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Biomimetic model of skeletal muscle isometric contraction: I. an energetic–viscoelastic model for the skeletal muscle isometric force twitch

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Abstract

This paper describes a revision of the Hill-type muscle model so that it will describe the chemo-mechanical energy conversion process (energetic) and the internal-element stiffness variation (viscoelastic) during a skeletal muscle isometric force twitch contraction. The derivation of this energetic–viscoelastic model is described by a first-order linear ordinary differential equation with constant energetic and viscoelastic coefficients. The model has been implemented as part of a biomimetic model, which describes the excitation–contraction coupling necessary to drive the energetic–viscoelastic model. Finally, the energetic–viscoelastic model is validated by comparing its isometric force–time profile with that of various muscles reported in the literature.

Keywords: Biomimetic; Skeletal muscle; Energetic; Viscoelastic; Isometric force twitch; Hill model

1. Introduction and background

The A.V. Hill model for skeletal muscle contraction is the classic macroscopic model that interrelates energetic events with intrinsic mechanical elements [1]. The model is directly applicable to skeletal muscle contractions in which external shortening occurs under constant external load (an isotonic contraction). However, this model is not applicable (either energetically or mechanically) to skeletal muscle contractions in which the external length remains constant during force development

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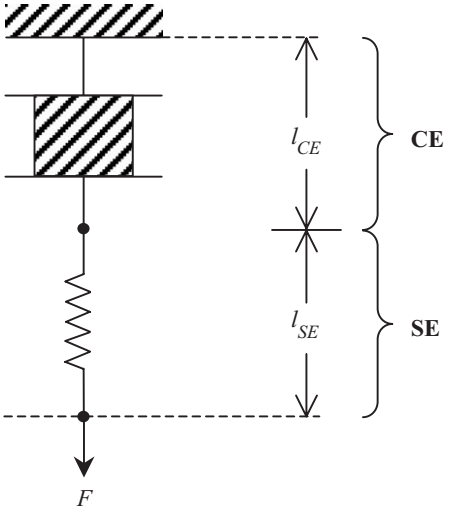

Fig. 1. A.V. Hill (1938) model.

(an isometric contraction). Nevertheless, the Hill model [1] can provide the essential conceptual basis for developing a mechano-energetic model for isometric skeletal muscle contraction.

An excellent review has already been written on the strengths and limitations of the A.V. Hill model of skeletal muscle mechanics [2]. Consequently, this section will only focus on those aspects of the Hill model that represent the foundational starting point for the development of the isometric model developed in this paper.

The classic A.V. Hill paper described muscle heat (energetic) measurements that were the basis of a model for the mechanical behavior of skeletal muscle [1]. The experimental technique itself involved muscle contractions experiencing external length changes (shortening) under a constant load (an isotonic type of muscle contraction). Hill's observations on heat liberation (energetics) can be directly converted into an equation on muscle mechanics:

$$(P + a)(V + b) = b(P_0 + a). \tag{1}$$

The mechanical variables are the muscle load force (P) and its velocity-of-shortening (V). P_0 is an active state force (defined below for Eq. (4)), while 'a' and 'b' are coefficients used to obtain Eq. (1). Derivation of Eq. (1) from heat measurements has been nicely reviewed by Katz [3].

The basic model that is described by Eq. (1) is known as the two-element model (Fig. 1) and is applicable to isotonic-type contractions. The first element is called the series elastic element (SE) and is assumed to be a spring, with a length and stiffness determined by the instantaneous muscle force. The second element is called the contractile element (CE) and is assumed to be characterized as the force–velocity relation between the muscle instantaneous speed of shortening (V) and the instantaneous muscle force (P), as per Eq. (1). In an isotonic contraction, muscle force is constant and the Hill model implies that the muscle shortening velocity is the same as the shortening velocity of the CE (V_{CE}):

$$V = V_{\mathrm{CE}}. \tag{2}$$

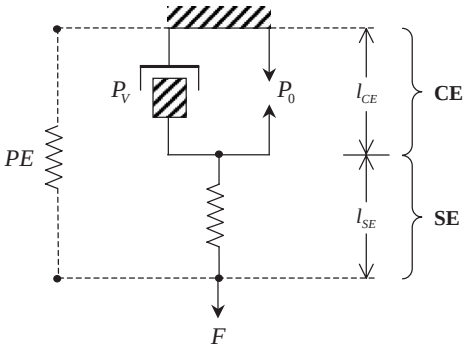

Fig. 2. Internal structure of the CE.

When Eq. (2) is substituted into Eq. (1), it is apparent that the force–velocity relationship of the CE is a restatement of Eq. (1):

$$(P + a)(V_{\mathrm{CE}} + b) = b(P_0 + a). \tag{3}$$

For the Hill model, the CE embodies the process of chemo-mechanical energy conversion. This separation of the elasticity and contractility of muscle into two different phenomenological entities connected in series is a fundamental characteristic of the Hill model. However, when one considers the generally accepted cross-bridge theory of muscle contraction, the separation is somewhat artificial because the cross-bridges are simultaneously contractile and elastic structures distributed uniformly throughout the contractile tissue. It is noted by Zalahak [4] that Eq. (1) can be rewritten so as to define internal structure to the CE:

$$P = P_0 - P_{\mathrm{V}} = P_0 - C(V; P_0)V, \tag{4}$$

where

$$C(V; P_0) = \frac{P_0 + a}{V + b}. \tag{5}$$

This algebraic manipulation can be interpreted as follows: the force (P) generated by the CE is the difference between an internal contractile force (P_0) and an internal viscous resisting force (P_{V}), which depends nonlinearly on the velocity. Fig. 2 is a diagram of the Hill model incorporating this internal decomposition of the CE. Note the internal arrangement of the structure of the CE of the Hill model showing the active state force (P_0) and the quasi-viscous internal resisting force (P_{V}). Shown in dashes is the parallel elastic element (PE), which may be added to model the passive elastic properties of unstimulated muscle.

Hill [5] defined the active state force (P_0) as the force that a muscle exerted when the CE was neither shortening nor lengthening—that is, when the CE velocity is zero. P_0 may be approximated as the isometric (tetanic) force (which is the force that a muscle exerts when the muscle length is neither shortening or lengthening)—that is, the muscle length velocity is zero. This is because a relatively high static stiffness is generally attributed to the SE (in series with the CE) when the muscle is maximally activated.

The Hill [1] model encounters its first problem during an isometric twitch contraction because the characteristic force–velocity relationship of the CE defined in Eq. (3), which embodies the process

of chemo-mechanical energy conversion, is only valid during an isotonic muscle contraction. To illustrate this point, consider an isometric muscle contraction so that the muscle force (P) is equal to the isometric force (P_0). Then substituting into Eq. (3):

$$(P_0 + a)(V_{CE} + b) = b(P_0 + a). \tag{6}$$

The result is that

$$V_{CE} = 0. \tag{7}$$

Hence, the force–velocity relationship reduces to an identity

$$(P_0 + a)b = b(P_0 + a). \tag{8}$$

The Hill [1] model encounters a second problem during an isometric muscle twitch contraction because there is a characteristic stiffness variation during the time course of the contraction. Gassar and Hill [6] found muscle to be especially rigid right after the initiation of stimulation (stiffness leading muscle force). Cecchi et al. [7] and Stein and Gordon [8] have carefully quantified changes in stiffness during isometric contraction, showing that stiffness leads force during the rising phase of tetanic isometric contraction, as if it was in part a function of activation. However, stiffness lags behind muscle force during relaxation (and by even more than it leads during activation). Such results are not consistent with the concept of stiffness being a static function of force and activation, and thus this important effect is very difficult to incorporate within the Hill model structure [2].

These two problems with the Hill [1] model relate directly to the purpose of this research paper. First, there is a need for an *energetic* Hill-type model that describes the chemo-mechanical energy conversion process when muscle contracts isometrically. Second, a need exists for a *viscoelastic* Hill-type model that relates P_0 to isometric muscle force in terms of the internal mechanical elements so that there is the characteristic stiffness variation during the time course of an isometric contraction. Consequently, this paper describes the development and validation of an energetic–viscoelastic Hill-type muscle model that satisfies these two needs.

2. Methods

2.1. Model development

2.1.1. Internal-element development

In the development of an energetic–viscoelastic model, the Hill [1] model (Fig. 1) is a good starting point. The internal structure of the contractile element is utilized as depicted in Fig. 2. The energetic–viscoelastic model (Fig. 3) introduces two additional elements into the model of Fig. 2. First, an active force generator (F_A) replaces P_0 of the Hill model. F_A defines the chemo-mechanical energy conversion during an *isometric* twitch contraction. F_A is a unidirectional active state force that generates physical shortening of the contractile element (l_{CE}) during the time course of an isometric twitch.

Second, a passive viscous element (c_2) is added in series with the SE (series elastic element) of the Hill model. c_2 (in combination with c_1) interacts with the series elastic spring (k) and defines a *second* viscoelastic time constant. The *first* viscoelastic time constant is defined by only c_1 (the

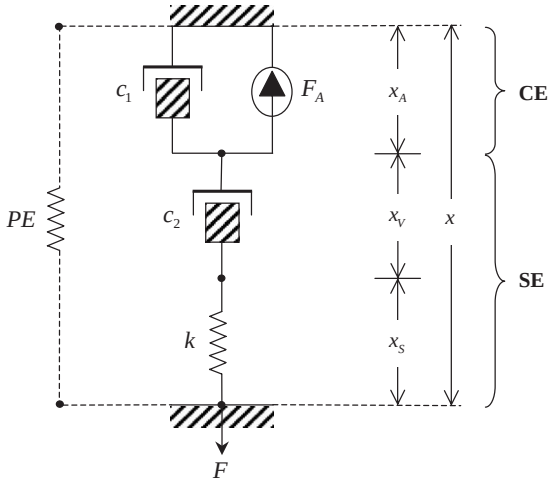

Fig. 3. Energetic–viscoelastic model (F_A is active state force).

Hill quasi-viscous element) interacting with k (the Hill series elastic element). The interaction of these *two* viscoelastic time constants accounts for variability of stiffness during an isometric muscle contraction.

Stiffness is considered a function of activation and in our model, F_A (active state force) is the force generated by calcium (C) activation of the contractile element. In other words, stiffness may be equated to activation force (F_A). c_1 is a Hill model "active" viscous "energy-dissipative" element that acts as follows. During the initial, rapid activation force (F_A) increase (or force development), F_A of the contractile element *leads* (i.e. is greater than) the actual isometric force (F) development (occurring at the end of the muscle) due to some c_1 energy absorption. This internal energy absorption occurs at a rate determined by the *first* viscoelastic time constant (defined above). Consequently, internal stiffness *leads* external force development. During the subsequent (more prolonged) activation force (F_A) decrease (or force relaxation), F_A of the contractile element lags behind (i.e. is lower than) the actual isometric force (F) development (at the end of the muscle) due to stored energy in the SE spring (k) being absorbed by c_2 (in combination with c_1), as represented by the second viscoelastic time constant. Consequently, internal stiffness *lags* external force decay.

2.1.2. Mathematical development

Mathematical development of the energetic–viscoelastic muscle model is guided by the "law of parsimony" [9]. The most parsimonious skeletal muscle model to describe the energetics and mechanics of isometric force twitch dynamics would be a first order linear ordinary differential equation with constant coefficients. In the development of such a model, we refer to Fig. 3 in deriving the governing differential equation.

Relating the external isometric force (F) to the internal forces

$$F = F_S = F_V = F_C + F_A, \tag{9}$$

where F_S is the series elastic force stored in the spring, F_V is the series viscous force absorbed by the "passive" dashpot (c_2), and F_C is the viscous force absorbed by the "active" dashpot (c_1) of the contractile element.

Relating the external velocity (V) to the internal velocities (where x, x_A, x_V, and x_S are per Fig. 3):

$$V = \dot{x} = \dot{x}_A + \dot{x}_V + \dot{x}_S. \tag{10}$$

For an isometric contraction, invoke the internal velocity constraint

$$-\dot{x}_A + \dot{x}_V + \dot{x}_S = 0. \tag{11}$$

Substituting into Eq. (11), the internal velocities as a function of their respective force

$$-\frac{F_C}{c_1} + \frac{F_S}{c_2} + \frac{(dF_S/dt)}{k} = 0. \tag{12}$$

For an isometric contraction, invoking the internal force constraint

$$F_A - F_C - F_S = 0. \tag{13}$$

Rearranging Eq. (12):

$$\frac{dF_S}{dt} = \frac{k}{c_1} F_C - \frac{k}{c_2} F_S. \tag{14}$$

Rearranging Eq. (13):

$$F_C = F_A - F_S. \tag{15}$$

Substituting Eq. (15) into Eq. (14):

$$\frac{dF_S}{dt} = \frac{k}{c_1} F_A - \frac{k}{c_1} F_S - \frac{k}{c_2} F_S. \tag{16}$$

Rearranging Eq. (16):

$$\frac{dF_S}{dt} = \frac{k}{c_1} F_A - k\left(\frac{1}{c_1} + \frac{1}{c_2}\right) F_S. \tag{17}$$

Define

$$\frac{1}{c_{12}} \equiv \frac{1}{c_1} + \frac{1}{c_2}. \tag{18}$$

So

$$c_{12} = \frac{c_1 c_2}{c_1 + c_2}. \tag{19}$$

Substituting Eq. (18) into Eq. (17) and rearranging

$$\frac{dF_S}{dt} = \frac{1}{\tau_2^*} F_A - \frac{1}{\tau_3} F_S, \tag{20}$$

where

$$\tau_2^* = \frac{c_1}{k}, \tag{21}$$

$$\tau_3 = \frac{c_{12}}{k} = \frac{c_1 c_2}{k(c_1 + c_2)}. \tag{22}$$

Referring to Eq. (9), note

$$F = F_S. \tag{23}$$

Substituting Eq. (23) into Eq. (20) and rearranging

$$\frac{dF}{dt} = \left(\frac{1}{\tau_2^*}\right) F_A - \left(\frac{1}{\tau_3}\right) F. \tag{24}$$

Now, define

$$F_A = \left(\frac{dF}{dC}\right) C, \tag{25}$$

where dF/dC is the change in force (F) with respect to the change in the concentration of free calcium (C) in the sarcoplasm.

Substituting Eq. (25) into Eq. (24):

$$\frac{dF}{dt} = \left(\frac{1}{\tau_2^*}\right)\left(\frac{dF}{dC}\right) C - \left(\frac{1}{\tau_3}\right) F, \tag{26}$$

which can be rewritten as

$$\frac{dF}{dt} = k_2^* C - k_3 F, \tag{27}$$

where

$$k_2^* = \left(\frac{1}{\tau_2^*}\right)\left(\frac{dF}{dC}\right), \tag{28}$$

$$k_3 = \frac{1}{\tau_3}. \tag{29}$$

2.2. Model implementation

In order to implement Eq. (27), it is necessary to define the time course of C. This was done by developing a second differential equation for the free calcium (C) in the sarcoplasm of skeletal muscle, as described in Part II of this study [10]:

$$\dot{C} = k_1^* P - k_2 C. \tag{30}$$

Since $\dot{C}$ is the central factor in the excitation–contraction coupling process of skeletal muscle, this required developing yet a third differential equation for the sarcoplasmic reticulum (SR) calcium

permeability (P) in response to an action potential (V_0):

$$\dot{P} = -k_1 P \tag{31}$$

for which at $t = 0$:

$$P(0) = P_0 = \alpha V_0. \tag{32}$$

The three differential equations (27), (30), and (31) and algebraic equation (32) constitute a phenomenological model of the excitation–contraction coupling process. The values of the coefficients (α, k_1, k_1^*, k_2, k_2^*, and k_3) of (27), (30), (31), and (32) were determined utilizing methods described by Neidhard-Doll et al. [10].

The energetic–viscoelastic model was then validated using MATLAB R12 for the solution of the three simultaneous differential equations (27), (30), and (31).

```
%C:\matlabR12\work\models\model_1.m
%Script file
tspan = [0 400]; %time in msec
x0 = [1; 0; 0];
[t, x] = ode45('diff_1',tspan,x0);
plot(t,x)

%C:\matlabR12\work\models\diff_1.m
%Function file
function xdot = model_1(t, x);
xdot = zeros(3, 1);
k1 = 0.1578;
k1star = 0.29;
k2 = 0.0628;
k2star = 0.0572;
k3 = 0.0172;
xdot(1) = -k1*x(1);
xdot(2) = k1star*x(1) - k2*x(2);
xdot(3) = k2star*x(2) - k3*x(3);
```

The investigators determined two characteristic features of the model-generated isometric force twitch curve: the contraction time (t_c) and the half-relaxation time ($t_{1/2}$). The values were then compared to known values in the published literature.

3. Results

Fig. 4 shows the SR calcium permeability (P), free calcium (C) in the sarcoplasm of skeletal muscle, and isometric force twitch (F), as a sequentially coupled system. The energetic–viscoelastic model is driven by SR membrane permeability changes and the resultant calcium flux. Note that P, C, and F are normalized with respect to their peak amplitudes.

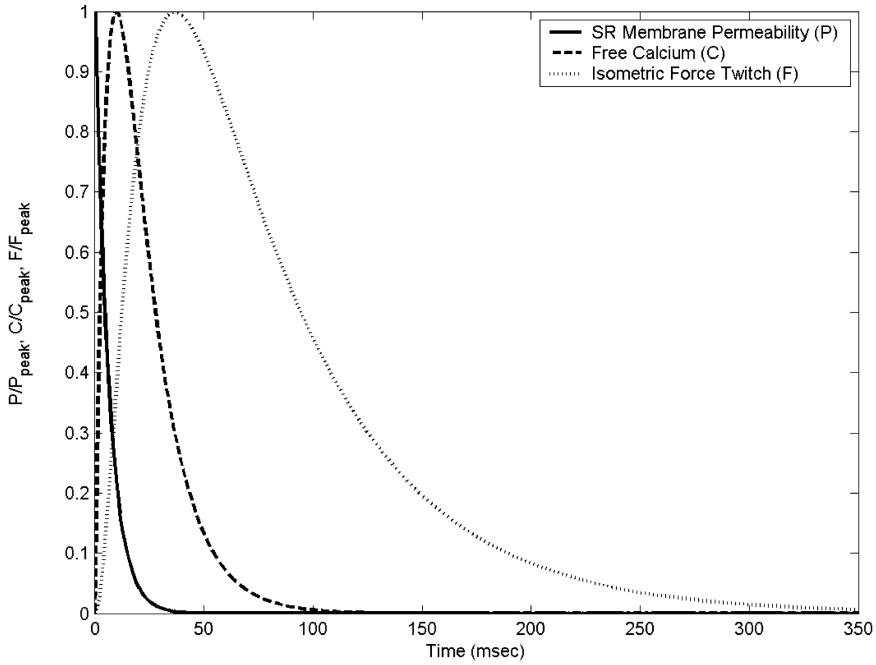

Fig. 4. SR membrane permeability (P), free calcium (C), and isometric force twitch (F) coupled in the energetic–viscoelastic model.

Fig. 5 illustrates the isometric force twitch generated by the energetic–viscoelastic model. Note that two isometric force twitch characteristics have been identified: t_c is approximately 36.2 ms and $t_{1/2}$ is 58.4 ms. Recall that t_c is the isometric muscle contraction time interval (i.e. time-to-peak), measured between the stimulus onset and the peak force amplitude. $t_{1/2}$ is the half-relaxation time, which denotes the interval of the isometric force twitch between peak amplitude and 50% of its peak amplitude.

Table 1 represents the isometric force twitch–time characteristics (t_c and $t_{1/2}$) for various skeletal muscles, animal species, and test temperatures. Where the literature has permitted, Table 1 also identifies the muscle fiber-type composition.

4. Discussion

The objective of this paper was to revise the 1938 Hill model [1], so that it described the chemo-mechanical energy conversion process (energetic) and internal-element stiffness variation (viscoelastic) during muscle contraction. This objective has been realized by deriving, implementing, and validating an energetic–viscoelastic model. The derived model is described by a first-order linear ordinary differential equation (Eq. (27)) that consisted of a constant energetic parameter (Eq. (28)) and a constant viscoelastic parameter (Eq. (29)). This is the most parsimonious form of the energetic–viscoelastic model. The law of parsimony [9] states that when considering an array of

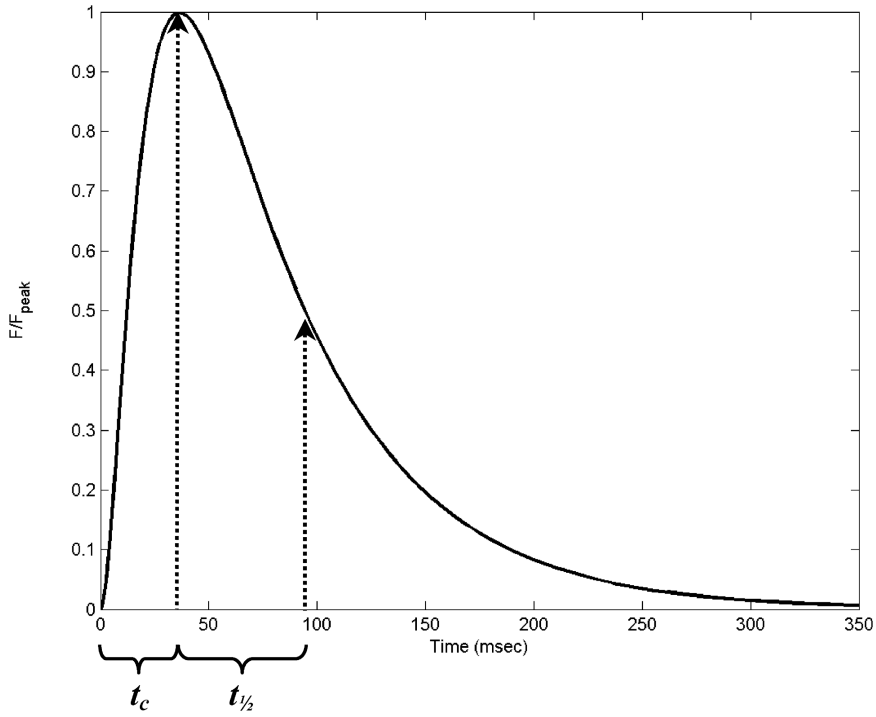

Fig. 5. Isometric force twitch generated by biomimetic model (t_c is 36.2 ms; $t_{1/2}$ is 58.4 ms).

alternative models, one should chose the simplest model that satisfactorily describes the characteristics to be modeled.

Because the energetic parameter depended upon a time-varying intra-cellular calcium flux, implementation of the model required that it be driven by a pair of simultaneous differential equations. These equations described SR membrane permeability changes (Eq. (31)) and the resultant calcium flux (Eq. (30)). The collective set of the three differential equations ((27), (30), and (31)) represents a biomimetic model of isometric muscle contraction. The determination of the differential equation k-parameters was the essential and critical element in the actual implementation of the model (Fig. 4). The details of the derivation of these various k-parameters are presented in Part II of this paper by Neidhard-Doll et al. [10]. Validation of the model was then performed by determining two characteristic features (t_c and $t_{1/2}$) of the model-generated isometric force twitch (Fig. 5) and comparing these values with known values in the published literature (Table 1). In general terms, the t_c of the model (36.2 ms) is definitely within the minimum–maximum range (3.8 to 187 ms) from Table 1 with respect to various muscles, various species, and various temperatures. Also, the $t_{1/2}$ of the model (58.4 ms) is definitely within the minimum–maximum range (2.5 to 180 ms) from Table 1 when viewed across the board.

Closer examination of Table 1 allows a more specific identification of the muscle and species representative of the isometric force twitch shown in Fig. 5. The characteristic k-parameters of Eq. (27) represented by Eqs. (28) and (29) were derived from the experimental data of Sun et al. [13] and Cannell and Allen [14], both of which utilized frog anterior tibialis muscle. For details,

Table 1
Isometric force twitch–time characteristics for various skeletal muscles, animal species, and test temperatures

Muscle	Species	Fiber type	F_{Peak}	t_c (ms)	$t_{1/2}$ (ms)	Notes	Source
Anterior tibialis	Cat	81% Fast[a] (20% FOG, 61% FG) 19% Slow[a]	—	53	52	37°C	[11]
Anterior tibialis	Frog (Rana esculenta)	—	—	101.54± 4.62	143.08± 4.62	4.5°C (low temp.)	[12]
Anterior tibialis	Frog (Rana temporaria)	—	$(0.67 \pm 0.02)F_0$	73.2 ± 2.1	180.0 ± 10.6	2–4°C; F_0 = max tetanic force	[13]
Anterior tibialis	Frog	—	51.0 ± 1.8 mg	24.0 ± 2.0	18.0 ± 2.0	20°C	[14]
Anterior tibialis	Frog (Rana temporaria)	—	5.24 ± 0.07 mN	72.73 ± 3.6 ms	127.28 ± 3.6 ms	5–6°C	[15]
Dorsal interosseus (first)	Human	—	0.7 g	45 ms	25 ms	37°C (in vivo)	[16]
EDL	Rat	59% FOG[a] 38% FG[a] 3% SO[a]	0.275 mN	33.33 ms	46.67 ms	27 ± 2°C	[17]
Extraocular	Human	100% Fast[b]	—	3.80 ± 0.63	2.53 ± 0.63	37°C (in vivo)	[18]
Flexor digitorum longus (FDL)	Cat	32% FOG[a] 61% FG[a] 7% SO[a]	7.51 ± 0.68 N	23.3 ± 3.8	14.7 ± 3.9	37°C	[19]
Gastronemius	Human	50% Fast[b] 50% Slow[b]	—	12.66 ± 0.63	10.76 ± 0.63	37°C (in vivo)	[18]
M. peroneus longus (PerL)	Cat	Slow[c]	0.563 ± 0.093	29.6 ± 5.8	39.8 ± 9.1	37–38°C	[20]
M. peroneus longus (PerL)	Cat	Fast-fatigue sensitive[c]	3.0 ± 0.093	17.4 ± 2	17.7 ± 3.2	37–38°C	[20]
M. peroneus longus (PerL)	Cat	Fast-fatigue resistant[c]	—	19.5 ± 2.9	20.9 ± 3.3	37–38°C	[20]
M. peroneus longus (PerL)	Cat	28% FOG[a] 66% FG[a] 6% SO[a] 17% Slow[c]	—	23 ± 2	17 ± 2	37–38°C	[21]
Sartorius	Frog	—	—	76.47	41.18	0°C	[22]
Sartorius	Frog	—	51.7 ± 0.4	187.3 ± 2.5	268.3 ± 2.5	0°C	[23]
Soleus	Cat	0% FOG[a] 0% FG[a] 100% SO[a]	4.934 ± 0.484 N	73.4 ± 6.2	84.2 ± 8.6	37°C	[19]
Soleus	Human	80% Slow[b]	—	41.14 ± 0.63	34.18 ± 0.63	37°C (in vivo)	[18]

[a] [24] FOG: fast oxidative glycolytic; FG: fast glycolytic; SO: slow oxidative.
[b] [25].
[c] [26].

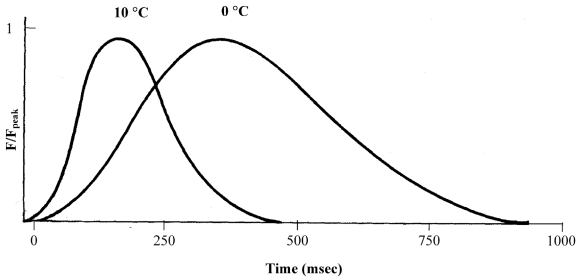

Fig. 6. Twitch force at muscle temperatures of 10°C and 0°C in the frog sartorius (normalized with respect to peak amplitude).

the reader should refer to Part II of this study [10]. Inspection of Table 1 indicates that the t_c for [13,14], respectively, is 73.2 and 24.0 ms. The $t_{1/2}$, respectively, for the same references is 180.0 and 18.0 ms. Recall that the t_c for the energetic–viscoelastic model is 36.2 ms, which is an intermediate value between 73.2 and 24.0 ms. Furthermore, the $t_{1/2}$ for the model (58.4 ms) is an intermediate value between 180.0 and 18.0 ms. This intermediate value is to be expected due to the difference in muscle temperatures between the two studies. Sun et al. [13] studied frog anterior tibialis muscle at 2–4°C, while Cannell and Allen [14] studied the same muscle at 20°C. This is a muscle temperature difference of 16–18°C.

The frog is a poikilothermic animal so that its core temperate rises or falls according to the surrounding ambient environmental temperature. As shown by Fig. 6, a muscle temperature difference of 10°C dramatically alters the time profile of an isometric force twitch [22]. The physiological explanation is that the reaction rate of enzymes is highly dependent on their temperature. This holds true for all enzymes including actin and myosin in skeletal muscle. Therefore, it is not surprising that contraction velocity for both skeletal and cardiac muscle is highly dependent upon the temperature of the contracting musculature. For example, a reduction in muscle temperature from 38°C to 28°C will cause approximately a 50% reduction in the contraction velocity for the medial gastrocnemius muscle of the cat, as shown in Fig. 7 [27]. In a similar manner, but for an isometric force twitch, a decrease in muscle temperature from 10°C to 0°C will cause an approximate doubling (slowing) of the isometric force-twitch time profile (Fig. 6). Consequently, Cannell and Allen [14] have the shorter t_c and $t_{1/2}$, while Sun et al. [13] have the distinctly longer t_c and $t_{1/2}$. In effect, the model predicted isometric force twitch (Fig. 5) would theoretically represent that of a frog anterior tibialis muscle at 10°C.

Knowing the specific muscle represented by the model allows us to approximate the fiber-type composition for the energetic–viscoelastic model. Table 1 does not directly provide such information for the two studies [13,14]. However, Ariano et al. [24] do indicate that cat anterior tibialis muscle is composed of 80% fast-twitch and 20% slow-twitch fibers. Although a different species than frog, the t_c of our model is about 70% of the characteristic t_c for Ruch et al. [11], and the $t_{1/2}$ of the energetic–viscoelastic model is about 110% of the $t_{1/2}$ for Ruch et al. [11].

The characteristic differentiation between fast- and slow-twitch muscle fibers is that the contraction time (t_c) has shorter duration for fast-twitch muscle fibers, when compared to slow-twitch muscle fibers. For example, Garnett et al. [25] reported contraction times ranging from 90 to 110 ms for

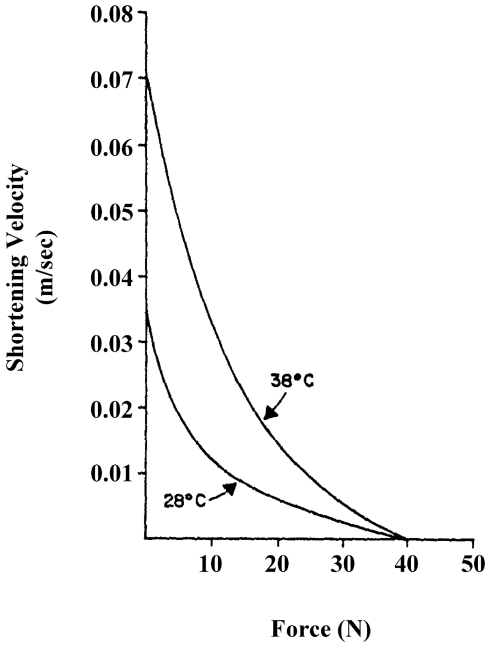

Fig. 7. The force–velocity relationship at muscle temperatures of 28–38°C in the cat medial gastrocnemius muscle.

slow-twitch motor units, and from 40 to 84 ms for fast-twitch motor units of humans. Consequently, it may be concluded that the isometric force-twitch time profile for the energetic–viscoelastic model is representative of a mixed fiber-type muscle of at least 50% fast-twitch and 50% slow-twitch fibers, and probably closer to 75% fast-twitch and 25% slow-twitch fibers.

In conclusion, this study has developed, implemented, and validated an energetic–viscoelastic model for the skeletal muscle isometric force twitch. However, the model implementation was dependent upon the biomimetic model of skeletal muscle isometric contraction with respect to the excitation–contraction coupling that precedes isometric force twitch development. Furthermore, the energetic–viscoelastic model validation was dependent (in part) on the specific k-parameter values utilized in the model differential equations. Consequently, in order to fully understand the implementation and validation of the energetic–viscoelastic model, it is necessary to understand the complete biomimetic model of skeletal muscle isometric contraction and the theory and methods utilized in determining the specific k-parameter values. Consequently, we now proceed to Part II of this paper [10].

5. Summary

This paper describes the development and validation of an energetic–viscoelastic muscle model that demonstrates the following features: (1) an *energetic* Hill-type model that describes the chemo-mechanical energy conversion proves when muscle contracts isometrically; and (2) a *viscoelastic* Hill-type model that relates active force P_0 to isometric muscle force (F) in terms of the internal

mechanical elements so that there is the characteristic stiffness variation during the time course of an isometric contraction.

Mathematical development of the energetic–viscoelastic muscle model has been guided by the "law of parsimony," which requires that the most parsimonious skeletal muscle model (describing the energetics and mechanics of isometric force twitch dynamics) should be a first-order linear ordinary differential equation with constant coefficients. The energetic–viscoelastic model then introduces two additional elements into the traditional Hill-type model. First, an active force generator (F_A) replaces P_0 of the Hill model. F_A defines the chemo-mechanical energy conversion during an isometric twitch contraction. F_A is a unidirectional active force that generates physical shortening of the contractile element during the time course of an isometric twitch. The result is a first-order linear ordinary differential equation consists of an energetic parameter constant and a viscoelastic parameter constant. The energetic–viscoelastic model has required that it be coupled with two other differential equations that describe the sarcoplasmic reticulum membrane permeability changes and the resultant calcium flux. The three differential equations constitute a biomimetic model of the excitation–contraction coupling process. MATLAB R12 was then utilized for the solution of the three simultaneous differential equations.

The resultant energetic–viscoelastic model was then validated with both general and specific data. In general terms, the contraction time of the model (36.2 ms) is definitely within the minimum–maximum range (3.8 to 187 ms) for various skeletal muscles in the reported literature. The half-relaxation time for the model (58.4 ms) is definitely in the minimum–maximum range (2.5–180 ms) for the reported literature. In specific terms, it was determined that the isometric force–time twitch of the energetic–viscoelastic model was reasonably approximated by that of frog anterior tibialis muscle contracting at 10°C. It was further determined that the model is representative of a mixed fiber-type muscle of at least 50% fast-twitch and 50% slow-twitch fibers.

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