# Consequences of simulated car driving at constant high speed on the sensorimotor control of leg muscles and the braking response

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**Accepted for publication**
Received 5 March 2016;
accepted 6 June 2016

**Key words**
braking emergency; car driving; leg muscles; sensorimotor control

## Summary

Due to the increase in time spent seated in cars, there is a risk of fatigue of the leg muscles which adjust the force exerted on the accelerator pedal. Any change in their sensorimotor control could lengthen the response to emergency braking. Fourteen healthy male subjects (mean age: 42 ± 4 years) were explored. Before and after a 1-h driving trial at 120 km $h^{-1}$, we measured the braking response, the maximal leg extension and foot inversion forces, the tonic vibratory response (TVR) in gastrocnemius medialis (GM) and tibialis anterior (TA) muscles to explore the myotatic reflex, and the Hoffmann reflex (H-reflex). During driving, surface electromyograms (EMGs) of GM and TA were recorded and the ratio between high (H) and low (L) EMG energies allowed to evaluate the recruitment of high- and low-frequency motor unit discharges. During driving, the H/L ratio decreased in TA, whereas modest and often no significant H/L changes occurred in GM muscle. After driving, the maximal foot inversion force decreased (−19%), while the leg extension force did not vary. Reduced TVR amplitude (−29%) was measured in TA, but no H-reflex changes were noted. The braking reaction time was not modified after the driving trial. Driving at constant elevated speed reduced the myotatic reflex and the recruitment of motor units in TA muscle. The corresponding changes were rarely present in the GM muscle that plays a key role in the braking response, and this could explain the absence of a reduced braking reaction time.

## Introduction

Due to the increasing dependence of humans on automobiles and thus the increase in time spent seated in cars, there is a risk of fatigue of the leg muscles participating in the driving task. Very few data are found on the occurrence of a muscle fatigue during prolonged car driving although it could quite significantly alter the driver's performance. Four studies have examined the consequences of prolonged (1-h) simulated driving on the trapezius, deltoid and vertebral muscles (Sheridan *et al.*, 1991; Kolic & Taboun, 2002; Hostens & Ramon, 2005; Durkin *et al.*, 2006). Only one study by Pääsuke *et al.* (2007) has explored the changes in electromyogram (EMG) and Hoffman reflex (H-reflex) in the plantar flexor muscles during and after repeated, brief low-intensity isometric contractions, which could mimic alternated actions on the accelerator pedal. They reported a decrease in the median frequency (MF) of the EMG power spectrum during contractions and reduced maximal voluntary contractions after these tasks.

Two leg muscles are involved while driving at a constant speed. The plantar flexor muscles (gastrocnemius and soleus muscles) play a key role to adjusting the force exerted by the foot on the accelerator pedal. The dorsiflexor muscles [tibialis anterior (TA), and tibialis posterior muscles] maintain the foot position on the pedal. No study has examined the consequences of driving at a constant speed on the sensorimotor control of these muscles.

The mechanisms of fatigue are complex (Enoka & Stuart, 1992; Gandevia, 2001). Peripheral muscle fatigue results in force failure due to a contractile fatigue. Central fatigue factors may be also present, decreasing the voluntary activation of the muscle from the primary motor cortex or modulating the propagation of the signal down the pyramidal pathways and the activation of the motor unit themselves (Gandevia, 2001). The modulation of the recruitment strategy of motor units constitutes a major component of the central fatigue. It precedes the peripheral muscle fatigue and results in a MF fall of the EMG power spectrum showing a reduced recruitment of high-frequency, highly fatigable, motor units as an attempt to delay the occurrence of force failure (Marsden *et al.*, 1983; Bigland-Ritchie *et al.*, 1986). Several human studies (Bigland-Ritchie *et al.*, 1986; Woods *et al.*, 1987; Garland & McComas, 1990)

 

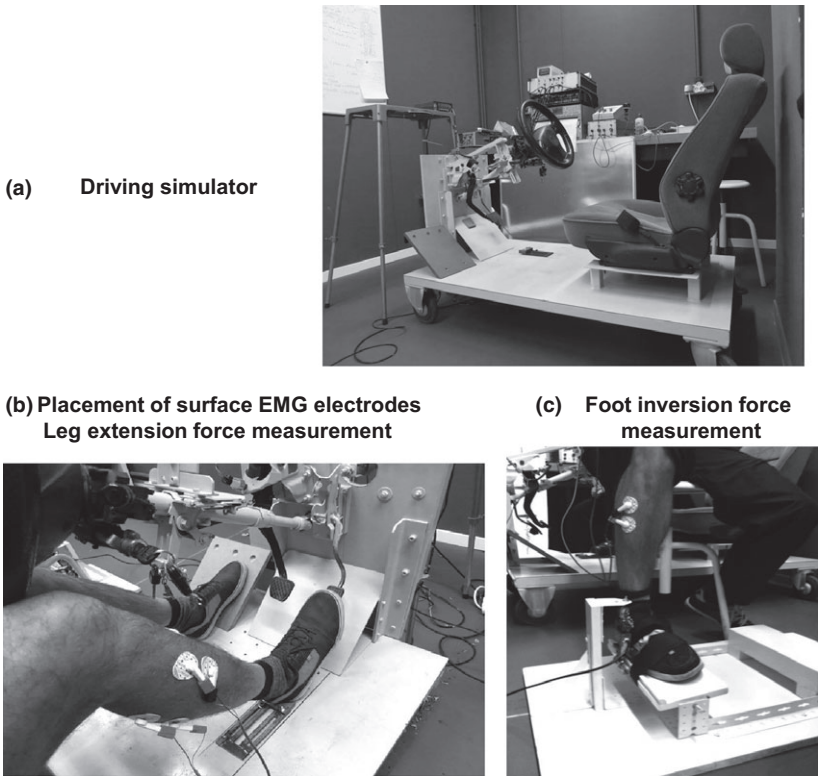


**Figure 1** The driving simulator (a) with details on the surface electrodes positioning (b). The maximal forces produced by the leg extensor and foot invertor muscles were, respectively, measured when the subject was asked to maximally push on the accelerator pedal (b) or to produce inversion movements in the frontal plane (c).

show that this EMG response results from a reflex mechanism due to the activation of the groups III and IV muscle afferents by the muscle metabolites, including the lactic acid. Animal studies also show that muscle acidosis depresses the facilitating influences exerted by the muscle spindles (Lagier-Tessonnier *et al.*, 1993; Decherchi *et al.*, 1998). This could be responsible for the fatigue-induced depression of the tonic vibratory response (TVR) noted by Brerro-Saby *et al.* (2008) and Vie *et al.* (2013a,b) in their study exploring the myotatic reflex in humans. Any TVR reduction in the leg muscles during driving could alter their sensorimotor control with the consequence of a lengthened reaction time of braking.

The present human study examines the driver's performances during a 1-h driving at a constant elevated speed (120 km $h^{-1}$) on a car driving simulator, hypothesizing that fatigue of the leg muscles participating to the driving task could occur. We chose a 1-h driving session to approach the mean time spent in cars in highway traffic and also because previous studies (Sheridan *et al.*, 1991; Kolic & Taboun, 2002; Hostens & Ramon, 2005; Durkin *et al.*, 2006) had shown fatigue of the upper extremities and vertebral muscles within the same period. The sensorimotor control of leg muscles was studied through the recordings of the TVR and H-reflex, and using the EMG power spectrum analysis. The braking reaction time was measured to evaluate the consequences of a possible fatigue of the leg and foot muscles. To estimate a possible bodily fatigue, fatigue scales [Pichot and Multidimensional Fatigue Inventory (MFI) tests] were proposed to each subject after the simulated driving trial had ended.

## Methods

### Ethical approval

This research adheres to the principles of the latest revision of the *Declaration of Helsinki*. The protocol was submitted to and approved by our institutional committee (CPP Sud Mediterranée 1). The procedures were carried out with the adequate understanding and written consent of the subjects.

### Participants

Fourteen healthy male subjects (mean age: 42 ± 4 years; mean weight: 81 ± 3 kg) were explored. All were free of foot pain and had no antecedent of trauma or surgery of the feet and legs. None were involved in an exercise programme. Their driving experience was superior to ten years (20 ± 6 years).

### Instrumentation

A home-made apparatus was built using auto parts including a driver's seat, a wheel, a steering column, brake and

accelerator pedals (Fig. 1a). The subject was asked to maintain constant the force exerted on the accelerator pedal at 20 N. This 20 N value is proportional to the car speed when driving at 120 km $h^{-1}$ a Volkswagen Golf car (data given by IDIADA Automotive Technology SA, Tarragona, Spain). The articulated support of the accelerator pedal was connected to a load cell (Scaime model ZF 100, linear from 0 to 1000 N; AS Technologies, Langlade, France) fixed on the upper third of the pedal arm. Calibration with a 2-kg weight applied on the pedal allowed to correct the data given by the load cell to really develop a 20 N force during the 1-h driving task. Before and at the end of the driving period, the subject received the signal by an intermittent red flash light to push with the right foot on the brake pedal. Electrical contacts with the accelerator and brake pedals gave signals of the loss of pressure exerted on the accelerator pedal and hitting the brake pedal. Their signals were fed to an oscilloscope (Gould model DSO 400, Ballainvilliers, France), permitting to accurately determine the braking reaction time.

The foot placement was constantly adjusted. The ankle and femoro-tibial angles were measured with a goniometer when the subject had chosen his driving position. The mean value of the ankle angle was 103 ± 4° and that of the femoro-tibial angle 107 ± 5°.

## Measurements

### Maximal forces

The maximal forces were measured under isometric conditions. The subject was comfortably seated. To measure the maximal leg extension force, the subject was asked to maximally push on the accelerator pedal to the end of the pedal's range. The maximal foot inversion force was measured using a custom-built device already described (Vie *et al.*, 2013a,b; Fig. 1c). The subjects had no feedback of the developed force, and they were also not informed of the reference values so as not to influence their performance after the driving trial. At each epoch of the protocol, three 5-s maximal leg extension and foot inversion manoeuvres were executed to determine the highest force value. A 5-s rest period separated two successive manoeuvres.

### Electromyographic recordings and analyses

Bipolar (30-mm centre-to-centre) Ag–AgCl surface electrodes (model 13L20; Dantec Medtronic, Skovlunde, Denmark) were used to measure the EMG voltage from the right TA and gastrocnemius medialis (GM) muscles. The EMG signal was recorded (Myosystem 1400A; Noraxon Inc., Scottsdale, AZ, USA), amplified with a common mode rejection ratio = 90 dB, input impedance = 100 mΩ, gain = 5000, the frequency band ranging from 10 to 10 000 Hz. The software program allowed to obtain the power spectrum. The EMG signal was digitized with a sampling frequency of 2000 Hz using the data acquisition card mounted in the computer. The Noraxon software program calculated the root-mean-square (RMS) in two bands of low (L: 10–50 Hz) and high (H: 50–500 Hz) frequencies. The changes in H/L ratio give an indication of a leftward shift of the EMG power spectrum and bring further information on separate H and L changes which, respectively, correspond to the recruitment of highly fatigable and fatigue-resistant motor units. As in our previous studies (Badier *et al.*, 1993; Coulange *et al.*, 2006), a 15% H/L decrease was considered as significant.

### TVR recording

Mechanical vibrations were delivered by an electromagnetic vibration generator (Vibralgic 4; Electronic Conseils, Ales, France). The optimal TVR amplitude was measured for a vibration frequency of 80 Hz. The TVR magnitude was computed by measuring the RMS of raw TA and GM EMG signals and plateaued before the end of a 3-s period of vibration. The 3-s vibration periods were separated by 10-s epochs.

### Hoffman reflex and M-wave

To simultaneously record the muscle action potential (M-wave) and the Hoffman reflex (H-reflex) in the TA and GM muscles, we followed the recommendations of Palmieri *et al.* (2004). During M- and H-wave recordings, the subjects were sitting in a quiet environment because loud noise affects the amplitude of the H-reflex. The cathode was placed over the common peroneal nerve to the lateral aspect of the leg around the head of the fibula, whereas the anode was placed superior to the patella. Two adhesive skin electrodes were used to ensure that the stimulating sites to elicit the M-wave and H-reflex after the driving test were exactly the same as those for their predriving measurements. A neurostimulator (Grass S88, Quincy, MA, USA) delivered 1·0-ms rectangular pulses every 10 s through an isolation unit. The M- and H-waves were fed to an oscilloscope (Gould model DSO 400), permitting an average of 16 successive potentials to calculate peak-to-peak amplitude and latency. The maximal H-reflex amplitude was standardized to the maximal M-wave amplitude. Only the variations of the Hmax/Mmax ratio from control (predriving) were considered.

## Experimental procedure

Figure 2 gives the time schedule of the interventions in our protocol. Before the simulated driving task, three maximal leg extension and foot inversion manoeuvres were successively executed by the right and left foot, measurements from the left foot serving as controls. Control measurements of TVR in the right leg muscles and braking reaction time were then performed. During driving, continuous EMG recordings allowed measurements of the H/L ratio in TA and GM EMGs. After the 1-h driving challenge, we measured within maximally the first 3 min

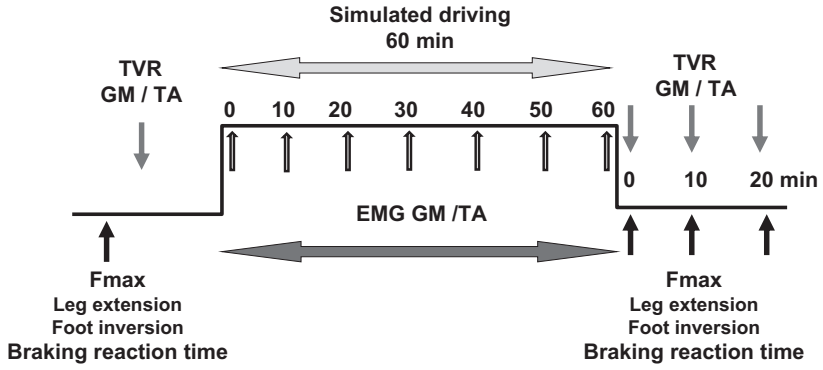


**Figure 2** The protocol of simulated driving. Before and after the 1-h challenge, maximal leg extension and foot inversion forces were measured and tonic vibratory responses recorded in both muscles [gastrocnemius medialis (GM) and tibialis anterior (TA)]. Surface electromyograms (EMGs) of GL and TA muscles were continuously recorded during the driving challenge.

the braking response, TVR in both muscles and the maximal forces (these early data were noted as measured at 'R0'). Then, TVR and maximal forces were measured at the 10th (R10) and 20th (R20) min. Both Pichot and MFI fatigue scales were checked between R0 and R10 epochs.

### Statistical analyses

Data are presented as mean ± standard error of mean (SEM). ANOVA for repeated measures of the TVR amplitude, M-wave and H-reflex characteristics, EMG energies in low and high frequencies bands, and forces, was used when the variables were normally distributed and a Friedman's test for repeated measures when they were not. Time differences were identified using the Tukey's multiple comparison test. Least-squares regression analyses were used to compare the maximal EMG changes measured at the end of the 1-h simulated driving to the maximal TVR variations which were always measured at R0. Significance of ANOVA or Friedman's test and of the R coefficient of regression was accepted if $P<0{\cdot}05$.

## Results

### The changes in the maximal leg extension and foot inversion forces after the simulated driving

Fig. 3 gives the statistical analyses of the maximal inversion and leg extension forces (Fmax) measured before (control) and after the simulated driving. No significant changes in maximal leg extension force were measured, while the maximal inversion force was significantly reduced in the right foot (−19 ± 6%) within the 5 min succeeding the 1-h driving trial.

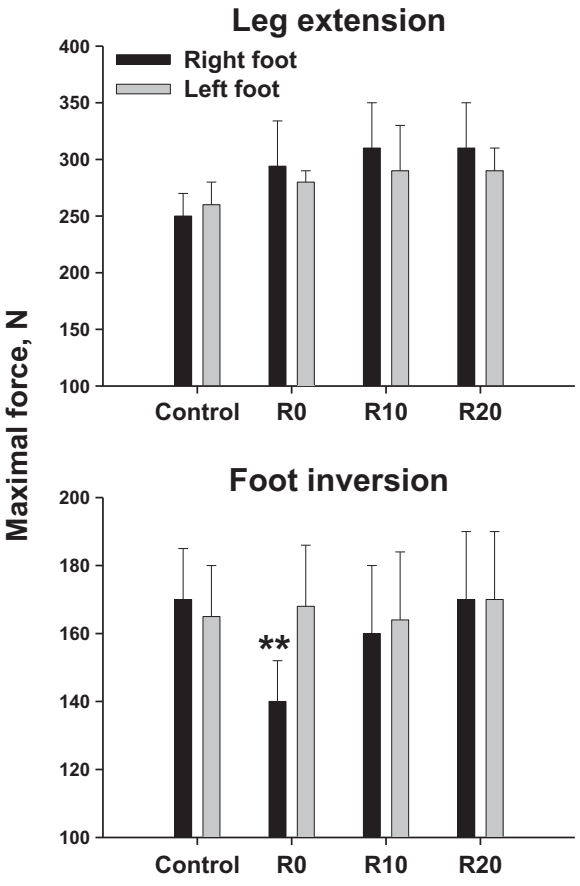


**Figure 3** Changes in the maximal leg extension and foot inversion maximal force of the right and left foot immediately after driving (R0) and at the 10th (R10) and 20th min (R20) postdriving recovery period compared to predriving control value. Data are mean ± SEM. **$P<0{\cdot}01$ compared to predriving control value.

### TVR, M-wave and H-reflex changes in TA and GM muscles after the simulated driving

Fig. 4 gives an individual example of the TVR recordings in both leg muscles, showing a marked fall of TVR amplitude in TA muscle at R0 (−29 ± 5%) and R10 (−21 ± 7%) and a modest, not significant, reduction in GM muscle at R0 in this subject. TVR reduction in TA and GM occurred in nine of 14 and six of 14 subjects, respectively. This explains that the TVR changes were statistically significant in TA but not in GM (Fig. 5).

No significant postfatigue test changes in M-wave amplitude and duration and in H-reflex duration and latency were noted. Also, the Hmax/Mmax ratio did not significantly vary after the fatigue test.

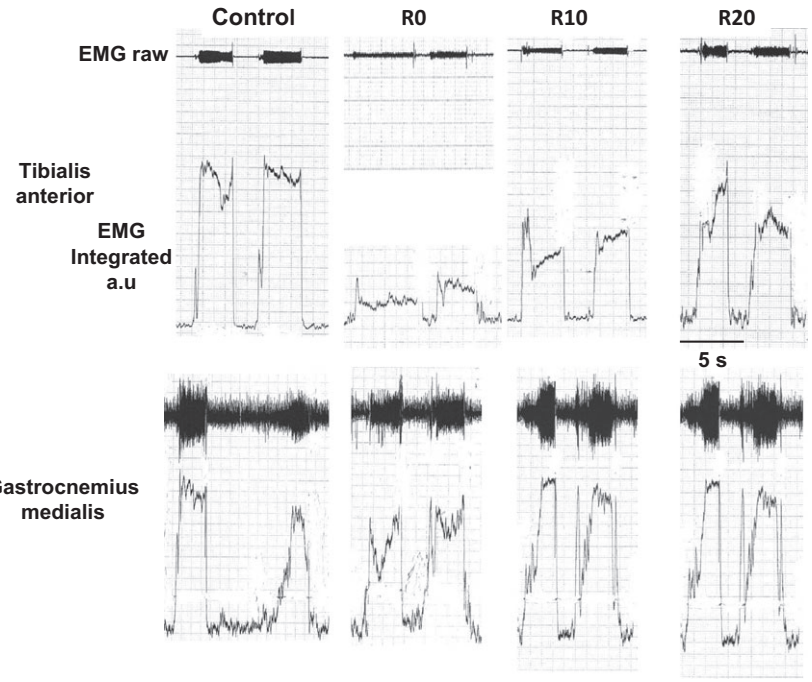


**Figure 4** Individual examples of tonic vibratory response (TVR) recording in the tibialis anterior and gastrocnemius medialis muscles before the driving challenge (control), immediately after driving had stopped (R0) and at the 10th (R10) and 20th min (R20) of the postdriving recovery period.

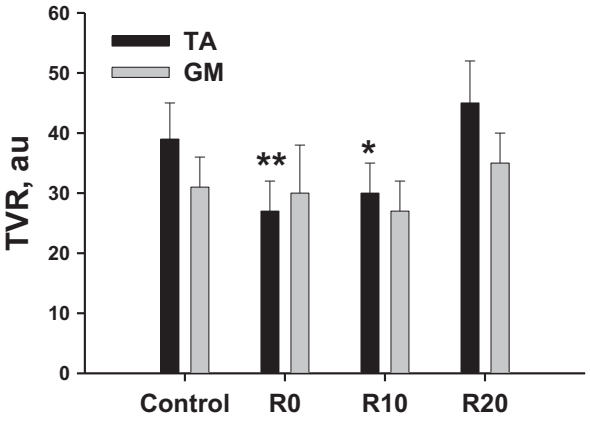


**Figure 5** Changes in tonic vibratory response (TVR) of the tibialis anterior (TA) and gastrocnemius medialis (GM) muscles immediately after driving (R0) and at the 10th (R10) and 20th min (R20) postdriving recovery period compared to predriving control value. Data are mean ± SEM. *P<0·05; **P<0·01 compared to predriving control value.

### EMG changes during the simulated driving

As shown in Fig. 6, a significant H/L reduction in TA occurred after 40 min of driving in 11 of 14 subjects, while the H/L reduction in GM was only noted in seven of 14 subjects. The mean H/L decrease was significantly higher (P<0·05) in TA (−28 ± 5%) than in GM (−7 ± 10%).

### Relationship between TVR and H/L changes

Significant linear correlations were found between the decreases in H/L ratio and TVR amplitude measured at the end of the simulated driving (Fig. 7). Thus, in both leg muscles, the highest depression of the TVR amplitude occurred in subjects having the highest reduction of the H/L ratio.

The age of the subjects and their driving experience were not correlated with the changes in forces, TVR and H/L ratio.

### Fatigue scales

Mean values of Pichot and MFI tests were, respectively, 6 ± 1 (extreme values: 0–15) and 9 ± 1 (extreme values: 7–13). No correlation was found between the fatigue scores and any physiological variables (maximal foot inversion force, TVR and H/L ratio).

### Braking reaction time

After the 1-h simulated driving, the latency of braking did not significantly vary (control: 145 ± 52 ms versus 130 ± 40 ms).

## Discussion

### Main results

The present study in healthy volunteers shows that a 1-h challenge of simulated driving at a high constant speed (120 km $h^{-1}$) induced a modest bodily fatigue assessed by elevated values of Pichot and MFI scores. After the driving trial, (i) the peak foot inversion force but not the leg extensor force was reduced, (ii) EMG signs of muscle fatigue (reduced H/L ratio) were noted in the TA muscle in the majority of our subjects but in only half the subjects in the GM muscle, (iii) there was a marked and prolonged (10 min) reduction of the TVR amplitude in the TA muscle (nine of 14 subjects),

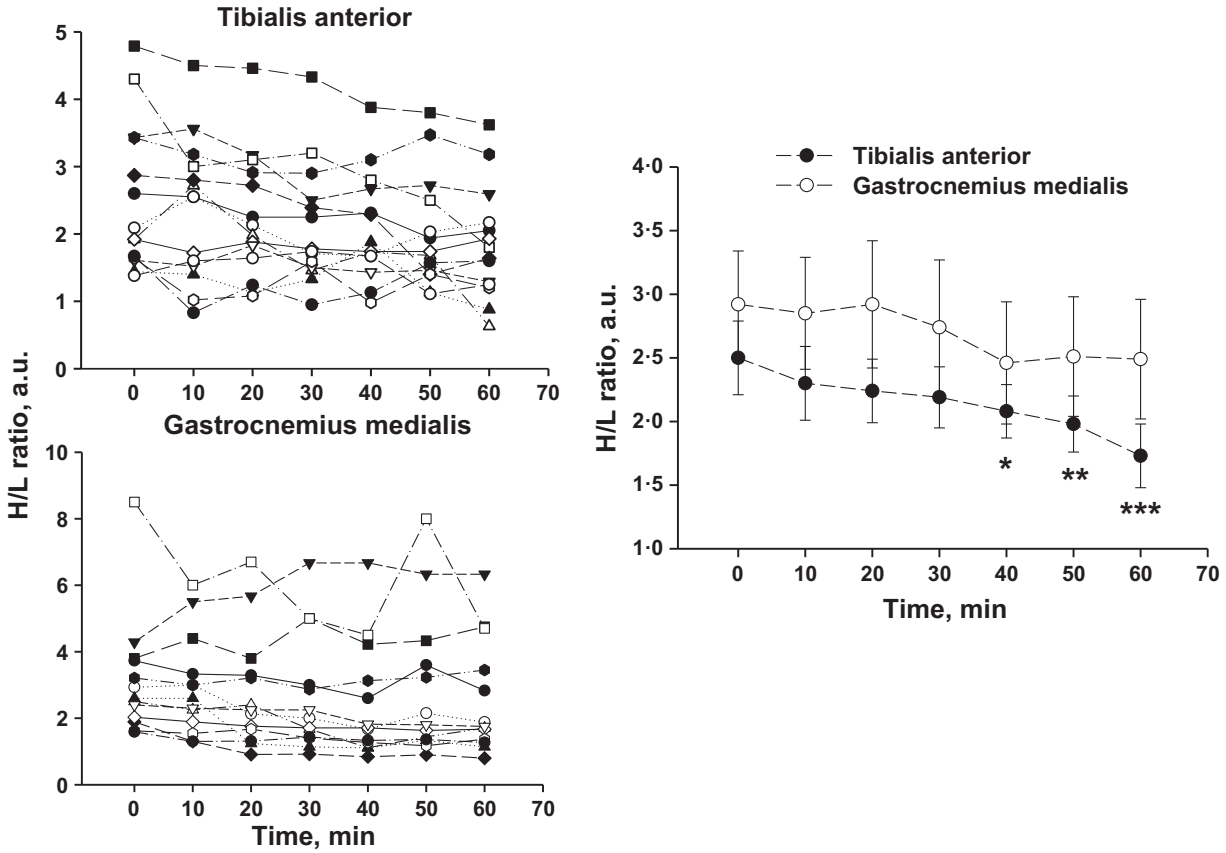


**Figure 6** Individual (A) and mean (±SEM) changes in high above low EMG energies (H/L ratio) in the tibialis anterior and gastrocnemius medialis muscles measured every 10 min during the driving challenge. *P<0·05; **P<0·01; ***P<0·001 compared to data measured at the onset of the trial.

while in the GM muscle, the TVR depression only occurred in six of 14 individuals, (iv) the magnitude of TVR decrease was positively correlated to that of H/L, (v) no significant post-drive variations of the H-reflex and M-wave were noted, and (vi) the braking reaction time was not affected.

### Breaking reaction time

The braking reaction time here measured was in the range of values reported by Hofmann *et al.* (2014). The braking reaction time involves several mechanisms, including the displacement of the leg to the brake pedal as well as the sensorimotor control of the leg's extensor muscles with the participation of both their central command and peripheral reflexes. The absence of any significant variations of the braking reaction time with time suggests that the different steps of the braking action were not affected. Indeed, the leg displacement was the same at the successive epochs of the protocol (pre- and post-driving sessions).

### Bodily fatigue

The modest increase in bodily fatigue scores related by our subjects could simply indicate a perception of discomfort due to the sustained static contraction of the TA muscle which apparently had no consequences on the central command to the leg muscles.

### Leg extension and foot inversion strength

No contractile fatigue of the leg extensor muscles was measured, whereas the maximal foot inversion force declined after the 1-h driving task. It merits to be underlined that fatigue of foot invertor muscles often occurs after maximal running (Vie *et al.*, 2013a) or maximal static efforts (Vie *et al.*, 2013b). The TA muscle, a foot invertor, contains a very large proportion of fast-twitch, highly fatigable fibres (Johnson *et al.*, 1973), and this could explain that the foot inversion force was often affected in the aforementioned studies including the present one. On the other hand, the gastrocnemius comprises a mixture of type I (fatigue resistant) and type II (fatigable) muscle fibres with a slight tendency towards a greater proportion of type I muscle fibres (Johnson *et al.*, 1973) explaining the absence of its contractile fatigue after the driving task (and also after running). Moreover, the target force sustained by the foot muscles was very low (20 N) compared to other protocols when the subjects had to sustain maximal or submaximal forces (Bigland-Ritchie *et al.*, 1986; Woods *et al.*, 1987;

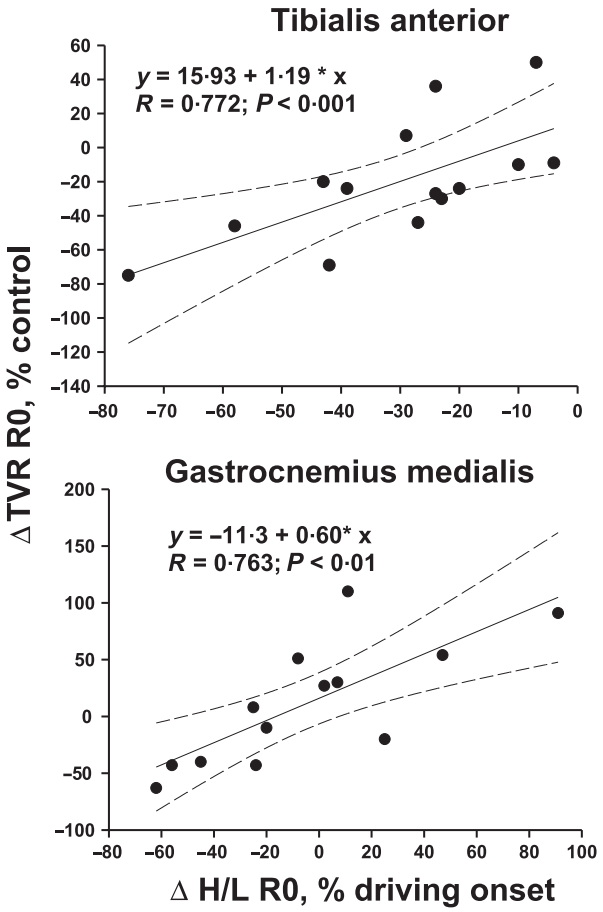


**Figure 7** Correlations between maximal H/L and tonic vibratory response (TVR) variations measured at the end of the 1-h driving challenge in tibialis anterior and gastrocnemius medialis muscles. Regression lines with 95% confidence intervals are drawn.

Garland & McComas, 1990; Brerro-Saby *et al.*, 2008; Vie *et al.*, 2013a,b).

### Myotatic reflex

In our previous human studies focused on muscle fatigue during sustained isometric tasks, the different possible mechanisms of the postexercise TVR reduction had been extensively discussed (Brerro-Saby *et al.*, 2008; Vie *et al.*, 2013a,b). The two main processes are (i) the direct inhibitory effects of lactic acid on the muscle spindle discharge (Lagier-Teissonnier *et al.*, 1993; Brerro-Saby *et al.*, 2008) and (ii) a muscle thixotropy, that is the changes in muscle mechanical properties due to the passive muscle stretch, which inhibits for consecutive short periods (few minutes) the myotatic reflex (Trajano *et al.*, 2014). In our study, it seems doubtful that the modest force exerted on the accelerator pedal was sufficient to produce an amount of lactic acid sufficient to alter the muscle spindle discharge. Thus, the postdrive TVR reduction in TA muscle could simply result from muscle thixotropy.

### Surface electromyogram

In protocols of sustained muscle contractions at a high strength (Bigland-Ritchie *et al.*, 1986; Woods *et al.*, 1987; Garland & McComas, 1990; Brerro-Saby *et al.*, 2008; Vie *et al.*, 2013a,b), EMG variations (H/L or MF decrease) occurred a few minutes, and even less, after the onset of the trials. In all cases, these EMG changes indicated the activation of the 'muscle wisdom' phenomenon, which is considered as a tool protecting the muscle against a contractile failure (peripheral fatigue; Marsden *et al.*, 1983; Bigland-Ritchie *et al.*, 1986; Enoka & Stuart, 1992). Observations in humans (Woods *et al.*, 1987; Garland & McComas, 1990; Gandevia, 2001) and animals (Jammes & Balzamo, 1992) are in favour of a reflex origin involving the activation of the group IV muscle afferents by lactic acid and other muscle metabolites (Darques *et al.*, 1998; Decherchi *et al.*, 1998). This suggests that muscle metabolites could be released in the leg muscles in the present protocol of simulated driving. The group IV muscle afferents, which also carry pain sensory pathways, project on both the spinal and supraspinal structures, including the cortical areas (Almeida *et al.*, 2004). These supraspinal projections explain the sensation of muscle fatigue and thus the modest changes in bodily fatigue scores here noted after driving.

Whatever the mechanisms of the reduced myotatic reflex, the combination of decreased facilitatory influences exerted by the muscle spindle afferents on the motoneurons and reduced recruitment of high-frequency motor units should attenuate the contractile fatigue of the TA muscle, constituting an adaptive response of the neuromuscular system. This protective mechanism seems to be very effective to prevent peripheral fatigue during driving because the reduction of the foot inversion force was modest and that of the leg extensor muscles was often absent.

## Limitations of the study

First, the driving condition in our simulated driving model is quite far from realistic because the vibrations from the road, which were absent, are a contributing factor to muscle fatigue during driving (Wilder *et al.*, 1994).

Second, our EMG analyses consisted in separate quantification of EMG energies in high- and low-frequency bands. This was commonly performed in numerous previous studies (for examples: Edwards & Wiles, 1981; Bigland-Ritchie *et al.*, 1986; Enoka & Stuart, 1992; Badier *et al.*, 1993; Hug *et al.*, 2003, 2004). The H/L changes have the same dimension as the MF of EMG power spectrum but bring supplementary information on the pattern of control of the different motoneuron pools. In our study, a progressive decrease in high frequencies EMG activities prevailed throughout the whole driving challenge, while the low frequencies ones remained quite stable. The H/L changes have a sense on the condition that the muscle membrane conduction velocity measured by the M-wave does not vary. In our study, no M-wave changes were measured because the 20 N force applied on

the accelerator pedal corresponded to only 8% of the maximal leg extension torque and 12% of the maximal inversion torque.

We cannot explore the occurrence of central fatigue using the twitch interpolation method in our protocol, as in our previous study in which the subjects were asked to sustain a fatiguing foot inversion (Vie *et al.*, 2013a,b). Indeed, we did not want to disturb the attention of the subjects during the driving challenge by any interpolation of maximal electrical stimulations of the working leg muscles. Given the nature of the simulated driving task (low-intensity isometric contraction), we may suppose that it would more likely induce a central than a peripheral fatigue of the leg muscles. In any case, the reduced H/L ratio during driving and the reduced TVR amplitude after driving were frequently measured in the TA and rarely in the GM muscle. Thus, we may suppose that the foot position during driving predominantly affects the dorsiflexor and not the plantar flexor muscles.

When the knee is flexed, as in driving, the ability of bi-articular gastrocnemius muscles to produce force is reduced because it operates at a short length. Indeed, Cresswell *et al.* (1995) have shown that the maximal leg extension torque decreased significantly in a sigmoidal fashion with an increasing of the knee flexion to 60% of the maximum torque developed at 180 degrees of knee angle. We agree with these data, but the knee being always flexed during driving we decided to measure the maximal leg extension torque in the same position.

## Conclusions

This study may be considered as a pilot first because it shows that prolonged simulated driving at a high speed modifies the sensorimotor control of the TA muscle and also produces a contractile fatigue of this muscle. It seems important to underline that the neuromuscular changes were modest or absent in the GM muscle, explaining the absence of an altered braking response.

## Acknowledgments

This research was only supported by funds from UMR MD2 and UMR T24 (Aix Marseille University) and the School of Podiatry in Marseille.

## Conflict of interest

No conflict of interest exist.

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