PUBLIC HEALTH

Social disparities in COVID-19 prevention

Individuals with cardiovascular disease (CVD) are known to be at risk of severe COVID-19. Hagan and colleagues now reveal that patients with CVD who live with socioeconomic disadvantage face barriers in preventing the spread of SARS-CoV-2, the coronavirus causing COVID-19. This patient population was less likely to adhere to the recommended risk-mitigation guidelines than those with greater socioeconomic advantage.

Using the US National Opinion Research Center COVID-19 Household Impact Survey, the researchers analysed the responses from 2,036 adults with self-reported CVD. A cumulative index of social determinants of health (SDOH) burden, a composite of community integration, economic stability, education, health insurance, neighbourhood quality and food security, was linked to data on COVID-19 risk-mitigation practices. Over one-third of the participants with CVD had the highest SDOH burden. Fewer individuals with the greatest SDOH burden, compared with those with the least SDOH burden, reported adhering to all social distancing (42% versus 59%) and personal protection measures, including face-mask wearing and hand hygiene, (76% versus 89%) or had a flexible work schedule (26% versus 41%). These trends remained after adjusting for demographics and comorbidities.

This questionnaire was conducted in April–June 2020, during the first stage of the pandemic. Then, public health experts primarily recommended the abovementioned preventive strategies to slow the spread of SARS-CoV-2. Vaccines and health-related behaviours, such as COVID-19 ‘decision fatigue’, were not yet present.

“This study lends further evidence to social determinants of health as key drivers of SARS-CoV-2,” the researchers advise. “Prioritizing socially disadvantaged populations in public health policies and programmes, such as health messaging and vaccination delivery, may help reduce existing disparities in COVID-19 infection risk and outcomes.”

Ciarán Martin Fitzpatrick,
BMC Series

ORIGINAL ARTICLE Hagan, K. K. et al. Social determinants of adherence to COVID-19 risk mitigation measures among adults with cardiovascular disease. *Circ. Cardiovasc. Qual. Outcomes*. <https://doi.org/10.1161/CIRCOUTCOMES.121.008118> (2021)

RELATED ARTICLE Chin-Hong, P. et al. Pulling at the heart: COVID-19, race/ethnicity and ongoing disparities. *Nat. Rev. Cardiol.* **17**, 533–535 (2020)

FIBROSIS

Cardiac fibroblasts contribute to sexually dimorphic responses to cardiac injury

Cardiac fibroblasts (CFs) might have an important role in mediating the sex-specific differences in the response to cardiac injury. According to a new study, male rats are more susceptible to cardiac fibrosis in response to chronic adrenergic stimulation than female rats, probably as a result of the increased myofibroblast activation in male hearts. Moreover, the sex-specific differences in CF responses were not abrogated by gonadectomy, suggesting that sex hormones do not control the CF response to β -adrenergic receptor (β -AR) stimulation.

Chronic β -AR activation is known to result in cardiac dysfunction, including hypertrophy and fibrosis. Sex-specific differences in β -AR signalling have been observed and are associated with improved outcomes in women compared with men. To study the cellular basis for the sexually dimorphic fibrotic response to chronic β -AR stimulation, Peter and colleagues used a model of isoprenaline (ISO) administration (a β -AR agonist) in rats. Chronic β -AR stimulation caused increased

cardiac hypertrophy, fibrosis and death in male rats compared with female rats, and these sex-specific differences were present even after gonadectomy. Further experiments showed that CFs were major contributors to this sexual dimorphism. CFs from female rats proliferated more in response to β -AR stimulation than CFs from male rats, and male rats had higher activation of CFs to myofibroblasts. In addition, ISO treatment induced an increase in the expression of *ADRB1* and *ADRB2* (encoding β_1 -AR and β_2 -AR, respectively) in CFs from male rats but decreased the expression of both receptors in CFs from female rats. Moreover, ISO-treated CFs from male rats had higher protein kinase A (PKA) activity than vehicle-treated cells, whereas ISO treatment did not activate PKA in CFs from female rats.

Irene Fernández-Ruiz

ORIGINAL ARTICLE Peter, A. K. et al. Cardiac fibroblasts mediate a sexually dimorphic fibrotic response to β -adrenergic stimulation. *J. Am. Heart Assoc.* <https://doi.org/10.1161/JAHA.120.018876> (2021)

ARRHYTHMIAS

Finerenone prevents atrial fibrillation in patients with CKD and diabetes

Mineralocorticoid-receptor antagonism with the novel drug finerenone reduces the risk of new-onset atrial fibrillation or flutter (AFF) in patients with chronic kidney disease (CKD) and type 2 diabetes mellitus. This finding comes from a secondary analysis of the FIDELIO-DKD trial and was presented at the ACC Scientific Sessions 2021.

Finerenone is a selective, non-steroidal mineralocorticoid-receptor antagonist. In the FIDELIO-DKD trial, 5,674 patients with CKD and diabetes were randomly assigned to finerenone or placebo. The main trial outcomes (published previously) showed that finerenone significantly reduced the risk of cardiovascular events in these patients. In a new, prespecified, exploratory analysis, researchers investigated the effect of finerenone on the incidence of new-onset AFF, which patients with CKD and diabetes are at a high risk of developing.

During follow-up (median 2.6 years), AFF occurred in 4.2% of patients receiving finerenone and 5.4% of patients receiving

placebo. In patients without a history of AFF at baseline, new-onset AFF occurred in 3.2% of patients receiving finerenone and 4.5% of patients receiving placebo (absolute risk reduction 1.3%; HR 0.71, 95% CI 0.53–0.94, $P=0.016$). Finerenone was also associated with a reduction in the risk of kidney or cardiovascular events, compared with placebo. The effect of finerenone on cardiovascular and renal outcomes was not significantly influenced by a history of AFF at baseline (8.1% of patients).

Preclinical data indicate that finerenone might reduce myocardial remodelling, including prevention of left atrial dilatation and left atrial fibrosis. Whether finerenone can reduce the incidence of new-onset AFF in patients with heart failure with preserved ejection fraction is being studied in the ongoing FINEARTS-HF trial.

Gregory B. Lim

ORIGINAL ARTICLE Filippatos, G. et al. Finerenone reduces onset of atrial fibrillation in patients with chronic kidney disease and type 2 diabetes. *J. Am. Coll. Cardiol.* <https://doi.org/10.1016/j.jacc.2021.04.079> (2021)