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INTERACTIONS BETWEEN DAMAGE EVENTS AND BIOINSPIRED VASCULATURES EMBEDDED IN FIBRE REINFORCED COMPOSITES

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ABSTRACT

The susceptibility of fibre reinforced polymer (FRP) laminate structures to impact damage is well documented. This damage can manifest itself in such a way that significant reductions in mechanical performance result whilst leaving little visual evidence of the impact event (a situation termed *barely visible impact damage* BVID). Microvascular networks that can potentially deliver large volumes of healing resin from an external reservoir to regions of internal damage have been recognised as a means of imparting a self-healing functionality to load bearing FRP structures. Interactions of such vascules with propagating cracks and damage events is a necessity to ensure release of the contained healing resin, and as such is the subject of this investigation [1].

Unidirectional glass fibre reinforced epoxy (HexPly 913, Hexcel Composites) was selected due to its translucency, allowing easy monitoring of an internal crack front. Steel wire with a diameter of 0.5mm, pre-coated with a PTFE release spray to ease removal post-cure, was selected as the vascule preform material. For fabrication route A, the vascule preform wires were located at the laminate mid-plane between the two central plies. To incorporate the steel wires via fabrication route B, the four central plies were cut at the desired vascule location. Four plies were cut to create the recess necessary for 0.5mm wires based on the nominal ply thickness of the pre-impregnated tape (0.135mm, determined experimentally). The same techniques were used to prepare specimens containing vascules orientated parallel or transverse to the fibre orientation. Typical micrographs of the resultant vascule morphologies are provided in Figure 1. Embedding a vascule transverse to the fibre orientation, via fabrication route A, led to significant fibre waviness and the formation of large resin pockets. The corresponding vascule formed via fabrication route B shows the elimination of fibre waviness but resin pockets still persist at the corners of the square recess created during fabrication. Alignment of the vascule to the fibre direction eliminates fibre waviness and resin pockets as the fibres 'fit' around the wire during the laminate cure cycle. The effects of the different vascule microstructures on the Mode I (Double Cantilever Beam, DCB) and II (End Loaded Split, ELS) interlaminar fracture toughness were evaluated.

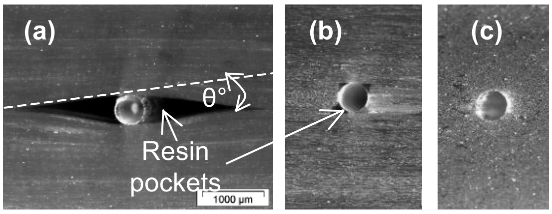

Figure 1: Vascule characterisation; (a) transverse fabrication route A, (b) transverse fabrication route B and (c) aligned vascule representative of both fabrication routes.

Vascules aligned parallel to the fibre and crack propagation direction were cleaved open regardless of fabrication route or opening mode and had little effect on the innate fracture toughness of the host laminate, Figure 2. However, transversely orientated vascules had significantly differing crack interactions:

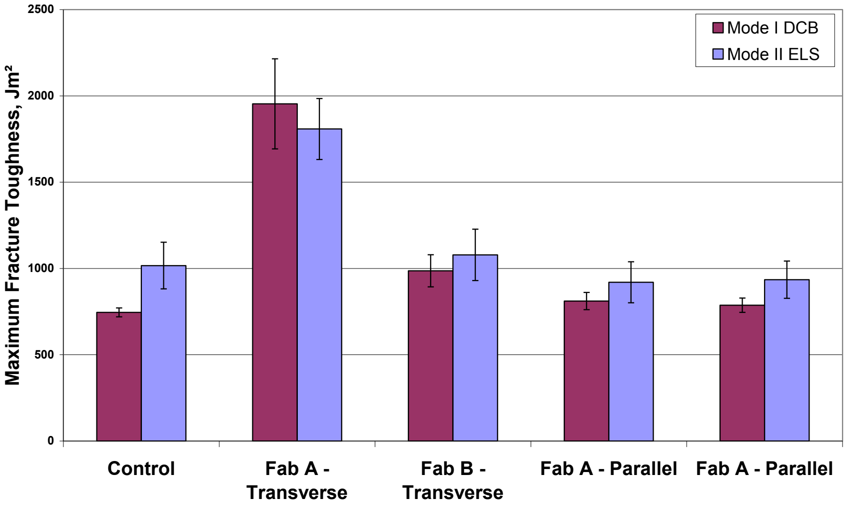

Figure 2: Modes I & II fracture toughness

Fabrication route A – Under mode I opening conditions these vascules were found to arrest cracks for significant periods leading to exceptional values of fracture toughness at the vascule locations. However, the build up of strain energy lead to rapid unzipping of the laminate fracture plane on propagation of the crack from the vascule backface. Under Mode II conditions the crack was arrested at the tip of the resin pocket, then jumped a layer of fibres leading to rapid unzipping of the specimen. This crack propagation path followed the fibre waviness around the vascule in such a way as resin release would not be expected (Fig 3(b)), and is in agreement with the damage layout formed around a vascule embedded within a quasi-isotropic carbon fibre laminate [2] using the same fabrication technique (Fig. 3(c)).

Fabrication route B – Cracks were found to breach the vascules under both Mode I & II conditions (Fig. 3(d) & (e)), leading to moderate crack arrest. Here, the stress concentrations expected at the corner resin pockets caused the vascule boundary to fail at lower strain energies. The energy stored due to crack arrest at each vascule was insufficient to cause rapid 'unzipping' of the specimens, with the crack being arrested at each subsequent vascule (if it jumped that far). The opening of the vascules is a pre-requisite for self-healing, as was confirmed under impact loading (Fig. 3(f)).

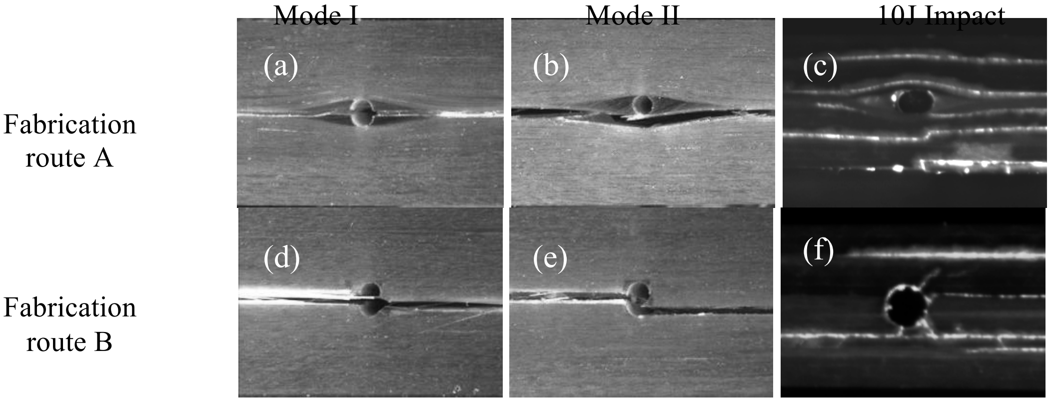

Figure 3: Vascule-damage interactions

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