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Journal of Biomechanics 41 (2008) 1184–1196

JOURNAL OF BIOMECHANICS

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Effects of mechanical compression on metabolism and distribution of oxygen and lactate in intervertebral disc

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Accepted 29 January 2008

Abstract

The objective of this study was to examine the effects of mechanical compression on metabolism and distributions of oxygen and lactate in the intervertebral disc (IVD) using a new formulation of the triphasic theory. In this study, the cellular metabolic rates of oxygen and lactate were incorporated into the newly developed formulation of the mechano-electrochemical mixture model [Huang, C.-Y., Gu, W.Y., 2007. Effect of tension-compression nonlinearity on solute transport in charged hydrated fibrosus tissues under dynamic unconfined compression. Journal of Biomechanical Engineering 129, 423–429]. The model was used to numerically analyze metabolism and transport of oxygen and lactate in the IVD under static or dynamic compression. The theoretical analyses demonstrated that compressive loading could affect transport and metabolism of nutrients. Dynamic compression increased oxygen concentration, reduced lactate accumulation, and promoted oxygen consumption and lactate production (i.e., energy conversion) within the IVD. Such effects of dynamic loading were dependent on strain level and loading frequency, and more pronounced in the IVD with less permeable endplate. In contrast, static compression exhibited inverse effects on transport and metabolism of oxygen and lactate. The theoretical predictions in this study are in good agreement with those in the literature. This study established a new theoretical model for analyzing cellular metabolism of nutrients in hydrated, fibrous soft tissues under mechanical compression.

Keywords: Computational biomechanics; Finite element method; Glucose; Mixture theory; Soft tissue mechanics; Transport

1. Introduction

Intervertebral disc (IVD) is the largest avascular structure in the human body with the nucleus pulposus (NP) being centered and surrounded on its periphery by the annulus fibrosus (AF), and superiorly and inferiorly by cartilaginous endplates (CEP) (Fig. 1). The NP is composed from randomly oriented collagen fibrils enmeshed in a proteoglycan gel while the AF is formed from a series of concentric lamellae consisting of collagen fibers (Hickey and Hukins, 1980; Marchand and Ahmed, 1990; Urban and Maroudas, 1980; Lundon and Bolton, 2001). Water is the major component of the IVD (65–90% wet weight). The other major components in IVD are collagen (15–65% dry weight), proteoglycan (10–60% dry weight), and other matrix proteins (15–45% dry weight) (Eyre et al., 1989; Johnstone et al., 1992; Gu et al., 1999a; Iatridis et al., 2007; Kraemer et al., 1985; Panagiotacopulos et al., 1987; Pearce, 1993). The IVD is primarily subjected to compressive load in vivo and has a strong propensity for swelling (Urban and Maroudas, 1981; Urban and McMullin, 1988; Yao et al., 2002). The osmotic pressure of the IVD is mainly due to the high density of charged carboxyl and sulfate groups on the glycosaminoglycans of the proteoglycans within the tissue (Urban and Maroudas, 1980; Urban et al., 1979). When an IVD is deformed under loading, interstitial fluid flow occurs, even though the hydraulic permeability of the tissue is very low (Best et al., 1994; Gu et al., 1999a; Gu and Yao, 2003; Houben et al., 1997; Iatridis et al., 1998; Perie et al., 2005). The electrical response of the IVD also changes when a disc is compressed (Yao and Gu, 2007b), due to the effects of diffusion potential and streaming potential (Lai et al., 2000; Gu et al., 1999b).

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doi:10.1016/j.jbiomech.2008.02.002

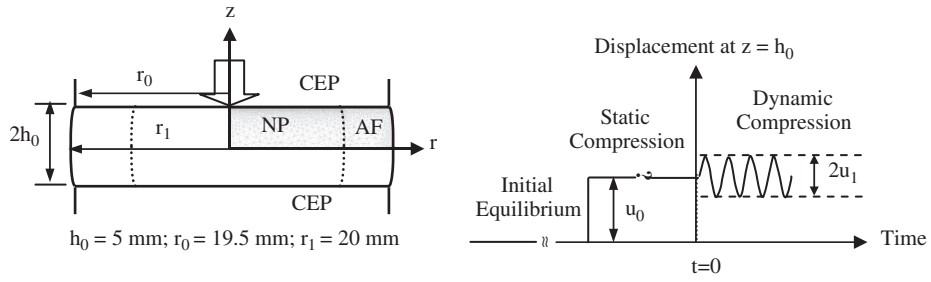

Fig. 1. Schematic of loading configuration for the finite element analysis of oxygen and lactate distributions. A constant displacement (10% of disc height) was applied during the static compression test. For dynamic compression test, a sinusoidal displacement ($u_1 = 2.5\%$ or 5% of disc height and frequency = 0.1 or 0.01 Hz) was imposed.

Vital nutrients are supplied to the IVD from the blood vessels located at the margins of the disc (Brodin, 1955; Brown and Tsaltas, 1976; Holm et al., 1981; Maroudas et al., 1975; Ogata and Whiteside, 1981; Urban et al., 1982). The transport of nutrients through the dense complex extracellular matrix to IVD cells relies mainly on diffusion. Therefore, poor nutrient supply has been suggested as a potential mechanism for disc degeneration (Bibby et al., 2002; Bibby and Urban, 2004; Horner and Urban, 2001).

There are two possible pathways for nutrient transport into (and waste transport out of) the IVD: the cartilage endplate route and the perianular route (Nachemson et al., 1970; Horner and Urban, 2001; Maroudas et al., 1975; Urban et al., 1977, 1978; Ogata and Whiteside, 1981; Holm et al., 1981; Crock and Goldwasser, 1984; Moore et al., 1992; Roberts et al., 1996). Since determining the in vivo distribution of nutrients in human IVD is often invasive and difficult (Bartels et al., 1998), the knowledge on nutrient transport in human IVD is limited. It is still unclear as to how the nutrition level in IVD is regulated by mechanical and biological factors, such as applied stress or strain, tissue degeneration, and endplate calcification. Recently, numerical analyses have been used to investigate the transport of nutrients and metabolites (e.g., oxygen, glucose, and lactate) within the human IVD (Selard et al., 2003; Soukane et al., 2005). In these previous theoretical studies, the complex geometry and nonhomogeneous properties of the human IVD were considered and the distribution of oxygen, glucose, and lactate within the IVD were shown to depend on solute diffusivity, cellular metabolic rates, the coupling effects between solute concentrations and consumptions/productions, and boundary conditions (Selard et al., 2003; Soukane et al., 2005). Most recently, Soukane et al. (2007) suggested that fluid loss and change in disc geometry which were assumed to be caused by static compression could affect the transport of nutrients and metabolites. However, only diffusion of solutes was considered in these theoretical studies.

It is well-known that dynamic compression augments the transport of large solutes in cartilaginous tissues (Bonassar et al., 2000; Evans and Quinn, 2006a, b; Huang and Gu, 2007; Mauck et al., 2003; O'Hara et al., 1990; Urban et al., 1982; Yao and Gu, 2004, 2007a). Recently, our theoretical study suggested that dynamic compression could promote transport of neutral solute in the anisotropic cartilaginous tissue by enhancing both diffusive and convective solute fluxes (Huang and Gu, 2007). However, the effect of mechanical compression on the distribution and metabolism of nutrients in the human IVD has not been studied. Therefore, the objective of this study was to examine the effects of static and dynamic compressions on distribution and metabolism of oxygen and lactate in the IVD using a new model based on the mechano-electrochemical mixture theory (Lai et al., 1991), due to the fact that IVD is a charged hydrated soft tissue.

2. Theoretical model

In this study, a new theoretical formulation was extended from our previous theoretical framework (Yao and Gu, 2004; Huang and Gu, 2007) by incorporating the cellular metabolic rates into the equation of balance of mass for solutes (Bedford and Drumheller, 1983):

$$\partial(\phi^{\mathrm{w}} c^{\alpha})/\partial t + \nabla \cdot (J^{\alpha} + \phi^{w} c^{\alpha} v^{\mathrm{s}}) = Q^{\alpha}, \tag{1}$$

where J^{α} is the molar flux of solute α relative to the solid phase, ϕ^{w} is the tissue porosity (volume fraction of water), c^{α} is the concentration of solute α (per unit interstitial water volume), v^{s} is the velocity of solid matrix, and Q^{α} is the cellular metabolic rate of solute α per unit tissue volume.

Two neutral solutes (oxygen (O_2) and lactate), sodium ion (+), and chloride ion (−) were considered in this study. The inclusion of charged ions is necessary to balance the fixed, negative charges on the solid matrix since it can affect the swelling pressure and swelling strain even at equilibrium under static loading. The consumption rates of oxygen were pH dependent (Bibby et al., 2005) and given as (Huang et al., 2007):

$$Q^{\mathrm{O_2}} = -\frac{V'_{\max}(pH - 4.95)c^{\mathrm{O_2}}}{K'_{\mathrm{m}}(pH - 4.59) + c^{\mathrm{O_2}}}\rho^{\mathrm{cell}}, \tag{2}$$

where the unit of $c^{\mathrm{O_2}}$ is μM, $V'_{\max} = 5.27$ nmol/million cells-hr for NP cells and 3.64 nmol/million cells-hr for AF cells, and $K'_{\mathrm{m}} = 3.4$ μM for NP cells and 12.3 μM for AF cells. Since there is no information about the lactate