The following research summaries were prepared by ASF's Research Director, B. André Weinstock, PhD, MSAS.

Le Bleu VS, Kanasaki K, Lovisa S, et al. Genetic reprogramming with stem cells regenerates glomerular epithelial podocytes in Alport syndrome. *Life Sci Alliance.* Published online April 1, 2024. **DOI:** 10.26508/lsa.202402664 (open access).
The concept of using stem cell transplants to cure Alport syndrome is not new but has encountered many challenges to making it a viable human therapy. This article makes some progress on this front by significantly improving a severe form of mouse Alport syndrome (COL4A3 knockout) with different types of genetically modified or specially prepared healthy stem cells via juvenile bone marrow transplant. The authors further confirm that podocyte cells are the sole source for collagen-IVα3,4,5 in the glomerular basement membrane and the specific target for future potential curative Alport therapies.

Mallawaarachchi AC, Fowles L, Wardrop L, et al. Genomic Testing in Patients with Kidney Failure of an Unknown Cause: a National Australian Study. *Clin J Am Soc Neph.* Published online May 3, 2024. **DOI:** 10.2215/CJN.0000000000000464 (open access).
This article represents further confirmation of the relatively high prevalence of Alport syndrome among undiagnosed and misdiagnosed kidney disease patients. In this Australian-based study, 100 chronic kidney disease patients with unknown (idiopathic) diagnoses had whole genome sequencing performed. Results showed that 25% were caused by genetic mutations, including four cases of Alport syndrome (both COL4A5 and COL4A4).

Puapatanakul P, Miner JH. Alport syndrome and Alport kidney diseases – elucidating the disease spectrum. *Curr Opin Nephrol Hypertens.* Published May 2024. **DOI:** 10.1097/MNH.0000000000000983 (open access).
This article (coauthored by Dr. Jeffrey Miner of ASF's Scientific Advisory Research Network) is a thorough overview of the very latest understanding of the diversity among Alport syndrome kidney disease genotypes and phenotypes, which range from clinically unrecognizable symptoms, to hematuria with or without proteinuria, to kidney failure. These outcomes are influenced by birth sex, genotype, and other genetic and environmental factors.