Afr. J Med. med. Sci (1999) 28, 117 - 119

Chondrocyte hypertrophy in the growth plate: A short communication on probable mechanism

Olusola O.A. Oni
The Glenfield Hospital, Groby Road, Leicester LE3 9QP, UK

Summary
The process by which chondrocytes hypertrophy has been deduced from observations on standard histology sections of the rabbit growth plate is reported Features suggestive of an osmotic process were observed, namely a hierarchy of cell sizes, cell rupture, an adjoining 'open' microcirculation and a permeable partition between cells and blood.

Keywords: *Chondrocyte, Hypertrophy, Growth plate, Osmosis*

Résumé
La processus par lequel l'hypertrophie des chondrocytes a ete deduite des observations sur des sections histologique standard des plats de croissance des lapins. Les traits suggerant un processus osmotique ont ete observes; moment, une hierarchie des tailles des cellules, rupture de cellulose, une ouverture de la microcirculation et une partition permeable entre les cellulos et le sang.

Introduction

Hypertrophic chondrocytes (HCs) are very important cells in bone biology. Being the mechanism by which cartilage is converted into bone [1]. HCs hold the key to endochondral ossification. The mechanism by which chondrocytes enlarge is not known. Hypertrophic chonodrocytes do not exhibit features normally associated with cell hypertrophy such as increase in number of organelles and in quantity of cytosolic materials [2]. There is evidence which suggests that chondrocytes in the growth plate enlarge primarily by accumulating water [3,4]. Cells that have accumulated excess water swell and this is a normal physiological occurrence in plant cells [5]. If this holds true for chondrocytes, evidence for osmosis such as cell swelling and cell rupture [5,6] would be apparent in standard histology sections and this is the object of this study.

Materials and methods

This study is part of a larger ongoing investigation into the biology of the growth plate. In previous reports, we have described osteocalcin expression in the groove of Ranvier [7] and the post-natal development of the physis [8]. In the current study, tibiae and femora obtaine from New Zealand white rabbits aged from 5 days to weeks were studied. The specimens were fixed in 1C buffered formalin for one week and then decalcified in Kristensen's solution for one week. Thereafter, they were routinely processed and later embedded in paraffin wax.

Correspondence: O.O.A. Oni, The Glenfield Hospital, Groby Road, Leicester LE3 9QP, UK.

Sections 6u thick were obtained, stained with haematoxylin and eosin (HE) and examined with an ordinary light microscope.

Results

The metaphyseal surface of the growth plate contained thin-walled sinuses. 'Microhaemorrhages' were observed in places where the lacunar wall had ruptured and red blood cells could be observed lying within the empty lacunae (Fig. 1).

Fig. 1: Photomicrograph of the physis showing red blood cells occupying empty lacunae.

Fig. 2: Photomicrograph showing hierarchy of cell sizes in both physis (P) and epiphysis (E).

There was a hierarchy of cell sizes with the largest, more rounded cells closest to the blood and the smallest, more flattened cells farthest away. A similar hierarchy of cell sizes could be observed in the epiphysis in specimens in which this structure had become developed (Figure 2). Chondrocytes in close proximity of one another or contained within the same lacunar walls or tissue zone had similar shapes and sizes.

Discussion

According to the findings from this study, the growth plate exhibits attributes (Fig. 3) which raise the possibility that an osmotic mechanism may be responsible for chondrocyte hypertrophy. First, the cartilage surface contains thin-walled vessels through which fluid could easily diffuse. The surface is also directly bathed in places by blood. The matrix is a 'porous' cell size from the swollen hypertrophic chondrocytes adjacent to the blood vessels to the flattened cells of the proliferation zone buried in the depths of the physis. Third, at the blood-cartilage interface, lacunae burst liberating their cell occupants which would later change phenotype and become osteoblastic [10].

All the cells do not simultaneously become hypertrophic because there is a water gradient across the cartilage. Diffusion is slow and effective in moving substances over short distances only [5,6]. Extracellular fluid formed as is usual from the microcirculation [11] is probably not within the immediate reach of every chondrocyte. To compensate for this, cartilage presumably uses a two-tier exchange process (Fig. 4); the first between blood and the cells closest to it and the second between the para-vascular and the remote regions of the cartillage. As a consequence of this, chondrocytes within the same tissue zone have similar sizes and shapes. Chondrocytes in close proximity may also exchange fluid with one another via channels similar to plasmodesmata [6], particularly as such cells would have arisen from a single ancestral cell [1]. By this means, a continuos fluid compartment is formed which has previously been called the 'symplast' (Fig. 5).

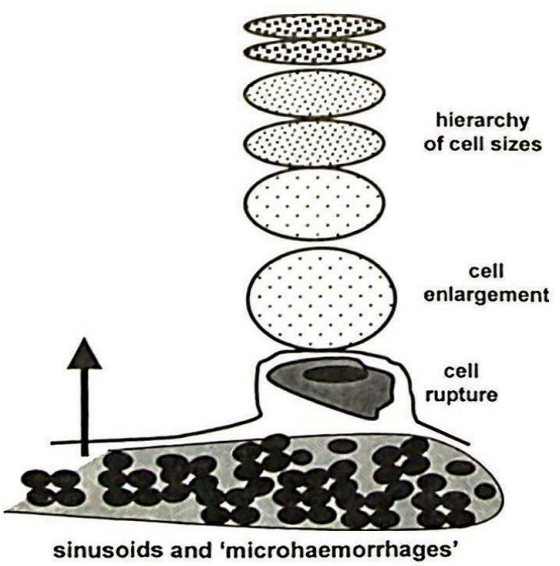

Fig. 3: The attributes of an osmotic phenomenon

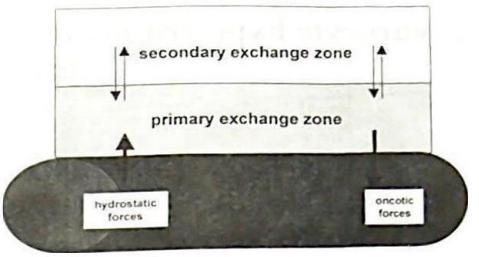

Fig. 4: The two-tier exchange concept

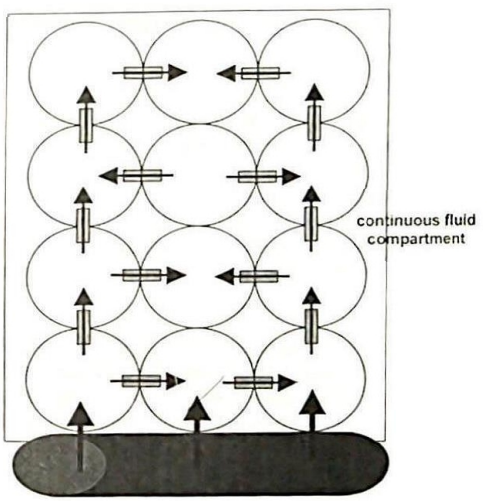

Fig. 5: The 'symplast' transport system.

References

1. Ham WH, Cormack DH. In Histophysiology of Cartilage, Bone, and Joints. Lippincott Co., Philadelphia, 1979.
2. Stevens A, Lowe J. In Pathology, Mosby Co., London, 1995.
3. Buckwalter JA, Mower D, Ungar R, Schaeffer J, Ginsberg B. Morphometric analysis of chondrocyte hypertrophy. J. Bone Joint Surg. 1986; 68: 243-255.
4. Hunziker EB, Schenk RK. Physiological mehanisms adopted by chondrocytes in regulating longitudinal bone growth in rats 1989; 414; 55-71.

5 Barker M. In GCSE Biology, Longman, Harlow, 1992.

6 Philips WD, Chilton TJ: In A-level Biology. Oxford University Press, Oxford, 1991.

7 Oni OOA. Osteocalcin expression in the groove of Ranvier of the rabbit growth plate. Injury 1997a; 28: 109-111.

8 Oni OOA The development of the growth plate after birth: a study by osteocalcin immunocytochemistry. 1997b; Injury 28: 199-202.

9. Maroudas A, Bullough P, Swanson SAV, Freeman MAR. The permeability of articular cartillage. J Bone Joint Surg 1968; 50B: 166-177.

10. Roach HI. Trans-differentiation of hypertrophic chondrocytes into cells capable of producing mineralized bone matrix. Bone Miner 1992; 19:1

11. Green JH. In An Introduction to Human Physiology, Oxford Medical Publications, Oxford, 1989.