The internal model of gravity for hand interception:

Parametric adaptation to zero-gravity visual targets on Earth

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Abstract

An internal model is a neural mechanism that mimics the dynamics of an object for either motor control or cognitive functions. Recent research focuses on the issue of whether multiple internal models are learned and switched to cope with a variety of conditions, or single general models are adapted by tuning the parameters. Here we addressed this issue by investigating how the manual interception of a moving target changes with changes of the visual environment. In our paradigm, a virtual target moves vertically downward on a screen with different laws of motion. Subjects are asked to punch a hidden ball that arrives in synchrony with the visual target. By using several different protocols, we systematically found that subjects do not develop a new internal model appropriate for constant speed targets, but they use the default gravity model and reduce the central processing time. The results imply that adaptation to zero-gravity targets involves a compression of temporal processing through the cortical and subcortical regions interconnected with the vestibular cortex, which has previously been shown to be the site of storage of the internal model of gravity.
**Introduction**

Fast interactions between our limbs and the environment cannot be performed solely under sensory feedback, because of delays and inaccuracies. To overcome these problems, the brain may acquire an internal model of the dynamics of the object to be controlled through motor learning, after which a movement can be performed in feedforward, in addition to feedback (e.g., Kawato 1999; Wolpert et al. 2001). Internal models are often thought to be unique for the objects that we have learnt to interact with. According to this view, a collection of specific internal models is stored in motor memory, and sensory or cognitive information provides the identifying cues to select the appropriate internal model (Flanagan and Wing 1997; Gordon et al. 1993; Wolpert and Kawato 1998). For instance, after subjects learn to manipulate two different tools (a rotated mouse and a velocity mouse), they can easily switch between the two, while distinct, spatially segregated regions are engaged in the cerebellum by each tool (Imamizu et al. 2003).

However, learning and switching between multiple models is not always easy or even possible. Thus, learning of two conflicting environments, such as opposing force fields or opposing visuomotor rotations, has been reported to be difficult or very slow, probably because of long-lasting interference in memory (Caithness et al. 2004; Krakauer et al. 1999). Osu et al. (2004) reported that subjects can learn two opposing force fields when provided with contextual cues and random, frequent switching. However, concurrent learning was slower and more difficult than separate learning. Karniel and Mussa-Ivaldi (2002) taught subjects, on separate days, to move in two different velocity-dependent force fields applied at the hand by a robot. Even after this experience, subjects were unable to move accurately when the same two fields
alternated after each movement. This suggests that subjects did not employ two separate models and switched between them according to the sequence context, but they tried to represent the alternating perturbations with a single internal model. From this and other evidence (see Martin et al. 1996), one is led to conclude that, in addition to multiple internal models specific for individual objects, there also exist single internal models that can cope with a variety of conditions.

Whether we learn a new model or tune an existing one might also depend on the ecological significance of the specific environment (Georgopoulos 2004; Gibson 1966; Shepard 1984). Natural selection favors adaptation to any biologically relevant property of the world, especially those properties that hold throughout all habitable environments. The 24-hour period of the terrestrial circadian cycle (consequence of the conservation of angular momentum) and the gravitational acceleration are examples of ecological constraints that are presumably internalized. On Earth, normally we experience an invariant gravity (1g), and the maximum variation that sporadically we encounter by changing latitude or altitude is <1%. There is increasing evidence that an internal model (called “1g model”) calculating the effects of Earth gravity on seen objects is stored in the brain. Indeed, the human visual system poorly estimates arbitrary image accelerations (Brouwer et al. 2002; Werkhoven et al. 1992), and these accelerations generally are not taken into account in timing manual interceptions (Port et al. 1997). Nevertheless, visually guided interceptions of objects falling under gravity are accurately timed by synchronizing the changes of anticipatory muscle activity, as well as those of reflex activity and limb impedance, to the time of collision (Lacquaniti and Maioli 1987; Lacquaniti and Maioli 1989; Lacquaniti et al. 1991; Lacquaniti et al. 1992; Lacquaniti et al. 1993a; Lacquaniti et al. 1993b; Michaels et al. 2001). Evidence
for internalized visual gravity was provided by the observation that, in the absence of gravity-determined sensory cues, astronauts initially expect the effects of Earth gravity on a dropped object (moving at constant speed) when they attempt to catch it in the Spacelab, and they adapt to the new environment only after few days of flight (McIntyre et al. 2001).

It should be noticed that a fixed model of free fall would not be appropriate to deal with a variety of natural conditions even on Earth. Thus, vertical motion of objects is accelerated by gravity and decelerated to a variable extent by air (or other fluid) friction depending on the object’s mass, size, shape, texture and fluid viscosity. Does the brain develop an internal model for each specific environment or does it adapt a single general model by tuning its parameters?

Here we addressed this issue by investigating how interceptive responses change with changes of the visual environment produced in the laboratory. In our paradigm, a virtual target moves vertically downward on a screen with different laws of motion. Subjects are asked to punch a hidden ball that arrives in synchrony with the visual target. Using this paradigm, it has previously been found (Zago et al. 2004) that subjects time their motor responses very differently when the visual target moves with an acceleration equivalent to gravity (1g) or when it moves at constant speed (0g). Motor responses generally are time-locked to the arrival of 1g targets, whereas the responses to 0g targets are premature. Here we used this paradigm to test between alternative strategies for learning to deal with the novel environment of visual targets descending at 0g. One possibility is that extensive training results in the development of a 0g model appropriate for the new situation. The alternative possibility is that subjects continue using the 1g model throughout the exposure to 0g targets, but adapt its internal
parameters. These two hypotheses entail completely different schemes of neural implementation for the temporal control of interception of falling objects. The first hypothesis predicts that, with training, the brain develops and stores multiple internal models of target motion in different neural populations, equivalent to modules (see Imamizu et al. 2003). The second hypothesis, instead, predicts that adaptation to a new target motion involves a different temporal processing through cortical and sub-cortical regions interconnected with the internal model of gravity stored in the brain. The following section is devoted to the formal treatment of the different model schemes for dealing with 1g and 0g targets.

**Modeling**

*The 1g model for timing interceptions*

The instantaneous height \( h(t) \) of a descending target above the interception point is given by:

\[
  h(t) = h_0 - v_0 t - 0.5 a_0 t^2
\]

(1)

where \( h_0 \) and \( v_0 \) are the initial height and velocity, respectively; \( a_0 \) is the constant acceleration of the target throughout its flight, \( a_0 = 1g \) for targets with an acceleration equivalent to gravity (Fig. 1A) and \( a_0 = 0 \) for constant speed targets (Fig. 1B).

When subjects start from a given initial position of the limb to punch a rapidly descending target, they generally make ballistic (explosive) movements of brief duration and relatively stereotyped waveform (Lee et al. 1983; Michaels et al. 2001; Zago et al. 2004; Zago et al. 2005). Moreover, target contact occurs close to the time of
peak arm velocity, and the deceleration phase is simply a follow-through. For this type of interceptions, it is generally considered that subjects time the movement based on a perceptual estimate of the remaining time-to-arrival at the interception point (referred to as time-to-contact, TTC) of the target (Fitch and Turvey 1978; Lee et al. 1983; Michaels et al. 2001; Tresilian 1999; Tresilian 2004). The movement is centrally triggered when TTC reaches a given criterion value (Lee et al. 1983; Michaels et al. 2001; Tresilian, 2004). The criterion value is also sometimes called time margin or time threshold relative to the expected collision, and will be denoted λ in the following. A time margin is necessary to compensate for signal transmission delays (typically, between 80 and 300 ms, see Port et al. 1997). Thus, λ includes the neuro-mechanical delays, and is equal to the sum of central processing time and peripheral movement time (Lacquaniti and Maioli 1989; Lee et al. 1983; Port et al. 1997; Tresilian 2004). Note that, for these TTC-models, the control of interception is under on-line visual control up to time λ: the visually derived estimate of target TTC is compared continuously (or intermittently) with the preset criterion value, as in a count-down process. When the time threshold λ is reached, the action is launched with little (if any) correction based on feedback (Tresilian 2004).

Within the class of these TTC-models, the 1g model specifically predicts that subjects estimate TTC using on-line visual information about target distance and velocity, under the assumption that it is accelerated by gravity (Lacquaniti and Maioli 1989; Lacquaniti et al. 1993a; McIntyre et al. 2001; Zago, et al. 2004). At time ε after the onset of target motion, the remaining time-to-arrival at the interception point is TTC(ε), target velocity is v(ε) and the height above the interception point is h(ε). Thus, according to the 1g model, when the target arrives at the interception point:
Equation 2 is simply obtained by setting $h(t) = 0$ on the left side of Eq. 1, with $v(\varepsilon)$ and $h(\varepsilon)$ as initial conditions. TTC$(\varepsilon)$ is then given by:

$$TTC(\varepsilon) = -\frac{v(\varepsilon) + \sqrt{v(\varepsilon)^2 + 2gh(\varepsilon)}}{g}$$

and the interceptive response is launched when TTC$(\varepsilon) = \lambda$.

**Predictions of the 1g model for 1g and 0g motions**

For a successful punch, the hand must reach the interception point at the same time as the target does. The hand arrives at time $\varepsilon + \lambda$ after the onset of target motion, whereas the target arrives at time $T$. For a successful interception: $\varepsilon + \lambda = T$. The 1g model correctly estimates the time of arrival of 1g targets if $h(\varepsilon)$ and $v(\varepsilon)$ are measured reliably by vision, and the punch is timed exactly on $T$ as a result (Fig. 1A). By contrast, the 1g model underestimates the time of arrival of 0g targets by $\delta$, and the punch occurs at time $\delta$ before target arrival even with correct measurements of $h(\varepsilon)$ and $v(\varepsilon) = v_0$ (Fig. 1B). The value of the temporal error $\delta$ is derived as follows. For a constant speed target:

$$T = \frac{h_0}{v_0}$$
\[ \varepsilon = \frac{h_0 - h(\varepsilon)}{v_0} \quad (5) \]

\[ \delta = T - \varepsilon - \lambda \quad (6) \]

By inserting Eqs. 2, 4 and 5 into Eq. 6, one obtains:

\[ \delta = \frac{g\lambda^2}{2v_0} \quad (7) \]

For simplicity, the above analysis neglects the existence of possible margins of error for the interceptive action. If present, these margins would result in a temporal tolerance window (\(\mu\)) around the ideal trigger time. This window depends on the spatio-temporal configuration of the moving hand relative to the moving target. Thus, for 1g targets, \(T - \lambda - \mu^- < \varepsilon < T - \lambda - \mu^+\). We will consider this aspect later (see Interception score in Methods).

**Learning**

Here we consider alternative strategies for learning to deal with 0g targets. Training might result in the development of a 0g model appropriate for the new situation. According to the 0g model:

\[ h(\varepsilon) = v(\varepsilon)TTC(\varepsilon) \quad (8) \]
Equation 8 follows from Eq. 2 by taking $g = 0$. TTC($\varepsilon$) is then given by:

$$TTC(\varepsilon) = \frac{h(\varepsilon)}{v(\varepsilon)}.$$  

(9)

and once again the interceptive response is triggered when $TTC(\varepsilon) = \lambda$. If subjects were able to use a 0g model with the 0g targets of the present experiments, they would estimate the time of arrival of these targets correctly, and produce motor responses time-locked to target arrival.

Alternatively, however, subjects might continue using the 1g model throughout the exposure to 0g targets, but they would adapt its parameters. An adaptive 1g model has two internal parameters, $\lambda$ and $\hat{g}$. $\lambda$ has been defined above as the time margin for triggering the interceptive action. $\hat{g}$ is an internalized estimate of target acceleration. In Bayesian estimation theory, $\hat{g}$ would represent the prior for target kinetics. In principle, the value of this prior could depend on the probability distribution of target accelerations within any one experiment. In the default case of targets accelerated by gravity, $\hat{g} = 1g$. Otherwise, $\hat{g}$ could take any arbitrary value, depending on the neural estimate of target acceleration. In the limit case that $\hat{g} = 0$, the adapted 1g model becomes identically equal to the 0g model considered above.

If we let the internal estimate of acceleration vary in the 1g model, Eq. 7 becomes:

$$\delta = \frac{\hat{g} \lambda^2}{2v_0}.$$  

(10)
Thus, as it is apparent from Eq. 10, for any given \( v_0 \), the error \( \delta \) with 0g targets could be reduced by decreasing either \( \lambda \) or \( \hat{g} \) or both. A decrease of \( \lambda \) could result from a compression of central processing times up to minimum values dictated by physiological constraints. In this case, one would expect that the error \( \delta \) with 0g targets decreased, but did not go below the minimum signal transmission delays (Fig. 2B). Performance with 1g targets should not be affected by a physiological decrease of \( \lambda \), as long as \( h(\varepsilon) \) and \( v(\varepsilon) \) continue to be correctly estimated (Fig. 2A).

In contrast with the adaptation of \( \lambda \), there are no \textit{a priori} physiological limits for the adaptation of the internal estimate of target acceleration (\( \hat{g} \)). In principle, the brain could adjust this estimate down to the true acceleration (\( \hat{g} = 0g \)), and succeed in zeroing the error \( \delta \) for 0g targets (Fig. 2D). Note, however, that progressive adaptation of \( \hat{g} \) to values smaller than 1g (as well as simultaneous adaptation of both \( \hat{g} \) and \( \lambda \)) would affect the responses to both 0g targets \textit{and} 1g targets. Responses to 0g targets would improve according to Eq. 10. At the same time, responses to 1g targets would worsen. An internal estimate of target acceleration to \( \hat{g} < 1g \) overestimates the time of arrival of 1g targets by \( \eta \), and the punch would occur at time \( \eta \) after target arrival (Fig. 2C). The value of this temporal error is derived as follows:

\[
\eta = \lambda + \varepsilon - T
\]  

(11)
\[ T = \frac{v_0 + \sqrt{v_0^2 + 2gh_0}}{g} \]  \hfill (12)

\[ h(\varepsilon) = h_0 - v_0 \varepsilon - 0.5g\varepsilon^2 = (v_0 + g\varepsilon) \lambda + 0.5\hat{g} \lambda^2 \]  \hfill (13)

\[ \varepsilon = \frac{-v_0}{g} - \lambda + \frac{\sqrt{v_0^2 + (g^2 - g\hat{g})\lambda^2 + 2gh_0}}{g} \]  \hfill (14)

By inserting Eqs. 12, 14 into Eq. 11, one obtains:

\[ \eta = \frac{\sqrt{v_0^2 + (g^2 - g\hat{g})\lambda^2 + 2gh_0} - \sqrt{v_0^2 + 2gh_0}}{g} \]  \hfill (15)
Methods

Subjects

In total twenty-nine healthy subjects (14 women and 15 men, 30 ± 6 years old, mean ± SD) participated in the study. The subjects were right-handed (as assessed by a short questionnaire based on the Edinburgh scale), had normal vision or vision that was corrected for normal, and were naïve to the purpose of the experiments. They gave informed consent to procedures approved by the Institutional Review Board of IRCCS Fondazione Santa Lucia, in conformity with the Declaration of Helsinki on the use of human subjects in research.

Experimental setup

Subjects sat on a chair placed in front of a vertical screen (3.94-m wide, 2.13-m high) attached to the ceiling of a dimly illuminated room. Subjects’ eyes were located at 0.5-m horizontal distance from the screen, 1.82-m below the top. The back of the chair, vertically inclined by 40°, supported the head and torso of the subjects. In that position, subjects could easily reach below and beyond the lower border of the screen by protracting their arm forward. Images were generated by a PC and displayed on the screen by a BARCO Graphics 808s (1024x768 pixels, 85-Hz refresh frequency). A black square box (width, 9 cm) was constantly displayed at the top of the screen against white background. In each trial, a red target sphere (diameter, 9 cm) moved vertically downward, emerging progressively from within the start box at the predefined initial speed and acceleration (see Protocols) and disappearing progressively at the lower border of the screen. Subjects did not perceive any transient acceleration related to the start of target motion, because the target appeared already in motion, as it would occur
when an object emerges after a period of visual occlusion. During the same time interval, a real ball was dropped by an electromagnet behind the screen, hidden to the subject until it emerged from below the lower border of the screen. The ball (diameter, 9 cm; weight, 70 g) was made of soft rubber, with a small steel washer (outer diameter, 1.4 cm) on the surface to ensure magnetic contact with a precision laminated electromagnet (G.W. Linsk Co. Inc., Clifton Springs, N.Y.). The electromagnet was driven by a PC-controlled device, held the ball and released it with an accuracy better than 1 ms. Spatial trajectories of the center of the virtual sphere on the screen and of the real ball were aligned in the vertical plane orthogonal to the seat of the chair, offset by 7.5-cm in depth. The release time of the real ball and the start time of virtual motion were set by the computer so as to result in synchronous arrival at an interception point located just below the lower border of the screen. The time of synchronous arrival will be denoted interception time in the following. Synchronous timing was determined by means of an optic calibration procedure that involved 1-KHz-shuttered video-recordings. The timing error was always less than the refresh rate of image display for all tested laws of virtual motion. Because the release of the ball from the electromagnet and its subsequent fall were noiseless and invisible to the subject till the emergence from below the screen, the visual information about the virtual target provided the only perceptually available time-to-contact information before the ball contact with the arm. A miniature accelerometer (either a custom-made or an Isotron Endevco Co., San Juan Capistrano, CA, depending on the experiment) was attached by means of a Velcro strap fastened to the wrist in rough correspondence with the midline between processus styloideus radii and processus styloideus ulnae. In this position, the device was not bent by wrist flexion-extension. In addition, three-dimensional motion of selected body
points was recorded by means of the Optotrak 3020 system (Northern Digital, Waterloo, Ontario, ± 3SD-accuracy better than 0.2 mm for \(x, y, z\) coordinates). Two infrared emitting markers were attached to the skin overlying the shoulder and elbow joint on the right arm. Two other markers were attached to the wrist strap at a close distance from the accelerometer. Three additional markers were fixed to the lower border of the screen to determine the screen plane. Accelerometer data were sampled at 1 KHz and Optotrak data at 200 Hz for 5 s, starting 0.4 s before target appearance in synchrony with trial start. Inter-trial interval was 14 s. Total duration of each session was 1h 20 min, with 10-min of rest allowed halfway.

**Task**

Before the experiment, subjects received general instructions and familiarized with the setup in front of the screen (they could not see behind it). They were told that they should punch a hidden falling ball as it emerged below the lower border of the screen. They were further informed that, to be successful, they had to monitor visually the motion of the sphere on the screen because it arrived at the same time as the hidden ball at the interception point. Between trials, subjects kept a free relaxed posture until an alert auditory signal instructed them to look at the box on the top of the screen and to recoil their arm in the starting posture: with the adducted shoulder, the upper arm was roughly vertical, the forearm horizontal, the wrist mid-pronated, the hand and fingers clenched in a fist. At trial onset, another auditory signal was followed by the visual target emerging from the box, after a random delay ranging from 1.2 to 1.7 s. Subjects were asked to punch with the finger knuckles so as to deviate the ball trajectory. According to the instructions, a valid interception required contacting any point of the
ball with any part of the finger knuckles. Instead, contacting the ball outside the
knuckles (such as with the metacarpus, carpus or forearm) represented an invalid trial.
Six practice trials were run before the experiment so that subjects could familiarize with
the task. In these trials, target motion \( (v_0=0, a_0 = 1g) \) was different from any motion
used during the actual experiment were projected.

**Protocols**

The visual target descended from the start box with pseudo-randomly assorted
initial speeds \( (v_0 = 0.7, 1, 1.5, 2.5 \) or \( 4.5 \) m s\(^{-1}\)) and accelerations \( (a_0 = 1g = 9.81 \) m s\(^{-2}\) or
\( a_0 = 0g) \). The randomization procedure avoided identical conditions in consecutive
trials. Each experimental session consisted of blocks of 55 or 50 trials (depending on the
protocol) repeated 4 times consecutively. The probability of a given \( v_0 \) was always 20%,
but the probability of 0g versus 1g acceleration could vary depending on the protocol
and the day of practice. We included five protocols that differed in the relative
probability of 0g versus 1g targets on any given day: P9, P50, P91, P100, and P0 (Table
1). The label denotes the probability (P) of 0g targets on day 1, P ranging between 0%
(P0) to 100% (P100). There were 7 subjects in group P50, 7 subjects in group P100, and
5 subjects for each group P0, P9, P91. All subjects of a group were exposed to identical
sequences of trials. Subjects of group P50 performed a one-day experiment with 50% of
0g targets and 50% of 1g targets. In the other groups there were two experimental
sessions 24h apart. Subjects of group P100 were exposed to an identical sequence of
trials on both days, involving only 0g targets (100% probability) except for the six
practice trials that preceded each session (see above). Subjects of group P9 were
exposed to 9% of 0g targets and 91% of 1g targets on day 1. On day 2, they were
exposed to an identical sequence of target \( v_0 \) but reversed accelerations relative to day 1, resulting in 91% of 0g trials and 9% of 1g trials. Subjects of group P91 practiced on day 1 (day 2) the same sequence experienced on day 2 (day 1) by subjects of group P9. Thus, they had 91% of 0g trials on day 1, and 9% of 0g trials on day 2. The sequence of target \( v_0 \) was identical for trials at the same acceleration in P9, P91, and P100.

**Data analysis**

**Motor timing.** Accelerometer data were numerically low-pass filtered (bi-directional 10-Hz-cutoff 2\(^{\text{nd}}\)-order Butterworth filter) to eliminate impact artifacts. Motor timing was quantified from the time of occurrence (denoted as cross-time) of the zero-crossing relative to the interception time. This is the time when the acceleration first crosses the zero-line after the first positive peak (see Fig. 3 for typical recordings). In correctly intercepted trials, the zero-crossing occurs very close to the interception time. Therefore, the cross-time reliably reflects the subject’s estimate of the arrival time of the target at the interception point. We previously showed that the timing of the zero-crossing co-varies tightly with that of other landmarks of the acceleration trace, such as the first positive peak and the first negative peak: the \( r^2 \) between each pair of landmarks is always > 0.90 (Zago et al. 2005). By correlating the motor timing with the spatial trajectories of the limb, we also showed that the temporal landmarks of wrist acceleration are good predictors of the wrist position measured at interception time (Zago et al. 2005). Thus, when the zero-crossing occurs earlier relative to the interception time (negative cross-time) the hand has moved past the interception zone when the ball arrives. Conversely, when the zero-crossing is later than the interception time.
time (positive cross-time), the hand has not reached yet the interception zone when the ball arrives.

**INTERCEPTION SCORE.** The cross-time provides information about motor timing relative to the nominal interception time. However, subjects had a temporal tolerance window ($\mu$) around this nominal time, because they could contact any point of the ball with any part of the finger knuckles. To provide a global score of performance that takes $\mu$ into account, we first determined the presence of any measurable contact between the ball and the limb in each trial. To this end, we filtered the raw acceleration signal with bi-directional 25-Hz-high-pass Butterworth filter and computed the time of initiation of high-frequency oscillations. Detailed analysis of the combined data set of wrist position and wrist acceleration showed that, when the hand passed close to the expected interception point around the expected interception time, contact oscillations invariably started between the positive peak and the negative peak of acceleration. Accordingly, we assigned a score of 1 (intercepted ball) to trials in which the contact oscillations started between the first positive peak and the first negative peak of acceleration, and a score of 0 (no valid interception) if there were no detectable oscillations or they started outside this interval. For each protocol, global interception score was computed as the fraction of all intercepted trials out of the total number of trials (over all subjects of a given group) of all $v_0$ for a given $a_0$.

**ADAPTATION RATE.** An exponential function was fitted to the series of repetitions of the chosen parameter $\xi$ (cross-time or interception score) for each condition ($a_0$ and
to characterize the rate at which subjects adapted during an experiment. The function has three free parameters: offset $b_0$, gain $b_1$, learning-constant $b_2$. The equation was

$$\xi_i = b_0 + b_1 \exp(-i/b_2)$$ (16)

where $\xi_i$ was the $\xi$ value estimated for repetition $i$.

**STATISTICS.** Differences between conditions were assessed using single- or multiple-factor ANOVA. The threshold for statistical significance was set at $\alpha=0.05$. Statistically significant differences of interception scores were assessed using the exact test for matched pairs (McNemar 1947). Statistics on correlation coefficients was performed on the normally distributed, Z-transformed values (Kendall and Stuart 1969).

**MODEL FITTING.** We took the cross-time (time of occurrence of the zero-crossing of hand acceleration) as the subject’s estimate of the arrival time of the target at the interception point. Therefore, the cross-times reflect the timing errors ($\delta$ for 0g trials or $\eta$ for 1g trials). Estimates of model parameters ($\lambda$ and $\hat{g}$) for any given repetition of a given condition were obtained by least-squares fitting the experimental values of cross-times averaged over all subjects of a group. All analyses were implemented in Matlab code (The MathWorks, Inc., Natick, MA).
Results

The aim of this work was to discriminate between two different hypotheses of how subjects learn to deal with the novel environment of visual targets descending at constant speed. One hypothesis is that subjects learn a new internal model specific for the 0g targets. The alternative hypothesis is that subjects do not develop a new model but adapt the pre-existing 1g model by stretching its parameters. We will compare the results of different experimental protocols with the predictions stemming from each hypothesis.

Interception of 0g versus 1g targets

In agreement with the previous results obtained with a similar paradigm (Zago et al. 2004; Zago et al. 2005), we found that the initial performance with 0g targets was very different from that with 1g targets. Motor responses generally were time-locked to the arrival of 1g targets, whereas the responses to 0g targets often were very premature, and the hand passed through the interception point well in advance of the target. As a result of premature timing in 0g trials, the falling ball often hit the forearm instead of the hand, generating an error feedback. Close scrutiny of the changes of the acceleration waveforms with prolonged exposure to 0g targets provides critical clues to discriminate between the two hypotheses of learning outlined above. If subjects learned a new internal model specific for the 0g targets, they should become able to estimate the target TTC correctly and, as a result, produce responses to 0g targets with the same timing with respect to impact as for 1g targets. Accordingly, one would expect that the
amplitude of the early, inappropriate response gradually diminished while another, distinct response developed time-locked to the nominal interception time, becoming increasingly predominant (Witney et al. 1999). The alternative hypothesis that subjects adapt the pre-existing 1g model by stretching its parameters predicts that the original response does not diminish but, with learning, it progressively migrates to a later time, closer and closer to the nominal interception time.

Figure 3 shows the time profiles of hand acceleration of three repetitions (1st, 2nd, and 12th in blue, green and red, respectively) of target $v_0=0.7 \text{ m s}^{-1}$ for a representative subject. It can be seen that the responses to 1g targets were time-locked to the nominal interception time (time 0 on the abscissa) from the first repetition and varied little in the following repetitions (Fig. 3, left). Thus, the zero-crossing of hand acceleration occurred very close to the interception time, indicating that subjects generated maximum momentum to punch the incoming ball at the right time. By contrast, when subjects were first exposed to the 0g target, their responses started too early (Fig. 3, right). With training, the early, inappropriate response did not disappear. Instead, it shifted closer to the nominal interception time, but remained premature as compared with 1g responses. This finding is consistent with the adaptation of a pre-existing model rather than the development of a new model.

Note that the waveform of hand acceleration was fairly stereotyped, independent of target initial velocity, acceleration and repetition. Hand movements always consisted of a brief acceleration followed by an equally brief deceleration, each phase lasting about 130 ms. To quantify the temporal relationship between different landmarks of wrist acceleration as a function of target acceleration, we computed the time interval between a given landmark in each 1g trial and the same landmark in the corresponding 0g trial.
Thus, $\Delta PP$ was the time interval between the positive acceleration peak in each 1g trial and the same landmark in the corresponding 0g trial, whereas $\Delta ZC$ was the time interval between the zero-crossing in the 1g trial and the same landmark in the 0g trial. When the data were pooled together ($n=3700$, all trials of all subjects in P9, P50, P91 and P0), they appeared scattered through a wide range because the timing of the responses varied across 0g trials (see Fig. 3). Importantly, however, the best-fitting regression line of $\Delta ZC$ versus $\Delta PP$ ($r^2 = 0.88$, intercept -0.58 ms, slope 0.98) was similar to that one would expect if the landmarks on the acceleration waveform maintained the same temporal relationship in 0g trials and 1g trials. Moreover, the time interval between the positive peak and the zero crossing of each acceleration trace varied very little across all conditions (by pooling all trials of all subjects, the mean SD was only $16 \pm 2$ ms).

**Learning rate**

Learning rate was quantified from the changes of two parameters as a function of repetition: i) the time of occurrence (denoted cross-time) of the zero-crossing of hand acceleration relative to the interception time, and ii) the interception score (see Methods). Mean cross-time values and interception scores are plotted as a function of repetition number in the top and bottom row of Fig. 4, respectively. These data were obtained in two immersive protocols: 100% of targets were either 1g trials over one session (day 1 of P0, Fig. 4, left column) or they were 0g trials over two sessions performed one day apart (day 1 and day 2 of P100, Fig. 4, right column). The timing of the responses to 1g targets did not change significantly with repetition (cross-time= -4 ± 8 ms, mean ± SD, $n = 40$; one-factor ANOVA, P=0.8). The interception score was high from the outset and improved only slightly with repetition. On average, it was $88 \pm 12\%$
in the first four repetitions and 98 ± 6% in the last four repetitions (the difference between these two mean scores was statistically significant, P<0.005, McNemar’s exact test for matched pairs). By contrast, cross-time and interception score changed drastically over the first few repetitions of 0g targets, reaching plateau levels which remained substantially lower than those for 1g targets. The changes of these parameters with repetition were fitted by an exponential function (Eq. 16), with learning constants of 1.3 and 2.2 repetitions for cross-time and score, respectively. Mean cross-time over the last four repetitions of day 2 was -89 ± 23 ms, and mean score was 45 ± 24%, both values being significantly (P<0.001) lower than the corresponding values of 1g targets.

In sum, the performance with 0g targets changed very rapidly after initial exposure, but even after extensive practice it remained well below the performance level with 1g targets. These observations provide further evidence in favor of the hypothesis that the pre-existing 1g model is adapted, and against the alternative hypothesis that a new 0g model is developed.

**Adaptation of the 1g model to immersive 0g exposure**

As explained in the Modeling section, the 1g model has two internal parameters, \( \lambda \) and \( \hat{g} \). \( \lambda \) is the time margin necessary to trigger the interceptive action before the expected collision, so as to compensate for neuro-mechanical delays. \( \hat{g} \) is the internalized estimate of target acceleration. The error \( \delta \) that would result by applying the 1g model to 0g targets might be reduced with training by adaptively decreasing either \( \lambda \) or \( \hat{g} \) or both parameters (Eq. 10). A decrease of \( \lambda \) could result from a compression of signal transmission delays up to a minimum value dictated by physiological constraints. In this case, one would expect that the error \( \delta \) with 0g targets
decreased, but did not go below a minimum value. In contrast with the adaptation of $\lambda$, there are no \textit{a priori} physiological limits for the adaptation of the internal estimate of target acceleration ($\hat{g}$). In principle, one could expect that the brain adjusts this estimate from the initial default of gravitational acceleration ($\hat{g}=1g$) up to the true acceleration ($\hat{g} = 0g$). If so, subjects should ultimately succeed in zeroing the error $\delta$ for $0g$ targets (Fig. 2D).

The values of cross-time measured in protocol P100 (Fig. 4) can be fitted equally well by one or the other adaptation scheme (Fig. 5). Based on modeling, either $\lambda$ or $\hat{g}$ decrease exponentially as a function of repetition. The steady-state value of $\lambda$ is 123 ms, a value that is compatible with the known visuo-motor delays for interception (Lacquaniti and Maioli 1989; Port et al. 1997). The steady-state value of $\hat{g}$ is 0.35g. Because this was an immersive protocol with prolonged exposure to $0g$ targets over two sessions one-day apart, it appears strange that $\hat{g}$ never approached the true $0g$-value of the visual targets. Nevertheless, both adaptation schemes are compatible with the data of this protocol. The data from the next protocol help discriminating between the two schemes.

\textit{Adaptation of the 1g model to concurrent, randomized presentation of 0g and 1g targets}

Adaptation of both $\lambda$ and $\hat{g}$ predict a progressive decrease of the error $\delta$ with $0g$ targets. However, progressive changes of $\lambda$ should not affect the response timing with predictable $1g$ targets presented concurrently with $0g$ targets within the same session (see Model). Instead, progressive changes of $\hat{g}$ do affect the response timing with these
1g targets. An internal estimate of target acceleration equal to \( \hat{g} < 1g \) overestimates the time of arrival of 1g targets by \( \eta \), and the punch will occur at time \( \eta \) after target arrival (see Fig. 2C). The smaller the value of \( \hat{g} \) as a result of adaptation, the more delayed will be the punch relative to the correct time and the lower will be the interception score. In other words, the performance in 1g trials should progressively worsen, in parallel with the improvement of the performance in 0g trials. Therefore, to discriminate between the hypothesis of \( \lambda \) adaptation and the hypothesis of \( \hat{g} \) adaptation, we should scrutinize the responses to 1g targets in a protocol where target acceleration randomly switches between 1g and 0g from trial to trial.

In protocol P50, 1g and 0g trials were randomized with the same probability (50%) within the same session (Fig. 6). Thus, subjects expected either type of target equally. Adaptation of \( \lambda \) with repetition (thick line) fits the cross-time values of 0g trials slightly better than does adaptation of \( \hat{g} \) (dashed line, mean \( r^2 = 0.82 \) and 0.79 for \( \lambda \) and \( \hat{g} \) adaptation, respectively, see Fig. 6B). However, the major difference between the predictions of the two adaptation schemes involves the 1g data (Fig. 6A). Adaptation of \( \hat{g} \) with repetition (dashed line) predicts progressive, systematic delays of the cross-time for 1g trials that should parallel the changes of timing for 0g trials as a function of repetition. No such trend was present in the experimental data (one-factor ANOVA, \( P=0.21 \)), in contrast with the hypothesis of adaptation of \( \hat{g} \) and in agreement with the hypothesis of adaptation of \( \lambda \) (thick line).

By the same token, the interception scores measured for 1g targets in this experiment failed to show the progressive decrement of performance with repetition predicted by the adaptation of \( \hat{g} \). In fact, the data showed the opposite trend (Fig. 7).
The interception score for 1g targets was low initially (60 ± 29% in the first repetition), and increased exponentially with repetition (the difference between the mean score in the first four repetitions and that in the last four repetitions was significant, P<0.0001, McNemar’s exact test for matched pairs). Performance with 0g targets was similar to that of the other protocols, with an exponential increase and a plateau lower than that for 1g targets.

**Relationship between adaptation and probability of 0g targets in any one session**

In Bayesian estimation theory, $\hat{g}$ represents the prior for target kinetics. In principle, the value of this prior should depend on the probability distribution of target accelerations within any one experiment (Kording and Wolpert 2004). If subjects acted in a Bayesian way in the face of uncertainty, they should combine *a priori* information about the most plausible target acceleration with evidence about the real acceleration experienced in previous presentations. Therefore, one would expect that the higher the probability of 0g trials in any one session, the greater the extent of adaptation of $\hat{g}$ and the lower the residual error $\delta$ with 0g targets at the end of practice. A similar prediction stems from a different but related hypothesis, namely that subjects learn to use a combination of two distinct internal models, the original 1g model and a new 0g (or fractional g) model, the relative weight of each one changing as a function of the context (Davidson and Wolpert 2004). If this were the case, one would expect that the higher the probability of 0g trials in any one session, the greater the weight of the corresponding 0g model and the lower the residual error $\delta$ with 0g targets at the end of practice.
We tested these two hypotheses by comparing 5 different protocols with different probabilities of 0g trials in any one session. Figure 8A plots the mean values of cross-time over the last 4 repetitions of the same 0g target in sessions with 50% probability (day 1 P50), 91% probability (day 1 P91, day 2 P9, and day 2P0), and 100% probability (day 2 P100). Contrary to the Bayesian prediction, there was no significant correlation ($r^2 = 0.15$, $P= 0.52$) between these cross-times (reflecting the error $\delta$) and the corresponding probability of 0g trials during that session. This negative finding provides further evidence against the hypothesis of adaptation of $\hat{g}$ as a strategy for dealing with 0g trials.

On the other hand, the residual error $\delta$ with 0g targets was significantly ($r^2 = 0.91$, $P < 0.02$) related to the probability of 1g trials on the first experimental day (Fig. 8B): the higher the previous exposure to 1g targets, the better the steady-state performance with 0g targets (the lower the residual error $\delta$). Obviously, the ranking co-varied in the opposite direction with the probability of 0g trials on the first experimental day, with the apparent paradox that the steady-state performance with 0g targets was worse in case of a higher previous exposure to 0g targets.

**Adaptation of $\lambda$ in the 1g model**

The bulk of the evidence presented above is against the adaptation of $\hat{g}$ and in favor of the adaptation of $\lambda$. Figure 9 shows the changes of the estimated $\lambda$ values as a function of repetition, plotted separately for all protocols. As predicted by the hypothesis of 1g model adaptation, $\lambda$ values tended to decrease monotonically with 0g repetitions. Exponential fitting of the changes of $\lambda$ with repetition showed a rapid decrement from initial values of around 200 ms to steady-state values that varied as a
function of protocol, according to the same ranking noticed in the previous section. Thus, the higher the previous exposure to 1g targets, the greater tended to be the extent of $\lambda$ adaptation (corresponding to lower steady-state values). Steady-state $\lambda$ values were 143, 137, 96, 104 and 78 ms for P100 (0% of 1g targets on day 1), P91 (9%), P50 (50%), P9 (91%) and P0 (100%), respectively.

**Discussion**

*Comparison of the 1g model with other interception models*

The 1g model belongs to the class of threshold-based TTC-models (Lee et al. 1983; Port et al. 1997; Tresilian 1999; Tresilian 2004). It predicts that the interceptive movement is centrally triggered when TTC reaches a given threshold value $\lambda$, equivalent to a time margin before the expected collision time. The control of interception is under on-line visual control up to $\lambda$, by comparing the visually derived estimate of target TTC with the preset threshold value. The threshold value is specified by information about target distance and velocity, under the assumption that the target is accelerated by gravity. At time $\lambda$ before the expected collision, the action is launched with little (if any) correction based on feedback. We showed that the punching movements of the present experiments were compatible with the 1g model. Indeed, all movements started from a fixed initial position, and involved brief and stereotyped acceleration waveforms. It should be noted, however, that although we found no evidence for modifications of the interceptive action once initiated, we cannot exclude subtle on-line changes that may have gone undetected in our analysis. Thus, the arguments we develop about adaptation of the 1g model strictly apply to the trigger of the movement up to the time of maximum momentum (corresponding to the measured
cross-time of hand acceleration), and we make no claim about the control of other aspects of the action. In principle, a modified version of the model should be applicable to other types of interception actions, such as those that involve slower responses developing under more variable conditions than those of the present experiments (e.g., variable starting position of the limb, movement duration, or final position of the target). These latter types of interception actions presumably rely on visual feedback till the end of the movement (Bootsma et al. 1997; Brenner et al. 1998; Dessing et al. 2002). In particular, the RRVITE model predicts that TTC information is combined with gaze-centered velocity and position information about both the target and the hand to provide continuous control of interception of slow targets (Dessing et al. 2005). As hypothesized by these authors, prior knowledge about gravity might be a direct input to the TTC stream. Thus, the $1g$ model could be coupled with the RRVITE model for the control of slower, more variable interceptions than the present ones.

A different model has been proposed that may also account for gravity effects: the $\tau$-guide model (Lee 1998; Georgopoulos 2002; Grealy et al. 2004). This model assumes that an action is coupled to an intrinsic second-order guide akin to gravity. It departs from the original $\tau$-model that ignores accelerations (Lee et al. 1983) and formally resembles the $1g$ model. However, the current version of the $\tau$-guide model does not take into account signal transmission delays (see Brouwer et al. 2003), and therefore it should be modified to account for the present results showing adaptable delays. Still another model has been proposed by Tresilian (1993; 1999): this model assumes that, for short heights of fall, TTC can be estimated by using internalized gravity and a perceptual estimate of drop height ($H$) by solving the equation $\text{TTC}=(2H/g)^{0.5}$. Also this model fails to account for adaptable delays. Moreover, it requires that subjects are able
to see the target starting from rest. It cannot predict TTC when the target first appears in
view with an unpredictable initial velocity, as was the case for the targets used in the
present experiments.

Adaptation of the 1g model

The hypothesis that subjects apply the 1g model predicts that they should time the
interception of 1g targets correctly, but that they should make systematic timing errors
with 0g targets. In other contexts, it has been shown that practice of arm movements in
a novel environment leads to formation of the appropriate internal model (Flanagan and
Wing 1997; Gandolfo et al. 1996; Gordon et al. 1993; Imamizu et al. 2003; Wolpert and
Kawato 1998). Here, we considered the possibility that training with 0g targets in our
experimental context might result in the development of a 0g model appropriate for
these targets. No evidence for this 0g model was found, even in an immersive protocol
with prolonged exposure to 0g targets over two sessions performed one-day apart (day 1
and day 2 of P100). This is striking given that: i) human vision is much more sensitive
to motions at constant speed than to accelerated motions (Brouwer et al. 2002;
Werkhoven et al. 1992), ii) a 0g model is equivalent to a first-order estimate of target
TTC based on visual information about the ratio between target distance and velocity,
estimate that has been shown to be used in several other tasks that do not involve a
falling mass (Merchant et al. 2003; Port et al. 1997; Regan and Hamstra 1993; Rushton
and Wann 1999; Tresilian 1999), and iii) it has previously been shown that, when 0g
visual targets projected in front of the subjects under conditions identical to those used
here are intercepted virtually by clicking a mouse-button in the absence of the real ball,
the responses are correctly timed in accord with a 0g model (see Fig. 12 in Zago et al. 2004).

In the present experiments, the failure to develop a 0g model for 0g targets occurs in the face of potentially conflicting information about the physical laws of motion. Vision should have signaled to the subjects that the visible target descended at constant speed. However, previous experience with other falling objects should have informed the subjects that the hidden real ball to be punched was acted upon by gravity. Moreover, body graviceptors attested to the presence of the Earth’s gravitational field. Overall, subjects appeared to rely on an internal model of the physical world in which a downward moving target should accelerate. Remarkably, the same behavior occurs even in the microgravity conditions of orbital flight, when all body sensors (including the graviceptors) and cognitive information concur in signaling the lack of measurable gravity. Thus, astronauts trigger muscle responses earlier with respect to impact when catching a ball launched “downward” at constant speed, as if they continued to anticipate the effects of gravity (McIntyre et al. 2001). The responses were well accounted for by the 1g model over three different sessions, performed on day 3, 9 and 15 of flight, with limited adaptation over time.

Therefore, both the present experiments as well as the previous microgravity experiments indicate that the a priori of Earth gravity for objects moving along the vertical is highly resistant to changes, despite strong, compelling evidence that a specific visual target is not affected by gravity, and despite the feedback about performance errors repeated trial after trial. In the present experiments, visual and somatosensory error feedback was generated when the falling ball hit the forearm instead of the hand, as a result of premature timing of the movement toward 0g targets.
Here we were able to show that, not only was there no evidence for the development of a 0g model, but also there was no evidence for adaptation of the internalized estimate of target acceleration ($\hat{g}$) to values intermediate between 1g and 0g when subjects were exposed concurrently to both sets of targets. When these targets are randomly intermixed with equal probability (protocol P50), adaptation of $\hat{g}$ to values intermediate between 1g and 0g would guarantee a globally adequate performance, because the error with 0g targets would be drastically reduced at the expense of a modest deterioration of the responses to 1g targets. However, in this protocol we failed to observe the predicted progressive deterioration of the responses to 1g targets in parallel with the improvement of the responses to 0g targets (Fig. 6). In fact, the interception score for 1g targets showed the opposite trend, tending to improve with repetition (Fig. 7).

The present results also rule out the possibility that subjects learned to use a combination of two distinct internal models, the original 1g model and a new 0g (or fractional g) model, the relative weight of each one changing as a function of the context (Davidson and Wolpert 2004). If this were the case, one would expect that the higher the probability of 0g trials in any one session, the greater the weight of the corresponding 0g model and the lower the residual error with 0g targets at the end of practice. No such relation was found by comparing five different protocols with different probabilities of 0g trials in any one session (Fig. 8A). Surprisingly, instead, the residual error with 0g targets depended on the probability of 1g trials on the first experimental day (Fig. 8B): the higher the previous exposure to 1g targets, the better the subsequent performance with 0g targets at steady-state. This result, as well as the progressive improvement of performance with 1g targets in the protocol P50, point to
the possibility that a pre-existing 1g model is calibrated for the specific task to be accomplished.

This calibration may involve, among other things, the precise setting of the internal parameter of the time margin $\lambda$ used to trigger the interceptive action. We showed that all the results were compatible with adaptive changes of $\lambda$. A decrease of $\lambda$ could result from a compression of central processing times up to a minimum value dictated by physiological constraints. Accordingly, the timing error with 0g targets decreases, but cannot go below the minimum signal transmission delay. By contrast, performance with 1g targets is not be affected by a physiological decrease of $\lambda$, as long as target distance and velocity continue to be correctly estimated by vision. The estimated value of $\lambda$ was $\approx 200$ ms before adaptation. Training with 0g trials resulted in a rapid drop to values between $\approx 80$ and 140 ms, depending on the protocol (Fig. 9). These values are close to the shortest visuo-manual delay times for ongoing target motion (Brenner et al. 1998; Lacquaniti and Maioli 1989; Port et al. 1997; Prablanc and Martin 1992). This may explain why the adaptation process tended to saturate early on, and the responses to 0g trials remained premature throughout the rest of training. A decrease of $\lambda$ results from a compression of signal transmission delays. Mechanical delays in ballistic interception are probably at a minimum from the outset. Central processing delays, instead, could be compressed by reducing reaction times through more efficient visuo-motor transformations (Dessing et al. 2002; Thorpe et al. 2001).

**Neural substrates**

A neural substrate for the 1g model has recently been identified by fMRI in man (Indovina et al. 2005). Visual acceleration coherent with represented gravity engaged
the vestibular cortex, most notably the parieto-insular vestibular cortex (PIVC), even though there was no time-varying stimulation of the vestibular sensors. The neural representation of gravity in this network is not only for motor tasks but also for cognitive functions. Indeed, activation of the network was observed irrespective of the motor task, suggesting that the activation depends on cognitive processing of gravity. We hypothesized that the 1g model is initially derived from graviceptors (vestibular and somatosensory receptors) in head coordinates, and is subsequently stored in PIVC as an abstract representation of gravity accessible by the visual system in world coordinates. Visual information is presumably relayed from the motion areas in the dorsal stream (mainly hV3a and hMT/V5+) into inferior parietal lobule (IPL) and PIVC. Target-motion and TTC information has been uncovered in the neural activity of the monkey IPL area 7a during interception of real and apparent motion (Merchant et al. 2004a; Merchant et al. 2004b). Area 7a is reciprocally connected with multiple visual areas including MST, FST, PO, STP (Cavada and Goldman-Rakic 1989). It projects to motor cortical areas either directly (Tanne-Gariepy et al. 2002) or via area 7m (Cavada and Goldman-Rakic 1989; Johnson et al. 1996). Also activity in motor cortex has been shown to reflect visual motion parameters in interception tasks (Lee et al. 2001; Port et al. 2001).

When response speed is at a premium (as in the present experiments), feedforward visual mechanisms may suffice without resorting to top-down feedback modulation (Fabre-Thorpe et al. 2001). Although the signal transmission delays estimated here are short (less than 200 ms), they appear compatible with fast processing of on-going visual stimuli. Thus, neurons in monkey MT area respond about 30 ms after a rapid change in a temporal sequence (Bair et al. 2002). If we consider that the conduction time from
human motor cortex to arm muscles is about 10 ms (Salenius et al. 1997), and the electro-mechanical delay 50 ms (Cavanagh and Komi 1979), cortico-cortical connections relaying information to motor cortex must take no more than few tens of milliseconds. It has been suggested that, under continuous conditions of stimulation, changes of target position can be extracted from an image on the basis of the first wave of information passing through the visual system, resulting in considerable efficiency and rapidity of processing (Van Rullen and Thorpe 2002).
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**Figure legends**

Fig. 1. Predictions of the 1g model for timing ballistic interceptions of a descending target. The instantaneous height of the target above the interception point is plotted as a function of time. $T$ denotes the total flight duration of the target. Subjects trigger the interception at time $\lambda$ before the expected collision, corresponding to time $\varepsilon$ after the onset of target motion. The dashed curve denotes the target trajectory extrapolated from current position $h(\varepsilon)$ and velocity $v(\varepsilon)$ on the basis of the 1g model. The punch occurs at the time of expected collision $\varepsilon + \lambda$ (corresponding to the intersection of the dashed curve with the time axis). A: The application of the 1g model correctly estimates the time of arrival of 1g targets. B: The application of the same model underestimates the time of arrival of 0g targets by $\delta$, and the punch occurs at time $\delta$ before target arrival.

Fig. 2. Simulation of adaptation of the internal parameters of the 1g model with prolonged exposure to 0g targets. $\lambda$ is the time margin before the expected collision. $\hat{g}$ is the internalized estimate of target acceleration. A-B: Simulations of adaptive reduction of the time margin $\lambda$, keeping $\hat{g} = 1g$. Red, green and blue traces correspond to $\lambda_1 = 300$ ms, $\lambda_2 = 200$ ms, $\lambda_3 = 100$ ms, respectively. Decreases of $\lambda$ do not affect the responses to 1g targets (A), but reduce the error $\delta$ with 0g targets (B). C-D: Simulations of adaptive reduction of $\hat{g} = \alpha g$, keeping $\lambda = 300$ ms. Red, green and blue traces correspond to $\alpha_1 = 0.7$, $\alpha_2 = 0.4$, $\alpha_1 = 0.1$, respectively. Decreases of $\hat{g}$
lead to overestimates of the time of arrival of 1g targets by $\eta$ (C), while reducing the error $\delta$ with 0g targets (D). In B, D $\delta_1 > \delta_2 > \delta_3$. In C, $\eta_1 < \eta_2 < \eta_3$.

**Fig. 3.** Interception of 1g versus 0g targets. Time profiles of hand acceleration of subject A.M. (protocol P0, $v_0=0.7$ ms$^{-1}$) for three repetitions ($1^{st}$, $2^{nd}$ and $12^{th}$, in blue, green, and red, respectively) of accelerated (left) and constant velocity (right) targets. Traces are aligned on interception time (vertical line) and negative time is before that time. Acceleration units are in g (1g=9.8 m s$^{-2}$).

**Fig. 4.** Effects of training for 1g versus 0g trials. A, C: The time of occurrence (cross-time) of the zero-crossing of hand acceleration relative to the interception time was averaged across all subjects for the condition $v_0 = 0.7$ m s$^{-1}$. Mean cross-time values (± 1SE) are plotted as a function of repetition number. B, D: Interception scores were computed as the fraction of all intercepted trials out of the total number of trials (over all subjects) over all five $v_0$. A-B: 1g data from day 1 of protocol P0. C-D: 0g data from day 1 (repetition 1-40) and day2 (repetition 41-80) of protocol P100. Thick line through interception scores of 1g trials corresponds to linear regression (P <0.05) as a function of repetition (linear regression through cross-time values was not significant). Thick lines through both cross-time and scores of 0g trials correspond to exponential fits (P <0.05).

**Fig. 5.** Adaptation of the 1g model in P100. Values of cross-time are the same as those of Fig. 4C. Thick line: Data are fitted (P <0.05) by letting the time margin ($\lambda$) of the 1g model decrease exponentially as a function of repetition. Initial and steady-state
values of $\lambda$ are 208 and 123 ms, respectively. $\hat{g} = 1g$ throughout. *Thin line:* Data are fitted (P <0.05) by letting the internalized estimate of target acceleration ($\hat{g}$) of the 1g model decrease exponentially as a function of repetition. Initial and steady-state values of $\hat{g}$ are 1g and 0.35g, respectively. $\lambda = 208$ ms throughout. The two fitting procedures yield indistinguishable results.

**Fig. 6.** Adaptation of the 1g model in P50. This was a one day protocol with 50% probability of 1g trials and 50% probability of 0g trials. Mean cross-time values are reported for the condition $v_0 = 1$ m s$^{-1}$, 1g and 0g data in A and B, respectively. Best fits (P <0.05) with exponentially decreasing values of $\lambda$ or $\hat{g}$ of the 1g model are plotted with *continuous red lines* or *dashed green lines*, respectively. Initial and steady-state values of $\lambda$ are 205 and 76 ms, respectively, learning constant of 1.8 repetitions. Initial and steady-state values of $\hat{g}$ are 1g and 0.2g, respectively, learning constant of 1.7 repetitions.

**Fig. 7.** Interception scores in group P50. Scores were computed over all subjects and all five $v_0$. 1g data and 0g data are plotted in the *left* and *right* panels, respectively. *Thick lines* correspond to exponential fits (P <0.05).

**Fig. 8.** Comparison of 0g learning across protocols. Mean cross-time values ($\pm$ 1SE) for target 0g, $v_0=0.7$ m s$^{-1}$ are plotted as a function of the probability of 0g trials on the same experimental day (A) or as a function of the probability of 1g trials on the first experimental day (B). Mean cross-time values were computed over the last 4 repetitions of day 2 P0, day 2 P9, day 1 P50, day 1 P91, and day 2 P100.
Fig. 9. Adaptation of the time margin $\lambda$ of the $1g$ model (see Fig. 2B) as a function of repetition in all protocols. For each protocol, values of $\lambda$ were computed over all $v_0$ and all subjects at incremental repetitions of $0g$ trials. Only the results for high probability ($\geq 50\%$) trials are presented, except for the $0g$ trials of day 1 of P9, which are plotted adjoined to those of day 2. Thick lines correspond to exponential fits ($P <0.05$). Learning constants are 1.4, 1.0, 0.9, 1.4 and 2.1 repetitions for P100, P91, P50, P9 and P0, respectively.
Table 1. Protocols.

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The table gives the number of 1g trials, the number of 0g trials, and the percentage of 0g trials for each day of the protocols.
Fig. 2
\[ a_0 = 1g \]

\[ a_0 = 0g \]

Fig. 3
Fig. 4
Fig. 7
Fig. 8