Chronic kidney disease (CKD) is an independent risk factor for cardiovascular disease (CVD), across all phenotypes. These includes atherosclerotic cardiovascular disease, heart failure, valvular heart disease, and arrythmias. The disproportionate burden of CVD seen in CKD represents the confluence of traditional risk factors (such as hypertension, diabetes ,and obesity), and pathophysiological mechanisms unique to the CKD milieu (such as inflammation, vascular/valvular calcification, and fibrosis). The elevated risk for CVD is present even in early stages of CKD, and is largely under-recognized given limited screening for CKD with eGFR measurement and albuminuria quantification, even in high-risk patients. The risk of CVD increases with progressive decline in kidney function, and while attenuated after kidney transplantation, still remains elevated when compared to the general population. Paradoxically, patients with CKD are routinely under-represented or excluded from cardiovascular trials, and are less likely to receive guideline directed therapies or procedures that may reduce CVD burden, a phenomenon termed “renalism.”

There have been several recent major advances in the cardiorenal space with the development of therapies such as the sodium glucose co-transporter inhibitors, glucagon like peptide-1 receptor agonists, and non-steroidal mineralocorticoid receptor antagonists, which have changed the therapeutic landscape for patients with CVD and CKD. These developments call for an urgent need for early detection of CKD to ensure appropriate and timely access to these life and organ saving therapies for patients with CKD. Finally, recognizing several systems-based barriers to appropriate care for patients with CVD and CKD is essential to save lives, reduce hospitalizations for heart/kidney disease, and improve quality of life.