Joseph Lagas, Doctoral Candidate in Molecular Biology (Washington University in St. Louis) and member of ASF's Emerging Leadership Council, generously agreed to provide a summary of Kohei Omachi's research.

Certain genetic mutations cause the formation of what is called a premature termination codon which result in the final protein only being partially made. This partially made protein cannot perform its normal function and can cause disease through the loss of this function; this occurs in some Alport Syndrome patients. This new class of drugs can cause the machinery making the protein to ignore the premature termination codon and make the entire protein like normal resulting in a complete normally functioning protein. A post-doctoral researcher in Dr. Miner's lab, Kohei Omachi, has found that, in cellular models of Alport Syndrome, this Read Through therapy may improve kidney cell function making it a promising new drug therapy for Alport Syndrome patients. Research into this therapy will now transition into mice and, if positive results are obtained, into clinical trials a few years down the road. This research highlights that much is being done in understanding and researching Alport syndrome, with some researchers thinking outside the box for new and exciting treatments.