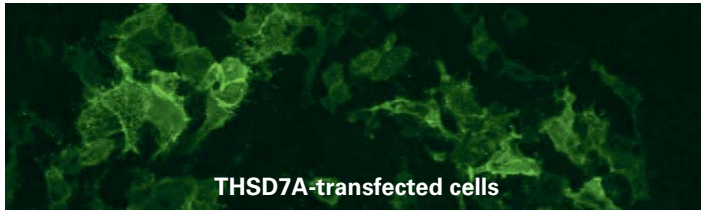




Anti-THSD7A IIFT



- Maximal specificity for primary membranous nephropathy (MN)
- Ideal supplement to the Anti-PLA2R IIFT for the differentiation between primary and secondary MN

Technical data

| | |
|--------------------------|--|
| Antigen substrate | Transfected cells and control-transfected cells (EU 90) |
| Sample material | Serum or plasma |
| Sample dilution | Qualitative 1:10; semiquantitative: 1:10, 1:100, 1:1000 etc. |
| Reagents | Ready for use, with the exception of the PBS Tween buffer |
| Test procedure | 30 min (sample) / 30 min (conjugate), room temperature |
| Microscopy | Objective: 20x; light source: EUROIMMUN LED, EUROStar Bluelight or mercury vapour lamp, 100W, Excitation filter: 450-490nm, colour separator: 510nm, blocking filter: 515 nm |
| Stability | 18 months from the date of manufacture when stored at +2°C to +8°C |
| Test kit format | 10 slides, each containing 3 or 5 test fields |
| Order no. | FA 1254-####-51 G |
| Related products | FA 1254-####-1 IIFT: Membranous Nephropathy Mosaic 1 (PLA2R-, THSD7A- and control-transf. cells) |

Clinical significance

Membranous nephropathy (MN) is a chronic inflammatory disease of the glomeruli which is accompanied by a progressive impairment of the kidney function. It is the most frequent cause of nephrotic syndrome in adults. MN is prevalent in all ethnic groups and genders, with men over 40 years of age and of white skin colour being more frequently affected. In young women with suspected MN, lupus nephritis should be considered. MN occurs very rarely in children. In around 20% of patients, MN is a secondary (accompanying) disease, which may result from infections, medication, drug or toxin intake, collagenosis, or other autoimmune diseases, and tumours. Secondary MN should be differentiated from primary MN (pMN). Whereas the therapy of secondary MN is based on the underlying disease, the treatment of pMN is aimed at the improvement of prognosis, especially with respect to nephrotic syndrome and hypertonia. If the origin of MN is unknown, which means it is neither autoantibody-associated nor secondary, it is called "idiopathic" (idiopathic membranous nephropathy, iMN). The underlying autoimmune mechanism of pMN is based on the production of autoantibodies against the transmembrane proteins phospholipase A2 receptor (PLA2R) and thrombospondin type-1 domain-containing protein 7A (THSD7A). These proteins are expressed on the podocyte surface. As a result of the binding of antibodies, the podocytes are damaged and protein enters the primary urine. While autoantibodies against PLA2R can be detected in the serum of up to 75% of pMN patients, the prevalence of anti-THSD7A varies from 2.5% to 14%, depending on the pMN cohort. In rare cases, autoantibodies against PLA2R and THSD7A may also occur together. The connection between THSD7A-positive pMN and the presence of malignant tumours is currently being researched.

Diagnostic application

The Anti-THSD7A IIFT is the ideal supplement to the Anti-Phospholipase A2 Receptor (PLA2R) IIFT for the serological screening of patients with suspected primary MN. The IIFT is suited for qualitative and semiquantitative determination of human autoantibodies of class IgG against THSD7A. The "IIFT: Membranous Nephropathy Mosaic 1" allows simultaneous determination of anti-PLA2R and anti-THSD7A autoantibodies, which increases the serological detection rate.



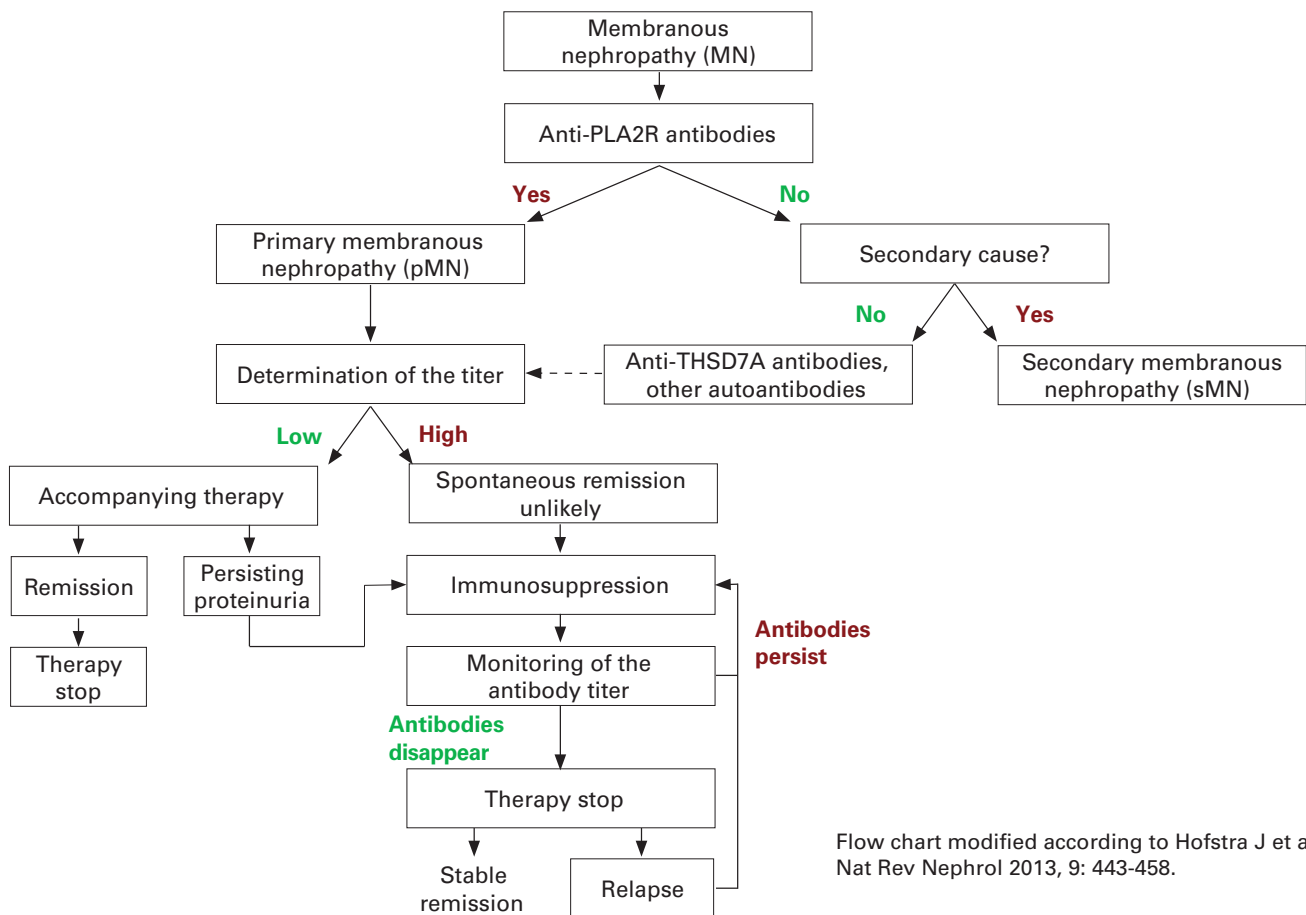
Test evaluation

Fluorescence pattern (positive reaction): Antibodies against thrombospondin type-1 domain-containing protein 7A (THSD7A) react with transfected cells of the test substrate, producing a fine-granular cytoplasmic fluorescence with an accentuated cell membrane. The cell nuclei remain unstained.

Reference range

Titer 1: < 10

Role of pMN-specific autoantibodies in diagnosis and therapy monitoring



Literature

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