The Human Gluteus Maximus and its Role in Running

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The human gluteus maximus and its role in running

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Summary

The human gluteus maximus is a distinctive muscle in terms of size, anatomy and function compared to apes and other non-human primates. Here we employ electromyographic and kinematic analyses of human subjects to test the hypothesis that the human gluteus maximus plays a more important role in running than walking. The results indicate that the gluteus maximus is mostly quiescent with low levels of activity during level and uphill walking, but increases substantially in activity and alters its timing with respect to speed during running. The major functions of the gluteus maximus during running are to control flexion of the trunk on the stance-side and to decelerate the swing leg; contractions of the stance-side gluteus maximus may also help to control flexion of the hip and to extend the thigh. Evidence for when the gluteus maximus became enlarged in human evolution is equivocal, but the muscle's minimal functional role during walking supports the hypothesis that enlargement of the gluteus maximus was likely important in the evolution of hominid running capabilities.

Key words: gluteus maximus, running, walking, locomotion, biomechanics, stabilization, human.

Introduction

Bipedalism has long been considered a characteristic feature of the hominid lineage (Darwin, 1859), and recent fossil evidence suggests that the very earliest hominids may have been bipedal in some manner (Haile-Selassie, 2001; Zollikofer et al., 2005). Not surprisingly, many aspects of the hominid musculoskeletal system, especially in the leg and foot, have undergone substantial reorganization for bipedal posture and locomotion (see Lovejoy, 1988; Aiello and Dean, 1990; Ward, 2002). One of these features may be the gluteus maximus (GM). The human GM is anatomically distinctive compared to other non-human primates in several respects, notably in its overall enlargement, in the expansion of its cranial portion and in the loss of its caudal portion. Since Cuvier (Cuvier, 1835), anatomists have speculated that the distinctive human GM is an adaptation for either walking or maintaining upright posture, but electromyographic (EMG) studies have shown that the GM has little or no activity in walking or normal upright standing (Joseph and Williams, 1957; Karlsson and Jonsson, 1965; Stern, 1972; Marzke et al., 1988). Instead, the human GM is primarily active during climbing (Zimmerman et al., 1994), as well as running and other activities that involve stabilizing the trunk against flexion (Stern et al., 1980; Marzke et al., 1988; McLay et al., 1990). Although there has been no systematic comparison of GM activity during walking and running (see below), the available evidence has led to the proposal that enlargement and reorganization of the GM may have played a role in, and possibly were selected for, the evolution of human endurance running capabilities (Bramble and Lieberman, 2004). In order to test this hypothesis, however, more data are needed on how the GM functions during running *versus* walking. This study therefore compares GM activity, combined with trunk and hindlimb kinematics, during bipedal walking and running in humans to test several hypotheses about the function and evolution of this distinctive muscle.

Comparative anatomy

To test hypotheses about GM function during locomotion in humans, it is useful to begin with a comparison of the muscle's anatomy and function in humans *versus* our closest relatives, the great apes. The human GM differs not only in relative size but also in its pattern of origin and insertion (Fig. 1). In apes, the GM has two very distinctive compartments with different origins and insertions. The more cranial portion, the gluteus maximus proprius (GMP), is a thin sheet of muscle that arises from the gluteal aponeurosis and the sacroilliac ligament, from the dorsal aspect of the sacrum, and from the upper portion of

the coccyx; the GMP inserts on the iliotibial tract (Stern, 1972; Sigmon, 1975; Aiello and Dean, 1990). The more caudal portion, the gluteus maximus ischiofemoralis (GMIF), comprises by far the greatest proportion of the ape GM. This thicker portion of the muscle arises primarily from the ischial tuberosity (Stern, 1972), and inserts along the entire lateral aspect of the femur all the way from the gluteal tuberosity to the lateral epicondyle (Stern, 1972; Swindler and Wood, 1973; Sigmon, 1975; Aiello and Dean, 1990; Lovejoy et al., 2002). The GMP and GMIF are considered two separate muscles in the orangutan (Sigmon, 1975).

The most substantial difference between humans and apes is that humans lack the GMIF and have only an enlarged GMP portion of the muscle (hereafter referred to as the human GM). The human GM arises from several sites including the broad, roughened surface on the superior margin of the posterior portion of iliac crest, the gluteal fascia that covers the gluteus medius, the fascial aponeurosis of the erector spinae on the sacrum, the posterior surface of the inferior portion of the sacrum, the lateral aspect of the upper coccyx, and the sacrotuberous ligament (see Aiello and Dean, 1990; Standring, 2005). The fibers from these various sites of origin unite to form a broad, thick, quadrilaterally shaped muscle with thick fascicular bundles. Fibers from the more cranial sites of origin primarily end in a thick laminar tendon that inserts on the iliotibial tract; some fibers from deeper portions of the muscle insert onto the gluteal ridge of the femur, generally on the proximal 25% of the femur (Stern, 1972).

Although humans lack a GMIF, the GM as a whole is relatively larger in humans because of considerable expansion of the muscle's GMP portion. The GM as a whole is approximately 1.6 times larger relative to body mass in humans compared to chimpanzees (Thorpe et al., 1996; Voronov, 2003). Dissections indicate that the GM comprises 18.3% of the total mass of the hip musculature in humans, compared to 11.7% and 13.3% for chimpanzees and gorillas, respectively (Haughton, 1873; Zihlman and Brunker, 1979).

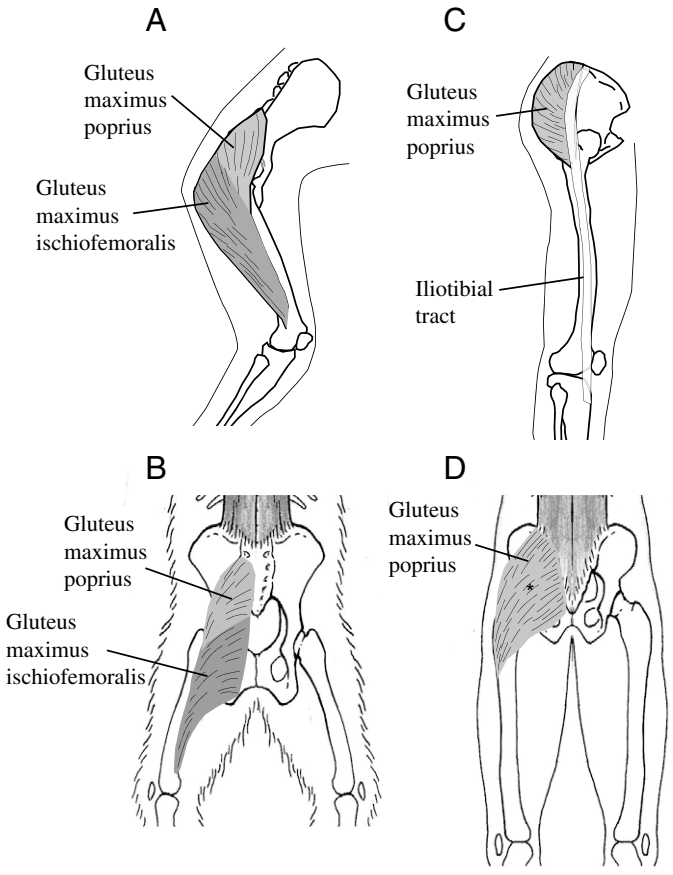

Fig. 1. Comparison of gluteus maximus anatomy in *Pan troglodytes* (A,B) and *Homo sapiens* (C,D). Note that the gluteus maximus in *Pan* has a cranial component, the gluteus maximus proprius (GMP), and a caudal component, the gluteus maximus ischiofemoralis (GMIF); humans have just the GMP, but it functions primarily like the ape GMIF. The GMP in humans is much thicker and larger than either portion of the GM in apes. The asterisk indicates the approximate location of GM electrodes used in this study.

Comparative function

A number of researchers have examined the functional implications of the anatomical differences between the human and non-human primate GM. In terms of function, the human GM is primarily an extensor of the hip, although its anterior and posterior fibers can be medial or lateral rotators, respectively. In contrast, the GMP in apes acts primarily as an abductor of the hip because it passes lateral to the hip joint. Despite differences in origin and insertion, the ape GMIF functions somewhat like the human GM as an extensor and lateral rotator of the hip (Stern, 1972; Tuttle et al., 1975; Tuttle et al., 1978; Tuttle et al., 1979). It has been suggested (Stern, 1972) that differences in femoral insertion between humans and nonhuman primates are mostly explained by pelvic reorganization for bipedality. In particular, the reduced and more proximal insertion of the GM on the femur in humans may be a function of the reduced resting length of caudal GM fibers when the femur is in line with the trunk, rather than at a 90° angle, as in a primate quadruped (Stern, 1972). Stern also noted (Stern, 1972) that angulation of the sacrum in upright humans increases the leverage of the GM during extension.

Since Cuvier (Cuvier, 1835), comparative anatomical and modeling analyses have led to many suggested functional explanations for the expanded cranial origin and enlargement of the human GM. Most of these studies hypothesize that the human GM functions either to extend and stabilize the hip, and/or to control flexion of the trunk relative to the stance leg during bipedal standing and walking (e.g. Washburn, 1951; Le Gros Clark, 1967; Lovejoy, 1988; Wolpoff, 2000; Anderson and Pandy, 2003; Jonkers et al., 2003a; Jonkers et al., 2003b). Importantly, there have also been many EMG studies of *in vivo* GM function in humans. In terms of non-locomotor function, these studies generally agree that the GM is quiescent during standing, and acts both as a hip extensor and as a stabilizer of the hip and sacroiliac joints during activities such as rising from a chair, stepping and lifting (e.g. Fischer and Houtz,

1968; Vakos et al., 1994; Oddsson, 1989; Millington et al., 1992; Noe et al., 1992; Snijders et al., 1993; Isear, Jr et al., 1997; Caterisano et al., 2002). Studies of GM function during walking have further shown that the all portions of the GM have absent-to-low levels of activity during level walking (Joseph and Williams, 1957; Karlsson and Jonsson, 1965; Stern et al., 1980; Marzke et al., 1988). GM levels were found to be higher during level walking with flexed hip and knee postures (Grasso et al., 2000); however, GM activity was found to be only minimally higher during uphill *versus* level walking, in which more flexed postures were used (Tokuhiro et al., 1985).

Although the GM has an apparently minor role during walking, several studies have found GM activity to be important in running. The GM was reported to be active at strong to moderate levels at the end of swing phase and during the first third of stance during running (Mann and Hagy, 1980a; Mann and Hagy, 1980b; Montgomery et al., 1984; Nilsson et al., 1985). In addition, it has been reported that GM activity during running peaks near the time of footstrike, and has a similar pattern of contract to the hamstrings (Jonhagen et al., 1996). Unfortunately, only one study has directly compared GM activity in walking and running (Stern et al., 1980), and only in a general qualitative way in one subject. Stern et al. nonetheless found that GM contractions during walking are 'minimal' compared to the 'considerable' increases in GM activity during jogging and running, particularly in the cranial portions of the muscle (Stern et al., 1980). In a comparison of level *versus* incline running, Swanson and Caldwell further showed that incline running (30°) at 4.5 m s^{-1} increased the intensity of GM contractions and resulted in an earlier onset relative to footstrike (Swanson and Caldwell, 2000). However, while EMG analyses of the GM generally indicate a far more important role in running than walking, there has yet to be a comprehensive and quantitative comparison of GM function in both gaits.

Running as a potential explanation for GM enlargement

When viewed in the context of comparative anatomy, the studies reviewed above suggest three functional explanations other than bipedal walking that may help account for the distinctive morphology of the GM in humans. The first is climbing, since the GMIF has been shown to contract in conjunction with the hamstrings to extend the hip during climbing in apes (Tuttle et al., 1975; Stern and Susman, 1981), whereas in humans the more cranial portions play a role in similar activities such as getting up from a chair or rising from a squatting position. Thus reorganization of the GM may have been necessary for the muscle to be involved in climbing in early bipeds. Although the climbing hypothesis has not received much attention, presumably because the activity does not appear to be a major part of the modern human locomotor repertoire, tree climbing may have been an important activity among early hominids (Susman et al., 1984). A second possibility, noted above, is that an enlarged GM evolved in bipedal hominids as a means to help control flexion of the trunk during foraging activities such as digging, throwing or clubbing that require leverage and/or stabilization of the trunk (Marzke et al., 1988). Since an upright trunk may be subject to greater flexion during uphill walking or carrying heavy objects, a related hypothesis is that the GM in humans is enlarged for controlling flexion of the trunk during non-steady bipedal walking.

A final hypothesized functional explanation for enlargement of the human GM is running, which is biomechanically very different from walking, and appears to stimulate higher levels of EMG activity than walking as noted above. As Bramble and Lieberman have argued (Bramble and Lieberman, 2004), humans are exceptional endurance runners compared to other mammals in terms of several criteria such as speed and distance. While many of the functional bases for human running clearly stem from adaptations for bipedal walking, humans have a number of derived features, such as elongated leg tendons, which may improve endurance running performance but play little or no role in walking (Bramble and Lieberman, 2004). The enlarged GM may be such a feature.

Three major biomechanical differences between running and walking are particularly relevant to GM function. First, running differs from walking in having an aerial phase that generates a much higher ground reaction force (GRF) at heel strike (HS) when the body collides with the ground. GRFs at HS in running are typically twice as high as during walking, and may exceed four times body weight at peak endurance speeds (Keller, 1996). In addition, during endurance running (although not necessarily sprinting), the trunk is more flexed at the hip than during walking, typically by approximately 10° (Thorstensson et al., 1984). Thus during running the hip extensors such as the GM and the erector spinae must counteract greater pitching forces that tend to flex the trunk anteriorly. In addition, the trunk may also be subjected to higher forces at HS in the coronal plane that tend to flex the trunk medially relative to the stance-side hip, and which are counteracted by the stance-side abductors of the hip (Stern, 1972; Stern et al., 1980).

A second potentially relevant biomechanical difference between running and walking is hip flexion during stance. During running, the hip tends to be more flexed than during walking, not only at HS but also during much of the stance phase, as the center of mass falls between HS and mid-stance (MS) and then rises between MS and toe-off (TO). Flexion of the hips, knees and ankles between HS and MS during running functions to store up elastic energy in the tendons of the legs; this energy is then released as kinetic energy through recoil during the second half of stance, helping to propel the body back into the air (Alexander, 1991). The higher impact forces experienced while running, combined with hip flexion during early stance phase, lead to a tendency of both the thigh and the trunk to collapse into flexion after heel strike. As already observed (Stern, 1972; Marzke et al., 1988), the human GM is uniquely suited to prevent both types of collapse in humans, whereas in apes, GMIF contractions only extend the thigh at the hip or prevent it from flexing.

A final difference between running and walking that is relevant to GM function is leg swing. During running, the swing leg is accelerated and then decelerated at much higher velocities than during walking. The GM in humans may thus act to slow the swing leg at the end of swing phase.

Hypotheses to be tested

This study uses EMG recordings of muscle contractions in conjunction with kinematic data on limb and trunk movements in human subjects while walking and running on a treadmill to characterize both the timing and magnitude of GM activity. Based on the above model, five specific hypotheses about GM function are tested:

Hypothesis 1. If the primary functional role of the GM is for running rather than walking, then overall normalized activity of the GM is predicted to be greater during running than during walking, including uphill walking.

Hypothesis 2. If contractions of the stance-side GM during running and walking function to control anterior flexion of the trunk, then normalized levels of GM activity should correlate positively with forward trunk pitch; in addition, timing of GM activity should correspond with differences in the timing of peak trunk flexion in walking (after MS) and running (at HS).

Hypothesis 3. If contractions of the stance-side GM during running function as hip stabilizers to control flexion of the thigh during bent-hip postures, then normalized levels of GM activity should be higher during bent-knee-bent-hip than normal gaits.

Hypothesis 4. If contractions of the stance-side GM during running are predicted to function as propulsive muscles to help extend the thigh along with the hamstrings during stance phase, then the timing and normalized levels of GM and hamstring activity should correlate strongly with each other.

Hypothesis 5. If contractions of the GM on the swing-side function to decelerate the swing-side leg prior to HS, then normalized levels of GM activity on the swing side should correlate positively with speed.

Materials and methods

Subjects

Nine volunteers participated in this study. The sample included five females and four males, all between the ages of 20 and 28. All subjects were Harvard University students who regularly do long-distance running, with no history of problems with their gait, and who participate in athletics on a regular basis. Mean stature was 172.4±8.9 cm (range: 164–186 cm); mean body mass was 87.7±5.1 kg (range: 83–95 kg). All subjects were barefoot during the experiment, and all recordings were made on the same treadmill (Vision Fitness T9250, Lake Mills, WI, USA). After the sensors and EMG electrodes (see below) had been attached, calibrated and tested, the subjects walked and ran at a variety of speeds in order to habituate themselves to the treadmill and the experimental conditions prior to recording. Once the subjects were comfortable and warmed-up, they were then recorded at three sequential walking speeds (1.0 m s^{-1}, 1.5 m s^{-1} and 2.0 m s^{-1}) followed by three sequential running speeds (2.0 m s^{-1}, 3.0 m s^{-1} and 4.0 m s^{-1}) during normal walking and running, and then while walking and running with a bent-hip and bent-knee (‘Groucho’ gait). The sequence of trials was generally the same for all subjects, but trials were repeated on a regular basis to test for signal similarity, and to ensure that the footswitches were operating properly. Repeatability was assessed by calculating the standard error of the mean for peak GM voltages and the onset of GM activity relative to foot strike at various speeds. These were found to be acceptable. For example, at a 3 m s^{-1} run for one subject, the standard error of the peak was 8.2% of the mean peak value, and the standard error of timing was 6.38 ms. Subjects were allowed to rest between trials. In order to ensure accurate normalization, no trials were used if EMG amplifications had been altered during the experiment. A subset of subjects also walked and ran both on level conditions and at a 12° incline (the maximum for the treadmill), a slope that has been shown to generate significant differences in the kinematics of the lower limb (Milliron and Cavanagh, 1990). All subjects signed informed consent forms, and all methods used were approved by the Harvard University Human Subjects Committee.

Electromyographic and kinematic data collection

Disposable, self-sticking, pre-wired surface EMG electrodes (Kendall, LTP, Chicopee, MA, USA) were placed over the center of their right and left GM approximately 5–6 cm below the cranial origin of the muscle (see Fig. 1). This electrode position corresponds approximately with the location of the muscle’s innervation zone (IZ) as determined by Rainoldi et al. (Rainoldi et al., 2004). Surface EMGs were used in this experiment because there are no nearby muscles likely to interfere with GM signal, and because they record from a number of motor units to give a general view of the muscle’s activity. Preliminary studies found that this location gave very consistent results that corresponded well to EMG signals from electrodes placed in various different locations of the muscle. Surface EMGs were also secured to the skin at the approximate midpoints between origin and insertion of the biceps femoris (hamstrings), and the gluteus medius. Electrodes were plugged into grounded preamplifiers worn on a waist belt connected *via* a lightweight fiber-optic cable to a MA300 EMG amplifier (Motion Analysis Inc., Baton Rouge, LA, USA). Loose wires were taped to the skin to prevent signal artifacts associated with wire movement during locomotion. The analog signal was passed through an A/D board (PowerLab, ADInstruments Inc., Colorado Springs, CO, USA) and data were captured at 4000 Hz and monitored in real time using Chart software (ADInstruments, Inc.).

In order to record kinematic data on the different portions of the stance phase, thin, flexible footswitches (Motion Analysis Inc.) were taped under the heel and under the head of the first metatarsal of each foot. Because the heel and toe footswitches have different voltage signals, the footswitches record for both feet the onset of heel-strike (HS), foot-flat (FF),

heel-off (HO), and toe-off (TO). A rate gyro (Watson Industries, Inc., Eau Claire, WI, USA) which outputs 0.31 V deg.$^{-1}$ s^{-1} was firmly taped to the upper back inbetween the vertebral borders of the scapulae to measure trunk pitch velocity.

Data analysis

Data from the footswitches were analyzed using custom designed software in Matlab (written by D.A.R.) to determine the timing of HS, FF, HO and TO. EMG data were also processed using custom-designed Matlab software (by D.A.R.) that performed the following functions. First, all raw data were filtered using a 4th order zero-lag Butterworth bandpass filter with frequency cut-offs at 60 and 300 Hz. After filtering, the onset of each muscle burst was determined using Thexton's randomization method (Thexton, 1996). First, the signal was rectified and then binned using a 10 ms reset integral (see Winter, 1990). A threshold was set at 1% of the maximum amplitude of the integrated signal, and the number of times the signal rose above this threshold ('runs') was calculated. The threshold was raised by 0.5% of the maximum amplitude and the number of runs above the threshold was recorded. This calculation was repeated until the threshold was equal to 100% of the maximum amplitude. Next, the signal was randomized and the threshold method was repeated on the new randomized signal. The threshold for the lowest value of the muscle signal was then calculated by subtracting the number of runs in the randomized signal from the number of runs in the original signal to find the maximum difference. All values below this threshold (e.g. values lower than random muscle activity) were eliminated from the original signal. The maximum value and time of onset for each muscle burst was determined from this processed signal. All maximum amplitudes were normalized to the maximum mean muscle burst recorded for each subject during the session.

Maximum anteroposterior rate of trunk pitching was determined as the maximum amplitude following heel-strike. For all EMG magnitudes and timing values, as well as kinematic variables, means were calculated from a minimum of five strides from each subject at a given velocity and experimental condition.

Statistical analyses

Means for each subject at each velocity and experimental condition were calculated using Excel. Since the standard errors of the pooled means for normalized GM levels differed significantly (P<0.05) between speeds and conditions (as determined by ANOVA), repeated-measures ANOVA (with a Tukey–Kramer *post-hoc* test to account for multiple comparisons) was used to assess the effects of individual differences on variance. By using the mean value of each individual for each trial, this method partitions variance attributed to differences between individuals within a given trial condition from variance attributed to difference between trials (Sokal and Rohlf, 1995). Additionally, means and standard errors for each individual were compared within each experimental condition to test for the effects of velocity on the variable of interest.

Results

As predicted by Hypothesis 1, the most salient characteristic of GM activity during locomotion is that the basic pattern and magnitude of GM contractions differ substantially between walking and running, as shown in Fig. 2 and Table 1. During a walk (Fig. 2A), the GM tends to contract at low levels following HS and throughout the ipsilateral stance phase with no obvious peak. During a run (Fig. 2B), the GM tends to contract biphasically with a first burst just prior to HS on the ipsilateral side, and a second, shorter burst prior to mid-swing about the time of HS on the contralateral side. In addition, normalized EMG magnitudes in the GM become higher with increasing velocity (at very high speeds the magnitude and duration of activity increases for both bursts blurring the distinction between these bursts in some individuals). During level walking (Fig. 3A), peak GM magnitudes around the time of ipsilateral HS are quite low, less than 10% of maximum amplitudes, but increase by about 2.5-fold between 1.0 and 2.0 m s^{-1}. Peak GM magnitudes at ipsilateral HS during running are approximately 50% higher (P<0.05) than for a walk at 2.0 m s^{-1} (a slow run, below the preferred walk–run transition for all subjects), and increase by approximately twofold between 2.0 and 4.0 m s^{-1}. As shown in Fig. 3B, peak magnitudes of GM activity during the swing phase also increase as a function of speed, and are significantly (P<0.05) higher in running than walking at the same speed (2.0 m s^{-1}). In addition, walking on an incline increased peak stance-side EMG magnitudes only slightly, well below levels for running; moreover, in contrast to level running, EMG magnitudes during uphill running do not significantly increase with speed (Fig. 3A).

Hypothesis 2 – that normalized levels of GM activity correlate positively with forward trunk pitch, and that timing of GM activity correlates with differences in the timing of peak

Table 1. *Comparison of kinematics and muscle activity in walking and running at 2.0 m s^{-1}*

Variable (% normalized maximum)	Walking	Running	Significance (paired *t*-test)
GM stance magnitude	0.42±0.04	0.53±0.04	0.002
GM swing magnitude	0.48±0.06	0.59±0.05	0.046
Trunk pitch rate (deg. s^{-1})	–67.61±20.61	–146.61±21.93	0.011

Values are means ± s.e.m. (N=9).

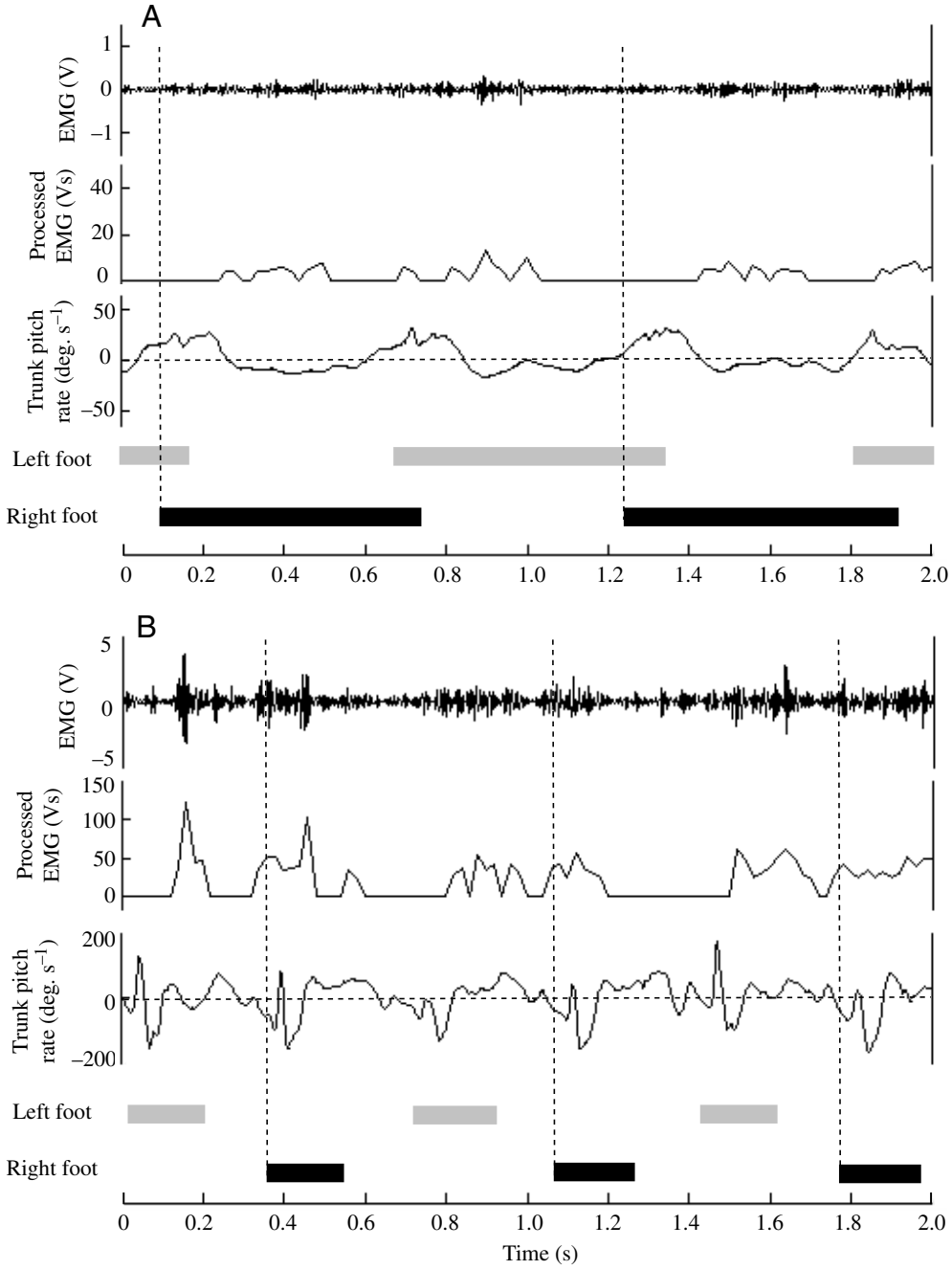

Fig. 2. Raw EMG traces of right-side gluteus maximus during walking (A; 1.5 m s^{-1}) and running (B; 3.0 m s^{-1}). Processed EMG is the filtered data (see text for details). Note that the scales for raw EMG, processed EMG and trunk pitch rate are different in walking compared to running. Note also that for both walking and running, forward trunk pitch rate is negative. Gray bars represent left foot contact and black bars represent right foot contact. Broken line indicates ipsilateral heel strike.

trunk flexion in walking (after midstance) and running (at heelstrike) – is also supported. Trunk pitching rate is much lower in walking than running, even at the same speeds. Maximum pitch rates during walking ranged from approximately –25 to –75 deg. s^{-1}, but were between –150 and –250 deg. s^{-1} during running (Fig. 4A). As hypothesized, trunk pitch correlates well (r^2=0.96) with normalized EMG magnitudes in both gaits (Fig. 4B). These results corroborate findings from earlier studies that the GM plays an active role in stabilizing the trunk against sagittal pitching (Marzke et al., 1988).

As noted above, Hypothesis 2 also predicts differences in the timing of the onset of GM contractions in running compared to walking. As illustrated in Fig. 2 and quantified in Fig. 5, the onset of GM always occurs *after* HS in a walk but always *prior* to HS in a run, with significantly earlier onset relative to HS as a percentage of stride duration with increasing speed during running. Note also that for both gaits (as predicted), the timing of maximum muscle activation occurs after the time of maximum trunk pitch rate (Fig. 5B).

Hypothesis 4 – that the GM also functions as a thigh extensor at the hip to help perform work – predicts that the timing and normalized levels of GM and the other major thigh extensor group, the hamstrings, should correlate well with each other. The magnitudes of maximum stance-side muscle activity for both GM and one of the hamstrings (the biceps femoris)

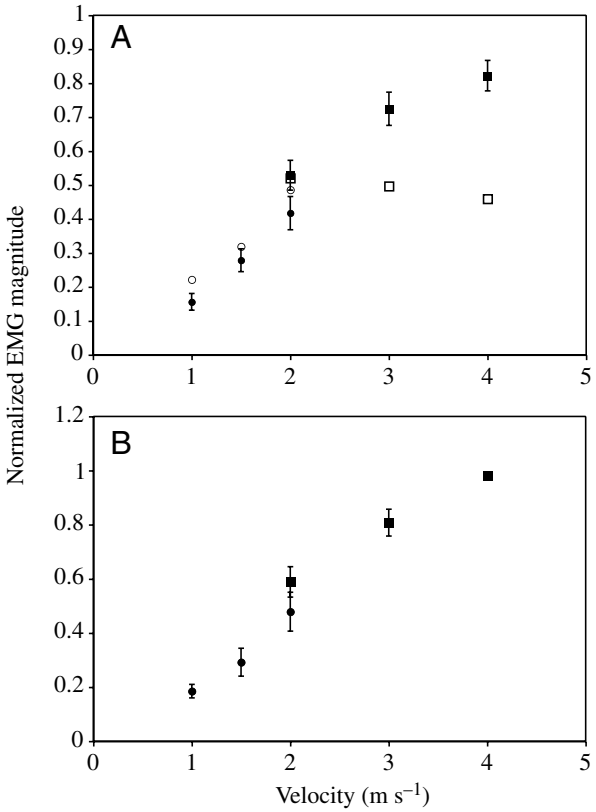

Fig. 3. (A) Normalized stance phase EMG magnitudes for the right-side gluteus maximus during level walking (filled circles), level running (filled squares), uphill walking (open circles) and uphill running (open squares). (B) Normalized swing phase EMG magnitudes during level walking (filled circles), level running (filled squares). Values are means ± 1 s.e.m.

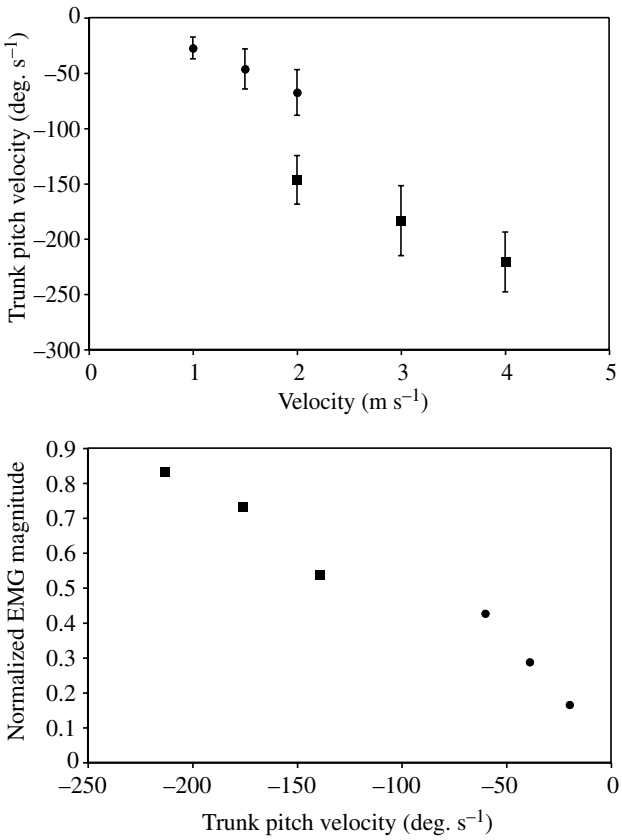

Fig. 4. (A) Normalized EMG magnitudes for the right-side gluteus maximus during walking (filled circles) and running (filled squares). (B) Trunk pitch rate (deg. s^{-1}) *versus* normalized EMG magnitude during walking (filled circles) and running (filled squares). Values are means ± 1 s.e.m.

were quite similar (F=0.593, P=0.705) as they both increased with velocity (Fig. 6A). Additionally, the time of onset for these two muscle groups (Fig. 6B) did not differ significantly (F=0.201, P=0.961).

Finally, subjects were asked to walk and run using a bent-hip bent-knee ('Groucho') gait in order to test the hypothesis that the GM may help to prevent the hip from collapsing into flexion during stance phase (Hypothesis 3). As Fig. 7A indicates, GM activity during 'Groucho' gaits was not significantly different from normal trials in walking, but was significantly lower compared to normal trials during running (F=5.549, P<0.05). Although these results suggest that the GM does not play an important role in resisting hip flexion (see below), maximum trunk pitch velocities also decreased significantly (F=2.952, P<0.05) during 'Groucho' running trials compared to control trials (Fig. 7B). As previously demonstrated (McMahon et al., 1987), ground reaction forces decrease significantly and subjects adopt a more vertical trunk posture during 'Groucho' running. Normalized EMG magnitudes during 'Groucho' running correlate very tightly (r^2=0.93) with the predicted relationship (Hypothesis 2) between EMG activity and maximum trunk pitch velocities noted above for normal and uphill walking and running (Fig. 7C).

Discussion

This is the first study to test quantitatively differences in GM function during walking *versus* running. The results reported in this study support some but not all of the five hypotheses outlined above. First, and most clearly, the GM is considerably more active during running than either normal walking or incline walking (Hypothesis 1). In particular, EMGs both are higher by several-fold and begin earlier relative to HS in walking *versus* running, supporting the findings of the only previous study that specifically compared GM activity for both gaits (Stern et al., 1980), as well as studies that solely examined walking (e.g. Joseph and Williams, 1957; Sutherland et al., 1960; Karlsson and Johnsson, 1965; Marzke et al., 1988). These results reported in the present study, however, do not indicate that GM has no functional role during walking. GM activity during walking in this study increased with speed, and as noted elsewhere by modeling analyses (Anderson and Pandy, 2003; Jonkers et al., 2003a), low levels of GM activity

may contribute to hip extension during stance, and to restraint of hip flexion during swing. While there is no simple relationship between normalized EMG magnitudes and muscle force production, the relatively lower levels of activity recorded here and in other studies do not support the hypothesis that enlargement of the GM in humans is primarily related to bipedal walking on flat surfaces (e.g. treadmills). Since larger muscles typically have greater force generation capabilities, other functional roles are needed to account for the relative expansion in humans compared to non-human primates in the absence of any apparent need to generate large forces during bipedal posture and walking.

One caveat, however, that requires more study is that GM activity may be important in walking up very steep inclines or very uneven terrain. The maximum incline in this study was only 12%, which is not particularly steep but nonetheless sufficient to induce noticeable changes in hindlimb kinematics (Milliron and Cavanagh, 1990), and which may require increased control of trunk flexion. Future experiments are needed to assess role of GM in such walking conditions, but there is some reason to suspect that they will be minor. Tokuhiro et al. found that GM activity is only subtly affected by uphill walking (Tokuhiro et al., 1985), and Swanson and Caldwell found that while the onset of GM contractions were relatively earlier in stance during running at a 30% incline (at 4.5 m s^{-1}), activity levels were not significantly higher (Swanson and Caldwell, 2000). In addition, the major added challenge of walking on uneven terrain is control of hip abduction, which is mostly accomplished by the gluteus medius and gluteus minimus (Soderberg and Dostal, 1978).

Although GM activity is demonstrably important in a wide variety of tasks including climbing and bending (see Marzke et al., 1988; Zimmerman et al., 1994), the above results support several specific hypotheses about the role of the GM during running (Stern et al., 1980; McLay et al., 1990; Bramble and Lieberman, 2004). Just as it was shown (Marzke et al., 1988) that the GM plays an important role in controlling flexion of the trunk during upright bipedal posture, the above results support Hypothesis 2 (above) that a major role of the GM is to extend the hip on the stance side to help control flexion of the trunk during running. Several lines of evidence support this hypothesis. First, as speed increases, so does trunk pitch rate and relative activation of the GM, leading to a nearly perfect correlation between maximum peak EMG magnitudes on the

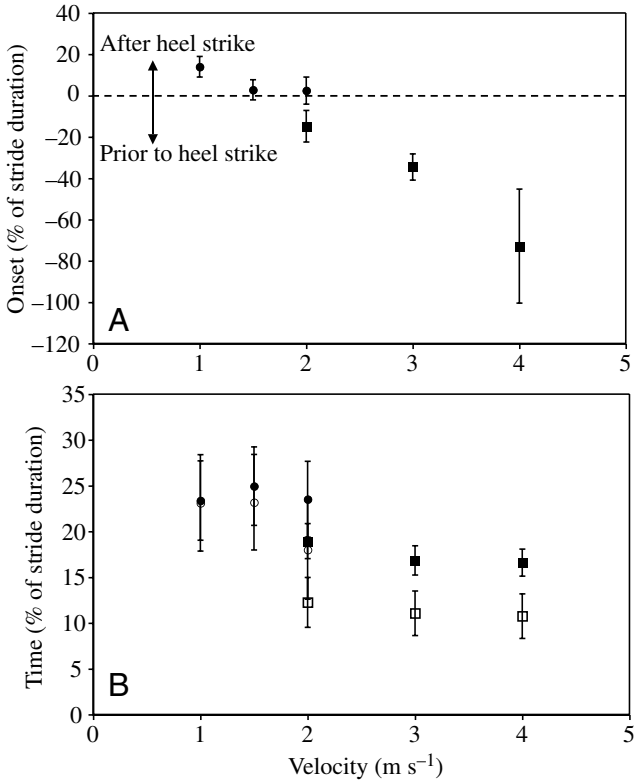

Fig. 5. (A) Time of gluteus maximus onset during walking (closed circles) and running (closed squares) as a percentage of stride duration. (B) Time of maximum gluteus maximus magnitudes (closed symbols) and maximum trunk pitch velocities (open symbols) during walking (circles) and running (squares) as a percentage of stride duration. Heel strike is broken line (0%). Values are means ± 1 s.e.m.

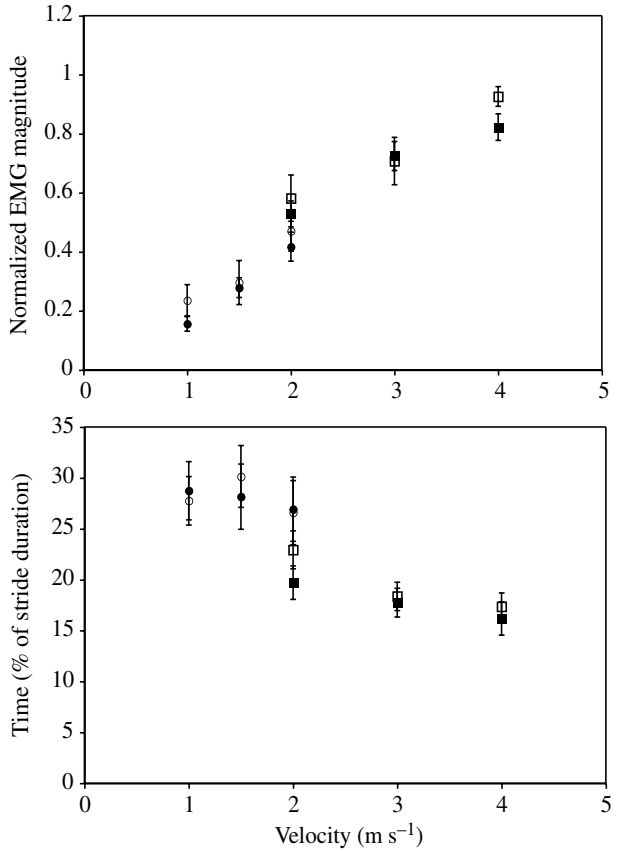

Fig. 6. (A) Normalized maximum magnitudes of gluteus maximus (closed symbols) and hamstrings (open symbols) during walking (circles) and running (squares). (B) Time of maximum magnitude of gluteus maximus (closed symbols) and hamstrings (open symbols) during walking (circles) and running (squares) trials. Heel strike is 0%. Values are means ± 1 s.e.m.

stance side and maximum peak trunk velocities across a range of speeds in both gaits. Importantly, this relationship is also true during 'Groucho' running when peak EMG activity was much lower relative to speed than during normal running (Fig. 7B), but at the level predicted for trunk pitch rate (Fig. 7C). The timing of GM activation also makes sense in terms of controlling trunk pitch rate. Peak flexion of the trunk in a walk occurs after MS as the body's center of gravity is beginning to fall, but in a run occurs at the time of HS. As Hypothesis 2 predicts, the stance-side GM contracts after HS in a walk but before HS in a run, thereby helping the GM extend the hip as the trunk pitches anteriorly. Additional evidence for the GM's role in controlling trunk flexion is provided by the results of the 'Groucho' gait trials. Although the timing of GM contractions during running could indicate that the stance-side GM functions as an antigravity muscle to resist flexion of the thigh relative to the trunk at HS, normalized peak magnitudes of the GM at HS were lower during 'Groucho' gaits than normal trials. This decrease in activity during 'Groucho' trials suggests that stabilizing the thigh to counteract flexion is not a major function of the GM. Instead, decreases in normalized peak EMG magnitudes during 'Groucho' trials relative to normal trials are predicted by the strong correlation between maximum trunk pitch rate and GM activation for normal walking and running (Fig. 7C). This result provides strong support for the hypothesis that GM functions largely as a trunk stabilizer during running.

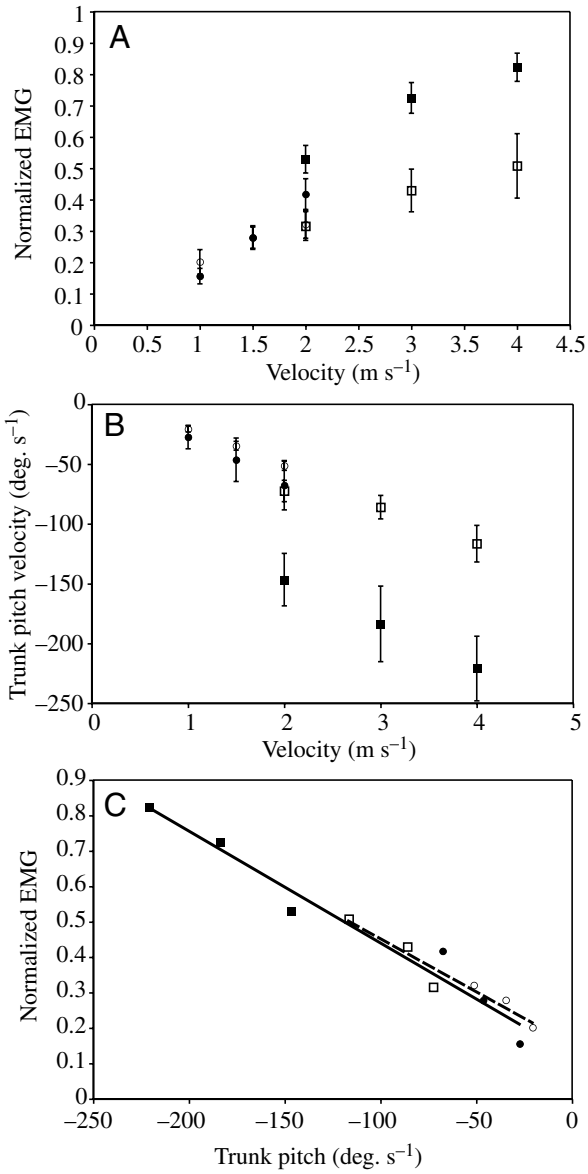

Fig. 7. (A) Normalized maximum magnitudes of gluteus maximus during control (closed symbols) and 'Groucho' (open symbols) walking (circles) and running (squares) trials. (B) Maximum trunk pitch velocity during control (closed symbols) and 'Groucho' (open symbols) walking (circles) and running (squares) trials. (C) Maximum trunk pitch velocity *versus* normalized EMG magnitude during control (closed symbols) and 'Groucho' (open symbols) walking (circles) and running (squares) trials. Values are means ± 1 s.e.m.

Although the above results do not support the hypothesis that the GM functions as a postural muscle to control flexion of the thigh during stance when the hip is flexed (Hypothesis 3), they do suggest that the GM has additional functions. One of these functions may be to help actively extend the thigh during stance (Hypothesis 4). In particular, the timing and magnitude of stance-side GM contractions were very similar to those of the hamstrings during both walking and running, confirming the results of several previous studies (Mann and Hagy, 1980b; Montgomery et al., 1984; Nilsson et al., 1985; Jonhagen et al., 1996). Such results are particularly interesting in terms of uphill locomotion. Roberts and Belliveau calculated that hip extensors such as the hamstrings and the GM may not produce much work output during horizontal running, but have increasingly high moments during uphill running (Roberts and Belliveau, 2005). It has also been shown (Sloniger et al., 1997; Belli et al., 2002) that the hip extensors have low moments and comparatively lower activity levels compared to the ankle and knee during flat running at normal speeds, but become increasingly important at very fast sprinting speeds. Further studies are needed to assess the contributions of the GM to hip extension during uphill running. As noted above, one explanation for the observed decrease in GM activation during uphill *versus* level running could be a decrease in trunk pitch caused by lower GRFs or possibly other changes in kinematics (e.g. contact time, or more vertical trunk postures). However, one other study that examined GM activity during running at an incline (Swanson and Caldwell, 2000) found earlier timing as well as higher levels of GM activity during uphill running, but at a much steeper incline (30°) and a faster speed (4.5 m s^{-1}) than examined in this study.

Finally, the GM is also active on the swing side during the aerial phase of running, when it can play little or no role either to control flexion of the trunk or to help extend the leg. As suggested (McLay et al., 1990), the most likely function of swing-side contractions of the GM is to decelerate the leg during swing phase. These results are also consistent with those reported by previous studies (Mann and Hagy, 1980b;

Montgomery et al., 1984; Nilsson et al., 1985; Jonhagen et al., 1996). While this hypothesis is difficult to test, it is consistent with data on both the timing and magnitude of normalized GM contractions at different speeds. In particular, the swing-side EMG contracts just prior to the midpoint of swing phase regardless of speed; in addition, as speed increases, so does the relative magnitude of the swing-side EMG. One possibility that needs further study is whether the braking action of the swing-side GM on the thigh also helps passively extend the knee.

Comparative function and evolution of the GM

The above results indicate that the reorganization and relative enlargement of the GM in humans does not give the muscle a major role in level bipedal walking. While we cannot discount the hypothesis that the GM was important for walking over uneven terrain (see above), the results of this study indicate that the GM has several critical functions that improve running performance. These data, combined with other results (Marzke et al., 1988) on bipedal postural control, raise several questions about the evolutionary origins of the unique anatomy of the human GM. To address these questions, it is useful to begin with a comparison of what is known about GM function in humans *versus* non-human primates, especially apes, in relation to their anatomical differences.

Non-human GM activity during locomotion has been examined using EMG in both chimpanzees (Tuttle et al., 1975; Stern and Susman, 1981) and in macaques (Hirasaki et al., 2000). These studies indicate that GM activity is generally similar during bipedal walking and vertical climbing in apes (Stern and Susman, 1981), both of which differ from activity during quadrupedal walking. In apes, the GMIF (which is absent in humans) and the middle and anterior portions of GMP (broadly homologous with the more cranial fibers of the human GM) are active during stance phase of both bipedalism and vertical climbing (Tuttle et al., 1975; Stern and Susman, 1981). Hirasaki et al. also noted GM activity during stance phase of climbing in Japanese macaques (Hirasaki et al., 2000). GM activity during swing phase is somewhat more variable in non-human primates, although the GMIF is active in apes at the end of swing during bipedal walking but not during vertical climbing (Stern and Susman, 1981). These results suggest three major functions of the non-human primate GM. First, the GMIF primarily acts as a thigh extensor during the stance phase in both climbing and walking (Stern, 1972; Tuttle et al., 1975; Stern et al., 1981). Second, the non-human primate GMP probably functions primarily as a thigh rotator (Tuttle et al., 1975; Stern and Susman, 1981), preventing the flexed femur from collapsing into lateral rotation during bipedal stance phase. Finally, the GMIF also helps decelerate the limb during swing phase in terrestrial locomotion (it is probably unnecessary to decelerate the limb during climbing).

As noted (Stern, 1972), evidence that the human GM and the ape GMIF both act primarily as hip extensors during the stance phase explains much of the derived configuration of human GM anatomy in terms of the reorganization of the human pelvis for bipedalism. In particular, apes use the more caudal fibers of the muscle, the GMIF, to extend the thigh during climbing and bipedal walking, and humans use the functionally equivalent cranial portion of the GM for bipedal running, and to a much lesser extent in walking. In addition, the ape GMIF and the human GM are both active towards the middle or end of swing phase, suggesting a shared role in swing-limb deceleration. The major functional contrast between humans and apes is that the ape GMP is primarily a medial rotator of the hip to counteract the tendency of the thigh to collapse into lateral rotation, and it may act additionally as an abductor of the thigh to prevent the tendency of the stance-side hip to collapse into adduction (Stern and Susman, 1981). Since GM activity is quite low during human bipedal walking on level surfaces, to the point of being absent in some subjects (Sutherland et al., 1960), it is reasonable to conclude that the expansion of the cranial portion of the GM is probably mostly related to its most dominant function in humans, the control of trunk pitch (Marzke et al., 1988). In order to test this hypothesis more fully, however, additional data are needed on the amount of work done by the muscle during hip extension (which would result in positive work), *versus* trunk flexion (which would result in negative work). It would also be useful to assess GM activity during walking and running on uneven substrates, although preliminary EMG data (unreported) during walking on uneven ground indicates no measurable increase in activity.

Another relevant point is that the human GM acts in conjunction with the erector spinae to control flexion of the trunk at two different joints. The erector spinae, which attaches to the sacrum and iliac crests, filling the trough between the left and right iliac tuberosities, extends the sacroiliac joint. The human GM shares part of the same area of attachment and aponeurosis as the erector spinae (Standring et al., 2005), but primarily extends the hip. Both muscles thus act in a complementary, combined fashion to control flexion of the trunk at the hip and the sacroiliac joint. Therefore, expansion of the human GM, which is essentially an expansion of the ape GMP, likely helped permit an important functional linkage across the two joints between the thigh and the lower back that is necessary to stabilize trunk pitch in a biped.

Another, related question is when the expansion and reorganization of the human GM occurred. Unfortunately, it is difficult if not impossible to reconstruct reliably the relative size and precise configuration of any muscle, including the GM, from its origin and insertion markings in fossils (Zumwalt, 2006). Although a human-like configuration of the pelvis is apparently present by at least 1.9 million years ago in *Homo erectus* (Day, 1973; Rose, 1984; Ruff, 1995), there is much disagreement over the organization of gluteal musculature in australopithecine species such as *Australopithecus afarensis* and *A. africanus*. Some researchers have suggested that muscle attachment markings on pelves of australopithecines are human-like (Lovejoy, 1988; Haeusler, 2002). In fact, Haeusler suggests that the australopithecine GM not only originated primarily from the ilium, but that it could have been as large as that of modern

humans (Haeusler, 2002). While several fragmentary pelves, notably AL 288-1 (*A. afarensis*) and Sts 14 (*A. africanus*), have roughened surfaces along the posterior iliac crests that may indicate an expansion of the GMP onto the iliac crest, the muscle's region of origin in these specimens appears to be limited to the medial third of the crest nearest the sacroiliac joint (Aiello and Dean, 1990). In several *H. erectus* innominates, as in modern humans, the attachment is much more extensive, comprising a widened, rough surface along the superior iliac crest that extends from the sacroiliac joint to the midpoint of the crest in some individuals. This expansion suggests a relatively larger cranial portion of the muscle in the genus *Homo*. Further evidence, however, is necessary to test the hypothesis that the australopithecine GMP was as expanded as in *Homo* (Toussaint et al., 2003).

Although the cranial origin of the GM in australopithecines may have been smaller and more ape-like than the expanded origin in *Homo*, the femoral insertion of the GM in *Australopithecus* appears to be similar to that of humans and derived from the ape pattern. Most notably, Lovejoy et al. pointed out that the insertion of the GM on the Maka femur (Mak-VP-1/1), attributed to *A. afarensis*, was restricted to the gluteal ridge on the proximal portion of the lateral femur (Lovejoy et al., 2002). As noted (Stern, 1972), this reorganization makes sense given the various derived adaptations of the pelvis for bipedalism that permit the GM to function as a hip extensor, and to accommodate differences in fiber resting lengths brought about by upright posture. It therefore seems likely that australopithecines lack a GMIF similar to that of non-human primates. However, the extent to which the GMIF was reduced or absent is not entirely clear and requires further study.

A final line of evidence comes from biomechanical models of hip muscle function in fossil hominids. The lines of action of the gluteal muscles on the pelvis of Lucy (AL 288-1, *A. afarensis*) were compared (Berge, 1994) using both more ape-like and human-like reconstructions. Berge concluded that an ape-like gluteal pattern, in which the caudal portion of GM is relatively large and the cranial portion is relatively small, would have provided australopithecines with the best leverage for powerful extension of the thigh, and would have allowed for the full range of thigh movements. Had australopithecines a human-like gluteal configuration, the GM would have had little leverage for extension of the femur, an important function in both human and ape locomotion (Berge, 1994; Berge and Daynes, 2001). In another modeling study (Nagano et al., 2005), it was estimated that if australopithecines had modern human-like gluteal attachments, then the GM would have needed to produce 30% higher forces than those of modern humans during walking. Although Nagano et al. primarily modeled the GM as an extensor of the hip (Nagano et al., 2005), they attributed its higher force production to its role as a hip abductor needed to maintain lateral trunk stability on a relatively wide pelvis. In view of the earlier analyses (Berge, 1994; Berge and Daynes, 2001), such estimated increases in GM activity during walking could also be attributed to the muscle's poor mechanical advantage as an extensor. In addition, none of the above studies explicitly considered the muscle's role as a trunk pitch stabilizer.

Considered together, the comparative and fossil evidence for the evolution of the GM suggests that australopithecines probably had an intermediate configuration between that of apes and humans. They clearly resembled humans and differed from apes in lacking expansion of the caudal GMIF portion of the muscle, but possibly did not have the same degree of cranial expansion evident in humans. It is therefore reasonable to hypothesize that australopithecines did not rely as heavily on the GM for trunk stabilization, either because they, like all other primates including chimpanzees, did not habitually run for long distances (Bramble and Lieberman, 2004), or because they compensated for the lack of strong cranial GM fibers with other muscles such as the erector spinae. Expansion of the cranial portions of the GM, however, would have been useful to australopithecines if they included a substantial portion of tree-climbing in their locomotor repertoires.

Future experimental and paleontological research is necessary to clarify the functional and evolutionary history of the human GM. Based on the above results, we offer several alternative scenarios that merit further study. As noted above, one possibility is that australopithecines had an intermediate configuration of the GM (Berge, 1994; Berge and Daynes, 2001), retaining some kind of caudal portion but with a less expanded cranial portion than is evident in *Homo*. If so, then the caudal portion would likely have been an effective extensor of the femur during climbing and perhaps walking, and the cranial portion would have helped to stabilize the sacrum, but probably would not have been a strong trunk stabilizer. An implication of this scenario is that the expansion of the cranial portion of the GM is a derived trait of *Homo* that would have been selected for control of trunk flexion during endurance running (Bramble and Lieberman, 2004) and/or foraging (Marzke et al., 1988). An alternative possibility, however, is that the configuration of the GM in *Australopithecus* was much like that of *Homo* in terms of the loss of the GMIF. Either the australopithecine GM as a whole was relatively smaller, as many researchers suggest, or possibly as large as in humans (Haeusler, 2002). As shown above, the GM in either case is unlikely to have played much of a role in level terrain walking, and is unlikely to have been selected for running given that the genus lacks many other features associated with running capabilities (Bramble and Lieberman, 2004). According to this scenario, the derived anatomy of the GM in *Australopithecus* was probably a reconfiguration of the gluteal musculature for climbing, or a novel adaptation for foraging tasks such as digging that involve flexion of the trunk (Marzke et al., 1988). We cannot discount the hypothesis that expansion of the GM might have been useful for walking on uneven terrain. However, it is clear that expansion of the GM in *Homo* would have benefited any activity that requires trunk stabilization, especially running. Regardless of which scenario is correct, the expansion of cranial portion of the GM is a uniquely hominid characteristic, perhaps distinctive to the genus *Homo*, which

played a vital role in the evolution of human running capabilities.

List of abbreviations

EMG	electromyographic
FF	foot flat
GM	gluteus maximus
GMIF	gluteus maximus ischiofemoralis
GMP	gluteus maximus proprius
GRF	ground reaction force
HO	heel-off
HS	heel strike
IZ	innervation zone
MS	mid-stance
TO	toe-off

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