Brain lesion

Brain lesions can arise from traumatic brain injury, infection, and craniotomy. Although injectablehydrogels show promise for promoting healing of lesions and health of surrounding tissue, enabling cellular ingrowth and restoring neural tissue continue to be challenging. Hu et al. hypothesized that these challenges arise in part from the mismatch of composition, stiffness and viscoelasticity between the hydrogel and the brain parenchyma, and tested this hypothesis by developing and evaluating a self-healing hydrogel that not only mimicked the composition, but also the stiffness and viscoelasticity of native brain parenchyma. The hydrogel was crosslinked by dynamic boronate ester bonds between phenylboronic acid grafted hyaluronic acid (HA-PBA) and dopamine grafted gelatin (Gel-Dopa). This HA-PBA/Gel-Dopa hydrogel could be injected into a lesion cavity in a shear-thinning manner with rapid hemostasis, high tissue adhesion and efficient self-healing. They tested this in an in vivo mouse model of brain lesions and found the multi-functional injectable hydrogel to support neural cell infiltration, decrease astrogliosis and glial scars, and close the lesions. The results suggest a role for extracellular matrix-mimicking viscoelasticity in brain lesion healing, and motivate additional experimentation in larger animals as the technology progresses towards potential application in humans [1)].

During surgery for intrinsic brain lesions, it is important to distinguish the pathological gyrus from the surrounding normal sulci and gyri. This task is usually tedious because of the pia mater-arachnoid membranes with their arterial and venous complexes that obscure the underlying anatomy. Moreover, most tumors grow in the white matter without initially distorting the cortical anatomy, making their direct visualization more difficult.

Focal brain lesions can alter the morphology and function of remote brain areas. When the damage is inflicted more slowly, the functional compensation by and structural reshaping of these areas seem to be more effective. It remains unclear, however, whether the momentum of lesion development also modulates the functional network topology of the remote brain areas. In this study, we compared resting-state functional connectivity data of patients with a slowly growing low-grade glioma (LGG) with that of patients with a faster-growing high-grade glioma (HGG). Using graph theory, we examined whether the tumour growth velocity modulated the functional network topology of remote areas, more specifically of the hemisphere contralateral to the lesion. We observed that the contralesional network topology characteristics differed between patient groups. Based only on the connectivity of the hemisphere contralateral to the lesion, patients could be classified in the correct tumour-grade group with 70% accuracy. Additionally, LGG patients showed smaller contralesional intramodular connectivity, smaller contralesional ratio between intra- and intermodular connectivity, and larger contralesional intermodular connectivity than HGG patients. These results suggest that, in the hemisphere contralateral to the lesion, there is a lower capacity for local, specialized information processing coupled to a higher capacity for distributed information processing in LGG patients. These results underline the utility of a network perspective in evaluating effects of focal brain injury [2)].

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2)
De Baene W, Rutten GJM, Sitskoorn MM. The Temporal Pattern of a Lesion Modulates the Functional Network Topology of Remote Brain Regions. Neural Plast. 2017;2017:3530723. doi: 10.1155/2017/3530723. Epub 2017 Aug 3. PubMed PMID: 28845308; PubMed Central PMCID: PMC5560088.