for COVID-19 clinical trials sponsored by Gilead, Regeneron, and Metro Biotech LLC.

DF has served on Advisory Boards for Gilead Sciences and AXCELLA.

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