Stroke is the third cause of death and the first cause of long-term disability. Ischemic stroke accounts for 87% of all strokes. A major cause for ischemic stroke is represented by atherosclerotic vulnerable plaques, whose clinical detection is an unmet priority. The complement system is an inflammatory process involved in plaque’s morphological evolution. STATEMENT aims at using the complement proteins as circulating bio sensors of plaque instability and stroke occurrence.

STATEMENT will focus on the pre-identified circulating complement biomarkers for plaque vulnerability, namely ficolins, initiators of the complement lectin pathway (LP). We will also analyze LP’s downstream active products and other complement pathway proteins seeking markers with enhanced sensitivity. We will retrospectively analyze available cohorts of atherosclerotic patients undergone endarterectomy. The identified biomarkers will be analyzed in vitro to define their functional interactions with cellular plaque components, like macrophages, platelets, neutrophils and smooth muscle cells. The candidate biomarkers, selected on the basis of the retrospective patient study and on the in vitro study, will be finally tested in patients in a multicentric study (Italy, Norway and France). STATEMENT will help advance prevention of neurologic complications and improve therapy by providing a marker for the early detection of rupture-prone atherosclerotic carotid plaques, bearing a risk for stroke.