Stroke is considered the most common and severely disabling neurological disease. It is one of the leading causes of death after heart disease and cancer, causing 10% deaths worldwide and involving risk factors such as smoking, obesity and nutritional disbalance. Disability affects 75% of stroke survivors through severe mental and/or physical impairment depending on the affected brain area (Go et al., 2014). Stroke treatment is limited to thrombolysis and antiplatelet therapy with a tissue plasminogen activator (tPA), which is only useful if administered within 3 hours of the appearance of early symptoms. This therapy is only used in 1–2% stroke patients and, has, indeed, limited clinical success, as it does not protect neurons from the hypoxic insult. Therefore, the development and evaluation of new drugs to reduce the life-threatening effects of stroke and hypoxia might prove extremely fruitful.

Stroke is produced by a decrease in blood supply due to different types of alterations in the brain blood vessels, which cause cerebral hypoxia/ischemia. In turn, the hypoxic damage produces several changes in gene expression patterns in brain tissue cells (neurons and glial cells). Rapid thrombolysis is effective in protecting the injured ischemic core, although preventing the pathological features that produce neuronal death requires the development of new treatments and the implementation of a dual therapy combining the disruption of the clot and the preservation of neuronal function. In this respect, many preclinical studies have been successful in finding neuroprotective agents, but subsequent clinical trials have rendered disappointing results.

Inflammation is one of the distinctive events in stroke, while microglial cells are the predominant inflammatory effectors in brain. Reactive astrocytosis and the formation of a glial scar in the boundary zone of the ischemic core are also critical events which can produce both positive and negative consequences.

Diet and fatty acid consumption are risk factors associated with stroke and are modified by the presence of triterpenoids. These results could be related to a reduction (Figure 1) in the activity of neuronal nitric oxide synthase (nNOS) and inducible nitric oxide synthase (iNOS) in microglial cells, both of which decreased with OA exposure in hypoxic animals. Therefore, acting through the activation of Nrf2, OA reduces (Figure 2) the expression of nNOS and iNOS in the hypoxic brain, protecting neurons against the oxidative damage triggered by microglial cells. We also showed an astrocytic reaction involving high levels of protein S100 which could stimulate the uptake of excitotoxic levels of glutamate and prevent neuronal damage, while also inducing the recovery of cytoskeletal protein assembly and thus stabilize cell morphology and function. Although experimental data is still required, growing evidence demonstrates the beneficial consequences of OA in the prevention and treatment of different neuropathologies. The enrichment of food and diet with this compound could prove a useful tool in health and quality of life improvement and decrease the incidence of frequent pathologies such as vascular diseases.

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