ISSN 1331-2820
UDK 616.9

# Riječ Uredništva

## 95 godina od prvog opisa Creutzfeldt-Jakobove bolesti

Bolest je prvi opisao neuropsihijatar Hans Gerhard Creutzfeldt 1920. godine, a sljedeće godine Alfons Jakob opisao je još dva slična bolesnika.

Bolest je karakterizirana propadanjem nervnih stanica u korteksu velikog mozga, ali i u drugim dijelovima subkorteksa. To je bio razlog da se govorilo o neurološkoj degenerativnoj bolesti. Opisivana je vakularna degeneracija u neuronima. Godine 1966., D. C. Gajdušek je dokazao da je Kuru kao i Creutzfeldt-Jakobova bolest prenosiva. Za to veliko otkriće dobio je Nobelovu nagradu.

Od tada se govori o transmisivnoj, degeneratinoj bolesti, a uzročnik bi mogao biti "*slow virus*". Bolest bi nastajala nakon produljene inkubacije zaraženih tim virusom. Otprije je poznato da neki virusi izazivaju kronične infekcije mozga, kao što je subakutni sklerozirajući panencefalitis, progresivni multifokalni encefalitis te AIDS.

Gajdušek i Gibbs (1977. god.) pretpostavljali su da je "nekonvencionalni" virus infektivni agens za Creutzfeldt-Jakobovu bolest, Kuru i scrapie.

Godine 1981., Stanley B. Prusiner je postavio novu teoriju tzv. prionsku, po kojoj Pr PSc izaziva bolest. Unatoč široko prihvaćenoj prionskoj etiologiji ostale su mnoge dileme u razumijevanju nove teorije.

PrP nema nukleinsku kiselinu koja je neophodna za razmnažanje infektivnog agensa. Novija istraživanja su pokazala da PrP nije infektivan i ne zadovoljava Kochove principe. U sadašnje vrijeme se ponovno piše o virusnoj teoriji etiologije ovih bolesti.

Manuelides L. tvrdi 2007. god. da su otkrivene virionske čestice veličine 25 nm koje uzrokuju transmisivnu spongioznu encefalopatiju.

Otkrićem virusa kao etiološkog agensa možda će se doći i do etiološke terapije, što bi moglo pružiti nadu bolesnicima s Creutzfeldt-Jakobovom bolesti i uspjeh liječnicima koji nažalost primjenjuju još i danas metode iz 1920. god.

***Prof. dr. sc. Ivan Beus***

ISSN 1331-2820
UDK 616.9

# Editorial

## 95 years since the first description of Creutzfeldt-Jakob disease

The disease was first described by neuropsychiatrist Hans Gerhard Creutzfeldt in 1920, and the following year Alfons Jakob described two similar cases.

The disease was characterized by deterioration of the nerve cells in the cerebral cortex, but also in other subcortical regions. That was the reason why the condition was referred to as neurological degenerative disease. Neuronal vacuolar degeneration was also described.

In 1966, D. C. Gajdušek recognized that Kuru, like Creutzfeldt-Jakob disease, was also a transmissible disease. For this great discovery he was awarded the Nobel Prize. It was believed that a "*slow virus*" was responsible for this transmissible, degenerative disease occurring after a prolonged incubation of those infected with this virus. It has been known that some viruses cause chronic infections of the brain, such as subacute sclerosing panencephalitis, progressive multifocal encephalitis and AIDS.

Gajdušek and Gibbs (1977) assumed that "unconventional" virus was the infectious agent for Creutzfeldt-Jakob disease, Kuru and scrapie.

In 1981, Stanley B. Prusiner proposed the new "prion" theory, according to which Pr PSc causes the disease. Despite the widely accepted prion etiology, many dilemmas remained in understanding this new theory.

PrP lacks nucleic acids required for the propagation of the infectious agent. Recent studies have shown that PrP is not contagious and does not fulfill principles of Koch's postulate. Recently, much is written about the viral theory on the etiology of these diseases. In 2007 Manuelides L. argued that virion particles 25 nm in diameter cause transmissible spongious encephalopathy.

The discovery of the virus as etiological agent might lead to the discovery of etiological treatment, which could offer hope to patients with Creutzfeldt-Jakob disease, and success to physicians who unfortunately still use methods from the 1920s.

***Prof Ivan Beus MD, PhD***