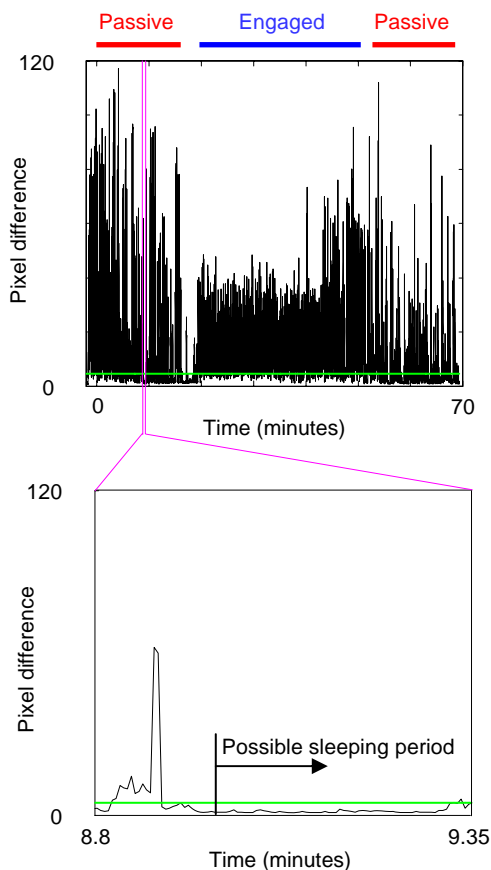
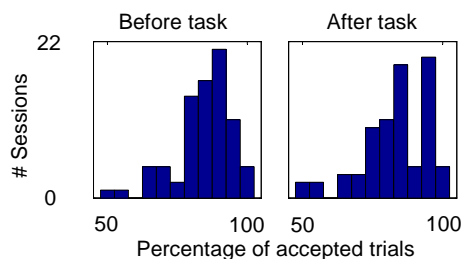


Figure S1  
S1a



S1b



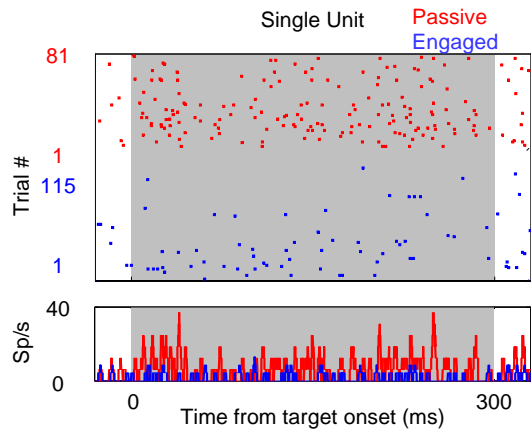
### Supplementary Figure S1

**Video analysis of movement.** A video was taken during all recording sessions at a frame rate of 30 Hz using an infrared camera located on top of the operant chamber. The video was re-sampled at 3 Hz and the difference in pixels between consecutive frames was taken as an index of the animal's movement. In order to set a threshold for movement for each recording session, we took the 10th percentile of the index of animal movement while the animal was performing the task and set it as our immobility threshold. Signals that fell below this were interpreted as the animal not moving. (**S1A**) Typical example of the index of animal movements during a recording session. The *green line* indicates the threshold of activity. This threshold was actually an underestimate of the animal's movement, because there are periods in which the signal goes below this threshold for long periods. The *inset* indicates the behavior of the signal during periods of extended immobility. Stimulus presentations that occurred after 5 seconds of immobility were excluded from the analysis of the passive condition. We used this conservative criterion to reduce the possibility of including periods of drowsiness or sleep in our analysis of the passive condition.

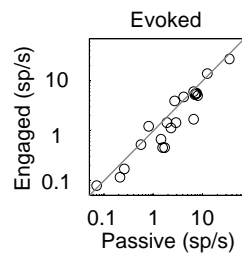
**Fig. S1B** shows the histogram of the fraction of trials (stimulus presentations) per session that were not rejected in the passive conditions due to animal immobility (8 animals). On average, for a given session, 16% of the stimulus presentations in the passive condition were rejected because the animal did not move during the presentation of the stimulus, in the period before the task performance and 17% were rejected after the task performance.

# Figure S2

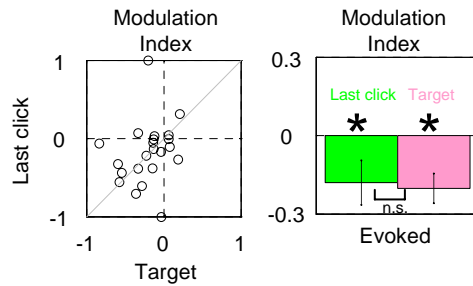
S2a



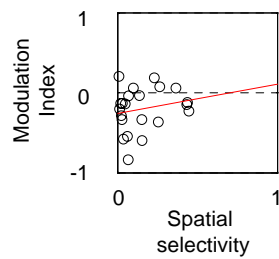
S2b



S2c



S2d

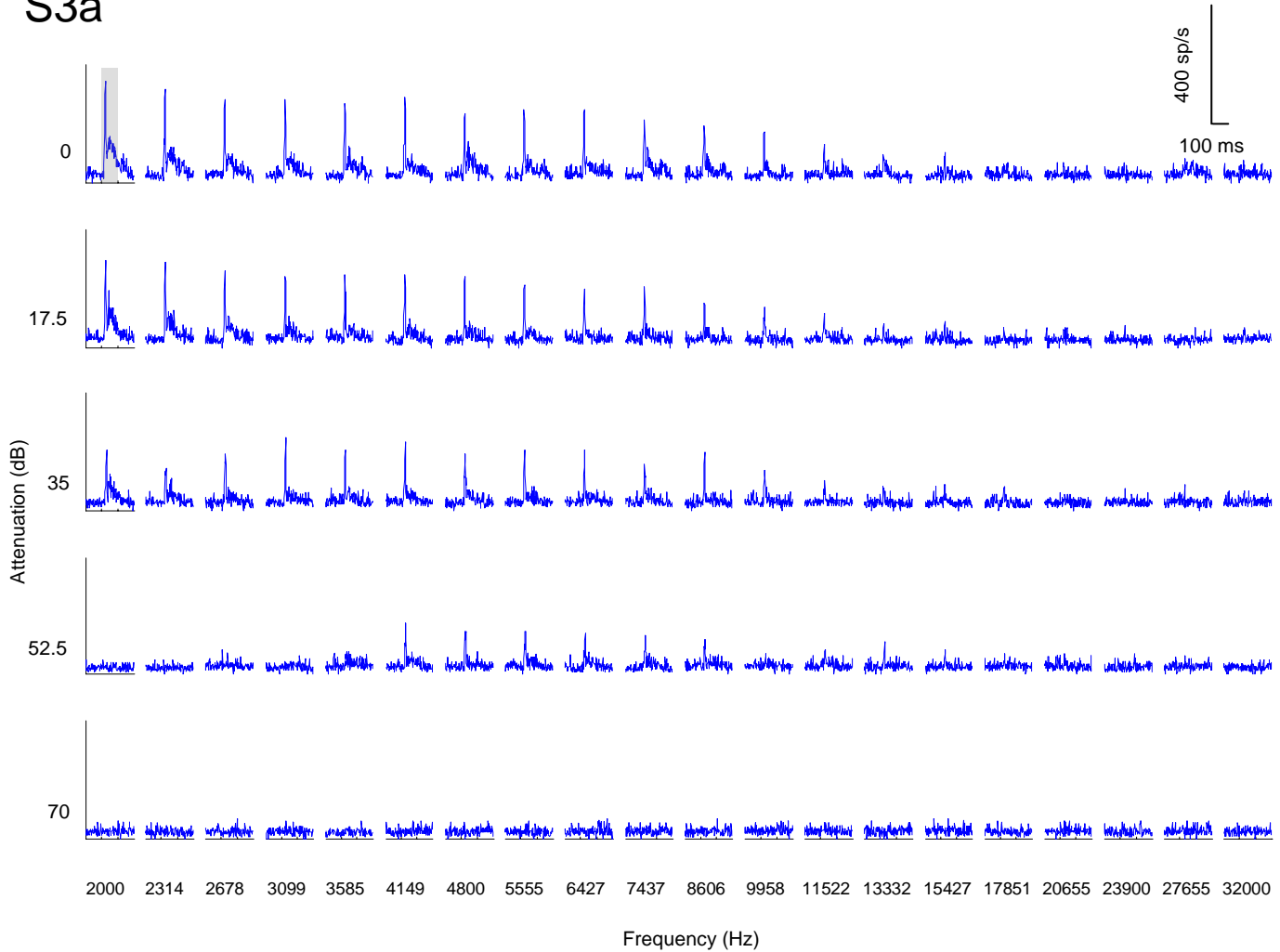


## Supplementary Figure S2

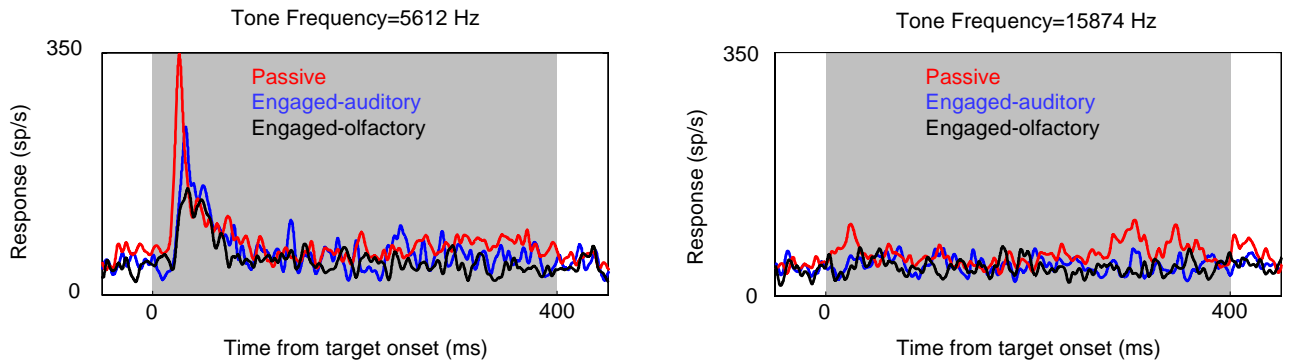
**Single unit responses to task relevant and task irrelevant stimuli are equally suppressed during the task.** This figure has a parallel structure to figure 2c-f, but is calculated using the data of 22 single units. For details see (SM3: Statistics: **Fig. 2 c-f and Fig. S2**).

# Figure S3

## S3a



## S3b

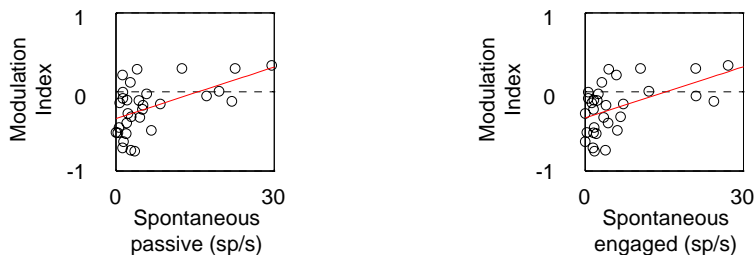


### Supplementary Figure S3

**Measurement of cortical tuning curves.** We measured the tuning curves before the first passive condition by presenting pure tones for 100 ms. We used 20 frequencies between 2KHz and 32 KHz and 4 sound levels, with 70 dB SPL as the maximum intensity (0 dB attenuation). The Best Frequency was defined as the frequency that evoked the largest response at the lowest intensity in which an evoked response was observed. **Fig. S3a** shows an example tuning curve for a cortical site. **Fig. S3b** shows responses for this site to two pure tones, showing that responses to these two frequencies were suppressed during task performance compared to the passive condition, although one frequency (5612 Hz) was closer to the best frequency (4149 Hz) than the other frequency (15874 Hz).

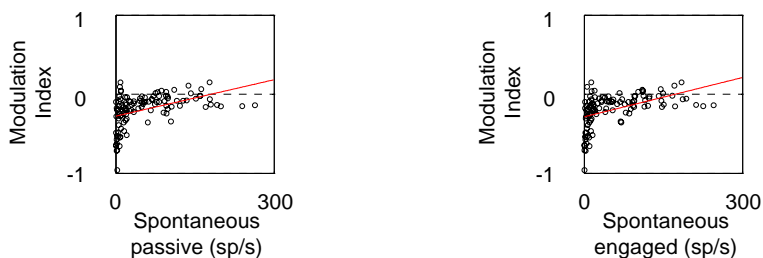
S4a

Single unit



S4b

Multiunit



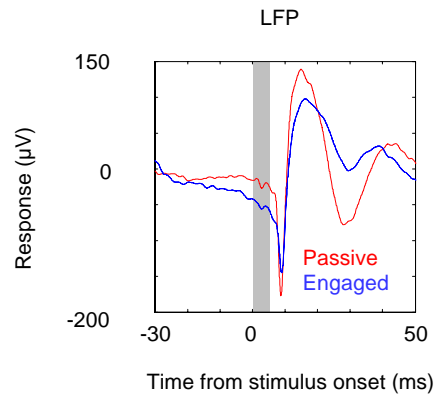
## Supplementary Figure S4

**Cortical units with high spontaneous firing rates show less suppression of the evoked response during the task. Fig.**

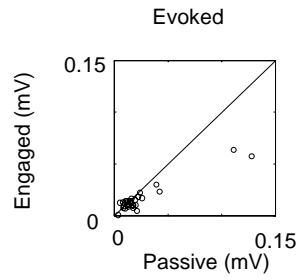
**S4a** shows that there was a significant positive correlation between the spontaneous firing rate, both in the passive and in the engaged condition, of cortical units and the modulation index  $\left( \frac{\text{Activity}_{\text{engaged}} - \text{Activity}_{\text{passive}}}{\text{Activity}_{\text{engaged}} + \text{Activity}_{\text{passive}}} \right)$  of the evoked response produced by task engagement. This data corresponds to the cells and multiunit sites shown in **Fig. 1**. The correlation was  $R=0.52$  ( $p=0.0023$ ) between the spontaneous firing rate in the passive condition and the modulation index, and it was  $R=0.50$  ( $p=0.0038$ ) between the spontaneous firing rate in the engaged condition and the modulation index. There was no significant correlation between the strength of the evoked response in the passive condition and the degree of suppression ( $R=0.13$ ,  $p=0.45$ ). **Fig. S4b** shows that this correlation could also be observed in multiunit recordings. The correlation was  $R=0.44$  ( $p=1.0e-6$ ) between the spontaneous firing rate in the passive condition and the modulation index, and it was  $R=0.48$  ( $p=8.6e-8$ ) between the spontaneous firing rate in the engaged condition and the modulation index.

# Figure S5

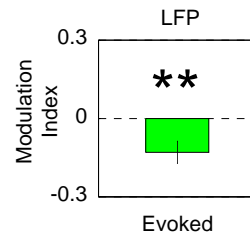
S5a



S5b

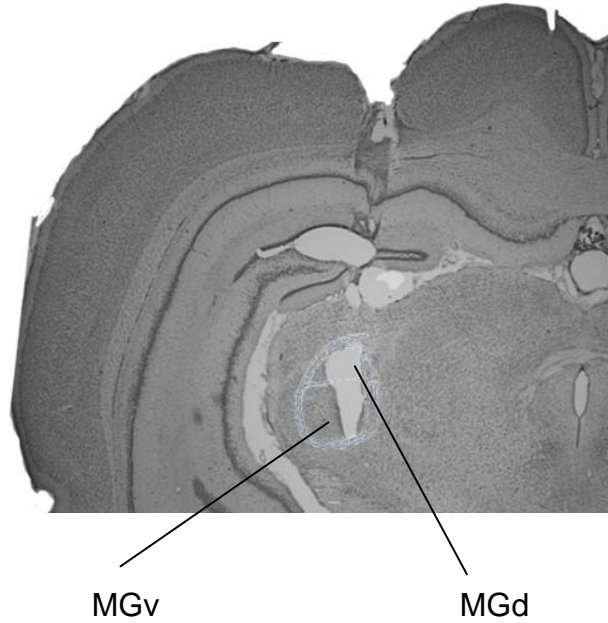


S5c



**Supplementary Figure S5**  
**Example (S5a) and population data (S5b-c) showing suppression of sound evoked LFPs in the head-fixed behavior**  
(Methods: task 4). For detailed caption see (SM3: Statistics: Fig. 5).

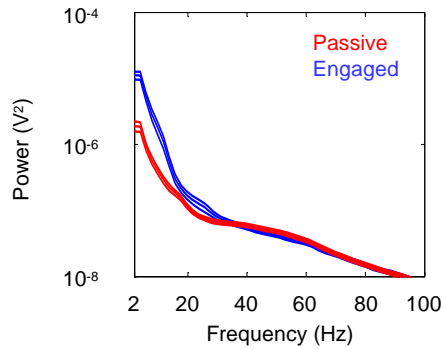
Figure S6



**Supplementary Figure S6**

**Histological reconstruction of recordings in the medial geniculate body (MGB).** We passed 50  $\mu$ A current, for 10 seconds, along the six electrode tracks, to mark the area where the recordings were made. Brains were sliced in 50  $\mu$ m sections. Recordings were from the dorsal (MGd) and ventral divisions (MGv) of the MGB (Paxinos and Watson 1986), which both project to primary auditory cortex in the rat (Roger and Arnault 1989; Kimura, Donishi et al. 2003). The lesion marks were visible between 5.2 and 5.8 mm posterior from bregma. The damage in the top cortical area was produced by the removal of a metal cannula that was implanted 2 mm into the brain. We could not distinguish individual electrode tracks.

# Figure S7

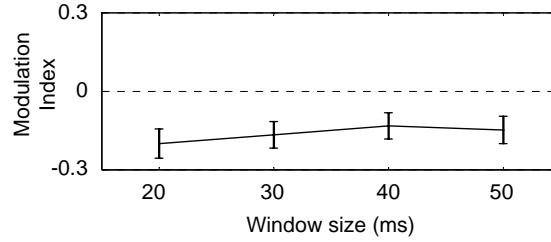


## Supplementary Figure S7

**Power spectrum of the spontaneous cortical activity in the passive and the engaged condition.** We recorded an EEG signal from one lead of each of the tetrodes. The signal was filtered between 1 and 475 Hz and acquired at 2016 Hz. In order to have a higher frequency resolution, we used a period of 400 ms preceding the stimulus onset. In the engaged condition, the animal would be in the center port waiting for the stimulus onset. The data was multiplied by a Hanning window of 400 ms. In each session, we calculated the power spectrum of each individual trial and averaged the spectrum to calculate the spectrum of each session. We have plotted the average power across all sessions (mean  $\pm$  s.e.m.). We found that the engaged condition has more power than the passive condition at frequencies below 30 Hz. The power spectrum is similar at frequencies above 30 Hz. The change in the power spectrum of the LFP recorded before stimulus onset indicates a subtler cortical state difference not reflected in the spontaneous firing rate.

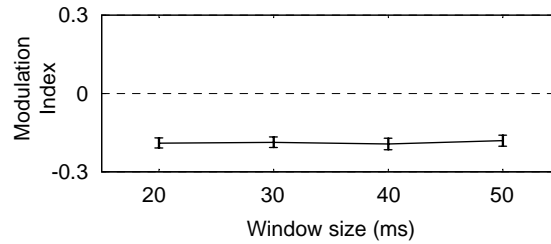
S8a

## Single unit cortical



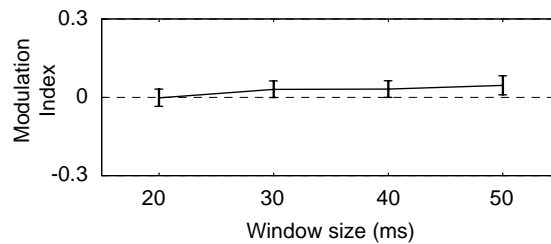
S8b

## Multiunit cortical



S8c

## Multiunit thalamic

**Supplementary Figure S8**

**Effects on modulation index in response to clicks were robust to perturbations in the window size.** We quantified evoked activity using a 20 ms window between 6 and 26 ms following the onset of the click, because most of the responses occurred in that early period in both auditory cortex and medial geniculate body. However, inclusion of a larger window of up to 50 ms for both cortical single units (**S8a**) and multiunit sites (**S8b**) and for thalamic multiunit sites (**S8c**) gave very similar results in terms of modulation index of evoked activity ( $(\text{Activity}_{\text{engaged}} - \text{Activity}_{\text{passive}}) / (\text{Activity}_{\text{engaged}} + \text{Activity}_{\text{passive}})$ ) between the passive and the engaged condition.



**Supplementary table 1: Experiment summary**

<b>Animal group</b>	<b>A</b>	<b>B</b>	<b>C</b>	<b>D</b>	<b>E</b>	<b>F</b>	<b>G</b>	<b>H</b>	<b>I</b>
<i>Number of animals</i>	2	2	2	2	5	1	4	4*	2*
<i>Task Number (see Methods)</i>	1	1	1	1	2	3	4	N.A	N.A
<i>Comparison engaged auditory vs. passive?</i>	Y	Y	Y	Y	N	Y	Y	N	N
<i>Comparison engaged olfactory vs. passive?</i>	N	N	N	N	N	Y	N	N	N
<i>Comparison engaged auditory versus engaged olfactory?</i>	N	N	N	N	Y	Y	N	N	N
<i>Comparison passive versus prolonged immobility?</i>	N	N	N	N	N	N	N	Y	N
<i>Comparison passive versus anesthetized?</i>	N	N	N	N	N	N	N	N	Y
<i>Number of different click rates tested</i>	6	2	2	6	0	0	1	2	2

<i>Target played in passive condition?</i>	N	Y	Y	N	N	Y	Y	Y	Y
<i>Number of sessions</i>	22	15	14	27	66	18	24	9	3
<i>Number of single units isolated</i>	59	28	0	13	709	0	0	0	0
<i>Number of multiunit sites</i>	96	84	90	156	180	66	72	45	14
<i>Number of LFP sites analyzed</i>	114	90	90	0	306	41	30	0	0
<i>Data used in figures</i>	1,2a-b	1,2c-f	2 c-f	6	3a-b, SM6	3c-f	5	4a-c	4d-f

\*Note: Animals in groups H and I were a subgroup of animals in groups B and C.

## Supplementary Material

### SM1: Behavioral monitoring

The animal EEG and a video of its behavior were monitored online, to confirm that the animal did not fall asleep during the passive condition. Long periods of immobility, and/or the onset of large amplitude, low frequency EEG were correlated with animal drowsiness. If these were observed, stimulus delivery was interrupted and the sound booth was gently tapped or the door was opened to keep the animal awake. Based on the recorded video, we excluded from the passive condition periods in which the animal was immobile for more than five seconds preceding the auditory stimulus onset; this represents a conservative criterion for avoiding the inclusion of periods of sleep in the dataset (see **Fig. S1**). We did not observe large low frequency oscillations in the power spectrum of the EEG signal in the analyzed portions of the passive period, indicating that the animal was not in slow-wave sleep (see **Fig. S7**).

### SM2: Stimulus response analysis

For the measurements described in **Fig. 1, 2a-b, 4, 5** and **6**: the responsiveness of a single unit or multiunit cluster to the click stimulus was determined by comparing the spike count in a 20 msec window before the onset of the stimulus with the response in another 20 msec window between 6 and 26 msec from the onset of the sound. Cells responded with a brief response of ~20 msec. The results reported here were not dependent on the length of the window used (see **Fig. S8**). We used a paired-sample sign test and established a level of 0.05 for significance in responsiveness in either the engaged or the passive condition. In the case of the LFP signal, it was always possible to see a short latency (<20 msec) deflection in response to the click stimulus, and no

selection based on responsiveness was performed. For both single units and multiunit clusters, spontaneous activity was quantified using the firing rate in the 20 msec preceding the first non-target stimulus in both the engaged and the passive conditions. The activity evoked by the non-target stimuli was quantified as the firing rate in a window between 6 and 26 msec from the sound onset. The LFP responses were quantified using the peak\_to\_peak value determined in the same response window (6 and 26 msec from onset). The evoked responses to the target stimulus (**Fig. 2c-f** and **Fig. S2**) were quantified for the duration of the stimulus (300 msec). For the task described in **Fig. 3a-b**, the responses were evaluated in a 40 msec window between 10 and 50 msec after stimulus onset and the spontaneous activity was quantified in a window of 40 msec preceding stimulus onset. We used a 40 msec window because the animals could withdraw from the center port after 50 msec, interrupting the stimulus delivery. For the measurements in **Fig. 3c-f**, we evaluated responsiveness in a window of 20 msec and quantified the evoked activity during the entire presentation of the stimulus (400 msec).

### **SM3: Statistics**

**Fig. 1 (Auditory cortex)** In 2 animals (15 recording sessions, task 1) we played the target in the passive condition and in another 2 animals (22 recording sessions) we did not play the target in the passive condition. The results were similar and are pooled together.

*Single units:* We recorded 87 well isolated stable units. We included for the analysis only those units (32/87; 7 were responsive only during the engaged condition, 12 only during the passive condition and 13 in both conditions) that showed a stimulus-locked response in either the passive or the engaged condition. Most of the units (24 out

of 32,  $p=0.0011$ , binomial distribution test) showed a suppression of evoked responses during the task, but no significant change in spontaneous firing rate (15/32 units showed an increase in spontaneous rate during the task;  $p=0.3$ , binomial distribution test). Fig. 1e is on a logarithmic scale and does not show the cells with zero firing rates in either the passive or engaged condition. The spontaneous plot shows 28/32 points and the evoked plot shows 31/32 points. The modulation index (  $(\text{Activity}_{\text{engaged}} - \text{Activity}_{\text{passive}}) / (\text{Activity}_{\text{engaged}} + \text{Activity}_{\text{passive}})$  ) for the evoked activity was  $-0.20 \pm 0.06$  and was significantly different from zero ( $p=0.0013$ ,  $n=32$ , double tailed t-test). The modulation index for the spontaneous activity was  $-0.010 \pm 0.08$  (mean  $\pm$  s.e.m.) and was not significantly different from zero ( $p=0.89$ ,  $n=32$ , double tailed t-test). Most responses (28/32) consisted of an elevation in firing rate compared to the background; results of these analyses were similar when we included only cells that elevated their firing rates.

*Multiunit sites:* One hundred eleven out of 180 multiunit sites were responsive to the target stimulus in either the passive or the engaged condition. Most of the sites showed a suppression of the evoked response during the task (98 out of 111,  $p < 1e-16$ , binomial distribution test). There was no general trend in the spontaneous activity (56 out of 111 showed suppression during the task,  $p=0.5$ , binomial distribution). The modulation index for the evoked response was  $-0.19 \pm 0.02$  (mean  $\pm$  s.e.m.) and it was significantly negative ( $p=1.90e-16$ ,  $n=111$ , double tailed t-test). There was no suppression for the spontaneous activity ( $0.0061 \pm 0.02$ ,  $p=0.80$ ,  $n=111$ , double tailed t-test).

*LFP sites:* We recorded LFP signal in 204 sites. Most of the sites showed a suppression of the evoked response during the task (166 out of 204,  $p < 1e-16$ , binomial distribution test). The modulation index for the evoked response was  $-0.12 \pm 0.01$  (mean  $\pm$  s.e.m.) and it was significantly negative ( $p=1.9e-19$ ,  $n=204$ , double tailed t-test).

**Fig. 2a-b (Auditory cortex)** We recorded in 2 animals across 22 recording sessions (task 1) in which we played regular trains of clicks (white noise bursts of 5 msec) with six different interclick intervals. The interclick intervals were 500, 200, 100, 66, 50 and 28 msec. Out of 96 sites recorded, we found 60 multiunit sites that were responsive to the first click. The modulation indexes for the steady state evoked response were significantly negative ( $p < 0.01$ ,  $n = 60$ , single tailed t-test) for all the repetition rates except for the two fastest trains of clicks (50 and 28 msec).

**Fig. 2c-f and Supplementary Fig. S2 (Auditory cortex)** The target stimulus was preceded by a train of clicks of either 5 or 20 Hz that lasted for 1.8 s. There were 93 multiunit sites and 22 single units responsive to the target stimulus, *i.e.* the stimulus used by the animals to make a decision in either the passive or the engaged condition. Responses are shown to the right target stimulus in the left auditory cortex in the passive versus the engaged condition. The responses to the left target stimulus were weak and found only in a few sites (33 out of 174 multiunit sites), so they were not further analyzed. Most of the sites and single units suppressed their responses during the task (59 out of 93 multiunit sites,  $p = 0.0062$ , binomial distribution test, 17 out of 22 single units,  $p = 0.0085$ , binomial distribution test). The modulation indexes were negative and significantly different from zero (multiunit:  $-0.073 \pm 0.02$ ,  $p = 1.2 \times 10^{-4}$ ,  $n = 93$ , double tailed t-test; single unit:  $-0.20 \pm 0.06$ ,  $p = 0.0019$ ,  $n = 22$ , double tailed t-test). The modulation indexes for the last click of the train were also negative and significantly different from zero (multiunit:  $-0.087 \pm 0.02$ ,  $p = 1.7 \times 10^{-4}$ , double tailed t-test,  $n = 93$ ; single unit:  $-0.18 \pm 0.09$ ,  $p = 0.046$ , double tailed t-test,  $n = 22$ ); however there was no difference between the modulation indexes of the last click of the train and the chord (multiunit:  $p = 0.18$ ,  $n = 93$ , paired t-test ; single unit:  $p = 0.81$ ,  $n = 22$ , paired t-test). The responses to the last click of the 5 Hz train and the 20 Hz train were similar and they were pooled together to compute the modulation index. The correlation coefficients between the modulation index and the absolute selectivity (see **Fig 2f** and **Fig. S2d**) were not

significantly different from zero (multiunit:  $R=0.082$ ,  $p=0.43$ ,  $n=93$ , double tailed t-test; single unit:  $R=0.21$ ,  $p=0.36$ ,  $n=22$ , double tailed t-test).

**Fig. 3a-b (Auditory cortex)** Five animals (66 recording sessions, task 2) performed blocks of trials of auditory discrimination (engaged-auditory) and blocks of trials of olfactory discrimination (engaged-olfactory). We compared the sound responses during the engaged-auditory block with the responses to the same sounds when the animal was in the engaged-olfactory block.

*Multiunit sites:* We analyzed 104 multiunit site responses (out of 612) that were responsive to the target stimulus in either the auditory or the olfactory block. The modulation index was defined as  $(\text{Activity}_{\text{auditory block}} - \text{Activity}_{\text{olfactory block}}) / (\text{Activity}_{\text{auditory block}} + \text{Activity}_{\text{olfactory block}})$ . The modulation index for the evoked response was  $0.015 \pm 0.02$  (mean  $\pm$  s.e.m.) and it was not significantly different from zero ( $p=0.34$ ,  $n=104$ , double tailed t-test). The modulation index for the spontaneous responses was  $-0.0022 \pm 0.02$  and it was not significantly different from zero ( $p=0.88$ ,  $n=104$ , double tailed t-test).

*LFP sites:* We recorded LFP signals in 306 sites, yielding 612 responses to tones. The modulation index for the evoked response was  $0.0059 \pm 0.008$  (mean  $\pm$  s.e.m.) and it was not significantly different from zero ( $p=0.49$ ,  $n=612$ , double tailed t-test).

**Fig. 3c-f** We recorded in one animal that performed task 3 (*Engaged-auditory vs. engaged-olfactory vs. passive*). We recorded 66 sites in 18 sessions.

*Multiunit sites:* Thirty-one sites responded to either of the two frequencies that we used for discrimination yielding 54 responses. *Spontaneous activity:* The modulation index between the engaged-auditory and the passive condition  $((\text{Activity}_{\text{auditory block}} - \text{Activity}_{\text{passive}}) / (\text{Activity}_{\text{auditory block}} + \text{Activity}_{\text{passive}}))$  was  $-0.0018 \pm 0.01$  (mean  $\pm$  s.e.m.)

and it was not significantly different from zero ( $p=0.88$ ,  $n=54$ , double tailed t-test). The modulation index between the engaged-olfactory and the passive condition  $((\text{Activity}_{\text{olfactory block}} - \text{Activity}_{\text{passive}}) / (\text{Activity}_{\text{olfactory block}} + \text{Activity}_{\text{passive}}))$  was  $-0.0012 \pm 0.01$  (mean  $\pm$  s.e.m.) and it was not significantly different from zero ( $p=0.92$ ,  $n=54$ , double tailed t-test). The modulation index between the engaged-auditory and the engaged-olfactory condition  $((\text{Activity}_{\text{auditory block}} - \text{Activity}_{\text{olfactory block}}) / (\text{Activity}_{\text{auditory block}} + \text{Activity}_{\text{olfactory block}}))$  was  $-0.0006 \pm 0.01$  (mean  $\pm$  s.e.m.) and it was not significantly different from zero ( $p=0.90$ ,  $n=54$ , double tailed t-test). *Evoked activity:* The modulation index between the engaged-auditory and the passive condition was  $-0.057 \pm 0.009$  (mean  $\pm$  s.e.m.) and it was significantly different from zero ( $p=9.6e-8$ ,  $n=54$ , double tailed t-test). The modulation index between the engaged-olfactory and the passive condition was  $-0.074 \pm 0.01$  (mean  $\pm$  s.e.m.) and it was significantly different from zero ( $p=1.05e-8$ ,  $n=54$ , double tailed t-test). The modulation index between the engaged-auditory and the engaged-olfactory condition was  $0.017 \pm 0.004$  (mean  $\pm$  s.e.m.) and it was significantly different from zero ( $p=8.9e-5$ ,  $n=54$ , double tailed t-test).

*LFP sites:* We have analyzed 41 LFP sites that responded to the pure tones used in the task. The modulation index between the engaged-auditory and the passive condition was  $-0.14 \pm 0.03$  (mean  $\pm$  s.e.m.) and it was significantly different from zero ( $p=9.2e-5$ ,  $n=41$ , double tailed t-test). The modulation index between the engaged-olfactory and the passive condition was  $-0.09 \pm 0.04$  (mean  $\pm$  s.e.m.) and it was significantly different from zero ( $p=0.016$ ,  $n=41$ , double tailed t-test). The modulation index between the engaged-auditory and the engaged-olfactory condition was  $-0.04 \pm 0.03$  (mean  $\pm$  s.e.m.) and it was not significantly different from zero ( $p=0.16$ ,  $n=41$ , double tailed t-test).

*Correlation analysis between stimulus discriminability and modulation index of evoked activity:* We calculated the discriminability between the two frequencies that we used during the task as:  $2 * \text{abs}((\text{area under the ROC curve}) - 0.5)$ . The correlation



coefficients between the modulation indexes of the evoked activity (engaged-auditory vs. passive and engaged-olfactory vs. passive) and the frequency selectivity were not significantly different from zero (engaged-auditory vs. passive,  $R=0.22$ ,  $p=0.10$ ,  $n=54$ , double tailed t-test; engaged-olfactory,  $R=0.19$ ,  $p=0.15$ ,  $n=54$ , double tailed t-test).

The correlation coefficients between the modulation indexes of the evoked activity (engaged-auditory vs. passive and engaged-olfactory vs. passive) and the distance to the best frequency in octaves of the recorded site were not significantly different from zero (engaged-auditory vs. passive,  $R=0.09$ ,  $p=0.52$ ,  $n=47$ , double tailed t-test; engaged-olfactory,  $R=0.16$ ,  $p=0.27$ ,  $n=47$ , double tailed t-test).

**Fig. 4a-c (Auditory cortex)** We detected periods of immobility of 15 seconds or longer in 4 animals by analyzing the video recordings. These longer periods of immobility were rare in our recordings, as we were actively monitoring the animal to keep him awake. There were only 9 sessions in which more than 10 stimulus presentations were made during a period of prolonged immobility. We compared the evoked response during these periods with our standard definition of passive, which required less than five seconds of immobility. In these 9 sessions, 33 out of 45 sites were responsive to the non-target stimulus in either the passive or prolonged-immobility condition. Spontaneous activity (*see Fig. 4b*) increased in the passive state compared to the prolonged-immobility state (23 out of 33 increased,  $p=0.017$ , binomial test), whereas evoked activity was unchanged (15 out of 33 increased,  $p=0.75$ , binomial test). The modulation index  $(\text{Activity}_{\text{passive}} - \text{Activity}_{\text{prolonged immobility}}) / (\text{Activity}_{\text{passive}} + \text{Activity}_{\text{prolonged immobility}})$  was significantly positive ( $0.20 \pm 0.06$ , mean  $\pm$  s.e.m,  $p=0.0036$ ,  $n=33$ , double tailed t-test) for the spontaneous activity and was not significantly different from zero ( $0.01 \pm 0.04$ , mean  $\pm$  s.e.m.,  $p=0.79$ ,  $n=33$ , double tailed t-test) for the evoked activity.

**Fig. 4d-f (Auditory cortex)** We recorded from two animals that were already implanted with a tetrode drive. We first recorded in the passive condition and then anesthetized the animals with an intraperitoneal injection of a mixture of ketamine (20 mg/kg) and medetomidine (0.17 mg/kg). Animals were lightly anesthetized and showed a strong pedal withdrawal reflex. We recorded in 14 sites across 3 sessions. We found 13 sites that were responsive to the non-target stimulus. The spontaneous activity was increased in all sites in the awake state compared to anesthetized state (13 out of 13). The evoked activity was suppressed in most sites (10 out of 13) in the awake state compared to the anesthetized state. The modulation index (  $(\text{Activity}_{\text{passive}} - \text{Activity}_{\text{anesthetized}}) / (\text{Activity}_{\text{passive}} + \text{Activity}_{\text{anesthetized}})$  ) was significantly positive for the spontaneous activity ( $0.53 \pm 0.06$ , mean  $\pm$  s.e.m,  $p=1.7e-6$ ,  $n=13$ , double tailed t-test) and significantly negative for the evoked activity ( $-0.19 \pm 0.05$ , mean  $\pm$  s.e.m,  $p=0.005$ ,  $n=13$ , double tailed t-test).

**Fig. 5 (Auditory Cortex)** We recorded in 4 rats during 24 recording sessions in the head-fixed behavior (task 4).

*Multiunit sites:* Sixty-four out of 72 multiunit sites were responsive to the target stimulus in either the passive or the engaged condition. Most of the sites showed a suppression of the evoked response during the task (53 out of 64,  $p < 1e-8$ , binomial distribution test). There was no general trend in the spontaneous activity (32 out of 64 showed suppression during the task,  $p=0.45$ , binomial distribution). The modulation index (  $(\text{Activity}_{\text{engaged}} - \text{Activity}_{\text{passive}}) / (\text{Activity}_{\text{engaged}} + \text{Activity}_{\text{passive}})$  ) for the evoked response was  $-0.20 \pm 0.03$  (mean  $\pm$  s.e.m.) and it was significantly negative ( $p=1.27e-8$ ,  $n=64$ , double tailed t-test). There was no suppression for the spontaneous activity ( $-0.012 \pm 0.04$ ,  $p=0.73$ ,  $n=64$ , double tailed t-test).

*LFP*: All 30 sites were responsive to the target stimulus in either passive or engaged condition. Most of the sites showed a suppression of the evoked response during the task (26 out of 30,  $p < 1e-5$ , binomial distribution test). The modulation index for the evoked response was  $-0.13 \pm 0.04$  (mean  $\pm$  s.e.m.) and it was significantly negative ( $p = 0.0031$ ,  $n = 30$ , double tailed t-test).

**Fig. 6 (Auditory thalamus)** We recorded from 2 animals in 27 recording sessions (task 1). We found 91 sites that were responsive to the first click, out of a total of 156 sampled sites. There was no difference in evoked activity (49 sites out of 91 showed suppression during the task,  $p = 0.80$ , binomial distribution test). Most of the sites showed an enhancement of spontaneous activity during the task (60 out of 91,  $p = 7.58e-4$ , binomial distribution test). The modulation index  $(\text{Activity}_{\text{engaged}} - \text{Activity}_{\text{passive}}) / (\text{Activity}_{\text{engaged}} + \text{Activity}_{\text{passive}})$  was  $0.16 \pm 0.04$  (mean  $\pm$  s.e.m.) for the spontaneous activity and was significantly positive ( $p = 1.49e-5$ ,  $n = 91$ , double tailed t-test), indicating enhancement during the task. There was no suppression of the evoked activity ( $-0.011 \pm 0.03$ ,  $p = 0.74$ ,  $n = 91$ , double tailed t-test).

#### **SM4: Comparison of neural activity between first and second passive block**

Because the first passive block occurred before the animal performed the task, whereas the second occurred after the animal was rewarded with water, we wondered whether the resulting differences in expectation, motivation, thirst etc. might lead to differences in evoked neural activity. To assess this, we defined a modulation index as:  $(\text{evoked}_{\text{passive before}} - \text{evoked}_{\text{passive after}}) / (\text{evoked}_{\text{passive before}} + \text{evoked}_{\text{passive after}})$ . Out of 111 multiunit sites, we found a modulation index of  $-0.001 \pm 0.02$  ( $p = 0.96$ , double-tailed t-

test). For 28 single units held for both passive blocks the modulation before and after was  $-0.075 \pm 0.06$  ( $p=0.23$ , double-tailed t-test). The LFP modulation index was  $-0.01 \pm 0.01$  ( $p=0.23$ , double-tailed t-test). We thus did not find any significant difference in the evoked cortical response between the two passive blocks.

### **SM5: Kurtosis of firing rate distribution**

As the distribution of firing rates across a population of cells becomes sparser, the number of cells with moderate responses is reduced, and the number of cells with very low and very high firing rates increases; this is called kurtosis (Vinje and Gallant 2000). We estimated the sparseness of the distribution of evoked firing rates across the population of the 32 responsive cells by calculating the kurtosis (Vinje and Gallant 2000). As a distribution becomes sparser, its kurtosis increases. The kurtosis is the fourth moment of the distribution; as a reference, the kurtosis of a gaussian distribution is 3. We found that  $\text{kurtosis}_{\text{passive}} = 4.1 \pm 0.7$  and that the distribution became sparser as the animals performed the task, with  $\text{kurtosis}_{\text{engaged}} = 8.4 \pm 2.4$ . Here we report the mean  $\pm$  standard deviation of a bootstrap estimator. The increase in kurtosis was significant (permutation test,  $p=0.028$ ).

### **SM6: Single unit responses during the intermodal auditory-olfactory task**

We recorded 709 well-isolated stable units. Of these, we included only those (189/709) that showed a stimulus-locked response in either the olfactory or the auditory block. We analyzed and combined responses for the low frequency tone and the high frequency tone, yielding 378 cell responses total. Most of the cell responses (212 out of

378,  $p=0.01$ , binomial distribution test) were larger in the auditory block compared to the responses in the olfactory block. The modulation index ( $(\text{Activity}_{\text{auditory block}} - \text{Activity}_{\text{olfactory block}}) / (\text{Activity}_{\text{auditory block}} + \text{Activity}_{\text{olfactory block}})$ ) was  $0.023 \pm 0.010$  for the evoked response and it was significantly different from zero ( $p=0.023$ ,  $n=378$ , double tailed t-test), consistent with the enhancement of activity by selective attention (Desimone and Duncan 1995; Fritz, Shamma et al. 2003). No significant change in spontaneous firing rate (104/189 units showed increase in spontaneous rate during the auditory block;  $p=0.095$ , binomial distribution test) was detected. The modulation index for the spontaneous activity was  $-0.0014 \pm 0.01$  and was not significantly different from zero ( $p=0.89$ ,  $n=189$ , double tailed t-test).

### **SM7: Bursts in thalamus**

We wondered whether the elevation in thalamic spontaneous rate might be associated with a switch from burst to tonic firing mode as is seen with large changes in arousal (Reinagel, Godwin et al. 1999; Swadlow and Gusev 2001; Weyand, Boudreaux et al. 2001; Sherman 2005; Bezdudnaya, Cano et al. 2006). In order to assess whether thalamic cells showed more bursts in the passive condition compared to the engaged condition, we isolated 13 single units from the thalamic recordings. We used a more stringent criterion for the cluster quality than in cortex: (1)  $<0.05\%$  refractory period violations; (2) an Isolation Distance (see *Electrophysiology and recording procedure* above) of more than 20. We found similar tendencies as in the multiunit recordings: most cells (9 cells out of 13) showed an increase in their spontaneous firing rate during the task, and we did not find a general trend for the evoked response (6 increased their firing rate, 7 decreased their firing rate). The mean spontaneous firing rate for these cells was  $8.3 \pm 1.7$  spikes/s in the passive condition, which is within the range of the

spontaneous firing rate reported in awake medial geniculate body (Massaux, Dutrieux et al. 2004). This value increased to  $12.7 \pm 2.4$  spikes/s in the engaged condition. When we looked at the fraction of bursts (bursts were quantified as a group of 2 or more spikes spaced less than 5 msec and preceded by a silent period of 100 msec or more), we found that the burst proportion was low in both conditions:  $2.9\% \pm 1.4\%$  in the passive and  $2.0\% \pm 0.9\%$  in the engaged condition, which agrees with previous reports of awake auditory thalamus (Massaux, Dutrieux et al. 2004). For each cell, we calculated whether the burst proportion exceeded what would be expected for a Poisson process with the same spontaneous firing rate. We only found two cells out of thirteen that exceeded the burst count of a Poisson process at the  $p=0.05$  level. The burst fraction is low in both states and we could not detect a difference between the passive and the engaged condition.

### **SM8: Thalamocortical depression model**

A simple model can reconcile our cortical and thalamic observations. In the cortex we observed no change in the spontaneous firing rate but a suppression of the evoked firing rate in the engaged condition, whereas in the thalamus we observed an enhancement of the spontaneous rate but no change in the evoked rate. We illustrate the thalamocortical depression model with a simple example. Suppose that the spontaneous thalamic firing rate in the passive condition is 10 Hz, and that the release probability at thalamocortical inputs is  $P_{\text{passive}} = 1/F_{\text{thalamic-passive}} = 1/10$ . If the spontaneous thalamic firing rate in the engaged condition is 20 Hz, then the release probability in the engaged condition is  $P_{\text{engaged}} = 1/F_{\text{thalamic-engaged}} = 1/20$ . We further assume that the transformation from thalamus to cortex is linear, *i.e.* that the product of the thalamocortical release probability and the thalamic firing rate determines the cortical firing rate ( $F_{\text{cortical}} =$

$P_{\text{release}} \times F_{\text{thalamic}}$ ). Under these assumptions, the spontaneous firing rate in the passive condition ( $F_{\text{cortex-passive}} = 10 \text{ Hz} \times 1/10 = 1 \text{ Hz}$ ) is equal to the firing rate in the engaged condition ( $F_{\text{cortex-engaged}} = 20 \text{ Hz} \times 1/20 = 1 \text{ Hz}$ ). Thus in the model, as in the data, cortical spontaneous firing rates are the same in the passive and engaged conditions.

Now suppose that the thalamic evoked response in both the passive and engaged conditions is 40 Hz. Because the spontaneous firing rate in the thalamus is higher in the engaged condition, thalamocortical synapses are more depressed, so the evoked firing rate in the cortex is lower ( $F'_{\text{cortex-engaged}} = 40 \text{ Hz} \times 1/20 = 2 \text{ Hz}$ ) than in the passive condition ( $F'_{\text{cortex-passive}} = 40 \text{ Hz} \times 1/10 = 4 \text{ Hz}$ ). Thus in the model, as in the data, cortical evoked firing rates are suppressed in the engaged relative to the passive condition.

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