1-7-2002

A theoretical model for the simulation of microdamage, repair and adaptation in compact bone.

David Taylor
Trinity College Dublin

Fergal J. O'Brien
Royal College of Surgeons in Ireland, fjobrien@rcsi.ie

T Clive Lee
Royal College of Surgeons in Ireland

Citation

Taylor D, O'Brien FJ, TC Lee. A theoretical model for the simulation of microdamage, repair and adaptation in compact bone. Meccanica. 2002; 37(4-5)397-406.

AIMETA '01
XV Congresso AIMETA di Meccanica Teorica e Applicata
15th AIMETA Congress of Theoretical and Applied Mechanics

A THEORETICAL MODEL FOR THE SIMULATION OF MICRODAMAGE, REPAIR AND ADAPTATION IN COMPACT BONE

D.TAYLOR[1], F.O'BRIEN[1,2], T.C.LEE[2]

[1] *Department of Mechanical Engineering, Trinity College, Dublin, Ireland.*
[2]*Department of Anatomy, Royal College of Surgeons in Ireland, Dublin, Ireland.*

ABSTRACT

This paper describes a new theoretical approach to bone microdamage, in which a population of cracks is explicitly modelled. A given sample of bone is assumed to contain a certain number of cracks, whose growth characteristics are described with an equation containing stochastic variables to create statistical differences from one crack to another. This type of model allows us to predict a wide variety of data. The present paper illustrates the different types of prediction which can be made, including: (i) standard damage parameters such as the number and length of cracks and the reduction in stiffness; (ii) fatigue test data such as the number of cycles to failure as a function of stress level, including scatter; (iii) effects due to the living system, including repair, remodelling and adaptation. A useful feature of the model is our ability to examine the statistics of the crack population in detail to find, for example, the number of cracks which are potentially dangerous as opposed to those which are dormant, and to investigate the reasons for increased crack numbers in the bones of older people. The potential also exists to use the model to investigate different theories of bone remodelling and adaptation.

1. INTRODUCTION

This paper is concerned with the prediction of damage in compact bone, which is a well-known problem in orthopaedic biomechanics. Damage, in the form of microcracks, is caused by cyclic (fatigue) loading; this can lead to stress fractures and has been linked to the physiological processes of repair, remodelling and adaptation [1,2,3]. We have recently obtained new data on the evolution of damage in test specimens by developing a series of dyes of different colours, enabling us to record the number and length of microcracks at various stages during a fatigue test [4,5]. The experimental details for this technique have been published elsewhere. In parallel we have been developing a theoretical model which attempts to simulate the evolution of damage. A first stage in developing this simulation has been to use it to predict the results of our experimental tests. Our first aim was to predict the number density of cracks as a function of time (expressed as the number of loading cycles) during a

fatigue test on a standard specimen. The advantage of a model of this kind is in the variety of results that it can predict, and in the fact that it can be extended relatively easily to incorporate features such as repair and remodelling. In the present paper we try to illustrate this by showing the various types of predictions which can be made.

2. THE THEORETICAL MODEL

The simulation has the following features:

a) Each crack in the specimen is modelled explicity. A typical test specimen will develop about 200 detectable cracks before failure.

b) Cracks are assumed to initiate on the first loading cycle, with an initial length (tip-to-tip in the transverse direction) of 1μm. The number of cracks which form is a function of the maximum stress in the cycle. No time is allowed for the initiation process. This is in line with current thinking on fatigue in other materials, which assumes that the amount of time needed to initiate cracks is small compared to the time taken for propagation through microstructural barriers (see below).

c) Crack growth is governed by the following equation:

$$da/dN = C(\Delta K - \Delta K_{th})^n + C'(\Delta K)^{n'}((d-a)/d)^m \quad (1)$$

...where da/dN is the rate of growth (in mm/cycle), ΔK is the stress intensity range which is related to the cyclic stress range, $\Delta\sigma$ and the crack length, $2a$, through the standard fracture mechanics equation:

$$\Delta K = F \Delta\sigma (\pi a)^{1/2} \quad (2)$$

F is a constant which depends on the geometry of the crack. The first term in eqn.1 describes the growth of the crack when it is relatively long; the second term describes short-crack growth, which is dominated by the presence of a barrier at a distance d from the initiation point. In practice cracks are slowed down (and often stopped) at the cement lines surrounding secondary osteons, so d represents the spacing of osteons. Each term is set to zero if it becomes negative. Values for the constants F, C, C', n, n', m and d have already been deduced in previous studies; the long-crack threshold ΔK_{th} has not been measured experimentally and so had to be estimated. The type of growth-rate curve produced by equation 1 is illustrated schematically in fig.1; a minimum in growth occurs when the crack reaches the barrier (a=d).

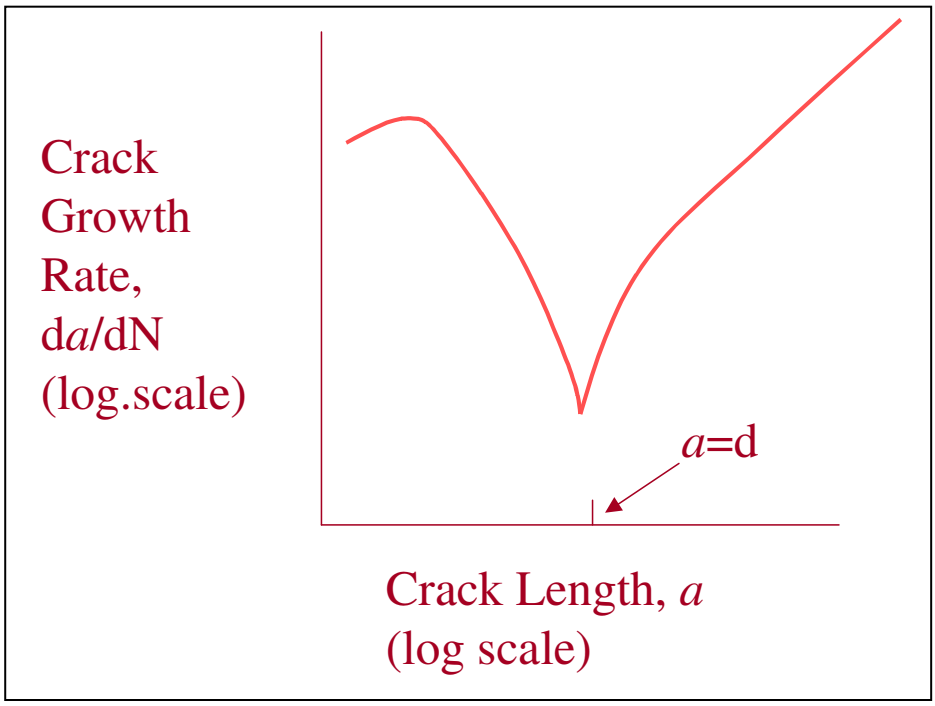
d) Each crack behaves differently in the simulation because the values of certain constants (C', d and ΔK_{th}) are allowed to vary stochastically, creating scatter which can be used in a Monte Carlo simulation of a fatigue test on a given specimen, containing a given number of cracks. Table 1 shows the values used for the various constants and their standard deviations.

e) Failure will occur when one crack reaches a sufficiently large length, set to 1mm. Cracks become detectable by our experimental technique if they are longer than 30μm, so estimates

of measured crack density must take this limit into account.

Parameter	Mean Value	Distribution	Std.dev./mean
C	3.46×10^{-7} (see note 1)	constant	
C'	1.51×10^{-4} (see note 1)	log-normal	0.05
ΔK_{th}	1.0 MPa(m)$^{1/2}$	normal	0.3
n	4.5	constant	
n'	5	constant	
m	3	constant	
d	57μm	normal	0.5
F	1.22	constant	

Table 1: Values used for the various constants, including statistical distributions



Note 1: Units for C and C' are such that da/dN is given in mm/cycle with ΔK in MPa(m)$^{1/2}$

Fig.1: Schematic illustration of the variation of crack growth rate with crack length according to equation 1.

3. RESULTS AND DISCUSSION

This type of model is capable of making predictions about a wide variety of phenomena. This is illustrated in the following sections; the emphasis here was on demonstrating the potential of the model to simulate different types of results, rather than on the numerical accuracy of the data, which we expect to be able to improve with future refinements. However, experimental data are shown for comparison, where available.

3.1. Crack density changes during a fatigue test

Our first aim was to predict the number density of cracks (i.e. the number of cracks seen per square millimetre on transverse sections), for comparison with new data obtained in our laboratories. The experimental technique, which has been described elsewhere [4,5] allows us to infiltrate coloured dyes into cracks located inside test specimens. This can be done at various stages during the life of a specimen, giving new information about the changing

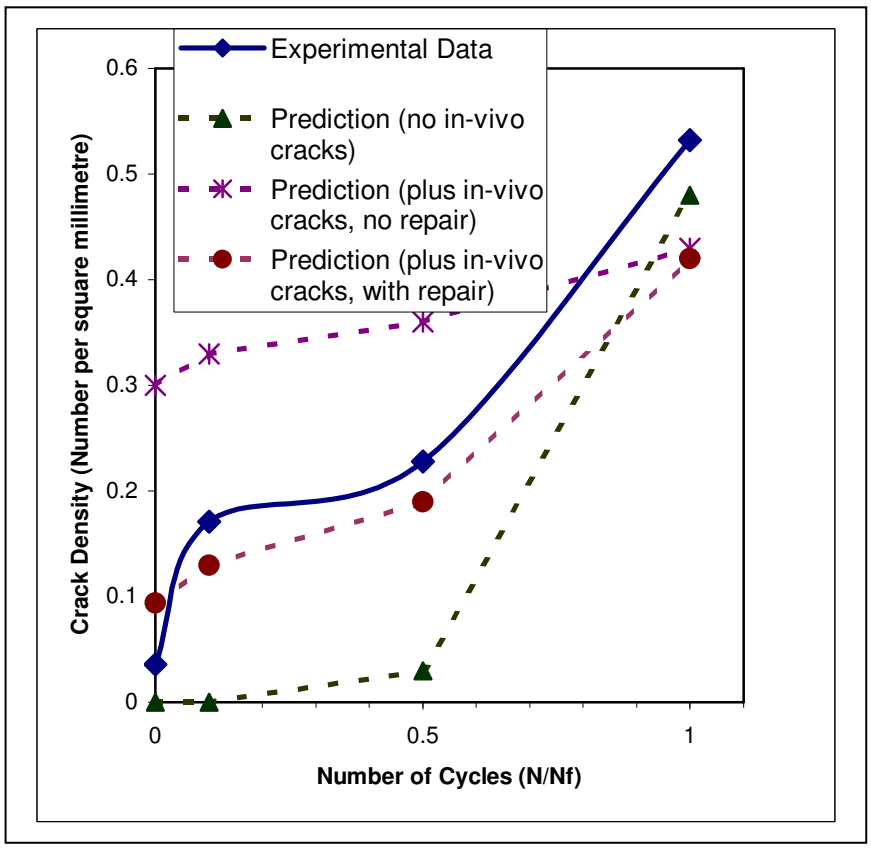
density and length of cracks as a function of number of cycles (N) during a fatigue test. Fig.2 shows the experimental data generated from specimens taken from bovine tibiae, loaded in cyclic compression at a stress range of 80MPa and a stress ratio (min.stress/max.stress) of 0.1.

Fig.2: Variation of crack density with number of cycles during a fatigue test.

The initial prediction (labelled 'no *in-vivo* cracks' on the figure) used a single population of cracks, all starting with an initial length of 1μm. The number of detectable cracks, defined as those greater than 30μm, increases very slowly at first, accelerating towards the end of the test. Such a population of cracks will clearly never give rise to a curve with a central plateau as seen in the experimental data. We assumed that this occurred because the specimens already contain cracks formed *in-vivo*, which would grow to become detectable at an earlier stage in the test. We tried to simulate this *in-vivo* behaviour, cycling a group of cracks at a stress range of 40MPa for 5 million cycles, to represent a few years of normal life. The stress range was then increased to the test value (80MPa) and more cracks added. This simulation improved the general shape of the curve, but was much more successful if we also simulated the selective repair of these *in-vivo* cracks by removing all *in-vivo* cracks longer than 100μm.

Best results were obtained by using 200 cracks/specimen at the *in-vivo* stage and a further 450 cracks at the testing stage. This raises a question about the total number of cracks and how this varies with stress level. In this model we assumed that all cracks are formed on the first loading cycle, that these cracks all have an initial length of 1μm, and that their number density is higher at higher stresses. This approach is clearly a simplification, but it is in line with current thinking on the fatigue of engineering materials, including metals, which holds that the initiation stage is a very small proportion of total life, and that the great majority of time is taken up in growth up to the first microstructural barrier. Given the two densities used here for

40MPa and 80MPa, one can propose a relationship between crack density (ρ) and stress level as follows:



$$\rho = A(\Delta\sigma)^{\alpha} \qquad (3)$$

Here A and α are constants, α=1.69. An equation of this form arises if one assumes that the number of regions in the specimen which fail (and therefore form cracks) depends on stress level according to a Weibull distribution, in which case α is related to the exponent in the Weibull equation. Alternatively one could note that, as a basic principal of fracture mechanics, the total cracked area should be proportional to the energy input and therefore to the stress squared, which would give a value of 2 for α, quite close to the value used here. Equation (3) was used to decide the number density of cracks in all the other simulations described below.

3.2 Stress/Life curves

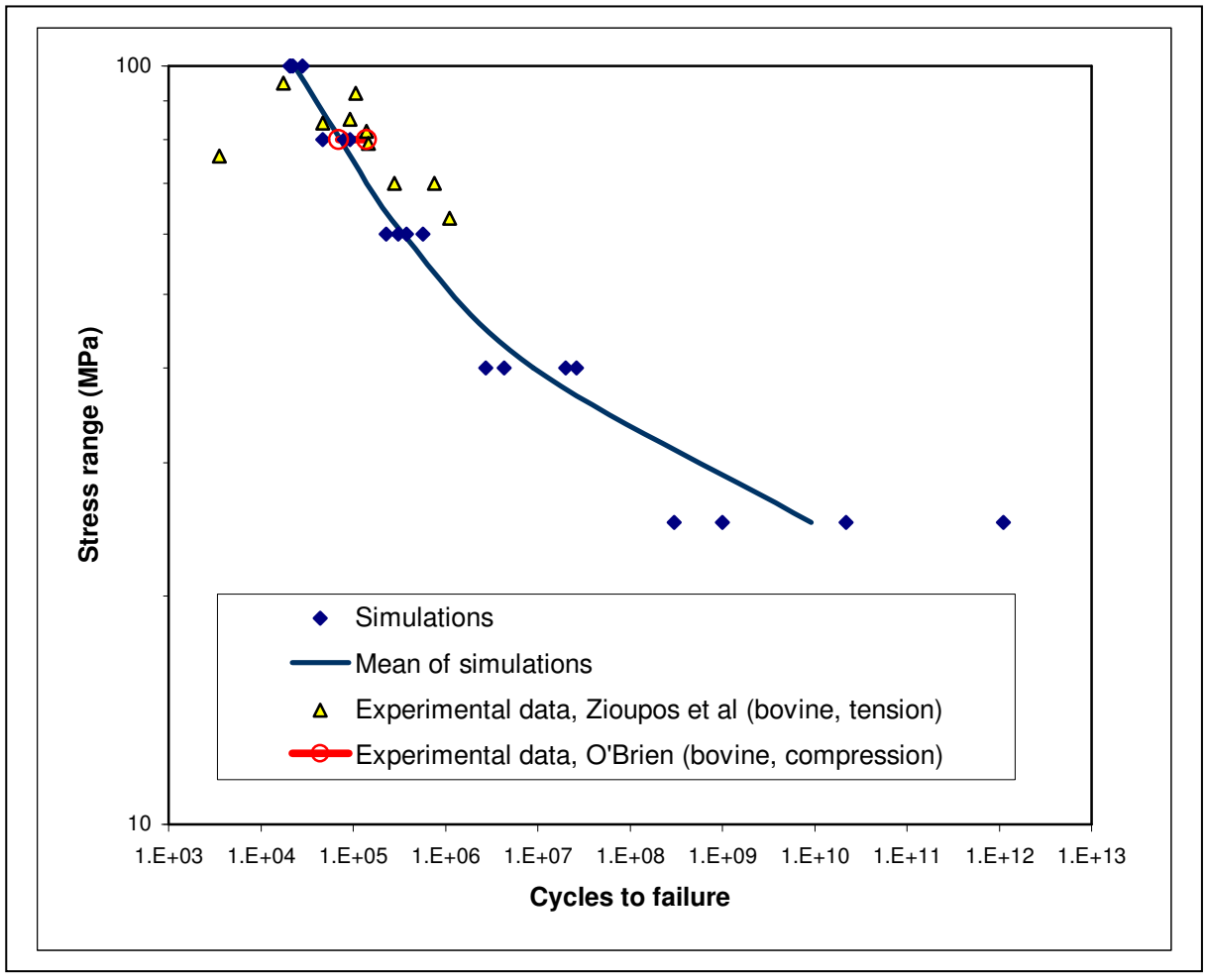
The effect of stress range on number of cycles to failure, N_f, for similar test conditions, can be easily found by allowing the simulation to run until one of the cracks becomes very large. We used a length of 1mm but this is not critical because at that stage the crack is growing very rapidly. As fig.3 shows, this enables us to predict not only the general dependance but also the

Fig.3: Simulated stress/life curve, plus experimental data

amount of scatter in test data. The simulations compare well with experimental data from our own work (O'Brien [4]) and also with data from Zioupos et al [8] who tested bovine bone specimens in cyclic tension. A great advantage of the simulations is that we are able to extend them down to stresses in the physiological range (below 50MPa) at which we expect more scatter and much longer lives, a point which will be returned to below.

3.3 Other damage variables

The model is capable of predicting any other parameters normally used to define damage, such as the lengths of cracks, the crack length density, etc. It is also capable of estimating the



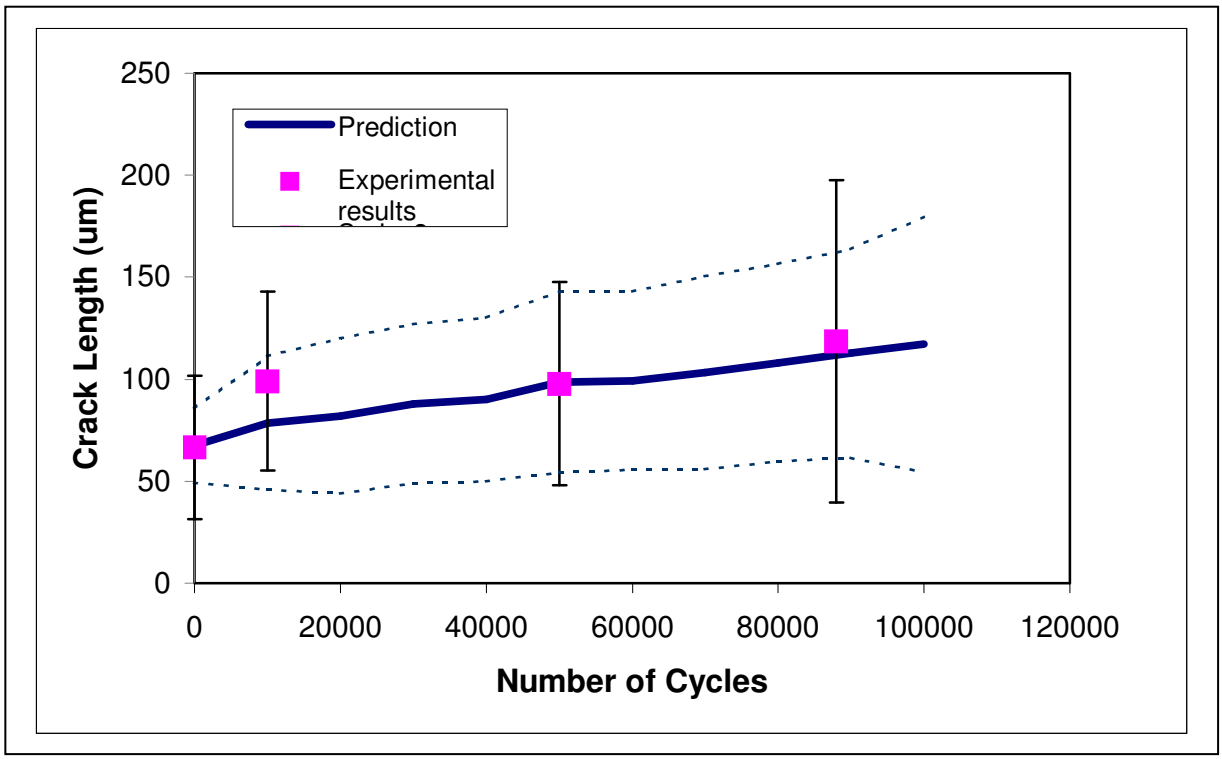
change in stiffness in the specimen by using fracture mechanics theory, since the growth of a crack at a given stress intensity implies a certain change in specimen compliance. This argument has been developed in earlier papers [6,7] and won't be discussed specifically here. We confine ourselves to showing one measure of damage: the mean length of cracks, which is presented in fig.4 as a function of N during a fatigue test at 80MPa. Both the mean value and

the standard deviation agree closely with our experimental data. It is interesting to note that the mean value changes only slightly during the test, a fact which has been recorded experimentally by several workers. This appears to be because most cracks become dormant at microstructural lengths around 100μm (see below).

Fig.4: Crack length variation during a fatigue test: mean and standard deviation

3.4 The effect of stressed volume

The stress/life curve (fig.3) would seem to imply that, at physiological stresses, failure is very unlikely during a person's normal lifetime. Below we represent *in-vivo* conditions by a stress range of 25MPa for 1.64 x 10^7 cycles per year, which implies that failure will take at least ten years and probably much longer. Others have estimated 2 million/year of 'significant' cycles (i.e. cycles of running, stair-climbing etc) at stress levels around 40MPa, which leads to a similar conclusion. But in practice bone does repair and remodel itself in order to prevent failure, and stress fractures do occur for people who have very active lifestyles or bone



deficiencies that prevent repair from being carried out. The present simulation suggests two reasons for this apparent anomaly, both of which relate to differences between data obtained from test specimens and the actual behaviour of intact bones:

a) A test specimen, taken from an animal after some years of life, will contain a population of cracks which is relatively safe because the most dangerous cracks have already been repaired. The remaining cracks will tend to be shorter and more likely to be dormant.

b) Another difference between the test specimen and the whole bone is size. Long bones in humans and other large animals have volumes of the order of 10^4-10^6 mm^3, of which approximately 20% is highly-stressed, but the stressed-volume of a typical test specimen is 100mm^3. All materials demonstrate a stressed-volume effect by which larger specimens tend

to fail more easily due to a statistical effect: the increased likelihood of weak regions leading to dangerous cracks being present.

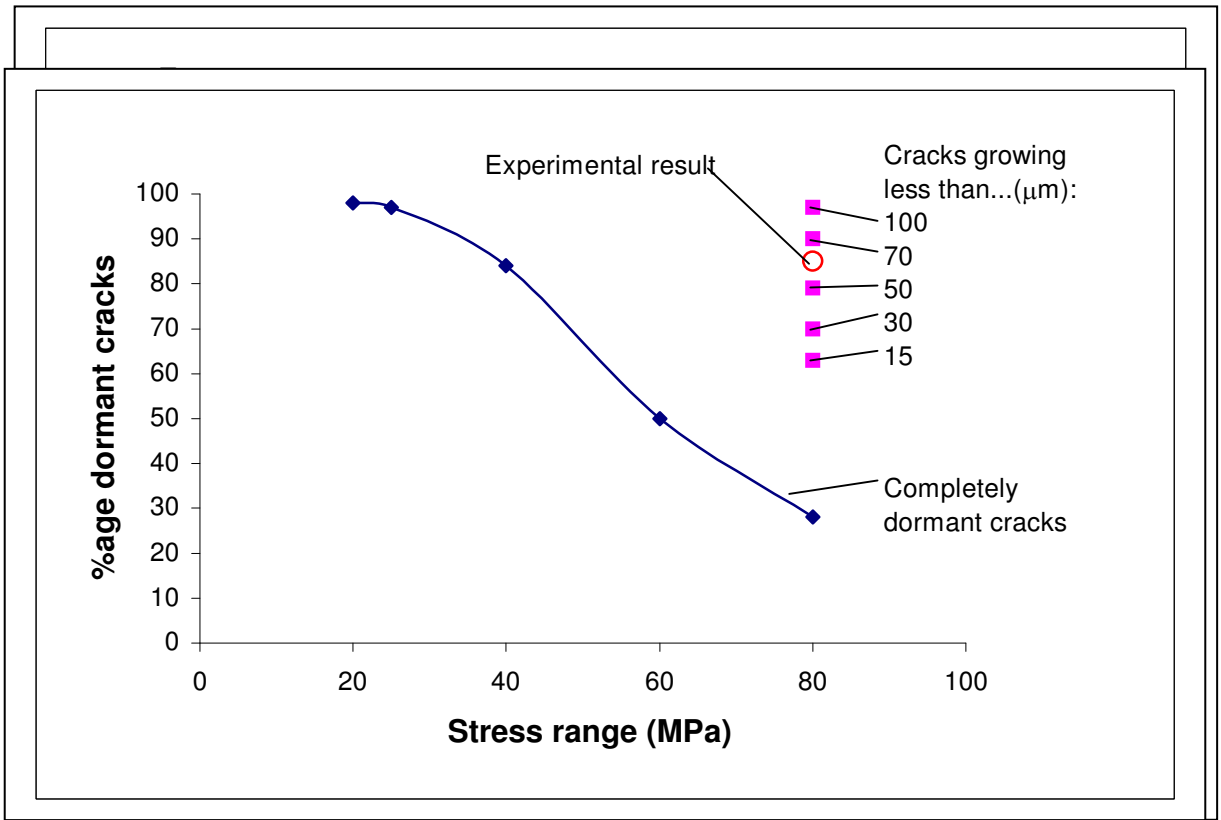
Fig.5 shows predictions of this effect from our model, plotting the number of cycles to failure as a function of specimen volume (varied by varying the total number of cracks) at a stress range of 40MPa. It is predicted that N_f will decrease markedly with increased size, the life of whole bones being less than that of specimens by about an order of magnitude. Also shown on the figure is a prediction made using a different approach, which was developed by the author

Fig.5: The effect of stressed volume on N_f at 40MPa.

in a number of previous papers [9,10]. This simpler approach uses the Weibull theory, which is capable of predicting volume effects based on the scatter in data from small specimens. It was able to predict results quite well within the range of measured data (specimen sizes from 10-1000mm^3) but no data from larger sizes was found. It agrees with the present simulation except at large volumes, where the simulation shows a weaker effect of volume. Of course we are only comparing two different theoretical models here, but the implication is that a simple Weibull model may not capture certain non-linearities of crack growth which will mean that the strength of whole bones, though less than that of specimens, is not as marked as we first thought.

3.5 Propagating cracks and dormant cracks

A feature of damage accumulation which is illustrated very dramatically by this model is the fact that most cracks are not dangerous, in the sense that they will never propagate to failure. This fact is implied by the minimal changes in average crack length (fig.4) which imply that, whilst one crack is extending itself over several millimetres in order to cause failure, most of the other cracks are not growing significantly. It is important to study the whole population of cracks, since it is from this distribution that the most dangerous crack emerges, but at the same time it should be recognised that most cracks are quite safe and do not need to be repaired. This may affect the approach taken by the living system when it is repairing and remodelling. The number of propagating and dormant cracks can be defined in different ways. For example, in our simulations when failure occurs (due to one crack reaching a length of 1mm) it is rare to find any other cracks with lengths greater than 300μm. This big difference



between the worst and second-worst cracks is due to the strong dependance of crack growth rate on crack length.

Fig.6: Percentage of dormant cracks

Another feature of the model is that certain cracks will never be able to grow to failure because both terms in equation 1 may go to zero. The first term, describing long-crack growth, will become zero when the applied stress intensity is equal to the threshold value (ΔK_{th}) , and the second term, describing short-crack growth, will approach zero as the crack length approaches the structure size, d. Since ΔK_{th} and d are both stochastic variables in our model, the condition for non-propagation, which is:

$$(\Delta K_{th}/F\Delta\sigma)^2 > d \qquad (4)$$

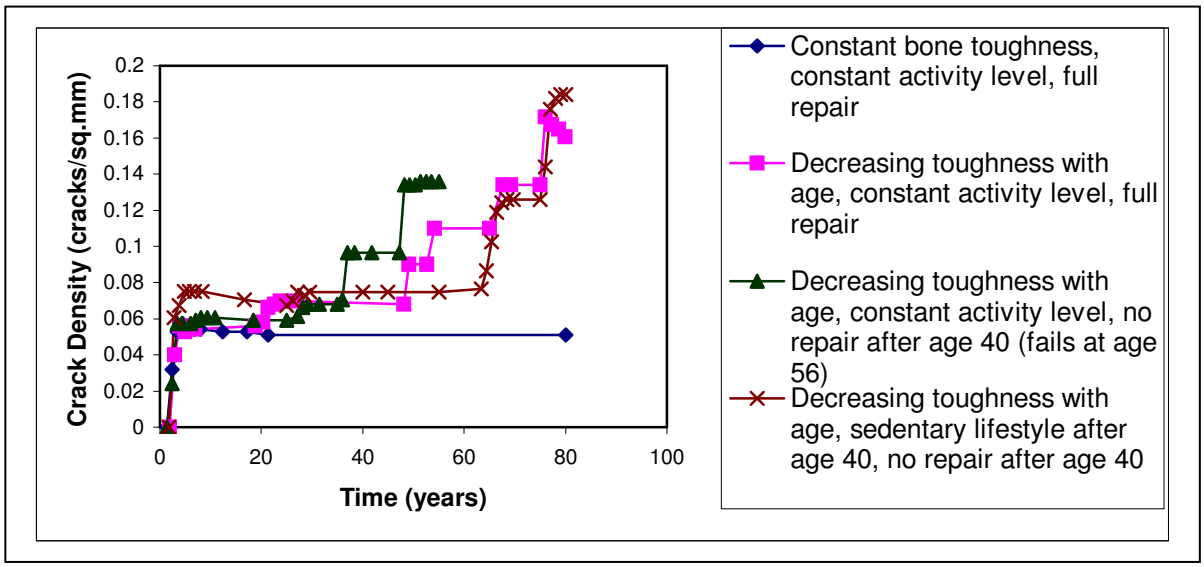
..will be met by a certain proportion of the crack population. Fig.6 shows that these 'completely dormant' cracks make up a large percentage of the population at low stress ranges. However when we tested at 80MPa using our sequential-dye system, we found only 8% of detected cracks had grown between 50,000 cycles and the end of the test (mean 88,000 cycles). This low figure is partly due to sectioning effects – we estimated a higher figure of 15% when this problem was corrected, giving 85% dormant cracks. This is still much greater than the value predicted using equation 4, which is 28%; the difference can be understood if we realise that many cracks, though not totally dormant, are growing by very small amounts. We recorded the number of cracks growing less than a given amount, Δa, during the simulation. This is much greater at 80MPa (fig.6), and the prediction agrees reasonably well with our experimental finding, given a detection level of around 50μm.

3.6 Crack density changes *in-vivo*

A more exciting feature of this model is that it can be used to predict the effect of *in-vivo* processes such as repair, remodelling and adaptation. The effect of repair is useful when considering the increase in crack density with age. Young people have relatively few cracks in their bones, but the number increases sharply from middle age. Our simulations (Fig.7) investigate various features of this change. We used standard activity levels defined by Whalen et al [11], and defined an effective stress range using a technique described in a previous paper [12]. Simulating a person having a normal, active lifestyle, and a strong repair process which removes all cracks greater than 100μm, we predict that the crack density will quickly stabilise around 0.05 cracks/mm^2 and remain constant. The onset of old age can be simulated by including three other features that may occur with aging: (i) decreased toughness and strength of bone; (ii) reduced ability to repair cracks, and; (iii) a more sedentary lifestyle. Simulations which include these features all show the upturn in crack density which has been reported experimentally [13,14]. If we cease repairing cracks at age 40 then failure (i.e. stress fractures) will occur around age 56, but if the person adopts a more sedentary lifestyle this can be avoided. Experimental data show curves of similar shape, but measured values for crack density vary widely, sometimes exceeding 5 cracks/mm^2. This is certainly due in part to variations in measurement technique, but also our simulation here is clearly simplistic in the way it treats repair, a feature which can be improved in future models.

3.7 Remodelling and adaptation

We can also incorporate into the model more sophisticated simulations of living processes. This has not been done up to now, as we have been concentrating on refining the model to the



point where it can make reasonable, quantitative predictions of the behaviour of dead bone. Future work will include a more realistic treatment of repair, which can take account of the known dynamic behaviour of resorption cavities (BMUs), and the inclusion of adaptation, whereby bone geometry and/or mechanical properties can be varied in response to changes in stress level. Damage variables can be used as the trigger for adaptation processes; it will be very interesting to see how the outcome can be affected by the choice of damage variable. Possible candidates are: crack number; mean length; total length; growth rate. The recognition that most cracks are dormant implies that an efficient adaptation scheme should focus on the small number of non-dormant cracks. This kind of theoretical model should, in its turn, suggest appropriate new experiments for its verification.

Fig.7: Changes in crack density with age

ACKNOWLEDGEMENTS

We are grateful to the Health Research Board and to the Royal College of Surgeons in Ireland for provision of funding for this project.

REFERENCES

[1] Martin, R.B., Burr, D.B. (1989): Structure, function and adaptation of compact bone. Raven Press (New York, USA) 1989.

[2] Martin RB: Mathematical model for repair of fatigue damage and stress fracture in osteonal bone. *J.Orthop.Res.* 13:309-316, 1995.

[3] Burr DB: Bone, exercise and stress fractures. *Exerc.Sport.Sci.Rev.* 25:171-194, 1997.

[4] O'Brien, F.J. Microcracks and the fatigue behaviour of compact bone. PhD thesis, Trinity College (University of Dublin) 2001.

[5] O'Brien FJ, Taylor D, Lee TC An improved labelling technique for monitoring microcrack growth in compact bone. J.Biomechanics (In Press).

[6] Taylor, D. Microcrack growth parameters for compact bone deduced from stiffness variations. *Journal of Biomechanics* 31, 587-592, 1998.

[7] Taylor D Fatigue damage in bone: links to adaptation. "*Synthesis in Bio Solid Mechanics*", Proc.IUTAM symp., Publ.Kluwer 175-186, 1999.

[8] Zioupos P, Wang XT, Currey J.D: Experimental and theoretical quantification of the development of damage in fatigue tests of bone and antler. J.Biomechanics 29:989-1002, 1996.

[9] Taylor, D: Fatigue of bone and bones: an analysis based on stressed volume. *J. Orthop.Res.* 16:163-169, 1998.

[10]Taylor D, O'Brien F, Prina-Mello A, Ryan C, O'Reilly P and Lee TC: Compression data on bovine bone confirms that a 'stressed volume' principle explains the variability of fatigue strength results. *J.Biomech*. 32:1199-1203, 1999.

[11] Whalen RT, Carter DR, Steele CR: Influence of physical activity on the regulation of bone density. *J.Biomech*. 21:825-837, 1988.

[12] Taylor D and Kuiper JH (2001) The prediction of stress fractures using a stressed volume concept. J.Orthop.Res. In Press.

[13]Schaffler MB, Choi K, Milgrom C: Microcracks and aging in human femoral compact bone. 40th Annual Meeting, Orthop.Res.Soc. pg190-32, 1994.

[14] Zioupos P: In vivo, in vitro and de novo microdamage in aging human cortical bone and its effect on toughness. Proc.European Society of Biomechanics, 2000.