

is to expand our group, support the formation of similar communities, and promote data science and AI literacy in biomedical and clinical contexts. We aspire to extend this knowledge to families, classmates, and eventually patients, facilitating a broader understanding of the role of AI in healthcare. **DISCUSSION/SIGNIFICANCE:** We believe diverse expertise and pedagogical theories can help demonstrate the potential of citizen science to democratize scientific experience. By nurturing collaborative networks our efforts aim to bridge gaps between disciplines and enhance the broader public's understanding of AI in healthcare.

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### The link between preexisting hypertension and COVID-19 severity in a hamster model

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**OBJECTIVES/GOALS:** Hypertension is a major risk factor for coronavirus disease 2019 (COVID-19) severity. Our goal was to determine if hypertension worsens lung pathology induced by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in hamsters. **METHODS/STUDY POPULATION:** Male hamsters (7-8 weeks old) were infused with angiotensin II (AII; 200 ng/kg/min via osmotic minipump) for 4 weeks to induce hypertension. During the last week of the infusion, the hamsters were inoculated intranasally with vehicle (V) or SARS-CoV-2 (S; 1 x 10<sup>5</sup> plaque forming units/ml). Half of the hamsters were sacrificed 1 day post-inoculation (dpi-1) and the other half on dpi-6. Two scoring systems were applied to lung tissue sections stained with hematoxylin and eosin to determine the degree and severity of lung pathology: the first system assessed all pertinent alterations in the lungs, while the second system only assessed the pathology related to the pulmonary vasculature. Lung histopathology scores were calculated as the sum of the airway and lung alveolar scores in arbitrary units (AU). **RESULTS/ANTICIPATED RESULTS:** Studies revealed that the SARS-CoV-2-infected hamsters exhibited a 76-fold higher total airway score compared to vehicle controls [(AU): V, 0.25 ± 0.1; S, 19.00 ± 1.35; p<0.05; n=4]. Total lung alveolar scores (27-fold) [(AU): V, 0.30 ± 0.11; S, 8.0 ± 4.1; p<0.05; n=4] and total vascular scores (17-fold) [(AU): V, 0.35 ± 0.2; S, 6.0 ± 1.4; p<0.05; n=4] were also markedly higher compared to controls on dpi-1. AII increased blood pressure, which was sustained through the 4-week infusion period. Under these conditions, body weight slightly dropped by 4.5%. Ongoing studies are assessing the effect of hypertension on the % of airway, alveoli and vessels affected, airway and alveolar severity, and bronchiolar epithelial and type II pneumocyte hyperplasia. **DISCUSSION/SIGNIFICANCE:** Establishing the hypertensive hamster as a small animal model of COVID-19 will facilitate investigations into why preexisting hypertension is a risk factor for disease severity. These studies could lead to the development of novel therapeutics for treating COVID-19 patients with hypertension.

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### Interleukin-6 protects renal dysfunction in mouse models of hypertension and salt-sensitive hypertension

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**OBJECTIVES/GOALS:** We are investigating the role of IL-6 in regulating renal function by measuring mean arterial pressure (MAP), renal plasma flow (RPF) and glomerular filtration rate (GFR) in wild type (WT) and IL-6-knockout (KO) mice in established mouse models of angiotensin II (AII)-dependent- hypertension and -salt-sensitive hypertension. **METHODS/STUDY POPULATION:** Twelve-week-old male WT and KO mice on the C57BL6 background strain were infused with vehicle (V; saline) or angiotensin II (AII; 200 ng/kg/min) for 12-14 days. Half of the AII-treatment groups were maintained on a high salt (HS; 6% NaCl) diet for the duration of the experiment, while the other half of the AII treatment groups and both vehicle groups were fed normal rat chow. MAP was continuously measured by a fluid filled catheter in conscious mice for the duration of the experiment. RPF and GFR were measured on days 12-14 in anesthetized mice by the para-aminohippurate, and fluorescein isothiocyanate-Inulin techniques, respectively. All data were analyzed by 2-way ANOVA; \*p<0.05 vs. WT, same treatment; #p<0.05 vs.V, same genotype; ^p<0.05, AII vs. AII+HS, same genotype. **RESULTS/ANTICIPATED RESULTS:** MAP was 31% lower in KO vs WT mice. AII increased MAP (1.2-fold) in WT but not KO mice. HS diet magnified AII-induced increases in MAP in WT and moderately increased MAP in AII-KO mice: [MAP (mmHg): WT+V, 130±7.0; KO+V, 91.0±4.0\*; WT+AII, 153±5.0#; KO+AII, 83.0±4.0\*; WT+AII+HS, 150±11#; KO+AII+HS, 93.0±4.0#]. AII infusion reduced RPF in the KO but not WT mice. Addition of HS reduced RPF in WT and exacerbated AII-induced reductions in RPF in KO mice [RPF (ml/min/g): WT+V, 1.82±0.23; KO+V, 1.91±0.40; WT+AII, 3.16±0.75#; KO+AII, 1.65±0.42\*; WT+AII+HS, 1.10±0.31#^; KO+AII+HS, 1.13±0.22#^]. The HS diet reduced GFR in AII-infused KO but not WT mice [GFR (μl/min/g): WT+V, 756±XX; KO+V, 788±XX; WT+AII, 1010±63\*#; KO+AII, 756±23\*; WT+AII+HS, 1100±150#; KO+AII+HS: 540±210\*#^]. **DISCUSSION/SIGNIFICANCE:** The absence of IL-6 in male mice attenuated AII- and/or AII+HS-induced increases in MAP; however, it exacerbated HS-induced reductions in RPF and GFR. These findings suggest inhibiting IL-6 has therapeutic potential as an antihypertensive but not as a renal protective agent in hypertension and salt-sensitive hypertension disease states.

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### Factors that impact the success of community-engaged research: perspectives from experienced researchers and community partners

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**OBJECTIVES/GOALS:** Involving community partners in translational research improves impact. Yet, community-engaged research