ASSIGNMENT 2

Preliminary Assignment Week 11 - Muscle Modelling Programming

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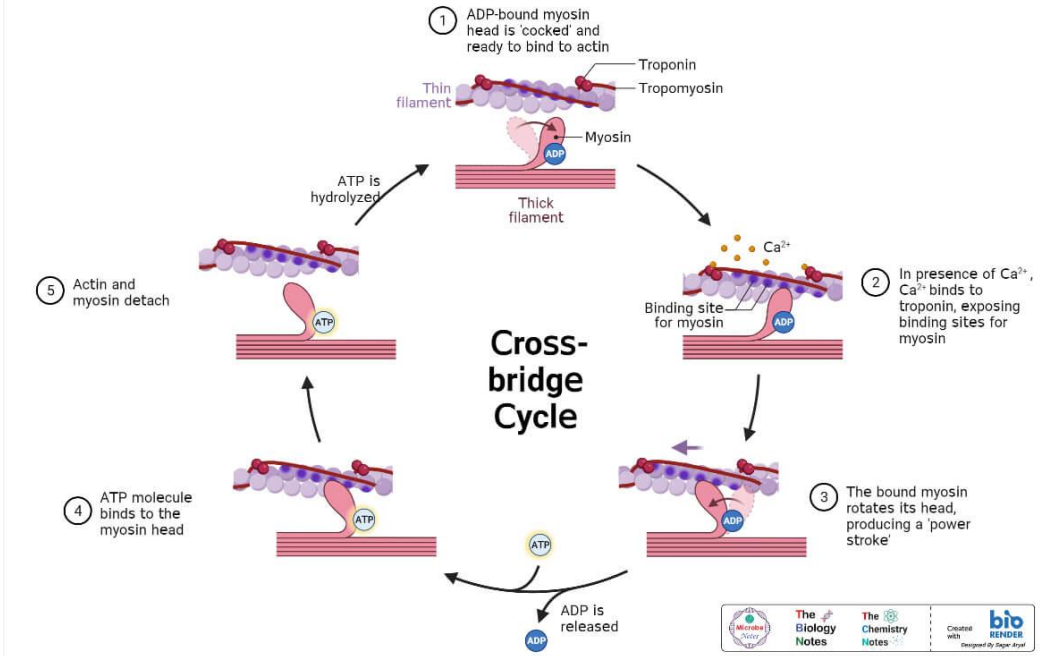
Assignment 3 – Preliminary Assignment Week 11: Assignment Project on Muscle Modelling

1. Describe what are Actin and Myosin, their functions, and mechanisms in controlling in muscle length change during contraction!

Actin and myosin are two main proteins involved in muscle contraction. Actin is a thin filament, while myosin is a thick filament. Both filaments are found in the sarcomere, the functional unit of muscle that enables contraction. When a muscle receives a signal to contract, the actin and myosin filaments interact and slide past each other, causing the muscle to shorten, or contract.

The primary function of actin and myosin is to generate force that enables muscle movement. During contraction, myosin heads bind to specific points on the actin, forming "cross-bridges." The myosin heads then "bend," or perform a power stroke, pulling the actin toward the center of the sarcomere, shortening the muscle. After one power stroke cycle is complete, the myosin releases the actin, ready to repeat the cycle if the signal for contraction continues.

Controlling changes in muscle length during contraction involves the repeated actin-myosin cross-bridge cycling mechanism. Energy from ATP (adenosine triphosphate) is required to move the myosin heads and to release them from the actin after the power stroke is completed. This mechanism allows the muscle to contract and relax in a controlled manner. The contraction process continues as long as there is a nerve signal and ATP supply. When the signal stops, the muscle returns to its original state. This mechanism allows the muscle to adapt to generating force at varying muscle lengths according to the needs of the body's activity. The general mechanism of the Actin-Myosin in controlling muscle length during contraction is like in the following picture:



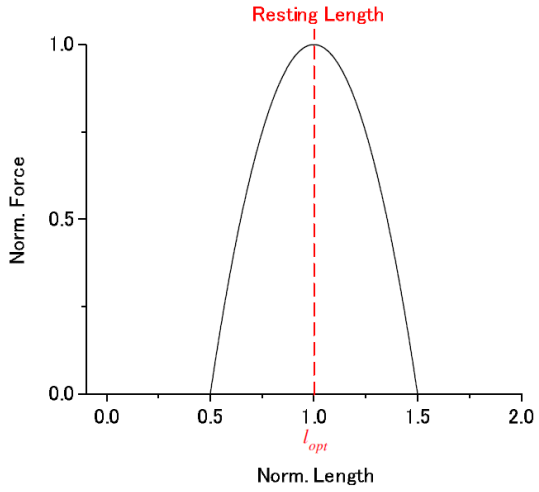
2. Explain about active and passive muscle force!

Active and passive muscle forces are two types of forces that play an important role in muscle contraction and movement. Active force is the force generated by muscle contraction directly due to the interaction between actin and myosin filaments. When a muscle receives a signal from the nervous system, calcium ions are released within the muscle fiber, allowing the myosin heads to bind to actin. This interaction produces an active force that shortens the muscle and allows movement. Active force is crucial in various physical activities such as lifting, pushing, or pulling.

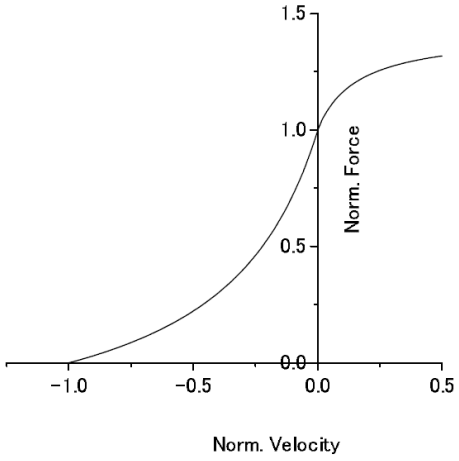
On the other hand, passive muscle force is the force generated when a muscle is stretched, without active contraction. This force arises from the elastic properties of the connective tissue structures surrounding the muscle fibers, such as tendons and elastin. When a muscle is stretched beyond its normal length, these passive elastic elements resist the stretch and create a force that tends to pull the muscle back to its resting length. Passive force plays a vital role in maintaining stability and posture and helps prevent muscle damage from overstretching.

3. Realize a computer program to produce a normalized f(l) and f(v) relationship curves, with c: 2.5, vmax= 3 m/det. 0.5lopt<=l(q)<=1.5lopt.

$$f(l) = 1 - \left(\frac{l(\theta) - l_{opt}}{0.5 l_{opt}}\right)^2$$



F-L Relationship



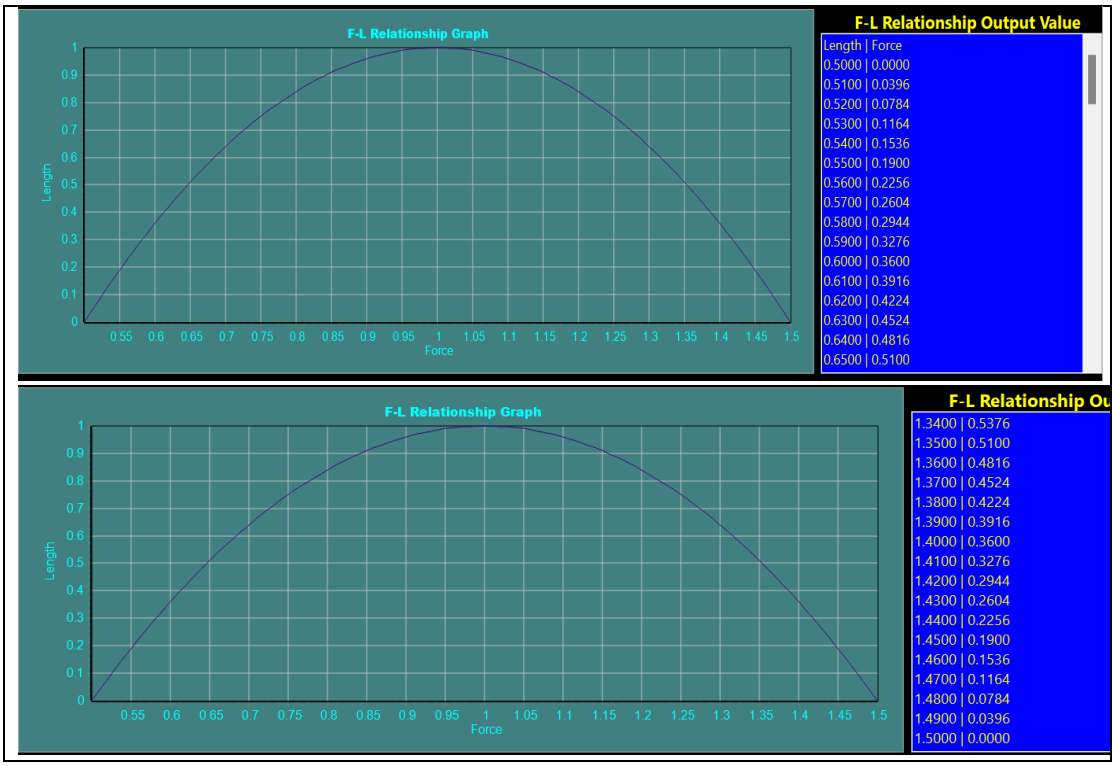
F-V Relationship

$$f(v) = \begin{cases} \frac{v_{max} - v}{v_{max} + 2.5v} & \text{if } l \leq l_{opt} \\ 1.3 - 0.3\frac{v_{max} - cv_{max}}{1 + c^2 v} & \text{if } l > l_{opt} \end{cases}$$

// Program code f(l)

```
procedure TForm2.Button1Click(Sender: TObject);
var
  n: Integer;
  ltheta, fl, lopt: Double;
  fls: string;
begin
  lopt := StrToFloat(Edit1.Text);
  Series1.Clear;
  ListBox1.Clear;

  ListBox1.Items.Add('Length | Force');

  for n := 0 to 100 do
  begin
    ltheta := (0.5 * lopt) + n * ((1.5 * lopt) - (0.5 * lopt)) / 100;
    fl := 1 - Power((ltheta - lopt) / (0.5 * lopt), 2);
    Str(fl:3:4, fls);
    ListBox1.Items.Add(FloatToStrF(ltheta, ffFixed, 8, 4) + ' | ' + fls);
    Series1.AddXY(ltheta, fl);
  end;
end;
```

//Program output, f(l) Computation Results in Graph and Output File.

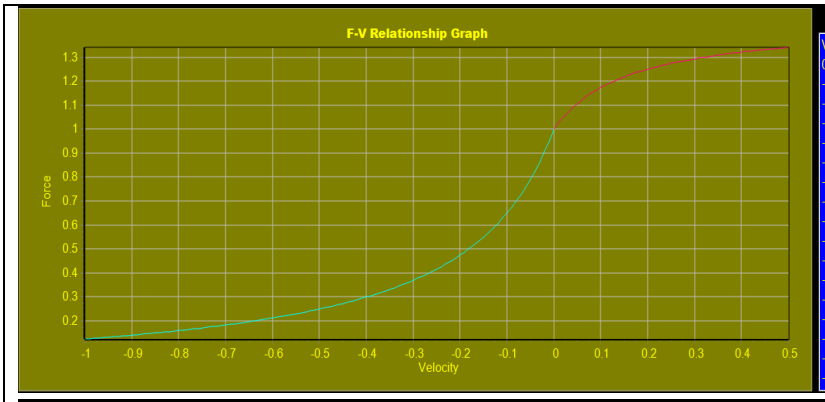


//Program Code f(v)

```
procedure TForm2.Button2Click(Sender: TObject);
var
  vmax, i: Integer;
  c, v, f_v: Double;
begin
  vmax := 3;
  c := 2.5;
  ListBox2.Clear;

  ListBox2.Items.Add('Velocity | Force');

  // Calculation for v from -1 to 0 (Concentric)
  for i := 0 to 100 do
  begin
    v := -1 + i / 100.0;
    f_v := (vmax - v) / (vmax + c * v);
    v := (-v - 1);
    f_v := f_v / 8;

    Series2.AddXY(v, f_v);

    ListBox2.Items.Add(Format('%.3f |%.3f', [v, f_v]));
  end;
```

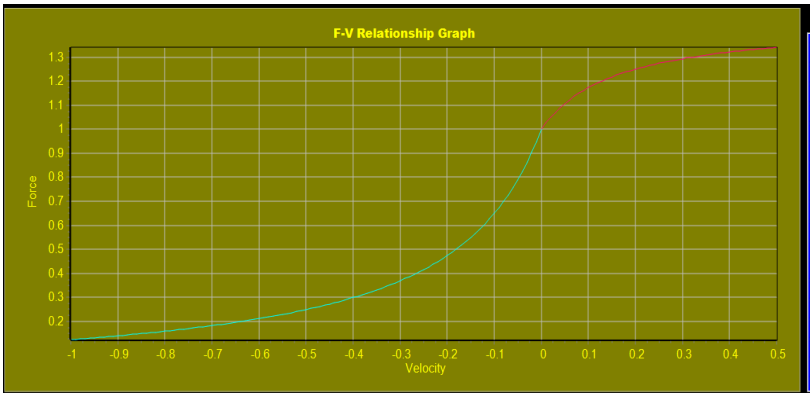
```
  // Calculation for v from 0 to 0.5 (Eccentric)
  for i := 0 to 50 do
  begin
    v := i / 100.0;
    f_v := 1.3 - (0.3 * ((vmax - c * vmax) / (1 + c * c * v)));
    f_v := (-f_v / 3) + 1.8833;

    Series3.AddXY(v, f_v);

    ListBox2.Items.Add(Format('%.3f |%.3f', [v, f_v]));
  end;
end;
```

//Program output, f(v) Computation Results in Graph and Output File.



F-V Relationship Output Value

Velocity	Force
0.000	1.000
-0.010	0.950
-0.020	0.905
-0.030	0.863
-0.040	0.825
-0.050	0.790
-0.060	0.758
-0.070	0.728
-0.080	0.700
-0.090	0.674
-0.100	0.650
-0.110	0.627
-0.120	0.606
-0.130	0.586
-0.140	0.568
-0.150	0.550
-0.160	0.533



F-V Relationship Output Value

0.330	1.303
0.340	1.306
0.350	1.309
0.360	1.312
0.370	1.314
0.380	1.317
0.390	1.319
0.400	1.321
0.410	1.324
0.420	1.326
0.430	1.328
0.440	1.330
0.450	1.332
0.460	1.334
0.470	1.336
0.480	1.337
0.490	1.339
0.500	1.341

ASSIGNMENT 2: MUSCLE MODELLING
REVISION

1.

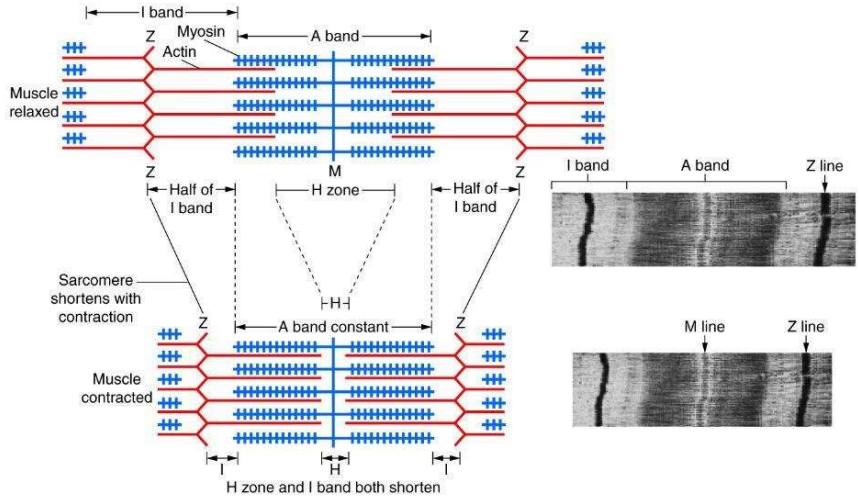


Figure 1. *Sarcomere when in Relaxion (above) and Contraction (below)*

Actin and myosin are the two primary myofilament proteins responsible for muscle contraction. Actin forms the basis of the "thin filaments," while myosin constitutes the "thick filaments." These filaments are meticulously arranged within the sarcomere, which is the fundamental contractile unit of a muscle fiber. The interaction between these filaments, governed by the sliding filament theory, generates the force required for movement. This process involves a detailed molecular mechanism known as the cross-bridge cycle, which directly controls the shortening and lengthening of the muscle.

Before examining the cycle, it is crucial to understand the structure of the sarcomere. The sarcomere is defined by Z-discs at its ends, which anchor the actin filaments. It features distinct bands: the **I Band** (Isotropic band) is a light-colored region containing only thin actin filaments, and the **A Band** (Anisotropic band) is a dark region spanning the entire length of the myosin thick filaments, including areas where actin and myosin overlap. Within the A band is the **H Zone** (from the German *heller*, meaning "brighter"), a central, slightly lighter area containing only thick myosin filaments. The change in muscle length is a direct result of changes within these bands. During contraction, the Z-discs are pulled closer together, causing the sarcomere to shorten. Consequently, the I Band and the H Zone both narrow, and in a maximal contraction, the H Zone can disappear entirely as the actin filaments are pulled towards the center of the sarcomere. The A Band, however, remains at a constant length because it is defined by the length of the myosin filament itself.

The mechanism driving this shortening is the **cross-bridge cycle**, a series of molecular events fueled by ATP. The cycle begins when a nerve impulse triggers the release of calcium ions (Ca^{2+}) from the sarcoplasmic reticulum. These calcium ions bind to troponin, a regulatory protein on the actin filament. This binding causes another protein, tropomyosin, to shift its position, exposing the myosin-binding sites on the actin filament.

Once these sites are exposed, the energized myosin heads (containing ADP and an inorganic phosphate, Pi) can bind to the actin, forming a "cross-bridge." This is followed by the "power stroke," where the myosin head pivots, releasing ADP and Pi. This action pulls the actin filament towards the M-line (the center of the sarcomere). For the muscle to continue contracting or to relax, the cross-bridge must detach. This critical step requires a new ATP molecule to bind to the myosin head, which breaks the bond between actin and myosin. Finally, the ATP is hydrolyzed back into ADP and Pi, which "re-cocks" the myosin head, returning it to an energized state, ready to form another cross-bridge if calcium is still present. This repeated cycling of cross-bridge formation, power strokes, and detachment pulls the thin filaments past the thick filaments, shortening the sarcomere and, ultimately, the entire muscle.

2. Muscle force can be categorized into two distinct types: active and passive force, which originate from different components and mechanisms within the muscle.

   Active force is the force generated by the muscle's contractile machinery through the cross-bridge cycle. This force is a direct result of the actin-myosin interactions as described previously. It is a metabolically active process, requiring a constant supply of ATP to fuel the formation and detachment of cross-bridges. The magnitude of the active force a muscle can produce is highly dependent on its length (the Force-Length relationship) and its velocity of contraction (the Force-Velocity relationship), which are the principles modeled by your computer program. This force is responsible for all voluntary movements, such as lifting, pushing, and pulling, as it is generated in direct response to signals from the nervous system.

   Passive force, on the other hand, is the force generated not by active contraction but by the stretching of the muscle's elastic components. This force does not require ATP and arises from the inherent elastic properties of the muscle's connective tissues (such as the epimysium, perimysium, and endomysium) and structural proteins like titin. Titin, a giant protein within the sarcomere, acts like a molecular spring, contributing significantly to passive force when the muscle is stretched beyond its resting length. Passive force is negligible at resting length but increases exponentially as the muscle is elongated. Its primary roles are to prevent the muscle from being overstretched and damaged, to store elastic energy, and to assist the muscle in returning to its original resting length after being stretched. This force is crucial for maintaining posture and stabilizing joints.

   [HILL-TYPE MUSCLE MODEL EXPLANATION]
   These two forces can be analogized with the Hill-Type Muscle Model. This passive force represents the viscoelastic properties of muscle and is modelled as the Parallel Elastic Component (PEC). Passive force occurs when the muscle is stretched beyond its resting length (Lopt or slack length) in the absence of neural activity. Anatomically, the main contributors are the Titin protein in the sarcomere and the connective tissue covering the muscle (epimysium, perimysium, endomysium). Passive force is non-linear and increases exponentially with changes in length. In the F-L curve, this force is zero at $L < Lopt$ and

begins to increase sharply when L > Lopt. This serves as a biological protection mechanism to prevent overstretching and sarcomere damage.

This Active Force is generated by the CC (Contractile Component) through the actin-myosin cross-bridge cycle powered by ATP hydrolysis and regulated by calcium (Ca2+) concentration. The magnitude of the active force is not constant, but rather a multivariable function of muscle length f(l) which follows a bell-shaped curve (Gaussian or parabolic). The maximum force (Fmax) occurs at the optimal length (Lopt) where the actin-myosin overlap is maximal. If it is too short (excessive overlap) or too long (reduced overlap), the active force decreases. Then there is the contraction velocity f(v) where the active force decreases when the muscle shortens rapidly (concentric) and increases drastically when the muscle is forced to lengthen (eccentric). And also the activation level a(t) which is the proportion of motor units recruited by the nervous system.

The total muscle force at time t can be formulated in general as:

$$Ftotal = [Fmax \cdot f(l) \cdot f(v) \cdot a(t)] + Fpassive(l)$$

This equation shows that the active force is the result of the modulation of the maximal capacity of the muscle by its position and movement, plus the passive restorative force of its elastic structure.

3. The new Force-Length (F-L) Relationship Graph can be seen as follows:

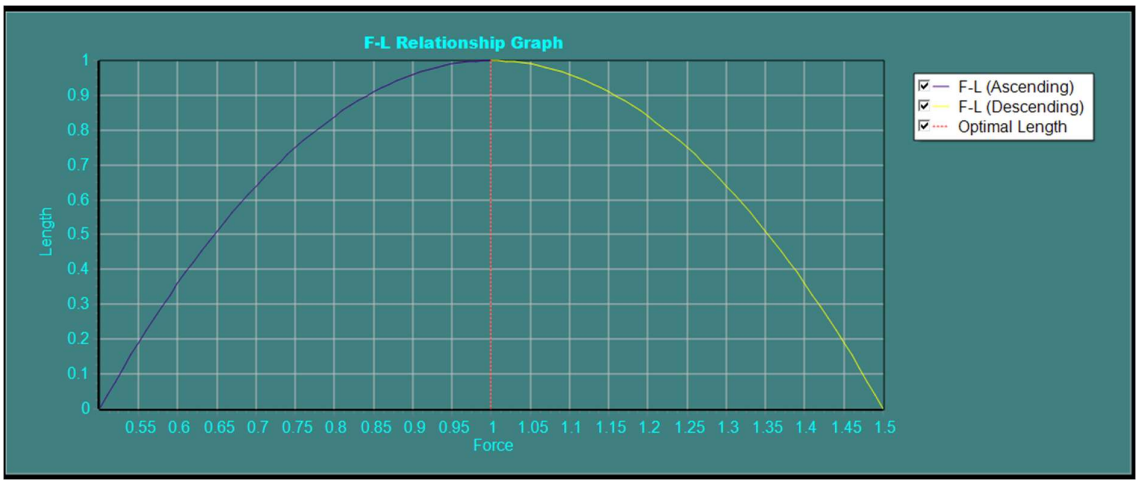


Figure 2. *Force-Length Relationship Graph*

4. The new Force-Velocity (F-V) Relationship Graph can be seen as follows:

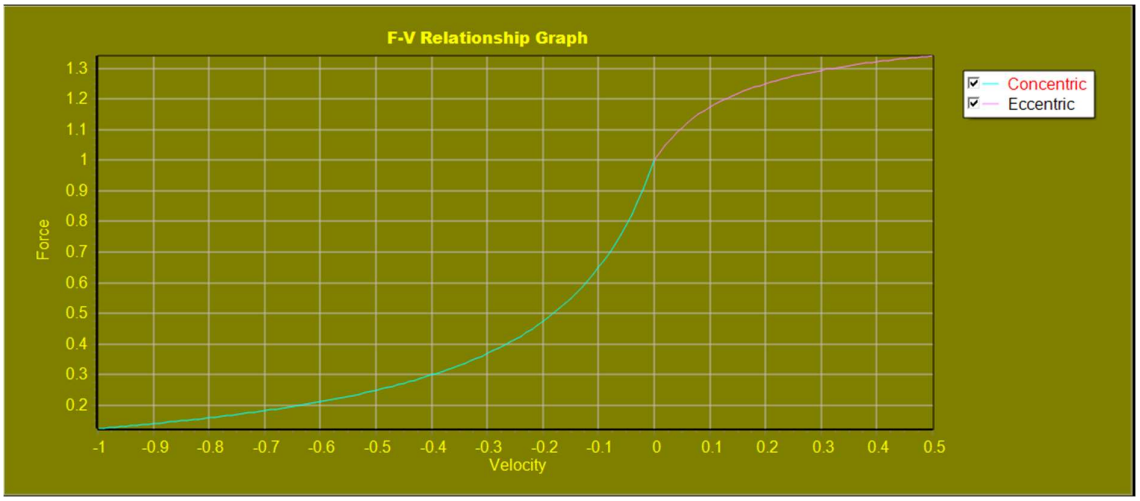


Figure 3. *Force-Velocity Relationship Graph*

ASSIGNMENT 2: MUSCLE MODELLING
REVISION 2

1. The new Force-Velocity (F-V) Relationship Graph can be seen as follows:

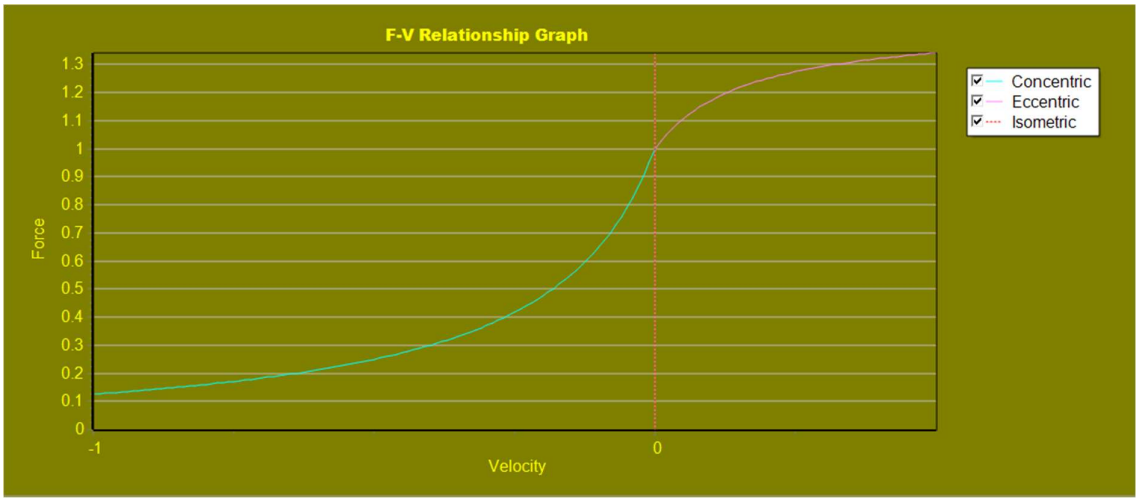


Figure 1. *Force-Velocity Relationship Graph*

Figure 1 illustrates the revised Force-Velocity (F-V) relationship graph, which is a fundamental concept in muscle biomechanics. This graph plots the amount of force a muscle can generate against the velocity at which it is contracting. The curve is divided into three distinct regions, representing the three types of muscle contractions. The vertical red line at a velocity of zero represents an **isometric contraction**. During this state, the muscle generates force without any change in its length, such as when pushing against an immovable object. The portion of the curve to the left, in the negative velocity range, depicts a **concentric contraction**, where the muscle shortens as it produces force. This region clearly shows an inverse relationship: as the speed of shortening increases, the maximum force the muscle can generate decreases. This explains why one can lift a very heavy weight slowly but can only move a very light weight quickly. The portion of the curve to the right, in the positive velocity range, represents an **eccentric contraction**, where the muscle is actively generating force while it is being lengthened by an external load. Critically, during an eccentric contraction, a muscle can sustain a significantly higher force than its maximum isometric force, which is vital for actions like controlled lowering of heavy objects and absorbing shock.

2. The Hill-Type Muscle Model Diagram that used in this assignment can be seen below:

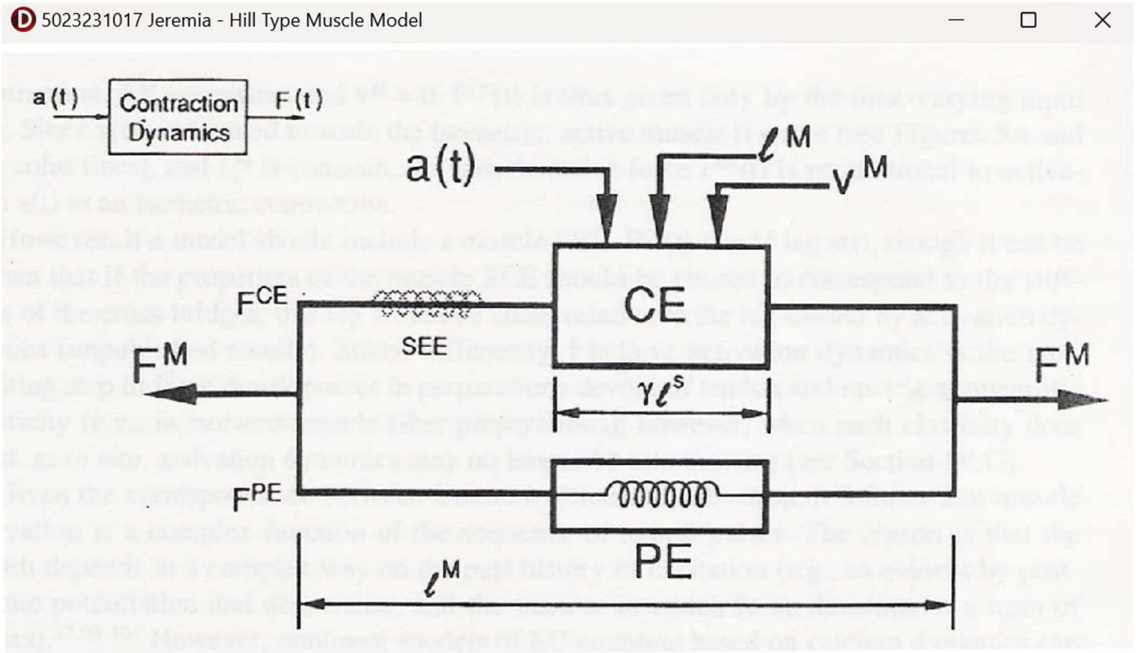


Figure 2. *The Hill-Type Muscle Model*

Figure 2 presents the Hill-Type Muscle Model, a widely used biomechanical construct that simulates the mechanical properties of a muscle-tendon unit. This model is composed of three primary elements. The **Contractile Element (CE)** represents the active, force-generating component of the muscle, driven by the actin-myosin cross-bridge cycle. The force produced by the CE is dependent on its length, its velocity of contraction, and its level of neural activation, a(t). In parallel with the CE is the **Parallel Elastic Element (PE)**, which models the passive elastic properties of the muscle fibres and their surrounding connective tissues (fascia). This element does not generate force at resting length but produces a resisting passive force when the muscle is stretched, preventing over-extension. In series with these two components is the **Series Elastic Element (SEE)**, which primarily represents the elastic properties of the tendon. The SEE transmits the force generated by the CE to the skeletal system. It also stores and releases elastic energy, which is crucial for efficient movement in activities like running and jumping. The total force generated by the muscle-tendon unit (F^M) is the sum of the forces from the contractile pathway (CE) and the passive pathway (PE).

3. The Force Generation Model Diagram can be seen below:

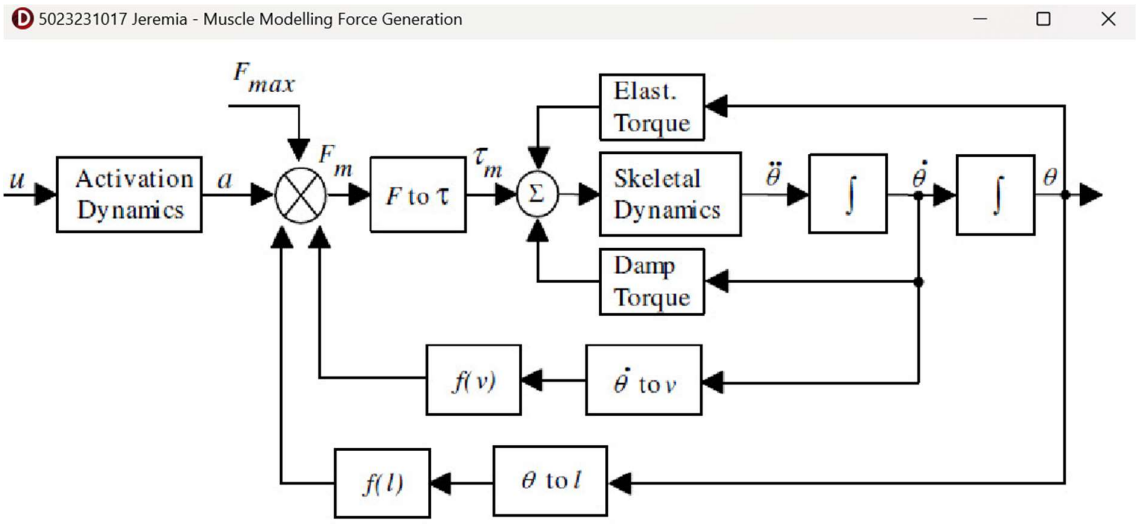


Figure 3. *The Force Generation Model Diagram*

Figure 3 provides a block diagram illustrating a more comprehensive model of force generation and its translation into movement, framed within a control systems perspective. The process begins with a neural command (u), which is converted into a physiological muscle activation level (a) by the **Activation Dynamics** block. The final muscle force (F_m) is then calculated by multiplying the maximum possible isometric force (F_{max}) by this activation level and two crucial, state-dependent scaling factors: f(l) (the Force-Length relationship) and f(v) (the Force-Velocity relationship). This calculated muscle force is then converted into a rotational torque (τ_m) around a joint. The **Skeletal Dynamics** block represents the physics of the limb, where the net torque (muscle torque minus any elastic and damping torques) produces an angular acceleration ($\ddot{\theta}$), according to Newton's laws of motion. This acceleration is integrated twice to yield the limb's angular velocity ($\dot{\theta}$) and angular position (θ). A key feature of this model is the feedback loop: the resulting motion (both angle and velocity) continuously determines the muscle's current length (l) and velocity (v), which are fed back to the f(l) and f(v) blocks. This closed-loop system accurately models the fact that the force a muscle can produce is constantly being modulated by the very movement it creates.