DAMAGE CONTROL WITHIN SKIN-STIFFENER DEBOND SPECIMENS UNDER FATIGUE CONDITIONS

R. Luterbacher*, R.S. Trask, I.P.Bond

Advanced Centre for Composite Innovation and Science (ACCIS), University of Bristol, Queen's Building, Bristol, BS8 1TR, United Kingdom

**Rafael.Luterbachermus@bristol.ac.uk*

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Abstract

The potential to redirect propagating delaminations in skin-stringer configurations away from critical failure paths and into dedicated self-healing zones has been experimentally investigated. Thermoplastic interleaves (for redirection) and a vascular network (for redirection and to ameliorate the damage) have been embedded into the structure. A variety of interleave – vascule configurations have been investigated, all successfully redirecting the propagating fatigue damage into the vascular network. The injection of a self-healing agent (low viscosity epoxy) into the vascular network demonstrated recovery of mechanical properties.

1 Introduction

Locally stiffened or "stringer"-skin structures are extensively used for lightweight applications. This design philosophy is efficient, as the skin takes the in-plane loads, whereas the stiffening elements provide increased bending stiffness and a reduction of the effective buckling length [1]. However, due to the localized stiffening, stress concentrations arise which can initiate damage e.g. debonding or delamination, within the structure. To mitigate against this risk, significant safety margins are used within the design phase in order to comply with the "damage non-growth" philosophy. This limits the potential weight savings offered by the application of advanced fibre reinforced composites. In addition, time consuming non-destructive inspections are necessary and, if damage is found, down-time of the aircraft and expensive repairs are required.

Self-healing materials are able to restore one or more functionalities e.g. mechanical properties [2]. Two different approaches can be distinguished. The first is a material with an intrinsic healing capability, such as remendable polymers. An external trigger is needed, e.g. heat, in order to activate the intrinsic healing mechanism. *Yang et al.* [3] reported on the use of mendable thermoplastic of carbon-epoxy T-joints. In the second case, an extrinsic healing agent is introduced into the material by means of microcapsules or a vascular network.

O'Brien et al. [4] incorporated microcapsules with a self-healing capability in skin/stringer flange debond specimens. However, the limited volume of healing agent was not sufficient to restore the mechanical properties.

The vascular network can either be hollow glass fibres which rupture in a damage event and release a healing agent (i.e. see [5,6]) or a network of hollow channels which is connected to an external reservoir (i.e. see [7,8]). The latter has the benefits that (1) the healing agent does not have to withstand the harsh curing environment (elevated temperature and pressure); (2) and the shelf life is not critical, as the healing agent is introduced into the laminate after the damage event;(3) if pressurized, the channels can be used for monitoring of the health state [9]; and (4) large damage volumes can be infused.

These vascular networks are manufactured by a "lost wax casting" procedure providing linear, branched and 3D configurations [10].

One major challenge facing the use of a vascular network for self-healing, is to guarantee connectivity between the vascules and the damage. Delaminations tend to propagate along the interfaces of plies with different ply angles. *Yasaee et al.* [11] controlled the interfaces along which delaminations propagate by selectively interleaving and arresting delaminations at interleaves. Interleaving consists of introducing a thin layer of thermoplastic, thermoset or chopped fibres between composite plies, thereby increasing the fracture toughness.

Vascules arranged transversely to the delamination propagation have also been reported to increase the fracture toughness [9,12] and can be used to control damage propagation.

The aim of this work is to prove the capability of redirecting propagating damage into a chosen area, where a self-healing feature is present which can then recover the mechanical performance of the structural part.

2 Materials and Methods

2.1 Specimen description

Testing of skin-stiffened structures is both technically challenging and expensive. In order to study the debonding of stringers, *Minguet et al.* [13] proposed simplified specimens, called "skin/stringer flange debond specimens". These specimens consist of a flange bonded to the skin and have a similar stress state and damage propagation as in a skin stringer bay configuration.

The geometry of the specimens is shown in Figure 1. Both the skin and flange were manufactured using a pre-impregnated E-glass/913 epoxy (Hexcel, UK) using the manufacturer's recommended curing cycle (60 min at 125ºC and 700kN/mm^2, heating rate 2-8ºC/min). The panels were manufactured by hand lay-up. The pre-cured flange $[-45_2/0_2/45_2/90_2]_S$ was cut to size and the edges chamfered to 20º. The skin $[-45_2/0_2/45_2/90_2]_S$ was laid up and assembled with the flange and cured. Prior to cutting the specimens to size, glass fibre end tabs were bonded.

The plies containing vascules had sections removed and stainless steel wires (diameter 0.56mm) coated with a release agent were placed between these cut-outs according to the manufacturing method proposed by *Norris et al.* [12].

The selected interleave material was a nominal 50 µm thick polyethylene-co-methacrylic acid (EMAA) film. The interleaves were cut to 3mm strips from a compression moulded sheet and

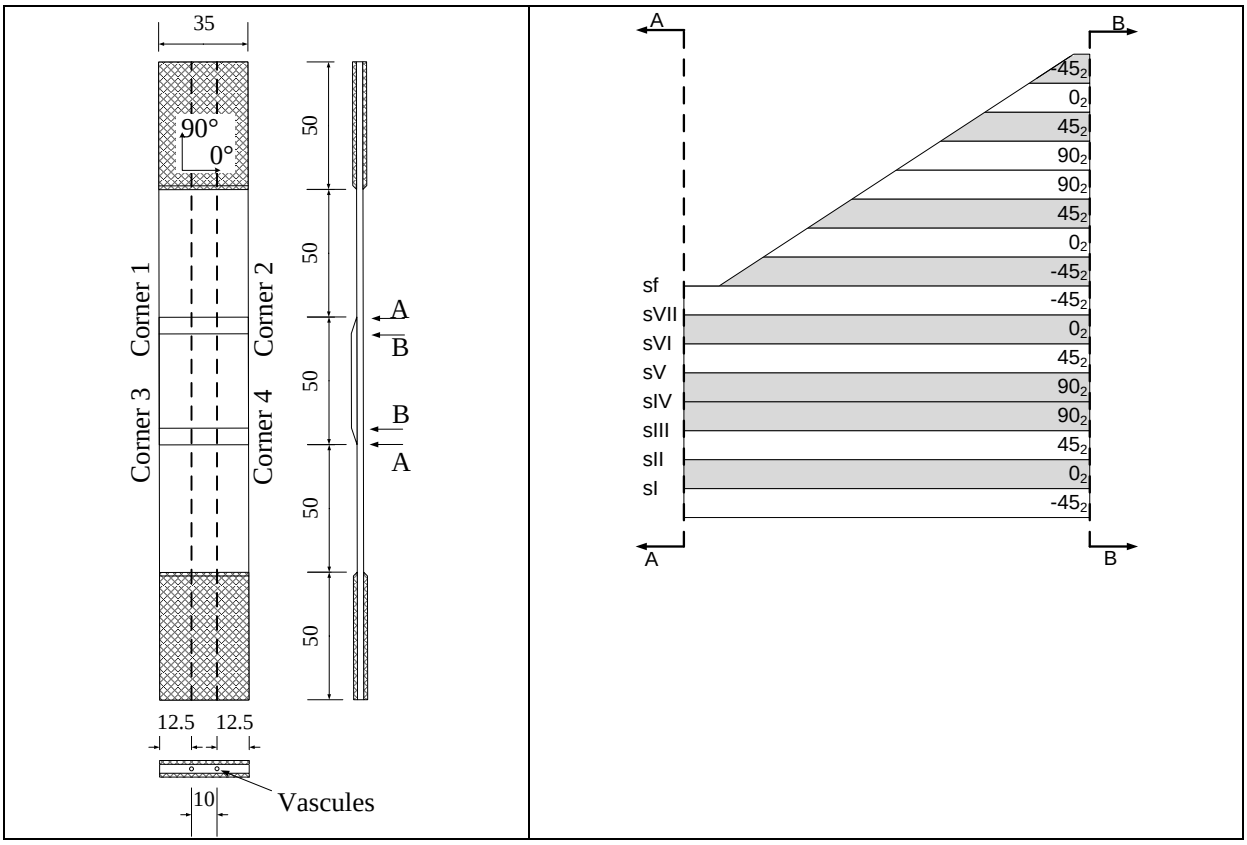

Figure 1. Left side: Specimen geometry and corner identification. Right side: Indentification of the interfaces.

Table 1 summarizes the different test configurations.

ID	Vascules	Interleave interfaces
A	Baseline	-
B	Longitudinal in 90_4	sf, sVII, sVI
C	Longitudinal in 90_4	sf, sVII, sVI staggered
D	Longitudinal in $45_2/90_2$	sf, sVII
E	Transverse in $-45_2/0_2$	sV

Table 1. Test configurations. Interleave interfaces defined in Figure 1**.**

located at ply interfaces. The sheet is hot formed from fused pellets (Sigma Aldrich Co.LLC). The composition of the copolymer is 15 wt% of methacrylic acid

Two different damage control mechanisms were studied, the first of which (ID B, ID C and ID D) aims to control the interfaces along which the delaminations propagate through interleaves, whereas in the case of ID E, the delaminations are redirected through the vasculature. In the latter case, the interleave was intended to slow the rate of delamination propagation.

2.2 Testing procedure

A Schenck Hydropuls® PSA universal testing machine, equipped with a calibrated load cell of 100 kN range, was used for the static and fatigue tests.

Static tests to failure were performed in accordance with ASTM D3039 – test rate 2mm/min. The stiffness was determined using the secant of the load-displacement curve normalized by the skin cross section as shown in equation (1).

$$E = \frac{F_2 - F_1}{d_2 - d_1} \cdot \frac{1}{w_{skin} \cdot t_{skin}} \quad (1)$$

F_1 and F_2 are the two load levels for the determination of the secant modulus and d_1 and d_2 the respective crosshead displacements. W_{skin} and t_{skin}refer to the width and thickness of the skin.

The dynamic tests were performed in accordance with ASTM D3479 (frequency 2Hz, R=0.1). During fatigue testing, the load displacement was measured every 100 cycles. Based on these data, the stiffness of the cycle was determined by the slope of the load displacement curve. The damage accumulation and healing performance was assessed as stiffness loss as a function of cycles.

A commercially available low viscosity resin (Resintech RT152) [7] was used as healing agent. In order to assess the healing performance, the specimens were cycled to a certain number of cycles, the resin was injected into the vascules, cured according to the manufacture instructions, and cycling was continued.

3 Results and discussion

Figure 2 shows a typical tensile test curve and the stiffness as function of cycles for the different configurations.

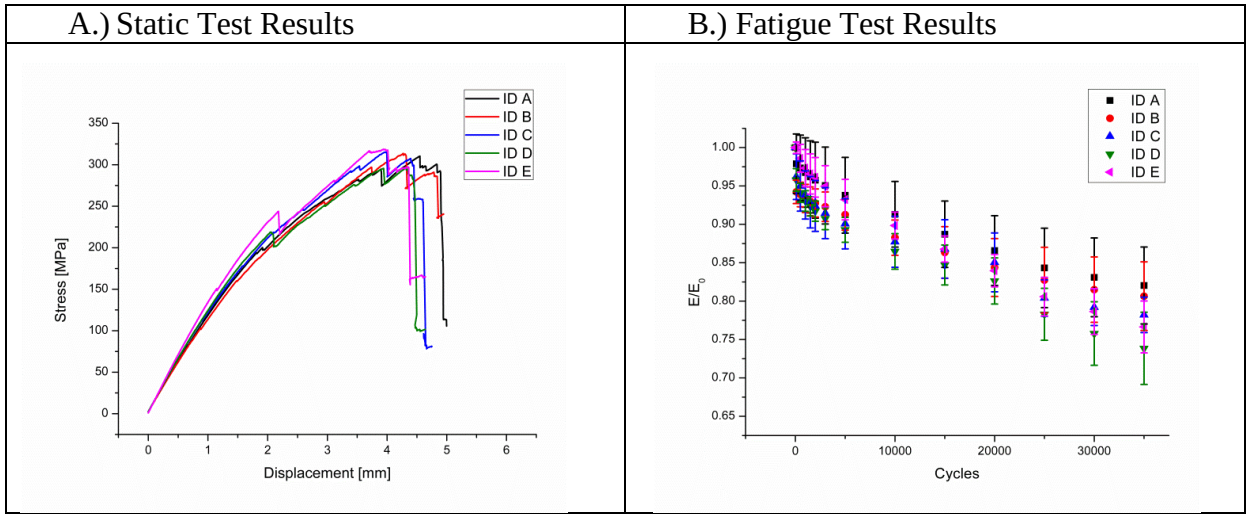

Figure 2. Left: Typical static curve for each configuration. Stress referred to skin cross section. Right: Stiffness decay as function of cycles. Error bars correspond to one standard deviation.

3.1 Static results

Two different damage mechanisms are observed: (1) Transverse failure in the non-load bearing plies and (2) delaminations in the skin of the flange area. The propagation of the delaminations leads to the ultimate failure of the specimens. These delaminations initiate at the tips of the flange and under mixed mode loading conditions propagate inwards along several interfaces. Since the stiffness decreases with increasing load and some load discontinuities are present prior to reaching the maximum load (Figure 2 A), the load-displacement graphs also indicate the progressive nature of the damage. Visual inspection

after testing revealed that the delaminations were arrested at the vascules. The introduction of the interleaves and the vascules does not alter the overall mechanical performance (Figure 2 A). This is important for the design, since any modification to redirect and heal the damage, should not decrease the initial properties.

3.2 Fatigue results

Fatigue tests were performed at approx. 40% of the ultimate tensile strength (F_{max}=10 kN) for all specimens. Figure 2 B presents the mean values of the progressive stiffness loss as a function of cycles

Two different regimes can be distinguished in the stiffness decay: (1) a rapid decay during the first 1000 cycles is followed by (2) a nearly linear one.

The initial steep stiffness decay is due to the presence of resin pockets at the tip of the flange, where the damage is initiated. This damage propagates as delaminations which reduce the stiffness.

The stiffness decay in the different types of specimen is similar to that of the baseline, except for configuration D. In the case of configuration D, the specimens are nearly "unzipped" along the interfaces sIV (90/90) and sV(90/45) where the vascules are present.

In all configurations, damage initiates at the resin pockets at the corners 1 and 4 (defined in Figure 1). In the case of corners 2 and 3, the damage starts due to ply splitting of the top -45_2 ply at 5 to 10 mm in front of the flange tip. These delaminations then migrate "downwards" along interfaces of different orientations. Figure 3 shows the damage pattern of the different configurations.

The damage initiates at the resin pockets because of transverse matrix cracking caused by the stress concentration at the flange tip. In the case where the damage initiates in front of the tip, it is due to damage accumulation in the -45 layers. This layer is not oriented in the loading direction and, therefore, fibre/matrix debonding and matrix cracking lead to transverse failure of the -45 layer. In both cases, the damage propagates as delaminations, which are subject to a mixed-mode loading condition [13] . These delaminations propagate in the resin rich interply zone and are subject to two concurrent damage propagation modes: (1) the delaminations propagate along the interply zone by coalescence of matrix voids created by tensile loading; or (2) the delaminations migrate to lower ply interfaces by ply splitting. However, this migration is arrested when the delamination reaches a ply oriented in the loading direction (90° layer), as further migration would cause fibre breakage or large scale plastic deformation

[14]. These mechanisms are enhanced by the translation of the load path from the skin into the flange.

Through optical microscopy, it has been concluded that when encountering an interleave (ID B, C and D), the delaminations migrate to a lower interface. This may suggest, that the local increment of the fracture toughness [11] enhances the "inherent" tendency of delaminations to migrate into lower plies.

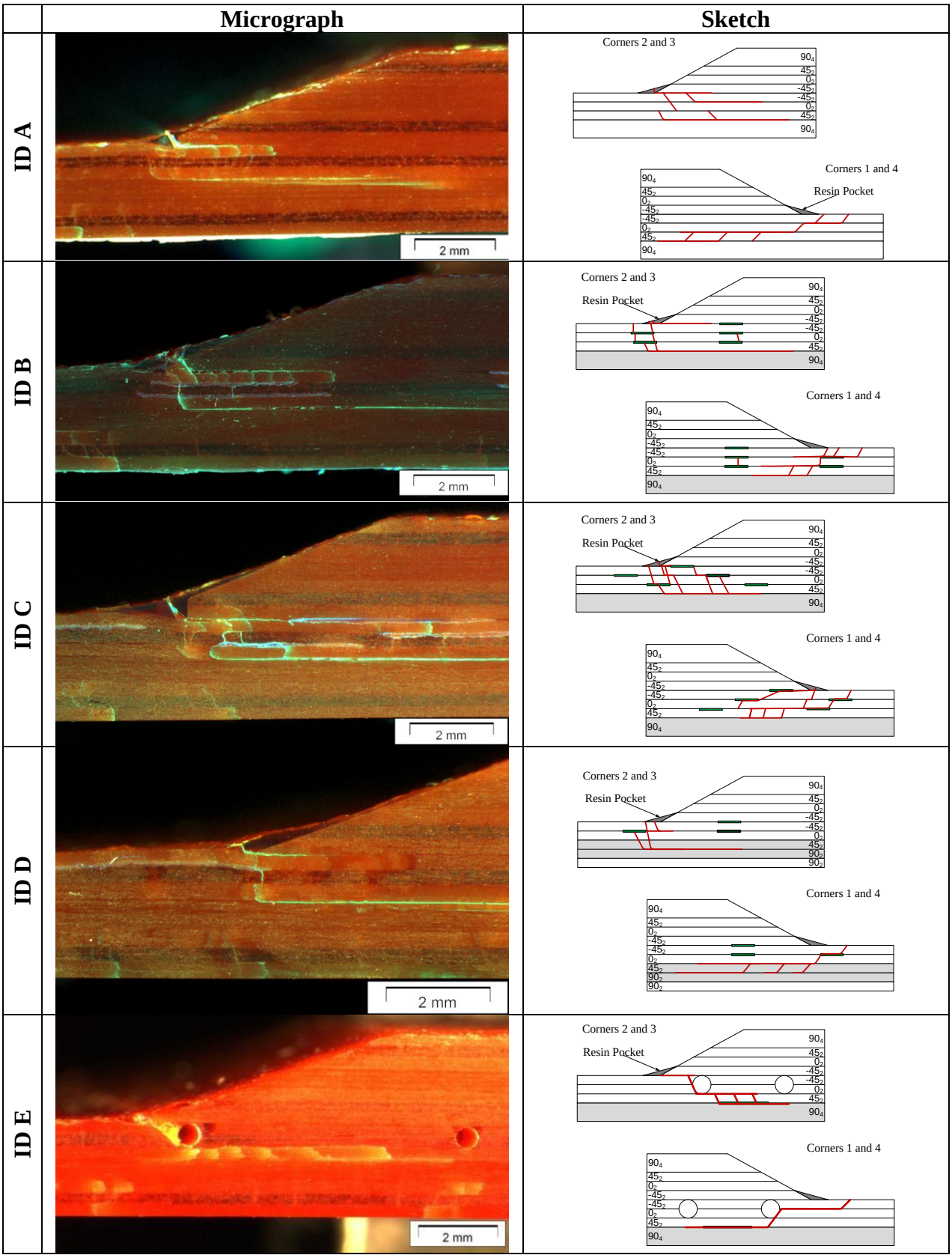

Figure 3. Damage pattern after fatigue damage. Left: micrographs. Right: sketches showing the different types of damage present

Greenhalgh et al. [14] explained this migration mechanism for a mixed mode bending test, which also applies in the case of the skin/flange debond specimens. The delaminations are "forced" to migrate into lower ply interfaces due to the resolved tensile stresses.

Norris et al. [12] reported that transverse vascules (ID E) arrest delaminations under static conditions. It is assumed that this also happens under fatigue conditions. The transverse vascules act similar to drilled holes used in metallic structures, first they arrest the delaminations and then redirect the delamination according to the resolved tensile stress.

3.3 Healing

Preliminary healing tests have been performed on the specimens (Figure 4).

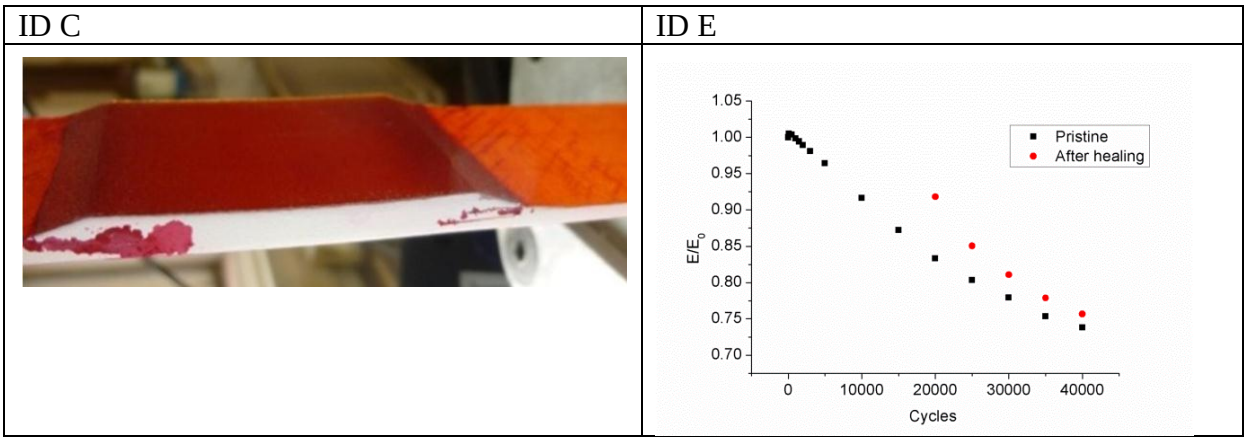

Figure 4. Left: "bleeding" composite showing connectivity between longitudinal vascules and damage site. Right: Stiffness recovery after injection. Specimen cycled to 20000 cycles, then healing agent injected through vascules and cured. Mean value of two specimens per configuration.

In order to show the connectivity between the vascules and the damage sites, a red dye penetrant was injected into the vascules of the specimens redirecting the damage through the interleaves (ID B,C and D). During fatigue testing, it was observed that the specimens started to "bleed" thereby demonstrating the connectivity between the damage sites and the vascules.

For the case of ID E, the commercially available low viscosity resin RT152 (Resintech) was injected in the vascules. A stiffness recovery from 83% to 92% of the initial stiffness was observed. Nevertheless, the mechanical properties decayed rapidly after healing due to the lack of toughness of the resin system used. An alternative 'tougher' system, which still meets all the requirements of an effective healing agent is currently being sought.

4 Conclusions

In this research, we have studied the damage growth in skin-stiffener debond specimens. The internal structure of the baseline configurations has been altered by discrete interleaving and the introduction of vascules, without substantially changing the global mechanical performance under static and dynamic conditions.

The delaminations paths have been steered successfully by the interleaves and vascules into the interfaces with vascules, thereby assuring the connectivity between the damage and the vascules.

The healing provided partial recovery of the mechanical performance of the structural element.

5 Acknowledgments

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6 References

[1] J. W. H. Yap, M. L. Scott, R. S. Thomson, and D. Hachenberg. The analysis of skin-to-stiffener debonding in composite aerospace structures, *Compos. Struct.*, 57(1–4): 425–435.

[2] B. J. Blaiszik, S. L. B. Kramer, S. C. Olugebefola, J. S. Moore, N. R. Sottos, and S. R. White. Self-Healing Polymers and Composites, *Annu. Rev. Mater. Res.*, 40(1): 179–211, 2010.

[3] T. Yang, J. Zhang, A. P. Mouritz, and C. H. Wang. Healing of carbon fibre–epoxy composite T-joints using mendable polymer fibre stitching, *Compos. Part B Eng.*, 45(1): 1499–1507, 2013.

[4] T. K. O'Brien and S. R. White. Assesment of composite delamination self-healing via micro-encapsulation. in *American Society for Proceedings: Composites 23rd Technical Conference*, 2008.

[5] R. S. Trask and I. P. Bond. Biomimetic self-healing of advanced composite structures using hollow glass fibres. *Smart Mater. Struct.*, 15(3): 704–710, 2006.

[6] J. W. C. Pang and I. P. Bond. 'Bleeding composites'—damage detection and self-repair using a biomimetic approach. *Compos. Part A Appl. Sci. Manuf.*, 36(2):183–188, 2005.

[7] C. J. Norris, J. A P. White, G. McCombe, P. Chatterjee, I. P. Bond, and R. S. Trask. Autonomous stimulus triggered self-healing in smart structural composites. *Smart Mater. Struct.*,21(9):1–10, 2012.

[8] C. J. Norris, G. J. Meadway, M. J. O'Sullivan, I. P. Bond, and R. S. Trask. Self-Healing Fibre Reinforced Composites via a Bioinspired Vasculature. *Adv. Funct. Mater.*, 21(19):3624–3633, 2011.

[9] A. Kousourakis, A. P. Mouritz, and M. K. Bannister. Interlaminar properties of polymer laminates containing internal sensor cavities. *Compos. Struct.*, 75(1–4): 610–618, 2006.

[10] R. S. Trask and I. P. Bond. Bioinspired engineering study of Plantae vascules for self-healing composite structures. *J. R. Soc. Interface*, 7(47):921–931, 2010.

[11] M. Yasaee, I. P. Bond, R. S. Trask, and E. S. Greenhalgh. "Damage control using discrete thermoplastic film inserts. *Compos. Part A Appl. Sci. Manuf.*, 43(6): 978–989, 2012.

[12] C. J. Norris, I. P. Bond, and R. S. Trask. The role of embedded bioinspired vasculature on damage formation in self-healing carbon fibre reinforced composites. *Compos. Part A Appl. Sci. Manuf.*, 42(6):639–648, 2011.

[13] P. J. Minguet and T. K. O'Brien, Analysis of Test Methods for characterizing skin/stringer debonding failures in reinforced composite panels. In *Composite Materials: Testing and Design (Twelfth volume)*:105-124. ASTM STP,1996.

[14] S. Singh and E. S. Greenhalgh. Micromechanics of interlaminar fracture in carbon fibre reinforced plastics at multidirectional ply interfaces under static and cyclic loading. *Plast. rubber Compos. Process. Appl.* 27(5): 220–226, 1998.