FATIGUE OF Ti-6Al-4V WITH A WIDMANSTATTEN MICROSTRUCTURE

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ABSTRACT

Fatigue crack initiation in an aligned structure of Ti-6Al-4V was investigated. Slip line cracking in the α phase was shown to be responsible for failure when surface origins dominated. It is suggested from fractographic and metallographic evidence that the same initiation mechanism produced internal origins during high cycle fatigue. The presence of an interface layer in the structure separating the α and β plates did not play a significant role in crack initiation. The local misorientation of adjacent α plates over an intercolony boundary was found to be the controlling factor.

Facets were noted on fracture surfaces whether failure initiated from a surface or a subsurface origin. No correlation between the exact location of the initiation site and facets was found.

KEYWORDS

Fatigue; titanium alloys; slip lines; internal origins; facets.

INTRODUCTION

The initiation mechanism of fatigue cracks, both internal and on the surface, which are responsible for failure during smooth specimen fatigue of titanium alloys is not clear. The applied stress and strain amplitude was reported to be a deciding factor of the operative mechanism of surface origins (MacDonald and Wood 1973). Slip line associated cracks and α/β interface cracks were reported when the cyclic stress and strain levels were above the static elastic values (Stubbington and Bowen 1974) while slipless cracking initiated cracks at levels below the static elastic range (Steel and McEvily 1976). The mechanisms of internal crack nucleation range from time dependent deformation (Harrison, Evans and Weaver 1979), cleavage of the α phase (Neal and Blenkinsop 1976) and the presence of internal defects (Eylon and Hall 1977). Recently the effect on crack initiation of an interface layer between the α and β phases in aligned structures has been questioned (Hallam and Hammond 1979).

The objective of this research was to identify crack initiation mechanisms in a Widmanstatten structure of Ti-6Al-4V over a large range of applied stresses and strains where several different mechanisms may have been responsible for crack

initiation, both surface and subsurface.

EXPERIMENTAL PROCEDURE

Ti-6Al-4V (IMI318) in thin sheet form, 1.58mm thick, and in rod form, 3.14mm in diameter, was heat treated for 3 hours at 1100°C followed by a furnace cool. A vacuum of better than 10^{-4} Torr was maintained throughout the heat treatment. A microstructure of aligned α and β plates in discrete colonies was produced. The colony size varied from an individual α plate up to colonies containing around 50 α plates. The prior β grain size was ~2mm.

Fatigue specimens were machined from heat treated blanks. Final specimen preparation before testing consisted of longitudinal grinding on SiC papers up to 600 grit followed by electropolishing in 94% glacial acetic acid and 6% perchloric acid at 15V.

Zero minimum direct strain fatigue tests on as-polished material were carried out using a sawtooth wave form at 1.8cpm on a Mand Servohydraulic fatigue testing machine. The specimens had a circular cross section with a diameter of 5.05mm and a parallel gauge length of 10mm. Stress controlled tests below the monotonic elastic limit were carried out on an Amsler Vibraphore fatigue machine at a frequency of 140Hz using thin sheet specimens which had an hourglass, waisted gauge length 12.7mm wide at the narrowest point and 1.5mm thick. The sheet specimens were etched prior to testing in 2%Hf, 10%HNO_3 and 88%H_2O so microstructural detail was present on the fatigued specimen surface. A replication technique (Brown 1980) was used to identify the crack which was responsible for failure during stress controlled testing. When internal origins occurred, no surface cracks were found on the replicas of specimen surfaces at 90% of the life. Tensile testing was carried out on electropolished thin sheet specimens using an Instron testing machine at strain rates comparable with strain controlled fatigue tests. Post fatigue metallographic sections of strain controlled specimens were prepared by longitudinal spark erosion cutting parallel to the gauge length and strain axis. The sections were ground on SiC papers up to 600 grit, then polished in slurries of 1μm and 0.25μm Al_2O_3, followed by etching in the same etchant as previously used in fatigue specimen preparation. Replicas, fracture surfaces and metallographic sections were examined in a Cambridge Instruments mark 2A SEM.

RESULTS

Crack initiation was associated with the boundary between colonies of differently oriented α plates during strain controlled fatigue when the peak tensile stress was above the monotonic tensile elastic limit of $1048Nm^{-2}$ for the alloy. Figure 1 indicates a crack at A and much slip activity in the region of the intercolony boundary. This was typical of many cracks which appeared on the specimen surface. Below the surface the cracks were not associated with intercolony boundaries, Fig. 2. After longitudinal sectioning of fatigued specimens, internal fatigue damage was found at intercolony boundaries, Fig. 3. Numerous areas of this type were found after fatigue, however unstressed material did not exhibit any internal damage after metallographic preparation in exactly the same manner.

The density of surface slip and cracks decreased and very few cracks were found when stress controlled tests were carried out when the peak cyclic tensile stress was below the monotonic elastic limit. However, intercolony boundaries still played an important part. The majority of slip was associated with these boundaries as were cracks, Fig. 4. A systematic study indicated that individual slip lines were responsible for crack initiation in this regime. A typical fatigue crack initiation process is shown in Fig. 5. After 3% of the life a slip line appeared in the α plate adjacent to a β plate that was also the intercolony

boundary. It was still present at 10.5% of the life, marked A, Fig. 5a. The slip line transformed into a crack in the α phase near the β plate after 20% of the life marked B, Fig. 5b. Early growth was accommodated by cracking along the slip line marked C, Fig. 5c and by α/β interface failure, marked D. The effectiveness of the β plates in retarding full crack tip opening is shown in Fig. 5d, when β plates remained intact, marked E, even though the intervening α plates had failed. This behavior was a constant feature of very early crack growth. Crack branching on a steplike manner occurred at 56% of the life, marked F, Fig. 5e. α/β interface failure and α plate failure normal to the plane of the α plates produced the steps. This failure morphology was reflected on the fracture surface after failure, marked G, Fig. 5f.

Facets, marked H, Fig. 6, did occur on the fracture surface of failures from surface initiated cracks. The origin in this case was at the position marked J. No specific topographical features identified the surface crack origin in this structure. Fatigue striations were the mechanism of Stage II crack growth. At long crack lengths near the onset of tensile failure, discrete areas of striations occurred mixed with tensile dimples, Fig. 7.

Internal origins were found at lives $>10^6$ cycles, Fig. 8. They exhibited all the features of a surface origin fracture surface with facets, stepped regions, striations and mixed striations and tensile failure. Facets were produced on this surface by crack growth normal to the plane of the α plates, Fig. 9. The initiation site was not characterized by any specific feature.

DISCUSSION

Surface crack initiation at cyclic stress ranges below the monotonic elastic limit during stress controlled fatigue tests occurred at an individual slip line in the α phase. The slip line intersected with a β plate that constituted an intercolony boundary. No other crack initiation mechanism was found in this regime. At high cyclic stresses investigated by strain controlled tests, the location of fatigue cracks was the same as the lower cyclic stress case. However, the increased peak tensile stresses during strain controlled testing induced substantially more slip in the area of the intercolony boundaries, typical of ductile material fatigue crack initiation. It is suggested that the same initiation process occurred in strain controlled, low cycle fatigue as at lower stress, high cycle fatigue. Cracks produced solely by α/β interface separation were not found during initiation. The only interface cracks which occurred in either stress regime were those necessary for accommodation of very early crack growth, after initiation of a crack in the α phase on a slip line.

Intercolony boundaries were a constant feature of surface initiation at both high and low cyclic stresses and were also the location of internal fatigue damage. Each colony represents a number of similarly aligned α plates present within a large prior β grain. Therefore, each individual colony can be thought of as a large single grain. Traversing an intercolony boundary is effectively crossing from a single α grain into another single α grain of different orientation. In this situation, slip lines can cross several α plates within a colony but they may find it extremely difficult to cross an intercolony boundary. The misorientation of α plates across an intercolony boundary is responsible for this behavior. Furthermore, if slip cannot be transmitted from one α colony to an adjacent one, a dislocation pileup will occur in the slip line contained in the α phase where it intersects the β plate which is an intercolony boundary. The increased stress due to a dislocation pileup may be enough to cause crack nucleation in the α phase at the head of the dislocation pileup. It is suggested that this is the operative mechanism for surface fatigue crack initiation in this structure.

An interface phase separated the α and β phases in this structure (Brown 1979). Evidence from the present investigation indicated that the local misorientation

between adjacent α plates was more important to crack initiation than the presence of the interface layer. The feature which was an obstacle to slip and ultimately produced cracking was the lack of a slip plane which could be activated in the α phase over an intercolony boundary rather than slip being blocked by the complex interface layer. In fact, within a colony slip occurred across several α plates without cracks or void formation which implied that the interfacial layer had no important effect on initiation. In this case, the stress at the head of a pileup in an α plate activated a parallel slip plane in the next similarly aligned plate. This stopped a stress buildup at the α/β interfaces within a colony which may have been sufficient to provide crack nucleation. At an intercolony boundary where cracks did initiate, no slip planes were activated in the colony adjacent to the one in which cracks nucleated. No stress relaxation was possible in this situation by slip. Crack nucleation was the method of relaxation.

Facets have previously been employed to identify the exact initiation site in titanium alloys (Harrison, Evans, and Weaver 1979). In this structure it is clearly not possible to do this. Facets appeared some way from the initiation site when surface origins were responsible for failure. The usual mechanism of facet production in the aligned structures was crack growth normal to the plane of the α plates, Fig. 9. None of the surface origins shown here could exhibit facets at the exact crack origin by this mechanism as newly initiated cracks which produced failure did not initially grow normal to the α plates. No specific topographical features appeared at the origin to differentiate it from the remainder of the early crack growth area.

Internal fatigue damage was found during high stress testing, evidence that internal dislocation processes occurred which would produce crack nucleii. The damage was identical to surface damage, and was present at the same location, intercolony boundaries. The fracture surface of an internal origin, apart from the site of the origin, was identical to that produced from a surface origin. It is, therefore, suggested that the same mechanism produced crack initiation in this structure irrespective of the location of the origin. Namely, slip lines in the α phase which intersected with an intercolony boundary was thought to be responsible for internal initiation.

An interesting feature of the later stages of crack growth was the mixed striation and dimples mode of failure. It demonstrated the highly anisotropic nature of crack growth behavior of the aligned structure of titanium alloys previously reported by Bowen (1970), and supported the view that colonies of similarly aligned α plates could be regarded as single α grains with the β phase having a small effect on crack growth. Throughout the investigation the β phase did not actively deter cracking of the α phase and it appeared to have little strengthening effect in terms of crack blocking, although the β phase had several roles affecting crack growth. It provided interfaces for very early interfacial crack growth during initiation when slip line growth was impossible and for the steplike fracture morphology. The latter process lengthened the crack path by permitting branching and retarded the growth rate. The β phase was intact at a crack tip during early slip line growth which prevented full crack opening, and the structure in this condition, although containing a crack in the α phase would still be load bearing. This would also retard crack growth.

CONCLUSIONS

(1) Slip lines in the α phase were responsible for surface crack initiation of Ti-6Al-4V.
(2) No correlation between facets and surface origin sites was found.
(3) It is suggested that internal crack nucleation was by the same process as surface initiated fatigue cracks.

(4) The interface phase did not play a significant role in crack initiation. The misorientation of α plates between adjacent colonies was the controlling microstructural factor.

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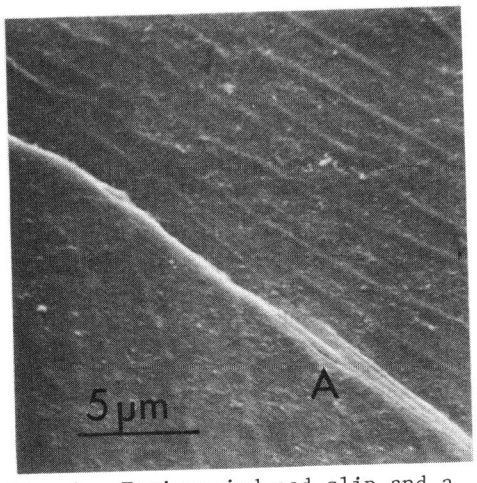

Fig. 1. Fatigue induced slip and a crack at A in the region of an intercolony boundary during low cycle fatigue.

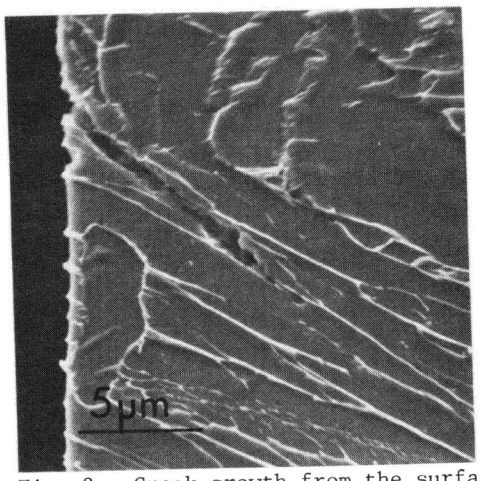

Fig. 2. Crack growth from the surface into the interior during low cycle fatigue. The crack ignored both the intercolony boundary and a growth direction normal to the plane of the α plates.

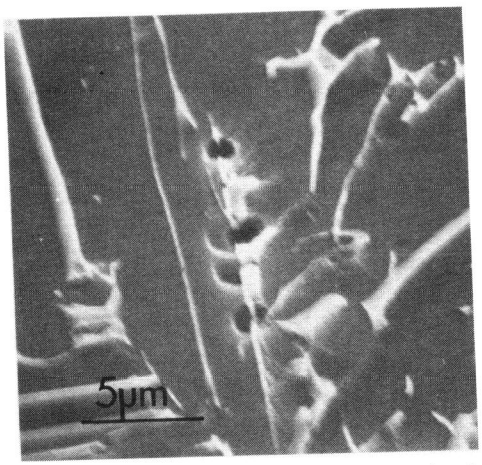

Fig. 3. Internal fatigue damage during low cycle fatigue located at an intercolony boundary.

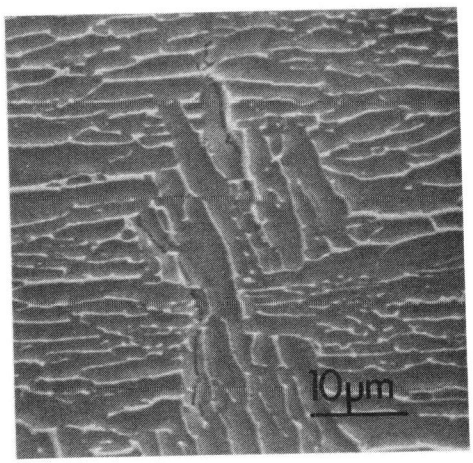

Fig. 4. Surface cracks at an intercolony boundary induced by stress controlled fatigue.

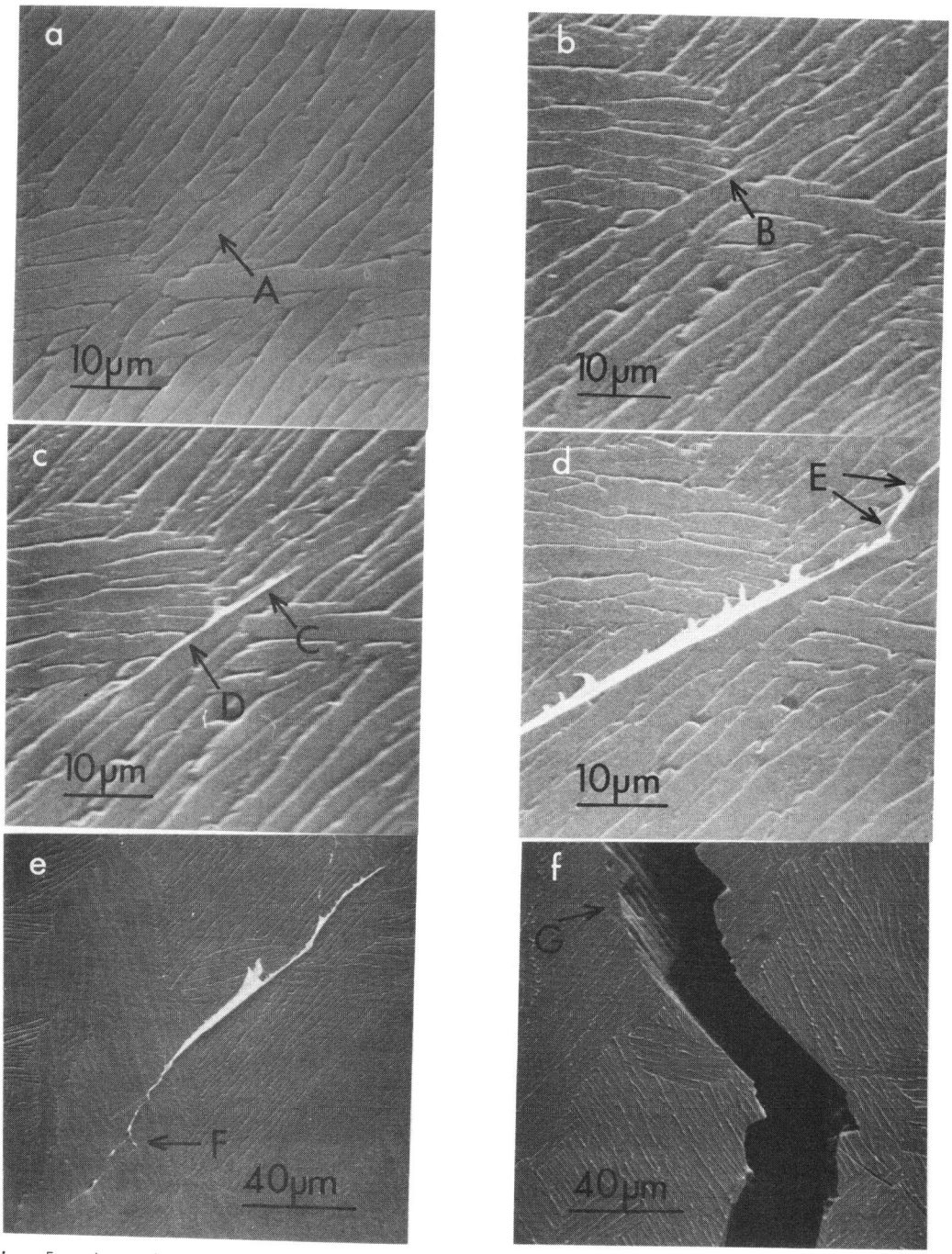

Fig. 5. A series of scanning electron micrographs following the crack initiation process during stress controlled fatigue tests; a) 10.5% of the life, b) 20%, c) 33%, d) 46%, e) 56%, f) 100%,

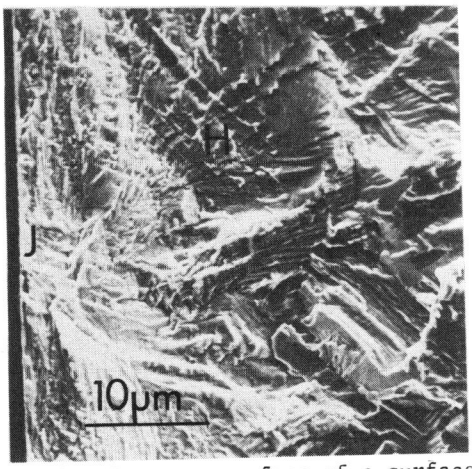

Fig. 6. Fracture surface of a surface initiated fatigue failure.

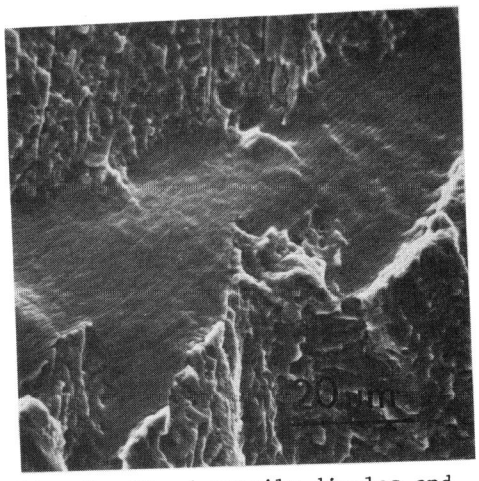

Fig. 7. Mixed tensile dimples and striation mode of crack growth at the late stage of crack growth.

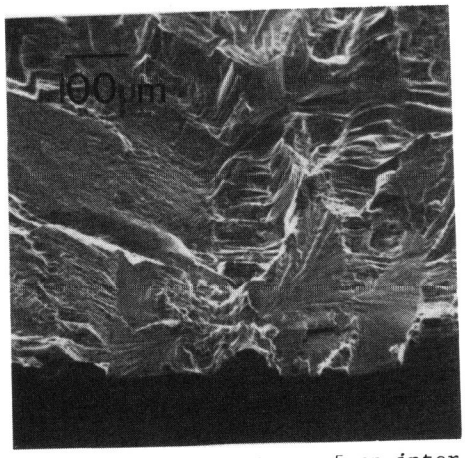

Fig. 8. Fracture surface of an internal initiation fatigue failure.

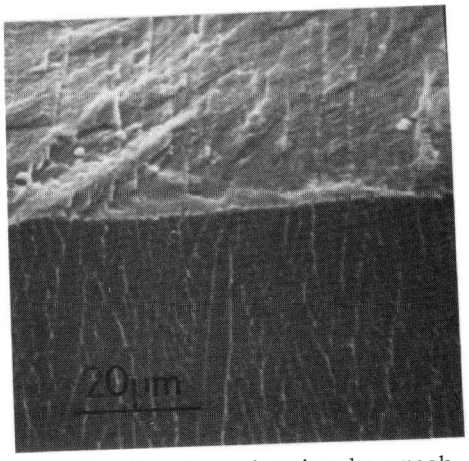

Fig. 9. Facet production by crack growth normal to the plane of the α plates.