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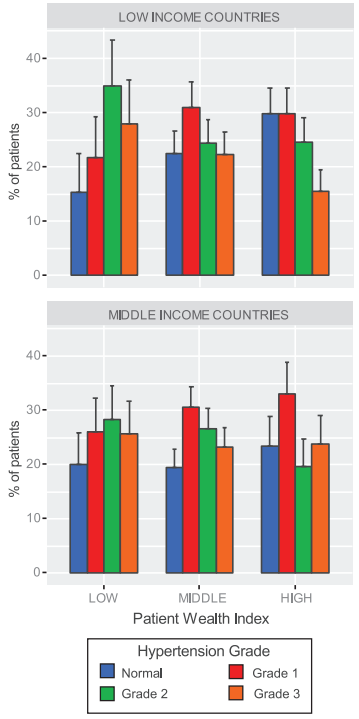
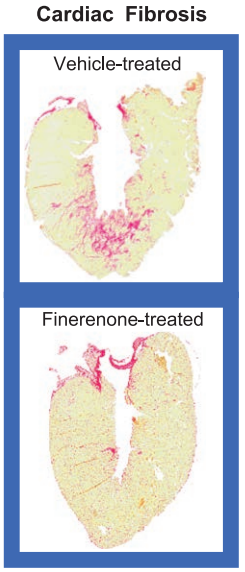
Socioeconomic Status and Hypertension in Africa (p 577)



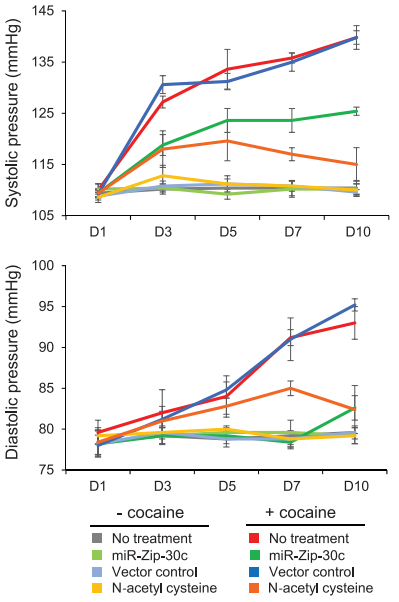
Systemic hypertension is a rapidly growing epidemic in Africa. Our study represents the first multination report on socioeconomic status and uncontrolled hypertension from Sub-Saharan Africa. Our results demonstrate the overall strikingly high proportion of uncontrolled blood pressure in low- and middle-income countries (77.4%), of which 61.7% had hypertension of grade 2 or 3. We further observed that the rate and severity (hypertension grade) of uncontrolled hypertension was proportional to individual wealth. This was observed in low-income but not middle-income countries, suggesting that the combination of individual poverty with a poor health system is particularly detrimental to achieving the goal of adequate blood pressure control. Hypertension is probably the easiest chronic noncommunicable disease to treat because blood pressure measurement for diagnosis and monitoring is simple, drug regimens are mainly once daily and inexpensive, and treatments generally do not need laboratory monitoring. According to World Health Organization estimations, the African region has the highest prevalence world rate of hypertension, afflicting 46% of the adult population aged ≥25 years. With >9 million deaths from hypertension worldwide each year, the potential effect of improved treatment of hypertension, particularly if combined with population-wide measures, would be substantial. Strategies for improving hypertension control in Sub-Saharan Africa should focus on people belonging to the lowest wealth groups with a particular focus on those living in low-income countries.

Antifibrotic Effects of Finerenone (p 599)



In this study, we demonstrate that the new nonsteroidal mineralocorticoid receptor antagonist finerenone prevents cardiac fibrosis and improves global longitudinal strain parameters in a mouse model of early subendocardial damage. In addition, we identify a new pharmacological mechanism of selective mineralocorticoid receptor modulation which distinguishes nonsteroidal mineralocorticoid receptor antagonists from steroidal mineralocorticoid receptor antagonists based on selective mineralocorticoid receptor–cofactor binding. It has been shown previously by other groups that finerenone also exerts antifibrotic action in the kidney. The efficacy and safety of finerenone in patients with type 2 diabetes mellitus and the clinical diagnosis of diabetic kidney disease is currently being investigated in 2 large phase 3 outcome trials in patients with diabetic kidney disease (FIGARO-DKD [Efficacy and Safety of Finerenone in Subjects With Type 2 Diabetes Mellitus and the Clinical Diagnosis of Diabetic Kidney Disease], URL: http://www.clinicaltrials.gov. Unique identifier: NCT02545049; FIDELIO-DKD [Efficacy and Safety of Finerenone in Subjects With Type 2 Diabetes Mellitus and Diabetic Kidney Disease], URL: http://www.clinicaltrials.gov. Unique identifier: NCT02540993). FIGARO-DKD is a 6400 patient trial that will examine the time to the first occurrence of the composite end point of cardiovascular death and nonfatal cardiovascular events, whereas FIDELIO-DKD is a 4800 patient trial that will compare the effects of finerenone versus placebo on the time to the first occurrence of the composite end point of kidney failure, decrease of estimated glomerular filtration rate ≥40% from baseline, or renal death. The marked cardiorenal antifibrotic effects of finerenone shown in our study and by others are likely to be substantial for finerenone's clinical effects currently tested in these trials.

MiR-30c-5p Pathway in Cocaine-Induced Hypertension (p 752)



The use of cocaine remains the leading cause for all illicit drug-related emergency department visits, with the majority of these visits resulting from cardiovascular complaints, including chest pain. Cocaine exposure has short-term effects on the cardiovascular system, such as increasing blood pressure and inducing arrhythmias, but also negatively impacts long-term cardiovascular health by increasing the risk for hypertension, aortic stiffness, and atherosclerosis. It is well-known that cocaine inhibits catecholamine reuptake at sympathetic nerve terminals, stimulates central sympathetic outflow, and increases the sensitivity of adrenergic nerve endings to norepinephrine. It has been assumed that these cocaine-associated sympathetic nervous system effects are wholly responsible for hypertension and other cardiovascular effects resulting from cocaine exposure. However, recent studies have suggested that other mechanisms may be governing the effects of cocaine on the cardiovascular system. To better understand the molecular mechanisms underlying cocaine-induced cardiovascular toxicities, we analyzed microRNAs whose expression was altered in mouse aortas in response to cocaine exposure. We have identified a key pathway, the miR-30c–Me1 (malic enzyme 1) axis, that is involved in regulating reactive oxygen species levels, crucial factors in the pathogenesis of hypertension, vascular aging, and atherosclerosis. Inhibition of this pathway suppresses cocaine-induced elevation of blood pressure and augmentation of aortic stiffness in mice, providing new molecular targets for the development of therapeutic strategies as well as the rationale for the use of an antioxidant, such as N-acetylcysteine, to treat cocaine-associated cardiovascular toxicities.

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