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Hierarchical Internal Variables Reflecting Microstructural Properties: Application to Cardiac Muscle Contraction

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Abstract

The formalism of internal state variables is proposed for describing the processes of deformation in muscles. Due to the complicated hierarchical microstructure of soft tissues where macroscopic stress states depend upon the sliding of molecules and ion concentrations, the internal variables are switched in successively forming a certain hierarchy. This hierarchy is a general property for complicated materials with different microscopic processes influencing the macroscopic behaviour. Based on that property a novel concept of hierarchical internal variables is defined (up to the knowledge of authors first time) and embedded into the framework of the existing formalism. The discussion based on an example of cardiac muscle contraction illustrates the advantages of this approach.

1. Introduction

The concept of internal varables has its origin in thermodynamics with first ideas in the description of reacting chemical systems [1]. Contemporary understanding has recently been reviewed by Maugin and Muschik [2] in the general context of non-equilibrium thermodynamics. It rests upon the assumption that the thermodynamic state is determined not only by observable variables χ like strain or stress but also by internal variables α hidden to the external observers. Such internal variables can be [2, 3]: a vector field d called direction in the case of liquid crystals; the damage parameter $D(0 \leq D \leq 1)$ in modelling the localization of damage; cumulated plastic strain in plasticity with hardening, etc. The formalism advocated in [2, 3] is based on continuum mechanics and includes a dissipation potential $\mathscr{D}$ from which the governing equations for internal variables are deductible. Given the Helmholtz free energy ψ and the dissipation potential $\mathscr{D}$, the constitutive equations can easily be derived. This concept shows also clearly the difference between the observable and

the internal variables. Observable variables are inertial governed by a balance law with a kinetic energy (i.e. an inertia), internal variables, however, do not possess inertia and are governed by the kinetic equations. The choice of an internal variable depends upon the level of observation, or as stated by Maugin [3] "is a matter of decision at outset from the part of the scientist". For example, temperature is usually considered as an observable variable, although the classical thermoelasticity is based on the Fourier law, the modified theories [4, 5] include thermal relaxation. However it has been shown [6] that treating temperature as in internal variable, a model evolution can be derived describing also the temperature shocks more accurately than the widely known Taylor shocks modelled by Burgers-type equations.

To the knowledge of the authors, all studies dealing with internal variables take them as opposed to observable (controllable) variables reciprocally. In other words, if a constitutive equation, say for stress σ is written like

$$\sigma = \sigma(\chi, \alpha), \tag{1.1}$$

then the evolution law for α is written like

$$\dot{\alpha} = f(\chi, \alpha). \tag{1.2}$$

The internal variable may be a scalar only or a vector with several components.

There are microstructured materials where such an approach does not hold. The reason is a hierarchical structure of a microstructure where processes differ between themselves due to different space and time scales. The best example of this kind is a living tissue – a muscle. Muscle contraction depends strongly on sliding filaments that in turn depend on oxygen and "food" supply, i.e. on cell energetics and muscle activation. Treating stresses in a muscle as observable variables, the internal variables form a hierarchy related to various structural hierarchies of the muscle. The main aim of this paper is to analyse such an example and to formulate for the first time the basic concepts of hierarchical internal variables. In Section 2 we represent the conventional formalism of internal variables following Maugin [3]. This is a preliminary basis for the following discussion. Section 3 describes first the hierarchical microstructure of muscles and the physiological background. Second, the extended model of the cardiac muscle contraction is given within the framework of internal variables. For this purpose, Huxley equations are used in the mechanochemical theory of the contraction [7]. Another variant is based on the distribution – moment approximation [8, 9]. The discussion in Section 4 leads to the formulation of the novel concept of hierarchical internal variables that bridges the formalism of continuum mechanics with the physiological modelling. The most important point of this concept is the clear thermodynamical basis applied to the various scales that gives the estimates of relaxation times and entropy fluxes for the various hierarchies.

2. Formalism of Internal Variables in Continuum Mechanics

In what follows, the general ideas of Maugin and Muschik [2] and Maugin [3] are used to represent the formalism of internal variables for later comparison. The notations are conventional [2].

We intend to define the thermodynamic state of a system that involves the observable state variables χ (e.g. elastic strain and temperature) and a certain number of internal variables α. The dependent variables (e.g., the stress) must be simultaneously a function of both

$$\sigma = \sigma(\chi, \alpha), \tag{2.1}$$

which must be complemented by an evolution law

$$\dot{\alpha} = f(\chi, \alpha) + g(\chi, \alpha)\dot{\chi} \tag{2.2}$$

describing the temporal evolution of the variable α.

The Clausius-Duhem inequality governing a mechanical process in a continuum is [2]

$$-(\dot{\psi} + S\dot{T}) + \boldsymbol{\sigma} : \dot{\boldsymbol{\varepsilon}} + \nabla(T\mathbf{k}) - (\mathbf{S} \cdot \nabla)T \geq 0, \tag{2.3}$$

where ψ is the Helmholtz free energy, $\boldsymbol{\sigma}$ is the Cauchy stress, $\boldsymbol{\varepsilon}$ the strain tensor, S the local entropy and T the temperature while

$$\mathbf{S} = \frac{1}{T}\mathbf{q} + \mathbf{k}, \tag{2.4}$$

where $\mathbf{k}$ is an extra entropy flux (later we see that it will be related to the internal variable) and $\mathbf{q}$ is the heat flux.

It is assumed that $\boldsymbol{\varepsilon}$ is split up in an elastic part $\boldsymbol{\varepsilon}^e$ and an anelastic part $\boldsymbol{\varepsilon}^p$

$$\boldsymbol{\varepsilon} = \boldsymbol{\varepsilon}^e + \boldsymbol{\varepsilon}^p. \tag{2.5}$$

The free energy function is assumed to be

$$\psi = \psi(\boldsymbol{\varepsilon}^e, T; \alpha, \nabla\alpha) \tag{2.6}$$

that is characteristic for plasticity, for example.

Calculating $\dot{\psi}$ from (2.6) and substituting it into (2.3), we obtain

$$\phi \equiv \boldsymbol{\sigma} : \dot{\boldsymbol{\varepsilon}}^p + \mathscr{A}\dot{\alpha} - (\mathbf{S} \cdot \nabla)T \geq 0. \tag{2.7}$$

Here the following derivatives are defined:

$$\boldsymbol{\sigma} = \frac{\partial \psi}{\partial \boldsymbol{\varepsilon}^e}, \quad S = -\frac{\partial \psi}{\partial T}, \tag{2.8}$$

$$\mathscr{A} = A - \nabla \cdot \mathbf{B} = -\frac{\delta \psi}{\delta \alpha}, \tag{2.9}$$

$$A = -\frac{\partial \psi}{\partial \alpha}, \quad \mathbf{B} = -\frac{\partial \psi}{\partial \nabla \alpha}, \tag{2.10}$$

$$\mathbf{k} = \frac{1}{T}\mathbf{B}\dot{\alpha}, \tag{2.11}$$

where

$$\frac{\delta}{\delta\alpha} = \frac{\partial}{\partial\alpha} - \nabla\cdot\frac{\partial}{\partial\nabla\alpha}. \tag{2.12}$$

Stronger conditions compared with (2.7) are

$$\phi_{intr} \equiv \boldsymbol{\sigma} : \dot{\boldsymbol{\varepsilon}}^p + \mathscr{A}\dot{\alpha} \geq 0, \tag{2.13}$$

$$\phi_{th} \equiv -(\mathbf{S}\cdot\nabla T) \geq 0, \tag{2.14}$$

where ψ_{intr} and ψ_{th} denote intrinsic and thermal dissipations, respectively.

Thermodynamical equilibrium is governed by (2.13) with

$$\dot{\varepsilon}^p = 0, \quad \mathscr{A} = 0, \tag{2.15}$$

assuming the temperature to be spatially uniform. We postulate now a dissipation potential

$$\mathscr{D} = \mathscr{D}(\boldsymbol{\sigma}, \dot{\alpha}, \boldsymbol{\varepsilon}^e, T, \alpha, \nabla\alpha) > 0, \tag{2.16}$$

which is convex in σ and $\dot{\alpha}$ so that

$$\dot{\boldsymbol{\varepsilon}}^p = \frac{\partial\mathscr{D}}{\partial\boldsymbol{\sigma}}, \quad \mathscr{A} = \frac{\partial\mathscr{D}}{\partial\dot{\alpha}}. \tag{2.17}$$

On the basis of (2.9) and (2.16) we obtain

$$\frac{\delta\psi}{\delta\alpha} + \frac{\partial\mathscr{D}}{\partial\dot{\alpha}} = 0, \tag{2.18}$$

that can be treated as an evolution equation (2.2) complementing the state law for the stress. Indeed, if $\mathscr{D}$ is quadratic in $\dot{\alpha}$ and ψ is first highly nonquadratic in α and second, quadratic and convex in $\nabla\alpha$. Then (2.17) yields

$$\dot{\alpha} = f(\alpha, \boldsymbol{\sigma}, \boldsymbol{\varepsilon}^e, T) + \nabla^2\alpha. \tag{2.19}$$

It has been shown [3] that depending on time scales (relaxation effects versus inertial ones), equation (2.18) can describe either dissipative or solitonic structures.

3. Internal Variables in Modelling Cardiac Muscle Contraction

3.1. Physiological background

We refer here to the fundamental treatises on cardiac performance [10, 11] focussing our attention to the mechanical behaviour of the heart. By contraction of heart muscles, the blood is pumped into circulation: the right ventricle is responsible for

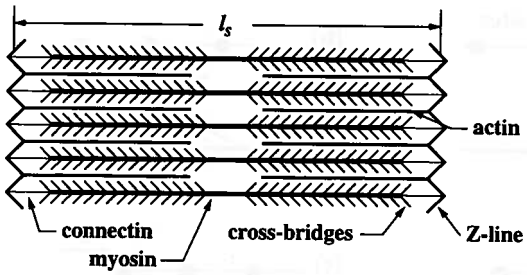

Fig. 1. A sarcomere of length l_s.

the pulmonary circulation, the left ventricle for the systemic circulation. In terms of continuum mechanics, the ventricles are thick-walked shells with complex geometry made of muscle-fibres with essential variation of their orientation, i.e. the muscle is a highly anisotropic material. Anisotropy of fibres is not the only important property – the fibres have a complicated microstructure. When activated by electrical signals originating from the sine-node, the stretching of fibres is caused by a complicated mechanism. In order to determine stresses in the walls of ventricles (myocardium) one has clearly to understand the leading factor of this mechanism.

For further discussion we need a more detailed description of muscle fibres [10–12]. The muscle fibre is made up by the bunches of myofibrils with a surrounding sarcotubular system. The myofibrils convert metabolic energy into mechanical energy and the sarcotubular system governs the behaviour of Ca^{2+} ions needed for activation. The structure of myofibrils is shown in Figure 1. A myofibril is composed of repeating units of myosin and actin filaments, called sarcomeres. The actin filament is made of a double helix of actin molecules with troponin molecules localized at every half-turn of the helix, the myosin filament consits of myosin proteins with certain spatially localized meromyosin molecules with heads resembling "golf-clubs". These molecules are shown in Figure 1 schematically as the "combs" called cross-bridges. The actin filaments are linked to each other at the Z-line, while a connecting molecule (connection) protects the sarcomere against overstretching linking also myosin filaments to this line.

The excitation of a muscle is triggered by an action potential from the conducting system. This potential releases Ca^{2+} ions that activate the troponin molecules at the actin filament so that they will be able to attach the heads of myosin molecules (see Fig. 2). The myosin molecules swivel and cause the sliding of filaments against each other, i.e. the contraction. The cycle is the following: attaching (influence of Ca^{2+} ions), swivelling (resulting in the sliding of filaments, i.e. powerstroke), detaching and resetting.

3.2. Microstructural models

It is clear that for modelling the mechanical stresses in such a complicated material as myocardium, its microstructure must be taken into account. The early phenomen-

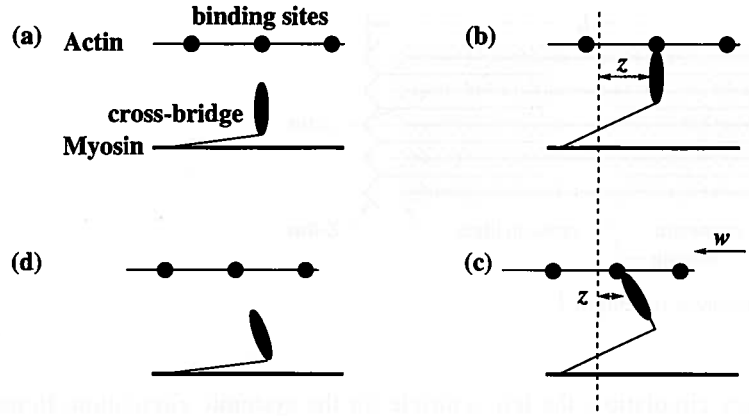

Fig. 2. A cycle of sliding the filaments (read clockwise): (a) to (b) – attaching; (b) to (c) – swivelling; (c) to (d) – detaching; (d) to (a) – resetting.

ological models have tried to describe the relationships between observed macroscopic data [13]. Contemporary microstructural models, however, try to take the physiological background and the structural peculiarities into account. Starting with Hill [13] and Huxley models [14, 15], contemporary modelling has put much emphasis on various modifications [8, 9, 12]. Continuum mechanics theory of the heart muscle has clearly reflected the physical meaning of microstructure (see, for example [12, 16, 17]) together with numerical solutions. Here we represent the basis models within the framework of the concept of internal variables, that will enable us later to generalize the basic ideas.

3.2.1. Huxley models

The total stress in the tissue can be split up into two parts

$$\boldsymbol{\sigma} = \boldsymbol{\sigma}_p + \boldsymbol{\sigma}_a, \tag{3.1}$$

where $\boldsymbol{\sigma}_p$ and $\boldsymbol{\sigma}_a$ denote passive and active stresses, respectively. Here $\boldsymbol{\sigma}$ is the Cauchy stress tensor. The passive stress $\boldsymbol{\sigma}_p$ results from elastic deformation of the myocardial tissue. For the sake of simplicity, the viscoelastic behaviour is neglected (for that see [17]). This is justified by the fact that the typical loading cycle is much shorter than the characteristic relaxation time of the myocardium [10]. The active stress is generated by sarcomeres and is directed parallel to the fibre orientation. Therefore we assume

$$\boldsymbol{\sigma}_a = \sigma_a \mathbf{e}_1 \mathbf{e}_1. \tag{3.2}$$

The passive stresses are determined from the Helmholtz free energy ψ (c.f. (2.8))

$$\boldsymbol{\sigma}_p = \frac{\partial \psi}{\partial \boldsymbol{\varepsilon}^e}. \tag{3.3}$$

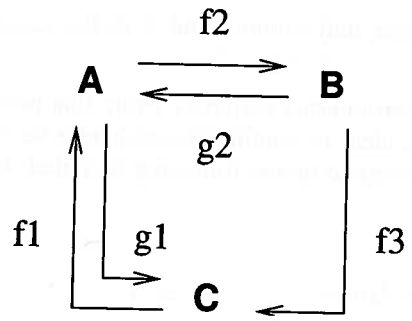

Fig. 3. Interaction between the binding states.

It is generally understood (Fung [18]) that ψ should be an exponential function of $\boldsymbol{\varepsilon}^e$, describing a hyperelastic material. For example,

$$\psi = -\frac{C}{2}(e^Q - 1), \tag{3.4}$$

where C is a material constant and

$$Q = b_1(\varepsilon_{11} + \varepsilon_{22} + \varepsilon_{33})^2 + b_2\varepsilon_{11}^2 + b_3(\varepsilon_{12}^2 + \varepsilon_{23}^2 + \varepsilon_{31}^2 - \varepsilon_{11}\varepsilon_{22} - \varepsilon_{22}\varepsilon_{33} - \varepsilon_{33}\varepsilon_{11}) \tag{3.5}$$

where b_1, b_2, b_3 are positive material constants and ε_{ij} the components of $\boldsymbol{\varepsilon}^e$.

The active stress $\boldsymbol{\sigma}_a$ must depend on the microstructure and in terms of continuum mechanics, is affected by internal variables. First, the force on actin molecules depends on the distance z between attached cross-bridge and the nearest acting site. Denoting by A and B the two "strong" binding states and by C the "weak" binding state [19] we recognize that cross-bridges produce mechanical forces in states A and B. The corresponding interaction scheme is shown in Figure 3 with kinetic constants f_i and g_i between the states. The forces are calculated by

$$F_A = K_A z, \tag{3.6}$$

$$F_B = K_B z, \tag{3.7}$$

where K_A, K_B are elastic constants. Further we denote $K_A = K$, $K_B = K$. The total force developed by myofibrils depends on the distribution of the cross-bridges. Let $n_A(z)$ and $n_B(z)$ be the relative amounts of cross-bridges between z and $z + dz$ in states A and B, respectively. Due to the independence of cross-bridge and actin sites distributions, the cross-bridges are uniformly distributed in z over an interval d. The active stress σ_a is then found from the expression

$$\sigma_a = \frac{ml_sK}{2d}\left(\int_{-d/2}^{d/2} n_A(z)dz + \int_{-d/2}^{d/2} n_B(z)dz\right), \tag{3.8}$$

where m is the number of cross-bridges per unit volume and l_s is the sarcomere length.

The variables n_A and n_B are nothing else than internal variables. From that point, the physiological model will be built along the ideas of continuum mechanics leading to the novel concept. Internal variables are governed by the following (coupled) kinetic equations [7]

$$\frac{\partial n_A}{\partial t} + w\frac{\partial n_A}{\partial z} = f_1 n_C + g_2 n_B - (g_1 + f_2)n_A, \tag{3.9}$$

$$\frac{\partial n_B}{\partial t} + w\frac{\partial n_B}{\partial z} = f_2 n_A - (g_2 + f_3)n_B, \tag{3.10}$$

where w is the velocity of lengthening and n_C is the amount of cross-bridges in the state C that does not produce force. The whole relative amount of activated cross-bridges in A must satisfy the summation rule

$$A = n_C + n_A + n_B. \tag{3.11}$$

This activation parameter A is again an internal variable but it affects not the observable variable σ_a directly but within kinematics of internal variables n_A and n_B. The activation parameter is in turn governed by the kinetic equation [15]

$$\frac{dA}{dt} = c_1(l_s)[Ca^{2+}](1 - A) - c_2(l_s)A \tag{3.12}$$

with

$$c_1 = const., \tag{3.13}$$

$$c_2 = c_{2MX} + c_{2F}\frac{l_{s(\max)} - l_s}{(l_s - l_{s(\min)})}. \tag{3.14}$$

Rewriting (3.12) in a form used in the formalism of internal variables [2, 3]

$$\frac{dA}{dt} = -(c_1(l_s)[Ca^{2+}] + c_2(l_s))A + c_1(l_s)[Ca^{2+}], \tag{3.15}$$

it is easy to conclude that the relaxation time τ_A and the equilibrium value of the internal variable A depend on a new variable, namely upon the relative Ca^{2+} concentration $[Ca^{2+}]$. This must be governed by its own kinetic equation

$$\frac{d[Ca^{2+}]}{dt} = f([Ca^{2+}]). \tag{3.16}$$

Instead of solving equation (3.15) with a known function $f(\ )$, the approximations of experimental curves can be used, for example:

$$[Ca^{2+}] = \begin{cases} \exp(5\frac{t-t_p}{t_p}), & \text{if } t \leq t_p, \\ \exp(-(\frac{t-t_p}{t_d})^2), & \text{if } t > t_p, \end{cases} \tag{3.17}$$

where t_d is the duration of one cycle of calcium changes and t_p the time to the peak.

To sum up, the active stress in myocardium is influenced

- directly by internal variables n_A, n_B;
- the internal variables n_A and n_B are influenced by another internal variable A;
- the internal variable A is influenced by the internal variable $[Ca^{2+}]$.

This scheme shows a *hierarchy of internal variables* reflecting the hierarchy of the processes in the tissue. A more detailed description of such a model with some quantitative data is given in [20].

3.2.2. Distribution-moment approximation

Zahalak [8] and Ma and Zahalak [9] have put the Huxley model into a continuum context using the so-called distribution moments. The active stress σ_a is described directly by one distribution moment α

$$\sigma_a = \mathscr{K} l_s \alpha, \tag{3.18}$$

where $\mathscr{K}$ is a constant, but this distribution moment is described by its kinetic equation

$$\frac{d\alpha}{dt} = \beta_3(t) b_1 - G_1(\alpha, \beta_1, \beta_2) + v\beta_1, \tag{3.19}$$

that depends on two other distribution moments β_1, β_2 and on the relative amount $\beta_3(t) = A(t)$ of active cross-bridges. The governing equations for those are

$$\frac{d\beta_1}{dt} = \beta_3 b_0 - G_0(\alpha, \beta_1, \beta_2), \tag{3.20}$$

$$\frac{d\beta_2}{dt} = \beta_3 b_2 - G_2(\alpha, \beta_1, \beta_2) + 2v\alpha, \tag{3.21}$$

$$\frac{d\beta_3}{dt} = c_1(l_s)\gamma(1 - \beta_3) - c_2(l_s)\beta_2), \tag{3.22}$$

where $v = dL_s/dt$ and $\gamma = [Ca^{2+}]$ is the intracellular Ca^{2+} concentration (c.f. eq. (3.16)). Again, similarly to Section 3.2.1., there are three levels of internal variables.

4. Dicussion

In Section 2, the formalism of internal variables used in continuum mechanics (solid mechanics) has clearly demonstrated the need for determining elastic (free energy) and dissipative potentials. The variables in this formalism are either observable or internal. In Section 3, more complicated cases were described in order to model cardiac muscle contraction. The internal variables in this case form a hierarchy reflecting the hierarchy of microstructure. Based on that analysis we propose now the concept of hierarchical internal variables that reflects the ordering in the physical (physiological) structure of a material (tissue).

In general terms, the concept of hierarchical internal variables is the following:

1) A constitutive equation for a dependent variable, say σ, which depends on the observable variable χ and internal the variable α

$$\sigma = \sigma(\chi, \alpha). \tag{4.1}$$

2) The evolution law for α is

$$\dot{\alpha} = f(\chi, \alpha, \beta), \tag{4.2}$$

where β is another (second-level) internal variable influencing σ only through the dynamics of the first level internal variable α.

3) This evolution law for β is

$$\dot{\beta} = g(\chi, \alpha, \beta, \gamma), \tag{4.3}$$

where γ is the next (third-level) internal variable, influencing σ only through the dynamics of the second-level internal variable, etc.

4) The evolution law for γ is

$$\dot{\gamma} = h(\chi, \alpha, \beta, \gamma, \ldots), \tag{4.4}$$

etc.

Internal variables $\alpha, \beta, \gamma, \ldots$ from a hierarchy reflecting the hierarchical structure of the material (tissue in Section 3). In this hierarchy, the influence of a certain internal variable is defined at its own level and one level higher.

The internal variables $\alpha, \beta, \gamma, \ldots$ have all their own relaxation times $\tau_\alpha, \tau_\beta, \tau_\gamma, \ldots$. The process itself has a macroscopic time scale τ_p. It is useful to express the relation of τ_i, $i = \alpha, \beta, \gamma, \ldots$ over τ_p through the dimensionless Deborah numbers [21]

$$De_i = \tau_i / \tau_p, \quad i = \alpha, \beta, \gamma \ldots . \tag{4.5}$$

that serve as measures of the degrees of nonequilibrium of $\alpha, \beta, \gamma, \ldots$. The condition $De_i \ll 1$ should be satisfied [2, 21] when the dynamics of the internal variables has no influence on the process. The condition for hierarchical internal variables implies

the same order of all the τ_i's. In Section 3, the muscle contraction is described over one cycle of heart beat and therefore this condition is always satisfied. If, however, one of τ_i's is large, i.e. the change of this internal variable is slow, then the corresponding evolution law (kinetic equation) could be approximated by an algebraic equation. The internal variable is then "frozen" [2]. Such situations in cardiac dynamics may happen due to pathological changes in cell energetics, in other cases they could be influenced, for example, by nonmonotonous coupling effects or physical effects that change the structural behaviour.

The physiological model discussed here describes the cardiac muscle contraction. It is clear that a similar formalism can also be considered for skeletal muscle contraction as for example analysed by Piazzesi and Lombardi [22] using the original equations [7]. The latters, however, explain the rates of transmission between the several states only at one level of internal variables.

The cardiac output is related to the demands imposed on the heart during exercise. As pointed out by Alpert *et al.* [23], the heart may use several strategies to meet these demands (in our terms) at various levels of internal variables. Then the functional behaviour of the whole heart could be optimized either for all or some level of internal variables.

It is obvious that the extra entropy flux $\mathbf{k}$ depends also hierarchically on all the internal variables (cf. expression (13) and evolution laws (44), (45), and (46)). Since the force development by a cross-bridge in the force-producing state depends on the first derivative of the Gibbs free energy G with respect to the displacement variable z [7], G is related to the dissipative potential $\mathscr{D}$ through $\boldsymbol{\sigma}$. There are several interesting questions which need further analysis. For example, how to construct dissipative potentials $\mathscr{D}_\alpha$, $\mathscr{D}_\beta$, $\mathscr{D}_\gamma \ldots$ corresponding to each level of internal variables, what kind of models need the gradients $\nabla\alpha$, $\nabla\beta$, $\nabla\gamma, \ldots$ to be included into the evolution laws, etc. Generally speaking, further analysis with clear distinction between the hierarchical internal variables might cast light over the formation of dissipative structures.

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