Chapter 3.

The effect of fibre heterogeneity on the force-length relationship for a muscle

3.1 Introduction

Muscle modelling has been used to predict the changes torque generated by muscles about a joint in response to changes of the angle of that joint (eg. Hoy et al., 1990; Meijer et al., 1998a). Hoy et al. (1990) were unable to predict active torque across as large a range of joint angles as were measured experimentally during knee flexion and hip extension. While Meijer et al. (1998a) solved this problem for knee extension by adjusting tendon lengths for each muscle within the quadriceps group, in-vitro research has suggested that even within a single muscle, traditional models underestimate the range of lengths over which a muscle can exert active force (Ettema and Huijing, 1994). The purpose of this chapter is to investigate whether the assumption of fibre homogeneity is responsible for these observations.

The sliding filament theory of skeletal muscle provides a theoretical basis to explain the change in active force a muscle can produce at different sarcomere lengths (Gordon et al., 1966). While this theory works well at the sarcomere level, certain assumptions must be made regarding the change in sarcomere lengths as a function of whole muscle length. Muscle architecture affects the relative change in fibre length with muscle length (Huijing and van Lookeren, 1989; Kaufman et al., 1989). Even if architecture is taken into account, whole muscle lengths can only be scaled directly to sarcomere lengths if fibre and sarcomere lengths are assumed to be homogenous throughout the muscle. The purpose of this chapter is to consider the limitations of this hypothesis, and to explore the benefits of using a statistical distribution of fibre and sarcomere lengths on the predictive ability of muscle models.

While homogenous fibre and sarcomere lengths have been a common assumption in skeletal muscle modelling (eg: Delp et al., 1990; Herzog et al., 1990; Meijer et al., 1998a), a number of studies have found active force-length curves over a wider range of muscle lengths than can be predicted using the sarcomere force-length curve (Herzog and ter Keurs, 1988; Huijing et al., 1989). These authors have suggested that heterogeneous fibre and sarcomere lengths may account for a widening of the active force-length curve because at very long or short muscle lengths, when the average sarcomere is too long or short to produce force, there may be others still within their active force range. Subsequently, Willems and Huijing (1994) demonstrated that not only was there significant variation in sarcomere lengths within a single muscle, but those muscles with greater variation in sarcomere lengths produced the widest active force-length curves.

Models including distributions of fibre and sarcomere lengths within a muscle have previously been described in published literature (Ettema and Huijing, 1994; Savelberg and Schamhardt, 1995). The simulations of Savelberg and Schamhardt (1995) were based on whole muscle force-length relationships described by Kaufman et al. (1989). The alternative approach, used by Ettema and Huijing (1994), is to base the model on fibre length rather than muscle length and then use muscle architecture to calculate whole muscle force-length (eg Delp et al., 1990; Herzog et al., 1990; Meijer et al., 1998a). There is a benefit in this approach in that the force-length relationship can then be easily incorporated into the force-velocity relationship that is commonly expressed as a function of fibre shortening, rather than whole muscle shortening. This approach has been taken within the present model.

Ettema and Huijing (1994) constructed a variety of different models using different hypothetical distributions of fibres and compared these models against the measured force-length relationship of rat gastrocnemius muscle. Their results demonstrated that modelling a distribution of sarcomere lengths (both with and without a corresponding distribution in numbers of sarcomere per fibre) would widen the expected range of lengths able to generate active tension. This widening of the length-tension curve was most apparent towards extremes of muscle length. Modelling muscles with a distribution of sarcomere lengths improved the fit between modelled and measured relationships over that expected from models with homogenous lengths. Modelling a distribution of fibre lengths without differences in sarcomere length per fibre also increased the range of active lengths; however, the effect was not as great as when sarcomere lengths were varied.

Rat gastrocnemius muscle has a pennation angle greater than 20 deg and this has a significant effect on the whole muscle's force – length relationship (Huijing et al., 1989). Changes in fibre pennation with muscle shortening were included in the model used by Ettema and Huijing (1994) along with measurements of series elastic compliance to predict the whole muscle force-length relationship. Matches between measured and modelled data were therefore dependent upon the validity of the fibre architecture model as well as the fibre and sarcomere length distribution. Because the results of Ettema and Huijing (1994) were expressed as a percentage of whole muscle length and therefore dependent upon the architecture of the specific muscle studied, it is difficult to apply those results to other muscles with different architectures.

This chapter will extend the work of Ettema and Huijing (1994) to include both fibre and sarcomere length distributions within the one model. The resulting model will be compared with published data for rat semimembranosus from Huijing et al. (1989) to establish validity. The model will then be applied to data from in vivo human rectus femoris muscle, measured by Herzog and ter Keurs (1988), to test whether their results can be explained by a distribution of fibre and sarcomere lengths. Finally, the relative merits of the distributed fibre model of the whole muscle force-length relationship will be discussed in comparison to the simpler, single sarcomere model.

3.2 Model Development

Sarcomere force-length relationship

The force-length relationship for a single sarcomere will be explained using lengths labelled 1 to 5 on Figure 3.2.1. Calculations for fibre length and relative force at each corner of this figure are summarised in Table 3.2.1. The derivation of these formulae will be described below.

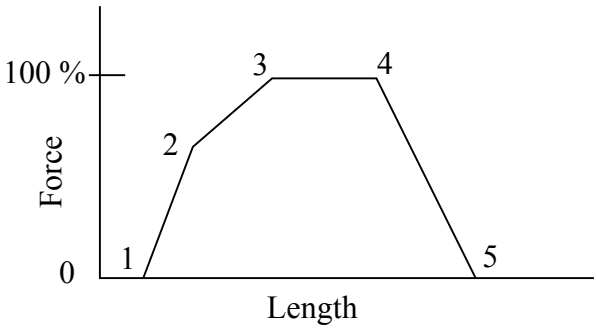


Figure 3.2.1 Force-length relationship for a single sarcomere.

Table 3.2.1 Definitions of force and length at each corner of Figure 3.2.1.

Corner number from Figure 3.2.1	Length	Force
1	$L_1 = 1.4\mu m$	$F_1 = 0$
2	$L_2 = l_m + l_z$	$F_2 = \frac{2 \times l_m + l_z - l_a}{l_m - l_H}$
3	$L_3 = l_a - l_H$	$F_3 = 1$
4	$L_4 = l_a + l_H$	$F_4 = 1$
5	$L_5 = l_a + l_m$	$F_5 = 0$
Optimum Length	$L_o = l_a$	$F_o = 1$

where: L_i = Length of sarcomere at corner i

F_i = Force exerted at corner i

l_a = Length of actin filament

l_m = Length of myosin filament

l_z = Width of z band

l_H = Width of H zone

l_o = Optimum sarcomere length

Length 1 was given by Zuurbier et al. (1995) from measurements taken on rat gastrocnemius and extensor digitorum longus muscles. It appears reasonable to use the same length 1 for all species because the force at very short sarcomere lengths is limited primarily by compression of the myosin filament (Stephenson et al., 1989), and myosin filament lengths appear to be constant between species (Walker and Schrodt, 1974).

Length 2 coincides with the Z lines contacting the myosin filament and the force at this length varies between species owing to different lengths of actin filaments (Walker and Schrodt, 1974). Stephenson et al. (1989) give the following equation for sarcomere lengths below the plateau:

$$F_i = \frac{l_m + l_i - l_a}{l_m - l_H}$$

Equation 3.2.1

Solving Equation 3.2.1 for $L_i = l_m + l_z$ gave the force at point 2.

Length 3 was chosen for the lower limit of the plateau based on the results reported by Stephenson et al. (1989) and by ter Keurs et al. (1981). Lengths 4 and 5 were taken from Gordon et al. (1966) as was the optimum fibre length given as $L_o = L_a$.

Myofilament lengths vary between species with consequent effects on the sarcomere force-length relationship (Walker and Schrodt, 1974). Table 3.2.2 gives filament lengths for rat and human muscle to be used in the above calculations. The lengths and forces calculated for these species are shown in Table 3.2.3.

Table 3.2.2 Myofilament lengths for Human and Rat muscle.

	Human *	Rat **
Myosin length	1.6 μm	1.6 μm
Actin length	2.64 μm	2.38 μm
H width	0.17 μm	0.17 μm
Z width	0.05 μm	0.05 μm

* Walker and Schrodt (1974) for human

** ter Keurs (1981)

Table 3.2.3 Force and sarcomere length calculated for human and rat muscle at each corner of Figure 3.2.1.

	Human		Rat	
	Sarcomere Length	Relative Force	Sarcomere Length	Relative Force
Length 1	1.4 μm	0	1.4 μm	0
Length 2	1.65 μm	43%	1.65 μm	61%
Length 3	2.47 μm	100%	2.21 μm	100%
Length 4	2.81 μm	100%	2.55 μm	100%
Length 5	4.24 μm	0	3.98 μm	0
Optimum Length	2.64 μm	100%	2.38 μm	100%

Distribution of fibres

Not all fibres within a muscle have exactly the same length. Similarly, not all fibres having the same length would be expected to contain the same number of sarcomeres. If the lengths

of individual fibres within a muscle are assumed to be normally distributed, then the relative number of fibres having particular lengths may be estimated from the normal curve. To simulate variance in both fibre length and number of sarcomeres, a model was developed containing 13 different fibre lengths distributed either side of the mean. Each fibre length was considered to have 13 possible sarcomere lengths, again normally distributed about a mean. Therefore, 13×13 or 169 different combinations of fibre length and sarcomere lengths were modelled. A smaller distribution of three fibre lengths and three sarcomere lengths is illustrated in Figure 3.2.2.

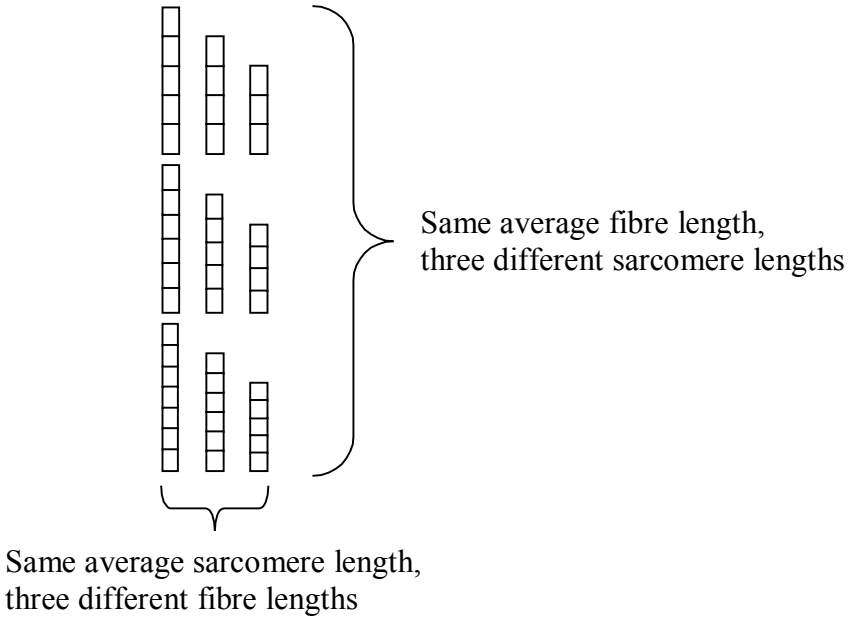


Figure 3.2.2 Illustration of the model's distribution of fibre and sarcomere lengths.

When specifying the distribution of sarcomere lengths, it was assumed that each group possessing one of the 13 different fibre lengths had the same average sarcomere length. This assumption is supported by Herzog et al. (1990) who found that fibres from different parts of human rectus femoris muscle varied in fibre length, but were consistent in average sarcomere length. By contrast, Willems and Huijing (1994) found significant differences in sarcomere length between proximal, intermediate and distal sections of rat semimembranosus muscle. Because the differences in mean fibre length between sections did not correspond to differences in sarcomere lengths (ie shorter fibres did not necessarily have shorter

sarcomeres), no satisfactory model could be derived from this data. Therefore, it was decided to assume constant average sarcomere length for all fibre lengths. Similarly, in the absence of any relevant data, the variance in sarcomere lengths was kept constant for all modelled fibre lengths.

A separate force - length relationship was developed for each of the 169 different combinations of fibre and sarcomere length. The fibre having the mean fibre length and mean sarcomere length will henceforth be referred to as the reference fibre. This reference fibre was given lengths varying between 0 and 300% of optimum length, calculated at 5% intervals. Every other fibre in the model was then given an appropriate length by assuming equal absolute length changes for every fibre. Therefore, when calculated as a percentage of the optimum length, those fibres with fewer sarcomeres in series had greater percentage length changes than longer fibres.

Force was calculated for each fibre/sarcomere combination as a function of its length according to the sarcomere force-length relationship described above. By specifying a mean and standard deviation for both fibre and sarcomere lengths, the normal distribution curve enabled total numbers of each fibre/sarcomere combination to be specified from an assumed population of 250000 fibres. Adding the force contribution of each of the 169 fibre combinations multiplied by the relative frequency of each combination gave a length-tension curve for the whole muscle. Modifying the mean and standard deviation for both fibre and sarcomere lengths within a muscle enables investigation of the effect these assumptions have on the whole muscle force – length relationship.

Rat Semimembranosus Muscle

Rat semimembranosus muscle provides a convenient test of the fibre distribution model because the muscle has a small angle of pennation of approximately 2 deg (Huijing et al., 1989). Fibre length changes are therefore almost directly proportional to whole muscle length changes and fibre force – length relationships may be inferred without reference to a model of muscle fibre length interactions.

Huijing et al. (1989) reported force – length relationships from six rat semimembranosus muscles against which the current model will be compared. Huijing et al. reported their fibres

to have a mean length of 2.96 cm and a standard deviation of 0.25 cm. The model for the present simulation will use this distribution of fibre lengths along a mean sarcomere length of 2.38 μm during a maximum contraction at the optimum muscle length (ter Keurs et al., 1981). The standard deviation of sarcomere lengths was set at either 12% or 41% of the mean to match measurements taken by Willems and Huijing (1994). These two standard deviations represented the most extreme examples from the 18 rat semimembranosus muscles that were examined by Willems and Huijing (values estimated from their figure 5B).

Model to match Herzog and ter Keurs (1988)

Herzog and ter Keurs (1988) measured the in-vivo force-length relationship of rectus femoris for six human subjects. To model this relationship using heterogeneous fibre and sarcomere lengths, data from Herzog et al. (1990) have been used. Herzog et al. (1990) measured fibre and sarcomere lengths from three portions of human rectus femoris while in the anatomical position. The standard deviations reported by Herzog et al. result from a combination of both within and between subject variation, however there were many more within subject points than between. Appropriate data describing the expected variance of sarcomere lengths within subjects are not yet available, however the results from Willems and Huijing (1994) indicate that it is likely to differ substantially between individuals. Table 3.2.4 shows the fibre and sarcomere lengths, together with standard deviations used in the present simulation. Note that these measurements were taken in the anatomical position rather than at muscle optimal length. This does not, however, affect the present simulation, because optimal length is assumed to occur at a sarcomere length of 2.64 μm, regardless of the anatomical length.

Table 3.2.4 Fibre and Sarcomere Lengths for Rectus Femoris from Herzog et al. (1990).

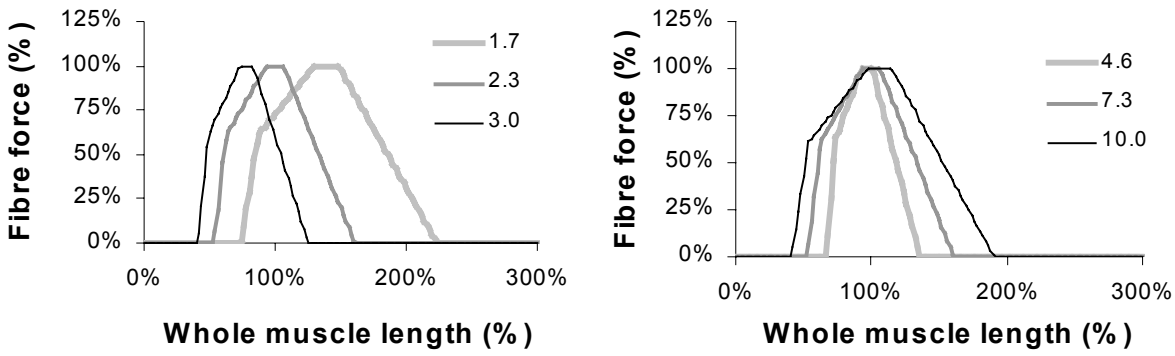
Compartment	Mean Fibre Length (cm)	SD	Mean Sarcomere Length (μm)	SD
Proximal *	7.9	0.55	2.29	0.10
Middle *	7.2	0.69	2.34	0.13
Distal *	6.9	0.74	2.33	0.09
Combined for whole muscle **	7.3	0.79	2.32	0.11

* Indicates values measured by Herzog et al., 1990

** Indicates values calculated by the present author from the data of Herzog et al., assuming equal fibre and sarcomere numbers in each compartment.

3.3 Results

Figure 3.3.1 illustrates the resulting force-length curves for a few fibres modelled using the human rectus femoris data of Herzog et al. (1990). The horizontal axes used for Figure 3.3.1 show the relative length of the reference fibre.



A B

Figure 3.3.1 Forces generated by individual fibres within the model. Part A illustrates 3 fibres, each having a length of 7.3 cm at the whole muscle resting length, but differing sarcomere lengths within each fibre. Part B illustrates 3 fibres, each having a sarcomere length of 2.3 μm at the whole muscle resting length, but differing total fibre lengths.

Figure 3.3.1A demonstrates that for modelled fibres that are equal in length at the anatomical position, those fibres with shorter sarcomere lengths at this length develop force at longer muscle lengths than do fibres with longer sarcomeres. Furthermore, those fibres with shorter sarcomeres at this particular length have more sarcomeres in series, and are therefore able to develop force over a wider range of lengths. Figure 3.3.1B illustrates three fibres having identical sarcomere lengths at the anatomical position, but with different fibre lengths. The longer fibres have more sarcomeres in series and therefore generate forces over a wider range of muscle lengths because a larger change in average fibre length is required to achieve the same proportional change in fibre length. One consequence of this widened active force range for fibres with more sarcomeres in series is that the model predicts a total force length curve skewed to the right.

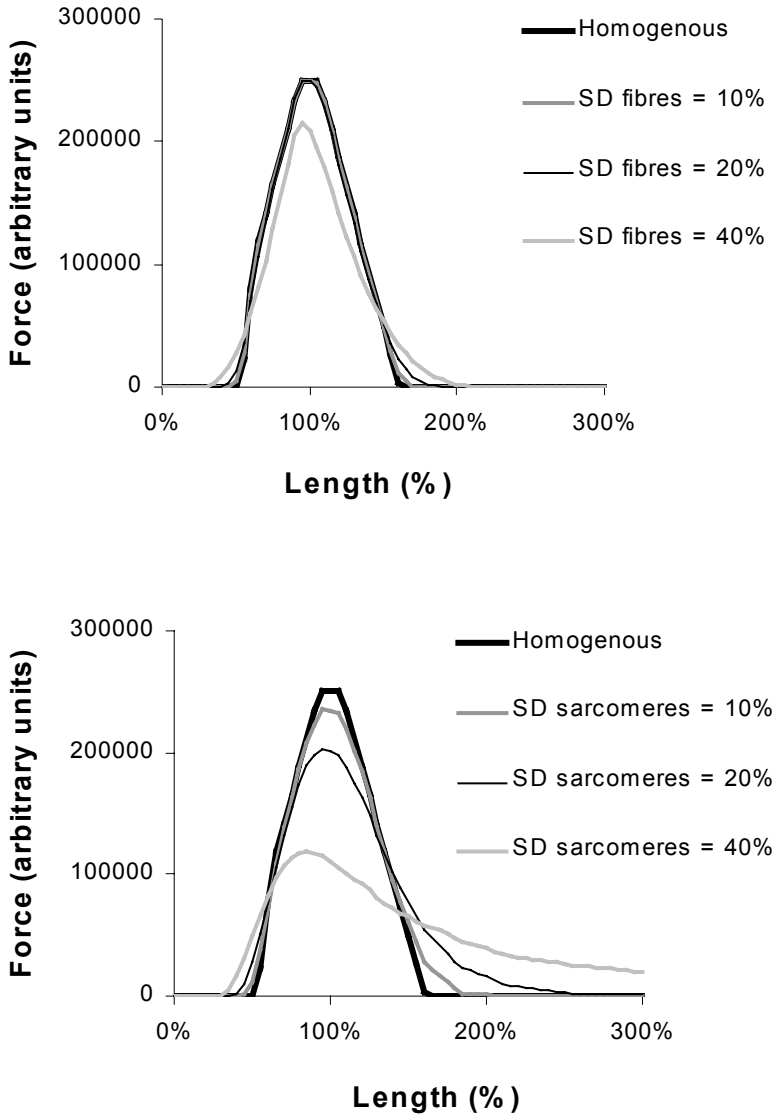


Figure 3.3.2 Forces generated by whole muscles having different distributions of fibre and sarcomere lengths (See text for details).

Total force produced by the sum of all fibres is illustrated by Figure 3.3.2. With 250,000 fibres and a nominal force of 1 unit per fibre, the maximum force is 250,000 units if all fibres produce maximum force simultaneously. Figure 3.3.2a illustrates the effect of varying the standard deviation of fibre lengths present within a muscle while holding sarcomere length constant for every fibre. Increased length variance results in wider ranges of active muscle lengths and less force at the optimal muscle length, as fewer fibres would be at maximum simultaneously. Figure 3.3.2b illustrates the effect of varying the distribution of sarcomere lengths while holding the standard deviation of fibre lengths constant at 10%.

Figure 3.3.2 illustrates that, for this simulation, a greater variation in fibre or sarcomere lengths results not only in a wider range of active forces as described by Willems and Huijing (1994), but also in a shift to the left where peak whole muscle force occurs at less than optimum length for the average fibre. This is because all fibres are assumed to have the same absolute length changes as the muscle shortens. Fibres with relatively long sarcomeres at 100% mean fibre length have fewer sarcomeres in series, and thus experience relatively larger percentage length changes for the same absolute change. As average fibre length decreases below 100%, those fibres having average sarcomere length or less decrease their force generating ability, while those fibres with initially longer sarcomere lengths get stronger. The different number of sarcomeres in series results in relatively greater percentage change in length for the longer fibres. The result of this is that the increase in force of longer fibres as the muscle shortens is greater than the decrease in force of the shorter fibres, and hence the whole muscle initially increases its force when it shortens below 100%. Figures 3.3.3 and 3.3.4 will therefore be normalised for both amplitude and optimal length to illustrate a peak force of 100% at a muscle length of 100%.

Model to match Huijing et al. (1989)

Figure 3.3.3 illustrates that modelling rat muscle using the two most extreme fibre distributions found by Willems and Huijing (1994) delineates the range of data reported by Huijing et al. (1989) at short fibre lengths. Between approximately 70 and 90% of optimum fibre length, however, the models predicted forces less than those measured experimentally. The measured plateau to the left of muscle resting length was wider than that predicted by the models.

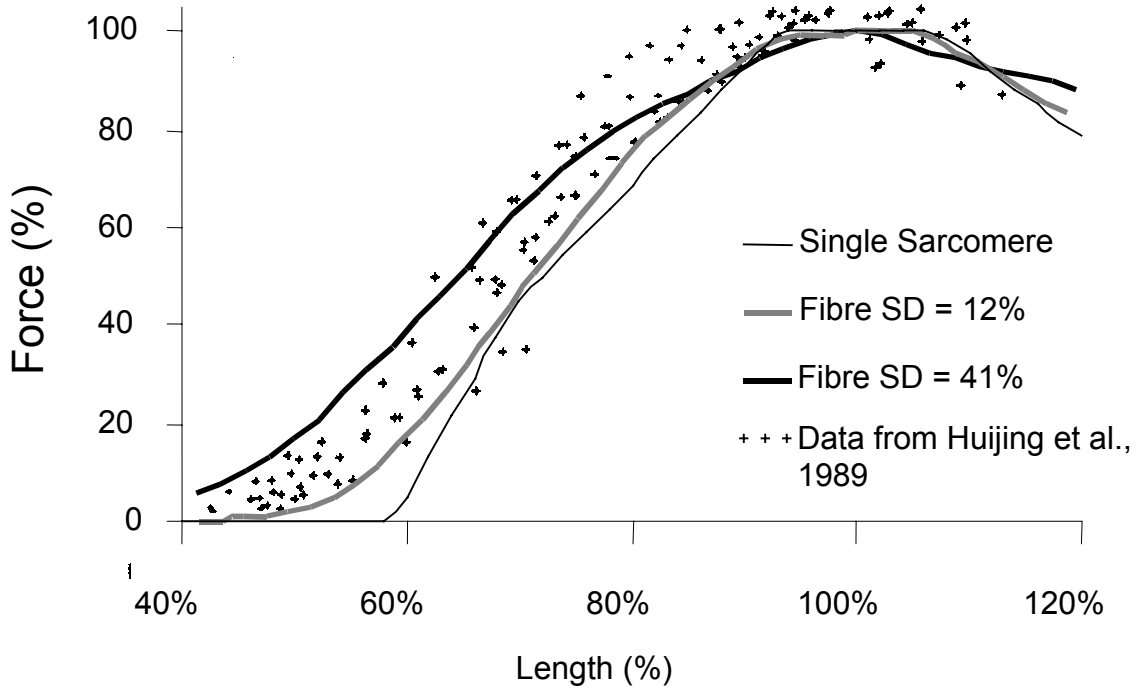


Figure 3.3.3 Data to match Figure 1a, Huijing et al. (1989)

Model to match Herzog and ter Keurs (1988)

The modelled force-length curve from Herzog and ter Keurs (1988) had the same basic shape as the present model; with a different active width resulting from different assumptions regarding myofilament length (Figure 3.3.4). Herzog and ter Keurs' measured force-length curve had a significantly smaller magnitude than the modelled curves, indicating either an error in the modelled force per unit physiological cross section or errors in the assumptions used to calculate in-vivo forces. Reasons for these differences are discussed by Herzog and ter Keurs (1988); the purpose of the present discussion, however, is to consider the shape and particularly width of the force-length curve rather than the absolute magnitude.

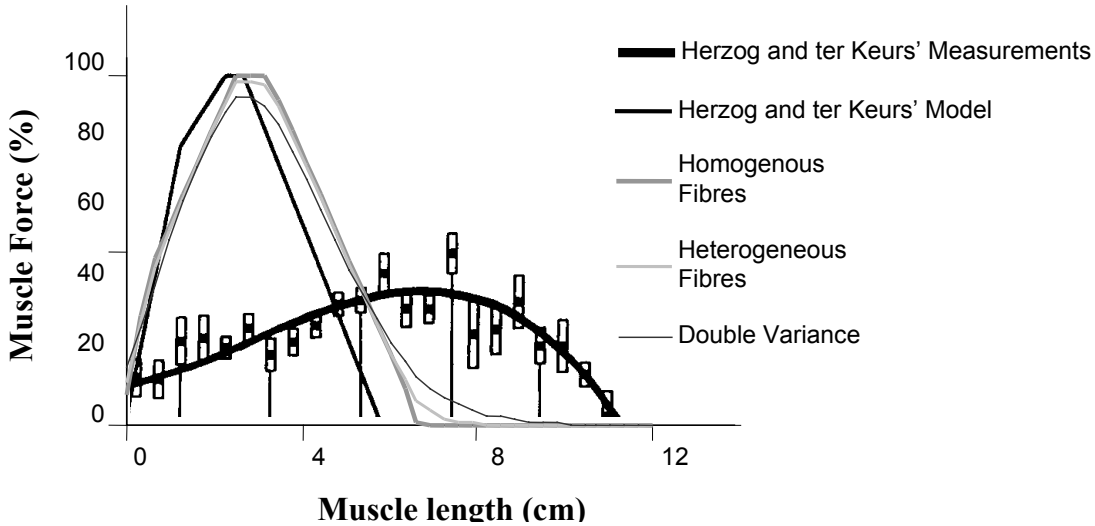


Figure 3.3.4 Data to match Figure 2, Herzog and ter Keurs (1988). The original figure from Herzog and ter Keurs contained a modelled curve shown as straight lines and a measured curve as a thicker, curved line. Curves from the present models are shown superimposed over those of Herzog and ter Keurs.

Allowing for a distribution of fibre lengths increases the range of lengths over which force can be made and reduces the maximum force produced by the muscle (Figure 3.3.4). The changes, however, are quite insubstantial compared to the difference between all models and Herzog and ter Keurs' (1988) in-vivo measured force-length data. Even doubling the expected values for fibre and sarcomere length variance does not increase the active range to that found by Herzog and ter Keurs. Furthermore, the shape of the active force-length curve is quite different, with Herzog and ter Keurs finding a curve skewed to the left, while the present model skews active forces to the right of optimum.

3.4 Discussion

There are a number of possible explanations for the difference between the present model and in-vivo force length curves measured by Herzog and ter Keurs (1988). Changes in fibre length were not measured directly by Herzog and ter Keurs. Rather, they were calculated using

assumptions about maximal activation across all joint angles, the moment arms of rectus femoris at the hip and knee, linear elasticity of the series elastic component and ignoring pennation angles of the fibres. If hip or knee moment arms were overestimated by Herzog and ter Keurs, this would have resulted in a corresponding over-estimation in the amount of fibre shortening during their experiment. Similarly, underestimating the series elastic component's stiffness would have resulted in an incorrectly large calculation of fibre shortening, particularly as the series elastic component of rectus femoris is very long compared to fibre length. Herzog and ter Keurs ignored fibre pennation and calculated change in fibre length as length of the series elastic component subtracted from whole muscle length. This assumption was also made by Huijing et al. (1989) for rat semimembranosus muscle, however the pennation angle for this muscle is much less (approx 2 deg). Pennation angles for human rectus femoris are about 14 deg (Friederich and Brand, 1990) and are likely to increase as a muscle develops tension and shortens (Fukunaga et al., 1997). By ignoring pennation angle, Herzog and ter Keurs are likely to have overestimated the range of fibre lengths in their experiments (Legreneur et al. 1997, Scott and Winter 1991). Finally, the possibility that the quadriceps may not have been maximally activated during all joint angles, thus leading to erroneous calculations of rectus femoris force-length must also be considered a possibility; even though the experimental procedure took rigorous steps to try and avoid this.

Several assumptions inherent in the distributed fibre model may also explain the failure to predict active force-length ranges found by Herzog and ter Keurs (1988). Key assumptions in the model are normal distribution of fibre and sarcomere lengths within the muscle, equal average sarcomere lengths for all fibre lengths and the actual values used for standard deviation of fibre and sarcomere lengths. While these assumptions are directly supported by the findings of Herzog et al. (1990) (except normality which was not discussed), it must be acknowledged that only a relatively few fibres and sarcomeres were examined from the total population within each muscle. If larger variations in lengths were present within each muscle then this would result in a wider range of active fibre lengths predicted by the model. This explanation is unlikely to lead to a full match against Herzog and ter Keur's data, however, as even doubling the assumed variance of fibre and sarcomere lengths did not produce wide enough ranges of fibre lengths (see Figure 3.3.4).

Willems and Huijing (1994) measured sarcomere heterogeneity in rat semimembranosus and also reported force – length curves for these muscles. While it was initially planned to

compare the model against these data, several factors made this difficult. Firstly, Willems and Huijing reported muscle force against length changes in millimetres from the muscle optimal length, without reporting what those optimum lengths were for each individual. This made it difficult to deduce relative length changes as a percent of optimum length. Secondly, they reported only polynomial curves fitted to raw data rather than the actual data points given by Huijing et al. (1989). Figure 3.3.3 from the present paper illustrates a pronounced "toe" region at short muscle lengths in both the data from Huijing et al. and in simulations by the present model. This "toe" is concave up whereas the polynomial curves presented by Willems and Huijing are convex. Whether this reflects the curve fitting process or actual differences in the data measured is impossible to tell without the original data points. For these reasons, data from Huijing et al. (1989) was used for comparison, even though sarcomere heterogeneity had to be estimated from another source.

Modelling a distribution of fibre and sarcomere lengths significantly increased the range of lengths over which a model can generate active force. This can be useful for simulating body movements at extreme joint angles where simpler models may lock-up, and not allow further muscle excursion should the force drop to zero. Predictions from the present model are consistent with those found earlier by Ettema and Huijing (1994). Again, like Ettema and Huijing (1994), the present model did not predict muscle forces as large as those found experimentally at lengths closer to optimum. The measured force-length curve appears broader near its optimum length and a normal distribution of fibre and sarcomere lengths does not significantly change this.

The assumption of normality forces the fibre force-length relationship to be dominated by the sarcomere force-length relationship of the reference fibre. There are relatively fewer fibres with lengths greatly different to the mean; therefore, these fibres cannot produce a large amount of force compared to maximum. Furthermore, the increase in force at length 80% from muscles having a longer initial sarcomere length would be balanced by the loss in force from an equal number of fibres having shorter sarcomere lengths. Therefore, the distributed fibre length force – length curves only differ from the single sarcomere curve towards the extremes of length where force is relatively small. It appears from Figure 3.3.3, that using the distributed fibre model only improves the match with experimental data for lengths below approximately 70%.

If the assumption of normality was not taken, then there could be relatively more fibres having lengths quite different to the mean. This could lead to a wider force – length curve than is anticipated from the present model. While this may then match the rat semimembranosus data from Figure 3.3.3, it is unlikely to explain the full width of the in-vivo measurements from Herzog and ter Keurs (1988). It seems likely that the measurements by Herzog and ter Keurs were flawed in some way; most likely in the assumptions used to estimate muscle length and force from the in-vivo torque measurements.

One factor in support of non-normality of fibre distributions is the finding that passive force is not generated until a muscle is stretched beyond its resting length. Figure 3.3.1A illustrates that when the whole muscle is at its resting length, some fibres are already at lengths greater than at rest. It would therefore seem likely that these fibres should be generating passive tension when the whole muscle is at resting length. Non-normality of fibre distribution offers a potential solution to this apparent contradiction. If, when the whole muscle was at rest, there were relatively few fibres at long lengths but a skewed distribution of fibres at shorter lengths, this would account for the lack of passive force development at muscle resting length while still allowing for a distribution of fibres to widen the active range of shortening below muscle optimum length. Unfortunately, there is no known data providing estimates of the normality of fibre distribution, so this point will have to remain conjecture at this point.

If shorter fibres had been modelled with correspondingly shorter sarcomere lengths, then this too would have increased the active range. While Herzog et al. (1990) found all fibre lengths within rectus femoris to have similar average sarcomere lengths, this was not found by Willems and Huijing (1994) for rat semimembranosus. Willems and Huijing did not, however, find a consistent trend in fibre to sarcomere lengths for use in the model and hence equal average sarcomere length was the only option available. Further anatomical measurements are required to validate the assumptions to be used in further modelling.

3.5 Summary

Modelling families of fibres with a normal distribution of fibre and sarcomere lengths predicts wider ranges of active fibre length that agree well with those measured for rat

semimembranosus muscle by Huijing et al. (1989) at very short fibre lengths. At lengths closer to optimum, however, the current model of normally distributed fibre and sarcomere lengths provides little benefit beyond the single fibre model. Further modelling is required to elucidate this further. Non-normal length distributions and changing the assumption that average sarcomere length does not vary proportionally with fibre length may widen the active force-length curve. Detailed anatomical measurements are required in order to validate the assumptions made by future models.

The present distributed fibre model does not agree well with force-length relationships measured in-vivo for human rectus femoris muscle by Herzog and ter Keurs (1988). It is unclear at this stage whether this poor fit of modelled to data results from inadequacies in the model or in the calculations performed by Herzog and ter Keurs. Further work is required in measuring distributions of fibre and sarcomere lengths to provide more confident values for input to the modelling process.

At this stage, it appears that the distributed fibre model does not offer a significant improvement to the prediction of whole muscle force within non-extreme ranges of fibre lengths. For this reason, along with the increased computational cost of the distributed model, only the single fibre model will be used for further modelling within the present study. This decision will be discussed further within Section 6.1.7.