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Polyethylene glycol-containing polyurethane hydrogel coatings for improving the biocompatibility of neural electrodes

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ABSTRACT

The instability of the interface between chronically implanted neuroprosthetic devices and neural tissue is a major obstacle to the long-term use of such devices in clinical practice. In this study, we investigate the feasibility of polyethylene glycol (PEG)-containing polyurethane (PU) hydrogel as coatings for polydimethylsiloxane (PDMS)-based neural electrodes in order to achieve a stable neural interface. The influence of PU hydrogel coatings on electrode electrochemical behaviour was investigated. Importantly, the biocompatibility of PU hydrogel coatings was evaluated in vitro and in vivo. Changes in the electrochemical impedance of microelectrodes with PU coatings were negligible. The amount of protein adsorption on the PDMS substrate was reduced by 93% after coating. Rat pheochromocytoma (PC12) cells exhibited more and longer neurites on PU films than on PDMS substrates. Furthermore, PDMS implants with ($n = 10$) and without ($n = 8$) PU coatings were implanted into the cortex of rats and the tissue response to the implants was evaluated 6 weeks post-implantation. GFAP staining for astrocytes and NeuN staining for neurons revealed that PU coatings attenuated glial scarring and reduced the neuronal cell loss around the implants. All of these findings suggest that PU hydrogel coating is feasible and favourable for neural electrode applications.

1. Introduction

Neuroprosthetic devices have been applied in clinical practice for many years to restore lost function in people who suffer from neurological disorders. For example, the cochlear implant is widely used to restore hearing in deaf patients, and deep brain stimulation (DBS) has become an accepted treatment for advanced Parkinson's disease [1–3]. The success of cochlear implants and DBS has promoted the research and development of other kinds of neuroprosthetic devices for clinical practice, such as visual prosthetics and brain–computer interfaces [4–6]. Neuroprosthetic devices replace lost neurological function by selectively stimulating the target neural tissue with the stimulation electrodes [7,8]. The electrodes are the key component in neuroprosthetic devices for establishing a functional electrical connection to record neural signals and to stimulate neural tissue. There are several types of electrodes designed to interface with the nervous system, such as intracortical needle electrodes, precision mechanics electrodes in cochlear implant and DBS, and micromachined subdural electrode arrays for recording electrocorticograms [9,10]. All these electrode approaches work well during acute implantation, but often suffer performance degradation after long-term implantation [11]. Although the precise mechanisms of this degradation are still not clear, the tissue response to implanted electrodes is believed to be a major contributing factor [12,13].

When a neural electrode is implanted into the brain for a long time, it initiates an inflammatory cascade involving protein adsorption, microglia/macrophage recruitment and activation, cytokine release, and glial scar formation around the electrode [13,14]. This will increase the distance between implanted electrodes and target neurons and decrease the efficacy of the neural stimulation, leading to deterioration in the performance of neuroprosthetic devices.

Many factors affect the tissue response to implanted electrodes [15–19], including the insertion trauma during the implantation, micromotion-induced mechanical strain between the electrode and the brain tissue around the implant [20,21], the chemical composition of electrode material, and the persistent presence of the electrode. When the electrode is implanted into the brain, it disrupts the blood vessels and ruptures the blood–brain barrier. A layer of host serum protein is then adsorbed onto the electrode surface, which activates the microglia/macrophages and initiates the acute tissue response. It has been found that major blood vessel disruption during implantation can induce greater tissue response

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