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What is the mechanism behind biological ferroelectricity?

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ABSTRACT

Biological tissues are soft, amorphous and often appear to possess a high degree of material symmetry. This would appear to preclude a phenomenon like ferroelectricity which, in the world of hard materials, only occurs in selected crystalline materials that are non-centrosymmetric. Recent experiments, however, indicate the presence of ferroelectricity in soft biological entities such as the protein elastin—a large biopolymer found in the extracellular domains of most tissues. In this letter, we present a model and an explanation for this intriguing observation. Based on a very simple physical hypothesis, we develop an analytical statistical mechanics model that, coupled with insights from molecular dynamics, provides a plausible mechanism underpinning biological ferroelectricity. Furthermore, we predict for the first time, piezoelectric properties of tropoelastin, a precursor/monomer of elastin—properties that are not easily obtained from experiments. Specifically we find that the piezoelectric constant of tropoelastin is larger than any known polymer.

1. Introduction

The presence of electromechanical coupling in biological materials and its physiological significance has been an active area of research over the last several decades. Piezoelectricity was first discovered in bone samples in 1957 [1]. Shortly after that, in 1966, the pyroelectric phenomenon was also discovered in bones [2]. However, ferroelectricity, the rarest electromechanical coupling, was not observed in biological materials until 2012 when it was shown that the tissue from porcine aortic walls does indeed exhibit all the tell-tale signatures of ferroelectricity typically associated with hard ceramics like Barium Titanate or Lead Zirconium Titanate [3]. The main component of these tissue samples is a protein called elastin.

Elastin is found throughout various connective tissues. Aptly named, elastin is responsible for the elastic response of tissues, allowing the tissues and the relevant organs to retain their original shape after loading [4]. Elastin proteins are in fact long polymers composed of many monomer proteins called tropoelastin. While we typically think of monomers as small molecular units, a single human tropoelastin protein consists of up to 792 amino acids, or roughly 10,000 atoms [5]. The form most commonly used in experiments, and here for comparison, is a recombinant form of tropoelastin which is composed of 698 amino acids (isoform SHELd26A) or about 8,700 atoms [3,6,7].

Despite the discovery of ferroelectricity in biological materials, its physiological role is still somewhat unclear although there are several speculations regarding its significance. There is a large amount of elastin present in arteries, so one proposed possibility is that ferroelectric switching may limit the shear stress due to pulsatile flow [8]. Another notion that has been advanced is that the dipole switching associated with ferroelectricity may provide some type of memory function for our tissues and organs to retain their original shape. Either way, we feel that it is unlikely that the occurrence of this phenomenon is purely coincidental. Indeed, somewhat tentatively, the experimental results show that biological ferroelectricity

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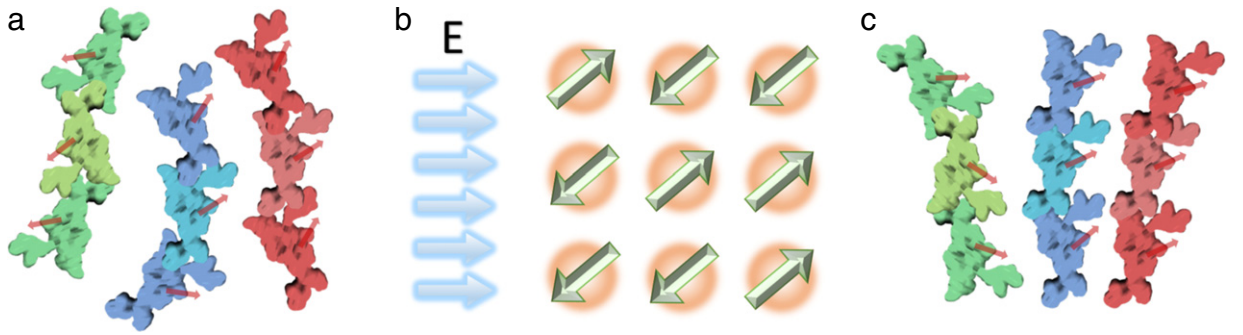

Fig. 1. (a) Two dimensional representation of amorphous elastin. Separate elastin polymers are shown in different colors, and shading is used to differentiate between tropoelastin monomers. The dipole moment associated with each monomer is shown by the red arrow. (b) Illustration of the two dimensional 2 site statistical mechanics model with general θ. Notice that $\phi_1 = 0$ and $\phi_2 = \frac{\pi}{2}$ are used as the angles made between a vector connecting two adjoining dipoles and the applied electric field. (c) Mostly aligned dipoles and monomers after/during application of an electric field sufficient for switching. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

in elastin is possibly linked to the aging process [9]. As our bodies age, elastin undergoes the slow process of glycation, i.e. the spontaneous, unassisted bonding of glucose to a protein. Once the concentrations of glucose bonded to elastin proteins are sufficiently high, such as during old age or in diabetics, elastin begins to degrade which may cause results such as lowered elastic response. This loss of elasticity can be easily viewed as wrinkles in the skin [10,11]. Coinciding with the change of mechanical properties, the ferroelectric response of elastin is also suppressed by the increasing severity of glycation [9]. Much research has been done on advanced glycation end-products (AGE), but none have focused on the effect of AGE on ferroelectricity [12,13]. The reason for the loss of ferroelectricity is again unknown, but as a first step, a reasonable starting point would be to first establish the mechanism underpinning biological ferroelectricity—this is the main goal of the current letter.

2. Hypothesis and central idea

Inspired from ideas already explored in the context of semi-crystalline polymers such as polyvinylidene fluoride (PVDF) [14], we propose the following picture of the likely mechanism for ferroelectricity in proteins like tropoelastin. We consider elastin as consisting of an amorphous array, or matrix, of tropoelastin monomers—each of which possesses a dipole moment. Alternatively, we could say that elastin is represented by a set of frozen dipoles which thermally fluctuate about some equilibrium angle. When subjected to an external electric field, the dipoles undergo some rotation and attempt to align with the field (Fig. 1). Ferroelectric switching, as will be elucidated in later sections, emerges as a cooperative effect. The overview of our modeling approach is as follows: (i) the statistical mechanics model of fluctuating frozen dipoles is adapted for the present case; (ii) molecular dynamics is used to estimate quantities like the monomer dipole moment (which feeds into the statistical mechanics model) and (iii) experimental observations are used to estimate the coercive field.

A comparison of the model with experimental observations provides reasonable assurance that our model and proposed mechanism is plausible. Furthermore, based on this model, we present numerical predictions of untested properties of tropoelastin—such as piezoelectric coefficient.

3. Biological ferroelectricity model

In the following (Section 3.1) we first present the statistical mechanics model that translates the physical picture of the preceding section into a simple analytical model. Molecular dynamics calculations, that connect with the analytical model, are presented in Section 3.2 and in subsequent subsections, we explore their implications (3.3 and 3.4).

3.1. Statistical mechanics framework

The model followed here was originally proposed by Broadhurst and Davis for PVDF [14]. They assume that the material is a collection of dipoles that thermally fluctuate; the dipoles interact only with the applied electric field, and the dipole–dipole interactions are purposefully neglected. In the general case, a dipole will have a probability, f_i, of being found in a preferred orientation specified by θ_i which is the angle between the applied electric field, E and the dipole orientation.

We start with the Helmholtz free energy of the system. While elastin is not crystalline, or even semi-crystalline, we approximate it as an evenly spaced set of dipoles allowing the use of terms such as the lattice energy of the crystal given by:

$$-U_0 \sum_{i=1}^{n} f_i^2 \tag{1}$$

where U_0 is the difference in energy between a filled lattice site and an empty site, and n is the number of preferred orientations in the "crystal". Here U_0 may be thought of as a material property which governs the ability of dipoles to change orientations. Our subsequent molecular dynamics results indicate that this scenario, even thought idealized, is a reasonable approximation (and price to pay) for analytical results. In the case of a two-site or two-orientation model, the system can be solved analytically.