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AN ANALYSIS OF THEORIES IN BIOMECHANICS [1)]

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The nature of the systems commonly thought of as coming into the remit of biomechanics range from 'mechanical' systems (concerned with the design of medical devices, such as implants) to 'mechanobiological' systems (concerned with the response of tissues, cells, and biomolecules to mechanical forces). In each case, a biomechanical system is characterised by the interaction of biological and physical elements. Like any other science, biomechanics advances by the development of theories and the testing of these theories by observation and experiment. The purpose of this paper is to analyse contemporary biomechanics research by providing a critique of the development of biomechanical theories. Only two are considered in detail: (i) the theory of hip prosthesis failure and (ii) the theory of adaptive-bone remodelling. In both cases, specific *basic statements*, or hypotheses, have been proposed and tested using computational and experimental methods. In the case of hip implants, there have been definite advances in the sense that many hypotheses that could have been rejected continue to be upheld despite severe testing. On the other hand, in the case of adaptive-bone remodelling where the dominant theory is 'adaptive elasticity', the tests of the theory have either been too lenient or they have smuggled in new assumptions that fundamentally alter it. The problems of testing adaptive-elasticity theory are described, and comparison is made with damage-based bone remodelling theories.

Key words: Prosthesis design, bone remodelling, mechanobiology, K. R. Popper.

1. INTRODUCTION

The earliest records of Arabic science show that mankind has long been fascinated by the idea of the human body as a machine. The thread of this idea

[1] This paper was presented, in part, when the author was a Visting Professor at the Institute of Fundamental Technological Research (IFTR), Polish Academy of Sciences, Warsaw, Poland.

has woven its way through the history of science, through Aristotle, to da Vinci, Galileo and Borelli, through Descartes and Boerhaave, reaching its clearest expression with the German anatomists of the last century [1]. With the growth of reductionist approaches to scientific investigation, interest in a mechanical understanding of biological systems begun to wane and eventually became overshadowed by achievements in molecular biology [2]. However, recent impulses, both scientific and societal, have forced a re-examination of the use of engineering principles to understand the mechanical behaviour of living systems.

On the scientific side, the impulses have come from the development of techniques that allow for more precise analysis of biological structures. These are (i) computational mechanics, (ii) high-resolution digital imaging systems, and (iii) biophysical measurement methods at the cell and molecular level. Societal motivations arise from the ageing nature of the populations of Europe and North America. Ageing diminishes the mechanical functionality of the body: reduced functionality occurs especially in the musculo-skeletal system (e.g., by arthritis or osteoporosis) and in the cardiovascular system (e.g., by stenosis of arteries or dysfunction of heart valves). Failure of the cardiovascular system accounts for 50% of all deaths in both 'developed' and developing countries [3]. As regards the musculo-skeletal system, one in seven women die when hospitalised as a result of bone fragility in old age [4]. Recognizing the epidemic proportions of osteoporosis and other musculo-skeletal diseases, the United Nations has officially declared 2000-2009 as the "bone and joint decade" [5].

Bioengineering solutions to musculo-skeletal diseases were developed in the 1970s in the 'first wave' of the application of engineering in biology and medicine. However, we are now moving away from those mechanics-led solutions to a biology-based engineering which involves direct integration of biological systems into implants. Inevitably biology will be brought, alongside physics and chemistry, into the core of engineering sciences [6].

For biomechanics to become part of engineering science, theories of use in the professional practice of engineering are required. In this paper, the methodology proposed by K. R. POPPER in *The logic of scientific discovery* [7] is used to examine the development of theories in biomechanics. In particular the author is interested in showing that the *testability* of theories, in the sense advocated by Popper, should be taken into more account in biomechanics research. The lack of testability of some of the most important theories of biomechanics is not, perhaps, sufficiently realised; and the lack of testability may be seen as, in some respects, an impediment to the emergence of theories with greater predictive power and internal consistency.

In summary, this paper is intended as a provocative look at the progress in biomechanics research, and the author hopes to persuade the reader that in one

area (prosthesis design) advances are being made, and that in another (bone remodelling) this is unfortunately not the case.

2. Tests on scientific theories

The contributions of K. R. Popper to the methodology of scientific investigation are well known. P. Medewar, who won the Nobel prize for medicine in 1960, has described *The logic of scientific discovery* [7] as one of the most important documents of the twentieth century. Popper argues that the empirical sciences (such as we hope biomechanics to be) are a system of theories. With respect to the construction of theories he distinguishes between two kinds of statements: *universal* statements, which are hypotheses with the "character of natural laws" and *singular* statements which apply to specific events and initial conditions [8]. A universal statement is about an unlimited number of cases, and may be thought of as an *all-statement* such as: 'All bodies attract each other with a force inversely proportional to the square of the distance between them'. An all-statement is not verifiable because it cannot be guaranteed that, at some time and in some place, a case will not be found to contradict it. However it is *falsifiable* if a single statement can be found which contradicts it - Popper called such statements *falsifying hypotheses.* Any singular statement, if it is self-consistent, can be a falsifying hypothesis. The more useful theories, such as those scientists call laws, will have more potentially falsifying hypotheses than weak theories. As Popper writes: "Not for nothing do we call the laws of nature 'laws': the more they prohibit the more they say" [9].

A measure of progress in biomechanics can be made by analysing explanatory power of some of its prominent theories. Applying Popper's ideas, progress may be considered to be high if theories with many potentially falsifying hypotheses have been developed, severely tested, and found to be upheld – or if they have been refuted they should be replaced by even stronger theories. Progress is not being made if all we continue to uphold are theories with few potential falsifiers – such theories will not much improve our ability to predict biomechanical phenomena.

The methods used to test hypotheses include both experimental and computational models. The rigour of the testing is highest if a theory can be subjected to independent tests of both an experimental and computational nature. Allied with the concept of testability is the notion that the degree to which a theory has been corroborated relies on the severity of the tests to which it has been subjected. Some insight into biomechanics can be obtained by looking at biomechanical theories from this perspective. Five theories are analysed and the results shown in Table 1.

Table 1: The relationship between falsfiability and predictive power of some biomechanical theories.

THEORY	FALSIFIABILITY (Number and 'severity' of potential falsifying experiments)	PREDICTIVE POWER
Relationship between elasticity and fabric in trabecular bone [1]	• Many potential falsifying experiments • High degree of independence - different bones, species, measurement methods (CT, serial sectioning, etc.) • Computational models and experimental measurements capable of high precision carried out by independent research groups.	Very high
Biphasic theory of cartilage viscoelasticity [2]	• Several potential falsifying experiments • Some degree of independence (different samples and species) • Computational models and experimental models.	High
Theory of stenosis formation by endothelial cell reaction to fluid shear stress [3]	• Few potential falsifying experiments • Low degree of independence (similar anatomical sites) • Difficult to measure shear forces without disturbing the flow reduces possibility for refutation.	Qualitative
Osteogenic index theory of endochondral ossification [4]	• Independent model parameters almost preclude any falsifying experiments • Some degree of independence (different ossification sites, and species) • Computational models only; lack of information on constitutive behaviour of tissues and of the loading regieme prevents rigorous tests.	Low
Trajectoral theory of trabecular bone remodelling (Wolff's law) [5]	• Hard to conceive of a falsifying experiment.	Almost none

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According to POPPER [7], the more opportunities a theory gives to be refuted by experience the better it is. This implies that we should prefer those theories that can be most severely tested. Furthermore, it might be inferred that a theory that cannot be severely tested is an impediment to scientific progress. These conclusions have the corollary that the refutation of a simple theory is a progressive step whereas the construction of a complex theory that is unrefutable (has no potential falsifiers) is the opposite. Theories that lack potential falsifiers leave little room for refutation and therefore have little predictive power[2]. Such a 'theory' may be considered merely as a collection of assertions and observations which are unconnected by any empirical relationship (Wollf's law of bone remodelling [10] being an example). To quote again from Popper: "An assertion which owing to its logical form is not testable can, at best, operate within science as a stimulus: it can suggest a problem" [11].

In that respect, it is the author's opinion that COWIN [12] and CURREY [13] do not appreciate the purpose of WOLFF'S book *Das Gesetz der Transformation der Knochen* [10].

3. THEORY OF HIP PROSTHESIS FAILURE

With the advent of modern hip replacement using polymethylmethacrylate for fixation and the 'low friction' concept for the articulation, several failure mechanisms for hip arthroplasty were observed [14]. These were mechanical in nature: e.g. stem breakage, cement fatigue, wear of the acetabular cup, 'radiographic' loosening, possibly exacerbated by proximal bone loss due to stress shielding. With the introduction of cementless prostheses of the press-fit or porous-coated type, new failure modes were observed whereby the implant failed to stabilize in the early post-operative period. Many hundreds of designs were released onto the market during this time; however no tests that could corroborate the hypothesised superiority of one design over another were available; such biomechanical tests as there were (e.g. strain analysis of bones to detect stress-shielding [15] or finite element stress analysis of implant components [16]) were not widely accepted as sufficiently precise to differentiate the performance of implants. Follow-up studies on patients did not allow inter-comparison of implants because of the differences in follow-up methodology (different outcome measures, patient selection proce-

[2]To illustrate this, Popper gives the example of Kepler who initially proved that the orbits of the planets were not circles and went on to show that they are, in fact, ellipses. Had he first set out with the objective of proving that they are ellipses he might have achieved nothing – the theory that orbits are circular was testable within the precision of the analyses available to him and therefore had the potential for falsification.

dures, surgical technique, etc.). The result was a proliferation of designs [17], and one authority has questioned whether or not "these new devices are marketed for the sake of the patients or to keep orthopaedic companies afloat" [18].

It can be recognised that proposing a new implant is a hypothesis, the hypothesis being that 'prosthesis A will give pain-free mobility to a patient for longer than prosthesis B'. The testability of this hypothesis was low for at least two reasons:

(i) the nature of the events by which pain-free mobility was lost was not fully understood and therefore experiments (physical or computational) could not be designed to test for it,

(ii) an end-point defining the 'loss of pain-free mobility' was not agreed.

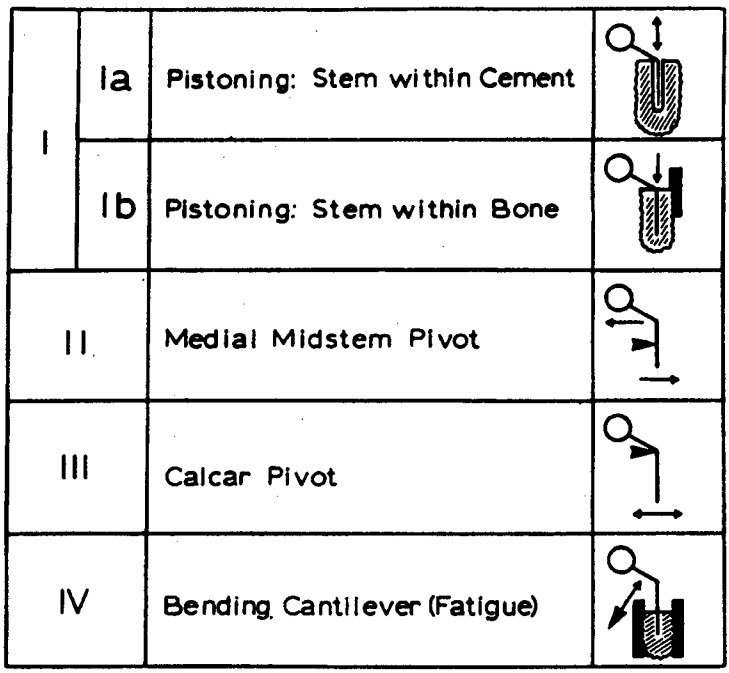


FIG. 1. "Failure modes" for femoral hip replacements, according to GRUEN *et al.* [19].

In the late 1970s, a step to resolving the first of these was given by GRUEN *et al.* [19] who proposed 'failure modes' for hip implants. Several researchers attributed failures observed in follow-up studies to one or other of these modes, shown in Fig. 1. However this theory did not serve to explain the failure in a way that they could be used to test the hypothesised superiority of one implant

relative to another; for example prostheses that underwent significant subsidence did not require revision [20] and bone loss "threatened" implant longevity but did not directly cause failure [21]. In summary, the theory of GRUEN *et al.* [19] may be seen to have failed to provide the basis for the emergence of the required tests: in fact the theory encapsulated all observations but did not connect them via physical processes that could be simulated in an experiment.

HUISKES [18] established a theory of failure of orthopaedic implants in the form of Huiskes' Failure Scenarios. These failure scenarios were hypothesised to cover all mechanisms leading to failure as follows: damage accumulation failure scenario, particulate reaction failure scenario, failed ingrowth failure scenario, stress shielding failure scenario, stress bypass failure scenario, and the destructive wear failure scenario. Therefore Huiskes' theory attempts to encapsulate in six statements the complex mechanisms of failure of an orthopaedic implant. These are testable because they present a sequence of physical events amenable to experimental and computational analysis. They re-organise the facts known about failure mechanisms of orthopaedic implants into a coherent form. Consider the damage accumulation failure scenario for example: it hypothesises that implants loosen by the accumulation of microcracks within the prosthetic materials, and that damage accumulation can be accelerated if stresses are increased as a result of adverse tissue adaptations such as soft tissue formation or proximo-medial bone loss. The scenario has been tested both numerically by VERDONSCHOT and HUISKES [22] and experimentally by MCCORMACK and PRENDERGAST [23], and in both cases it was corroborated. The damage accumulation failure scenario can also be used to discriminate the performance of prostheses [24, 25]. Other more rigorous approaches to simulation of multiaxial damage accumulation will allow even more severe tests of the damage accumulation failure scenario [26].

The issue of establishing an end-point has been addressed by the Swedish hip register [27]. It has facilitated inter-comparison of implants using time-to-revision as the measure of outcome. Of course there are problems with this measure such as the fact that not all surgeons (or patients) will agree that a revision is necessary for a particular state of loosening [14]. Nonetheless, it has established a definite end-point allowing comparison of implants in a precise way. The result has been that implants with below average performance can be unequivocally identified putting pressure on the surgeon to select an implant with a better performance. Due to the Swedish hip register, a mere five cemented implants now constitute 78% of the market in Sweden, without any suggestion of reduced quality of care [28]. This register, and others like it, give the information that biomechanicians require to test theories on the failure of implants relative to clinical outcomes [18, 29].

4. Theory of adaptative-elasticity

Roesler [30] gives a detailed review of the work of the 19th and 20th century German anatomists on problems in bone biomechanics. He shows that they established beyond doubt that bone tissue must adapt to mechanical loading, but that they failed to provide a mathematical description of the process. Cowin and Hegedus [31, 32] in 1976 used continuum mechanics to provide a description of adaptive-bone remodelling. This attempt to develop a theory from the axioms of mechanics has endeared adaptive-elasticity to many mechanicians in the field. Mass balance, momentum balance, energy balance, and a second law equation for an adaptive solid were proposed and an equation of the following form to describe the bone remodelling process was derived:

$$\frac{de}{dt} = a(e) + A_{ij}(e)K_{ijkl}(e)\sigma_{km} \tag{4.1}$$

where e is a measure of the change in volume fraction form its equilibrium value. To implement the theory for resorption/deposition on a bony surface, a homeostatic equilibrium strain denoted ε^0_{ij} is defined and

$$\frac{dX}{dt} = C_{ij}(\varepsilon_{ij} - \varepsilon^0_{ij}) \tag{4.2}$$

where X is the position of the surface. The theory was tested on simple problems relating to remodelling of a long bone diaphysis (modelled as a right circular cylinder). These were internal [33] and surface [34] remodelling induced by a medullary pin, devolution of inhomogeniety [35], and remodelling under constant load [36]. Cowin *et al.* [37] also tested the theory against bone adaptation experiments carried out in animals and found good agreement, but only if the remodelling constants were different for each animal, and different again for the periosteal and endosteal surfaces. A computational implementation was first developed using finite element modelling by Hart *et al.* [38] and this too corroborated the adaptive-elasticity theory.

4.1. Reduction in the number of potential falsifying hypotheses

After the tests described above, adaptive-elasticity theory was used as a basis of prediction of remodelling around orthopaedic implants, first by Huiskes *et al.* [39], and thereafter by several others, for example Weinans *et al.* [40] and Vander Sloten and Van der Perre [41]. In these papers, a rate of remodelling was hypothesised to be given as illustrated in Fig. 2, where the central region is a zone of equilibrium stimuli and the remodelling stimulus (S) was taken to be the strain energy density per unit mass. The adaptive bone remodelling equation was then re-cast as:

$$\frac{d\rho}{dt} = \begin{cases} C(S - k[1+s])^{\beta} & S > k(1+s) \\ 0 & k(1+s) \geq S \geq k(1-s) \\ C(S - k[1-s])^{\beta} & S < k(1-s) \end{cases} \tag{4.3}$$

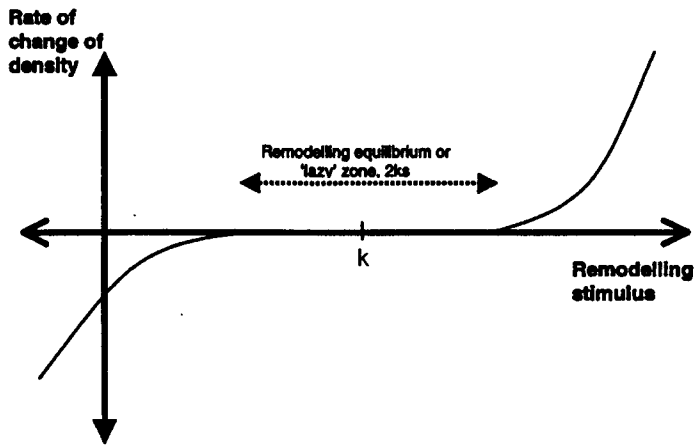


FIG. 2. The relationship between remodelling stimulus and rate of change of mass. The non-linearity of the equation is not employed as a material parameter but rather as a fitting parameter. This combined with the assumption of site-specificity of the remodelling reference stimulus, reduces the number of potentially falsifying hypotheses.

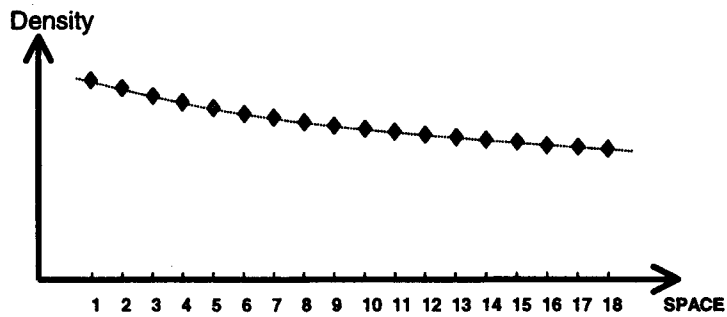
WEINANS *et al.* [42] and VAN RIETBERGEN *et al.* [43] carried out the first fully three-dimensional simulation using adaptive-elasticity theory. Since their bone-remodelling simulations of implants in beagle dogs gave approximately the same as that of the experiment, their results can be taken as a corroboration of adaptive elasticity theory. These papers also used the concept of the lazy zone, but it may be noted that the dimension of the lazy zone ($s = 0.75$) was different from that used by WEINANS *et al.* [40] ($s = 0.35$) and VANDER SLOTEN and VAN der PERRE [41] ($s = 0.1$). The dimension of lazy zone was, in effect, not a material parameter but a tuning parameter. In addition, in several simulations, the remodelling equilibrium constant, k, is assumed to vary from point-to-point in the bone (these are called site specific formulations of the theory). In such cases, k is determined from an analysis of the intact or pre-operative bone as it represents a spatial "target" controlling the adaptation process. Combined with the tuning capability offered by the lazy zone, this means that adaptive-elasticity theory almost impossible to falsify.

4.2. Introduction of an auxiliary hypothesis

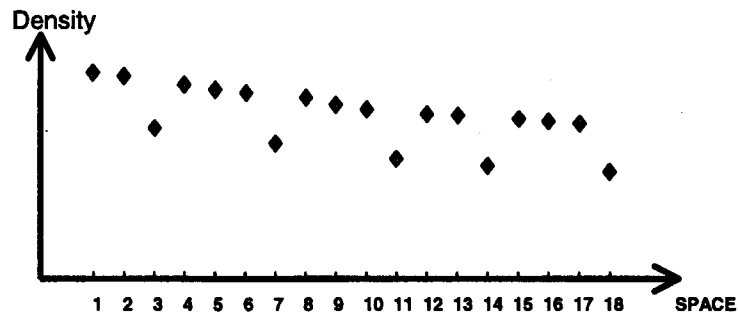
Elasticity theory is based on the continuum assumption, i.e. that all material points contain matter (there are no voids) but rather matter is 'smeared' to be continuous. Generalising the elasticity theory for adaptive materials, adaptive-elasticity assumes that each material point is capable of changing its porosity (i.e. density and therefore elastic response) depending on the mechanical stimuli acting on it. COWIN [44] states that adaptive elasticity theory permits the porosity at any material point to be an independent variable. In a finite element simulation of bone remodelling based on adaptive elasticity theory, WEINANS *et al.* [45] showed that an instability exists because when elements are perturbed from remodelling equilibrium, they either reach a maximum density or reduce to zero density. This is caused by a positive feedback whereby elements that achieve a higher stiffness than their neighbours attract yet more stress causing their density to increase yet further. The result is that voids (or pores) are created in those regions which do not attract the stress stimulus. The question arises whether or not this simulation falsified adaptive elasticity theory since a discontinuous structure (like reality) is produced only because it assumes the sensors are discrete whereby not every material points is 'capable of changing its porosity'. Even when certain numerical modelling issues (nodal averaging [46], etc) were resolved, the same instability still occurred. If mass will disappear from a region of the continuum so that a void is formed on the length scale of the mechanosensation process (i.e. millimetres), does this not pose problems for the continuum assumption?

Later studies made the remodelling algorithm mesh independent [47,48] and showed that a fully dense continuum would remodel into a trabecular pattern and that the trabecular pattern would alter in response to a change in the load. These simulations are undoubtedly representative of the real process, and rely on the same positive feedback mechanism as that found in WEINANS *et al.* [45]. No analytical continuum solution can provide these predictions – only a numerical one – and this fact alone serves to illustrate that the prediction relies on discrete sensors where certain material points control the adaptation of their neighbours [49].

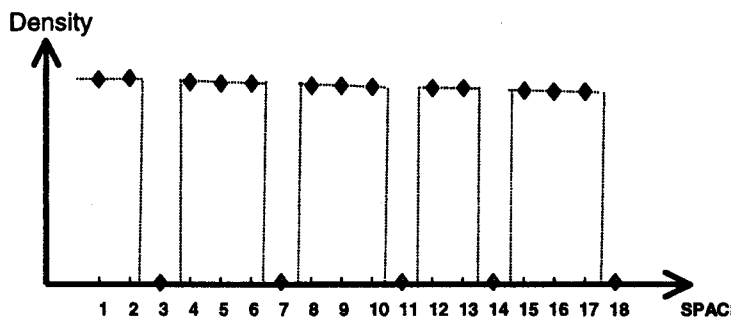
Therefore these simulations could have been construed as falsifying adaptive-elasticity theory because they show that the result of adaptation is a material that is not continuous at the scale of the mechano-regulatory process; that is to say, the results of the simulations are those of a discrete-dynamical 'state' model rather than remodelling of one continuum to another. Figure 3 attempts to illustrate this concept. Instead of this refutation becoming explicit, an auxiliary hypothesis was introduced which stated that the sensors were sufficiently close together to be assumed to be continuous which means that the gradient of the porosity should be continuous or smooth. However, this co-ordinating behaviour is not maintained on the cells in reality – the hypothesis is an auxiliary one.



(a) At remodelling equilibrium the material is modelled as continuous



(b) After a perturbation, positive feedback causes some regions to loose mass and others to gain mass. Rendering the density continuous at each iteration would prevent trabecularization.



(c) Eventually the positive feedback causes the emergence of a pattern (trabecularization). The positive feedback process occurs at the scale of the pores.

FIG. 3. A schematic illustration of how it because of positive feedback that the mechanism of trabecularization arises. The process generating the positive feedback occurs on a scale that may violate the assumption of continuity.

4.3. Testability of bone remodelling theories

It was concluded in Sec. 2 that those theories that have many potentially falsifying hypotheses are to be preferred over those that have few. We can use this criterion to compare two approaches to prediction of bone remodelling; the approach based on adaptive elasticity versus the approach based on fatigue microdamage [50]. Damage-adaptive remodelling is fundamentally different from strain-adaptive remodelling because in the latter the stimulus (damage) can accumulate, and the bone remodelling equation is written, following PRENDERGAST and TAYLOR [50] as:

$$\frac{d\rho}{dt} = C\Delta\omega \tag{4.4}$$

where ρ is the density and C is a remodelling rate constant. $\Delta\omega$ is the change in damage from the equilibrium level, and it is given by the integrated difference between the damage formation rate and the damage repair rate, as

$$\Delta\omega(t) = \int_{t_0}^{t} \frac{d\omega}{dt} - \frac{d\omega_{ss}}{dt} \tag{4.5}$$

There are several potentially falsifying hypotheses (i.e. statements that, if true, would refute the damage-adaptive remodelling theory). These statements are:

(i) static loads cause the same degree of remodelling as cyclic loads,

(ii) disperse microdamage does not exist in bone microstructure at remodelling equilibrium,

(iii) experiments on living bone show no change in the microdamage burden when the load is changed,

(iv) computer simulation of bone remodelling around implants needs to be 'tuned' to obtain physically-reasonable predictions of bone loss,

(v) no plausible mechanotransduction pathway exists.

As regards the first of these, RUBIN and LANYON [51], among many others, have shown the stimulatory effect of dynamic loads. Regarding the second, researchers as early as FROST [52] but more recently O'BRIEN *et al.* [53] have conclusively demonstrated that bone contains microdamage under normal daily activity. Thirdly, using the sheep forelimb model whereby the ulna is osteotomised to overload the radius, LEE *et al.* [54] demonstrated, that microdamage quantity increases when the load is increased, see Fig. 4.

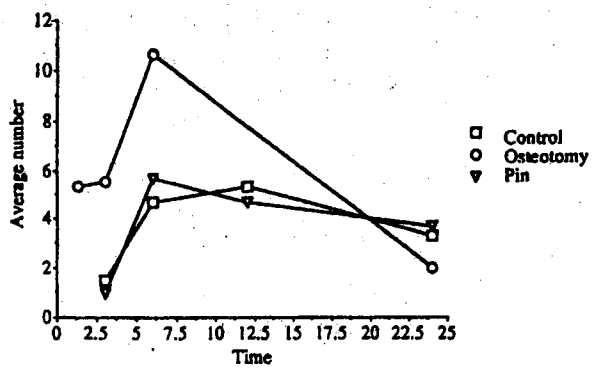


FIG. 4. Mean number of cracks vs. time (in weeks). A result form Professor T.C. Lee's experiment on the overloading (osteotomy) and underloading (pin) of the sheep ulna. The amount of microcracks in a bone increases when an overload is applied. This corroborates the hypothesis that microdamage may act as a bone remodelling stimulus, after LEE *et al.* [54].

Regarding the fourth possible refutation, computer simulations by PRENDERGAST and TAYLOR [55] and MCNAMARA *et al.* [56] showed that the nonlinearity in the relationship between stress and fatigue damage formation could predict bone loss patterns without the use of a 'lazy' zone. Regarding the fifth refutation (lack of a mechanotransduction pathway), several have been proposed including disruption of the canalicular network or damage sensation by strain changes in the neightbourhood of osteocyte cells [57]. From this it is clear that damage-adaptive remodelling is more testable – because of this it may have the potential to explain more bone remodelling phenomena than adaptive-elasticity theory.

5. RECAPITULATION AND CONCLUSIONS

What the author has tried to do in this paper is to take a closer look than is usual at the evolution of theories in biomechanics. This is done using the methods of scientific investigation proposed by Karl Popper. To the author's knowledge, no such an analysis has been presented in the literature. Before drawing to a conclusion, it should be noted that the analysis presented here is far from comprehensive as only two theories are considered in detail. Furthermore the outcome of the analysis is dependent to some considerable extent on the author's personal perspective. However, the author contends that the paper does show that Popper's concept of testability in terms of the number of potential falsifiers gives a useful insight into the value of biomechanical theories: if the paper convinces the reader that of the importance of the question "Can this theory really be falsified?" then the author will be happy.

Comparing the theory of hip prosthesis failure with that of strain-adaptive bone remodelling shows that the former is making significant progress because the concept of failure scenarios allows the testing of implants for specific failure mechanisms, and because the Swedish hip register allows valid clinical comparisons between implants to be made – the *falsifiability* of the hypothesis that one implant is superior to another is increasing. This is of considerable societal relevance given that up to one million are implanted annually meaning that perhaps as many as 20 million may be 'in service' at the present time.

The biomechanics of tissue adaptation is, on the other hand, not yet provided with a coherent biomechanical theory upon which the subject can be developed. The author does not want to argue that adaptive elasticity is wrong per se, but rather that it cannot be tested without the introduction of new assumptions and hypotheses. The theory is not sufficiently well-defined to make these new assumptions apparent. Perhaps an alternative to adaptive elasticity is required based on a discrete-dynamical system [49].

In conclusion, much research is being carried out on biomechanical problems so that theories of considerable predictive power are evolving in some areas (Table 1). In the mechanobiological side of biomechanics, the interplay of biological and physical reasoning has generated a host of theories that stand in the oft criticised [12, 13, 48] yet very influential tradition of Prof. Julius Wolff (1836-1902).

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