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## **EMBARGOED FOR RELEASE UNTIL 5:30 PM CT ON OCTOBER 30**

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October 27–28: ASN Management Office, America’s Center, Room 130, (314) 342-5511  
Friday, Oct. 29 – Monday, Nov. 1: ASN Media Room, America’s Center, Room 250,  
(314) 342-5508 (media room), 202-236-8142 (after hours)

### **ADVANCES MADE IN UNDERSTANDING THE PATHOGENESIS OF MEMBRANOUS GLOMERULONEPHRITIS**

**St. Louis, MO (Oct. 30, 2004)**— Although scientists have long suspected a link between a classic animal model in rats, Heyman nephritis, and the human disease, membranous glomerulonephritis, it’s taken more than 3 decades for Pierre M. Ronco and his colleagues of Hospital Tenon in Paris, France to connect the rat model to the human disease. Their groundbreaking research, the identification of the molecular mechanisms involved in the development of membranous nephropathy, a major cause of chronic renal failure, paves the way for numerous advances in understanding various forms of human glomerulonephritis. This landmark research, published in *The Lancet* on October 2, 2004, will be presented at the American Society of Nephrology’s 37<sup>th</sup> Annual Meeting and Scientific Exposition in St. Louis, Missouri.

An immune-mediated disease, membranous glomerulonephritis (MGN) is a common cause of nephrotic syndrome in adults and progresses to renal failure in about 40% of patients, eventually requiring dialysis or transplantation after 10 years. Although it can be associated with various infections, cancers, autoimmune diseases, and drugs, its cause in most patients has largely remained unknown.

Dr. Ronco and his colleagues analyzed a subgroup of pediatric patients with MGN and their mothers to understand the immune mechanism responsible for causing MGN in the fetus. They discovered that the disease was transmitted during pregnancy by maternal antibodies raised against a fetal kidney component, neutral endopeptidase. The mother recognized fetal neutral endopeptidase, present on placental cells, as a foreign antigen because she was genetically deficient in the neutral endopeptidase gene. The mothers antibodies then attacked the fetal kidney where they induced the disease. This process, which recalls Rh incompatibility, may also occur in neonatal diseases affecting other organs than the kidney.

“By understanding what causes the transference of MGN from mother to fetus, we have learned more about MGN and to some extent other types of human glomerulonephritis, including lupus nephritis,” says Ronco, Director of the Renal Division and INSERM Unit 489 "Remodeling and Repair of Renal Tissue" and Professor of Renal Medicine at Pierre et Marie Curie University. “It is important that we begin monitoring neutral-endopeptidase-deficient pregnant women and also develop therapeutic approaches to neutralize antibodies from the mother that cause kidney disease in the baby.”

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Dr. Ronco will present his findings during a lecture entitled, “Pathogenesis of Idiopathic MGN: Some New Clues,” as he joins several colleagues in a Clinical Nephrology Conference session devoted to membranous nephropathy during ASN’s Renal Week on Saturday, October 30 at 4:00 p.m.–6:00 p.m. in the Ferrara Theater of the America’s Center.

To encourage additional research on this disease and raise awareness of MGN in the scientific and lay communities, The Halpin Foundation and the ASN have created a research grant for scientists interested in studying MGN.

The ASN is a not-for-profit organization of 9,000 physicians and scientists dedicated to the study of nephrology and committed to providing a forum for the promulgation of information regarding the latest research and clinical findings on kidney diseases. ASN’s Renal Week 2004, the largest nephrology meeting of its kind, will provide a forum for more than 12,000 nephrologists to discuss the latest findings in renal research and engage in educational sessions relating advances in the care of patients with kidney and related disorders from October 27- November 1, 2004 at the America’s Center in St. Louis, Missouri.

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