European Journal of Neuroscience, Vol. 33, pp. 521–529, 2011

COGNITIVE NEUROSCIENCE

Development of auditory-specific brain rhythm in infants

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Keywords: auditory cortex, cortical plasticity, EEG frequency band analysis, event-related oscillations, maturation

Abstract

Human infants rapidly develop their auditory perceptual abilities and acquire culture-specific knowledge in speech and music in the second 6 months of life. In the adult brain, neural rhythm around 10 Hz in the temporal lobes is thought to reflect sound analysis and subsequent cognitive processes such as memory and attention. To study when and how such rhythm emerges in infancy, we examined electroencephaolgram (EEG) recordings in infants 4 and 12 months of age during sound stimulation and silence. In the 4-month-olds, the amplitudes of narrowly tuned 4-Hz brain rhythm, recorded from bilateral temporal electrodes, were modulated by sound stimuli. In the 12-month-olds, the sound-induced modulation occurred at faster 6-Hz rhythm at temporofrontal locations. The brain rhythms in the older infants consisted of more complex components, as even evident in individual data. These findings suggest that auditory-specific rhythmic neural activity, which is already established before 6 months of age, involves more speed-efficient long-range neural networks by the age of 12 months when long-term memory for native phoneme representation and for musical rhythmic features is formed. We suggest that maturation of distinct rhythmic components occurs in parallel, and that sensory-specific functions bound to particular thalamo-cortical networks are transferred to newly developed higher-order networks step by step until adult hierarchical neural oscillatory mechanisms are achieved across the whole brain.

Introduction

The human auditory system starts developing *in utero* (Kisilevsky & Low, 1998) and plays a fundamental role in acquisition of linguistic and musical knowledge in infancy. Six-month-old infants can discriminate phonetic contrasts (Werker & Lalonde, 1988), detect rhythmic pattern differences (Hannon & Trehub, 2005) and recognize a melody played at different pitch levels (Plantinga & Trainor, 2005). However, 12-month-olds do not recognize acoustic contrasts that are no longer meaningful in their native culture (Werker & Lalonde, 1988; Hannon & Trehub, 2005). Thus, infants appear to develop neural networks for higher-order sensory information processing in the second 6 months after birth.

Neural correlates of such perceptual skills have been probed using electroencephalography (EEG) or magnetoencephalography (MEG) rhythms, which are thought to reflect global and local neural networks operating at different frequency ranges (Varela *et al.*, 2001; Buzsáki & Draguhn, 2004). In adults, alpha-band rhythm around 10 Hz (8–12 Hz) is dominant and most pronounced across the brain in resting states, while its power decreases selectively at specific brain regions, according to sensory (visual/auditory/somatosensory), motor and cognitive (attention and memory) task demands (Klimesch, 1999). These modulations are hypothesized to reflect selective activation and

inhibition in specific neural networks regarding sensory processing and subsequent cognitive processes (Knyazev, 2007).

However, development of neural rhythms is not fully understood, mainly because to determine which infant rhythms correspond to those in adults, functional and spatial information should be taken into account (Kuhlman, 1980). Recent studies, using modern quantification methods with multiple electrodes, have shown that in infants between 8 and 12 months of age, the occipital-alpha rhythm is blocked by attending to visual stimuli (Stroganova *et al.*, 1999), and that the central-mu rhythm is blocked by movements (Orekhova *et al.*, 2006), both in a lower frequency range than in adults. These studies also confirmed earlier findings (Smith, 1939, 1941) that, as age increases, these rhythms gradually accelerate and become more spatially segregated.

Here, we examined for the first time in infants the development of 'temporal-tau' rhythm, the one remaining alpha-band rhythm originating from auditory cortex that is suppressed by sounds in adults (Tiihonen *et al.*, 1991; Lehtela *et al.*, 1997; Niedermeyer, 1997), and in 4- to 6-year-old children with already adult-like frequency (Fujioka & Ross, 2008). During the first 4 months the auditory system rapidly changes the way it processes acoustic inputs (Lutter *et al.*, 2006; Rivera-Gaxiola *et al.*, 2007), simple acoustic contrasts (Čeponiene *et al.*, 2000; Kushnerenko *et al.*, 2002), and more complex acoustic features such as periodicity pitch (He & Trainor, 2009) and sequential patterns (He *et al.*, 2009). EEG activity at rest also changes in spectral components (Mizuno *et al.*, 1970) and topography (Marshall *et al.*, *et a*

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Received 21 July 2010, revised 1 October 2010, accepted 28 October 2010

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2002) over the first year and beyond. Although sound suppresses overall EEG rhythms in sleeping newborns (Ellingson, 1958), its topographical information has not been examined. Therefore, we examined: (i) whether the spatially segregated tau-equivalent rhythm is present in infants younger than 6 months, and (ii) how it develops by 12 months of age, by using high-density EEG recording with and without sound stimulation. Further, we used Gaussian modeling of group and individual power spectra to examine how spatially segregated oscillatory neural networks develop.

Materials and methods

Subjects

Healthy, full-term infants with no known history of neurological or hearing problems were recruited as subjects. There were 12 4-monthold infants (five females, mean 141.8 days, SD = 6.07) and 13 12-month-old infants (eight females, mean 382.8 days, SD = 15.5). Written consent and a questionnaire on musical background were completed by a parent.

Stimuli and EEG recording procedure

Three 2-min episodes of EEG activity were recorded from each subject with and without auditory stimulation ('sound' and 'silent' conditions). The EEG data for these two conditions were analysed. The 4-month-old infants also participated in three episodes of another condition in which two piano tones (one 80% and the other 20% repetition) played in random order, but these data were not analysed here. In the sound condition, the auditory stimulus was a single piano tone repeated every 800 ms, resulting in 450 tones presented in total. The EEG was continuously recorded using 124-sensor HydroCel GSN nets (Electrical Geodesics, Inc., Eugene, OR, USA) referenced to Cz in a sound-treated room with background noise level < 29 dB(A). The sampling rate was 1000 Hz. To encourage infants to keep still, they watched a silent movie and puppet show on their caregiver's lap. Sound stimuli were played through E-PRIME software (v. 1.2; Psychology Software Tools, Inc., Pittsburgh, PA, USA) on a computer over a custom-designed speaker 1 m in front of the subject at a level of 70 dB(A). All study procedures were approved by the McMaster University Research Ethics Board. The McMaster Ethics Research Council follows the Canadian Tri Council Policy Statement: Ethical Conduct for Research Involving Humans.

Analysis of EEG power spectral density

The EEG data were first off-line filtered between 0.5 and 20 Hz, downsampled to 215 Hz and re-referenced using an average reference. To correct large-amplitude artifacts from movements and loss of electrode contacts, the artifact-blocking algorithm (Mourad *et al.*, 2007; Fujioka *et al.*, in press), implemented using MATLAB (Math-Works, Inc., Natick, MA, USA) was applied to the continuous data.

To calculate the power spectral density (PSD), the measured EEG data for each subject were stored in two matrices of the same size, one for each condition. The number of rows of each matrix corresponded to the number of electrodes, while the number of columns corresponded to the number of samples, *T*. However, *T* was chosen such that the stimulus frequency f_a (= 1.25 Hz) would also be equal to an integer multiple of the frequency bin Δf (= f_s/T , where f_s was the sampling frequency). This allowed us to examine whether there was an EEG power increase at the stimulus frequency and its harmonic

frequencies in the sound condition compared with the silent condition, as an indication of brain response specifically phase-locked to the stimuli (i.e. the event-related potential) in the frequency domain. The MATLAB function '*psd.m*' was applied to these matrices to obtain the PSD of each row. From these PSDs, the spatial distribution and the frequency band of the tau rhythm in each age group were examined as follows. First, the average PSD of all the subjects and all the electrodes in each group was calculated. The frequency band in which the PSDs between the two conditions (silent, sound) differed was visually identified as 4.0–5.5 Hz in the 4-month-old and 5.5–7.0 Hz in the 12-month-old groups. Topographic maps of the average power of the tau rhythm in the two conditions, as well as their difference, are shown in Fig. 2 below.

Estimation of group-based models of EEG rhythmic components

We examined how many separate rhythmic components could be identified in the PSD by using a modified version of a modeling technique previously proposed for automated adult EEG analysis (Chiang *et al.*, 2008).

The model was constructed in two steps. In the first, two approximations of the average PSD were obtained. The first approximation was a de-noised version of the average PSD, which was calculated as the approximate component at the 7th level of the *sym4* wavelet transform. The second approximation was the one that reflects the 1/f roll-off of the PSD. This approximation was calculated using a simple regression applied to the logarithm of the PSD. Visual inspection of the average PSD indicated that the peaks of interest existed in the frequencies between 3 and 8 Hz. Thus, only frequency bands outside of this range (e.g. between 1-2 and 9-10 Hz) were used for calculating the parameters of the regression approximation (Fig. 3A and D). The difference between the natural logarithms of



FIG. 1. Electrode clusters used for the power spectra comparison by ANOVA. Frontal (dark grey), LeftTemporal (open circles with solid line), RightTemporal (open circles with dotted line), Central (black) and Occipital (pale grey) clusters contained 22–30 electrodes each.

these two approximations produces a dimensionless spectral curve (Fig. 3B and E) termed the WR-approximate signal. In the second step of constructing the fitting model, we examined the minimum number of Gaussian pulses that can fit this WR-approximate signal obtained for each group with a relatively small residual error. Using the fit function in MATLAB, the number of Gaussian pulses was increased from one to eight. To prevent the fitting algorithm from fitting the residual noise, two constraints were imposed on the values of the parameters of the Gaussian pulses: (i) the center (mean) of each Gaussian pulse was constrained to lie within the frequency of interest (i.e. 3-8 Hz); and (ii) the minimum values of the amplitude and variance parameters were chosen to be more than those of residual noise outside the frequency of interest of the analysis (i.e. 1-3 and 8-10 Hz). These values were chosen to prevent the algorithm from fitting the residual noise within the frequency of interest. The minimum numbers of Gaussian pulses was two and three for the 4- and 12-month groups, respectively (Fig. 3C and F). Thereafter, the model for each age group was finalized as the corresponding Gaussian pulses after normalizing each to have unit norm. For example, the model for the 12-month group is the matrix $G_{12} = [g_1 g_2 g_3]$, where the column vectors $g_1 - g_3$ are the three Gaussian pulses shown in Fig. 3E after normalizing each to have unit norm.

Evaluation of individual EEG rhythmic components

Based on the model for the PSD of each group, we examined the number of peaks and the distribution of the tau rhythm in individuals. To simplify the exposition, we consider only the 12-month group in this section. A similar procedure was followed for the 4-month group. The objective of this part of the procedure was to identify the contribution of each normalized Gaussian pulse (in the model G_{12}) in the WR-approximate of the PSD (in the frequency range 1–10 Hz) at each individual recording electrode. For example, let the vector x_i refer to the WR-approximate of the PSD at the *i*-th recording electrode. An approximation of x_i can be calculated with the fitting model G_{12} using the following equation:

$$\hat{x}_i = a_{i1}g_1 + a_{i2}g_2 + a_{i3}g_3$$

where the vector $\mathbf{a}_i = [a_{i1} \ a_{i2} \ a_{i3}]^{\mathrm{T}}$ represents the contribution of the three Gaussian pulses $\mathbf{g}_1 - \mathbf{g}_3$ in \mathbf{x}_i , and it can be calculated as

$$a_i = (G_{12}^T G_{12})^{-1} G_{12}^T x_i.$$

After calculating \hat{x}_i , the values of the amplitude parameters $a_{i1}-a_{i3}$ were compared with those of the residual signal $\tilde{x}_i = x_i - \hat{x}_i$ using the *z*-test. If the value of any one of the amplitude parameters a_{i1} , a_{i2} or a_{i3} was not significantly different from those of the residual signal \tilde{x}_i , the value of this parameter was set to zero. This procedure was repeated for the silent and the sound conditions and for all $i = 1, ..., N_e$, where N_e is the number of the recording electrodes. For each subject, a topographic map was constructed for each of the amplitude parameters a_1-a_3 . Examples of the topographic maps are shown in the Figs 4 and 5.

Statistical analysis of EEG power spectra

The power value was \log_{10} -transformed to normalize the distribution. The relative power contribution in each frequency bin with 1.5-Hz width (2.5–4, 4–5.5, 5.5–7, 7–8.5 and 8.5–10 Hz) compared with the total power between 1 and 10 Hz was calculated, averaged over 22–30

electrodes within five clusters at frontal, central, occipital, left and right temporal areas (Fig. 1). The averaged relative power was then submitted to ANOVA. Three-way mixed ANOVAs were used with AGE (4 vs. 12 months) as between-subject factor, and STIM (sound vs. silent) and ELECTRODE (Frontal, LeftTemporal, RightTemporal, Central, Occipital) as within-subject factors for each frequency bin separately. Significance levels were set at 0.05, and Bonferroni corrections were used for multiple comparisons when applicable. ANOVAs were conducted using SPSS (ver. 16; SPSS Inc., Chicago, IL, USA). When Mauchley's sphericity was violated in any factor or interactions, Greenhouse–Geisser Epsilon was used to correct probability. Where the correction was applied, the original F values and corrected P-values are reported.

Results

EEG power spectral density and sound-induced suppression

In 4-month-old infants, there was a clear peak in PSD around 4 Hz in the silent condition and this was suppressed in the sound condition (Fig. 2A, top). Although the topographies of the average power of the silent and sound conditions appeared similar, the difference revealed clear bilateral patterns of temporal electrodes contributing to the sound-induced suppression (Fig. 2B, top). On the other hand, in the 12-month-old group, the spatial and spectral patterns were different. The average PSD showed two peaks at around 4 and 6 Hz (lower panel of Fig. 2A), in which the sound-induced suppression was evident only in the higher frequency peak, and the spatial distribution of the suppressed peak was concentrated in the temporofrontal electrodes (Fig. 2B, bottom). In the topography of the silent condition, the occipital and bilateral temporofrontal electrodes clearly showed higher power than the other electrodes. However, only the latter decreased in the sound condition as the power in the occipital site was not changed.

Only in the PSD corresponding to the sound condition (Fig. 2A, blue curve) were there multiple harmonic spikes every 1.25 Hz, which is equal to the regular sound stimulus rate (the stimulus was presented every 0.8 s). These reflect the broad-band auditory evoked (phase-locked) response which, interestingly, did not affect the sound-induced suppression of spontaneous EEG rhythmic activity.

To examine these differences in spectral and spatial characteristics of the PSD obtained (Fig. 2), we applied mixed ANOVAs separately to the relative power measures in each frequency band (1.5-Hz width) using one between-subject factor, AGE (4, 12 months), and two within-subject factors, STIM (sound, silent) and ELEC-TRODE (Frontal, LeftTemporal, RightTemporal, Central, Occipital). Our main interest was to see whether the spectral and topographical changes of stimulus-induced suppression between two age groups were statistically significant, as indicated by the interaction between STIM, ELECTRODE and AGE.

In the frequency band between 2.5 and 4.0 Hz, AGE was not significant, the interaction STIM × AGE approached significance (n.s., P < 0.06) and the interaction ELECTRODE × AGE was highly significant ($F_{4,92} = 11.976$, P < 0.001, $\eta_p^2 = 0.342$). The ELECTRODE × AGE interaction was caused by the topographical change between two ages – in 4-month-olds, the amplitude in temporal and occipital sites was larger than in central and frontal sites (P < 0.001), whereas in 12-month-olds, the amplitude in the frontal sites was larger than in the central and left temporal sites (P < 0.001). The main effect of ELECTRODE was also significant ($F_{4,92} = 10.882$, P < 0.001, $\eta_p^2 = 0.321$), because the amplitude at the central sites was smaller than at the right temporal site consistently across the groups.

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FIG. 2. Power spectral density (PSD) of EEG from 4-month-olds (top) and 12-month-olds (bottom) in silent and sound conditions. (A) The power spectrum across frequencies in all electrodes in silent condition is indicated by the red curves; this is suppressed in the sound condition, indicated by the blue curves. (B) Topography of the average power in the frequency range 4.0–5.5 Hz for 4-month-olds and 5.5–7.0 Hz for 12-month-olds, in silent conditions, sound conditions and the difference between the two.



FIG. 3. Left: power spectral density (PSD) approximation in the 4-month group from silent condition. (A) Original PSD (blue curve), 1/f regression (red curve) and wavelet smoothing (green curve). (B) As a result of the fit, the two components were identified. (C) The residual errors when a different number of Gaussian pulses was applied for the fitting. The minimum residual error was obtained in two pulses. Right: PSD approximation in the 12-month group. (D) Original PSD and two approximations. (E) The optimum estimate obtained with three components. (F) The residual error of fitting, which was smallest at three Gaussian pulses.

In the band at 4.0–5.5 Hz, the interaction STIM × ELE-CTRODE × AGE was highly significant ($F_{4,92} = 4.849$, P = 0.004, $\eta_p^2 = 0.174$), while the main effect of AGE was not significant. The

main effect of STIM ($F_{1,23} = 10.494$, P = 0.004, $\eta_p^2 = 0.313$) and the interaction STIM × AGE ($F_{1,23} = 11.546$, P = 0.002, $\eta_p^2 = 0.334$) were also significant. These data suggest that the suppression was



FIG. 4. Top: the model with the two Gaussian pulses (rhythmic components) applied to 4-month-old individuals. (A) Topography of an individual whose maps show a 'bilateral suppression' pattern for the narrow-band rhythm at 4-5 Hz (Gaussian 1, left panel) in the sound condition, compared with the silent condition (a contrast indicated by the red rectangle). (B) Another individual whose maps show a 'right-hemisphere dominant suppression' pattern. From silent to sound, the narrow-band 4-5 Hz rhythm shows suppression mainly in the right temporal electrodes (indicated by the red rectangle). For both individuals, there is no systematic change in topography for the broad-band 2-8 Hz rhythm between silent and sound (right column).

weak in the older group (n.s.) compared with the young group (P < 0.001) in this frequency band. The main effect of ELECTRODE ($F_{4,92} = 33.184$, P < 0.001, $\eta_p^2 = 0.591$, $\varepsilon = 0.760$) and its interactions ELECTRODE × AGE ($F_{4,92} = 3.893$, P = 0.012, $\eta_p^2 = 0.145$) and STIM × ELECTRODE ($F_{4,92} = 4.878$, P = 0.004, $\eta_p^2 = 0.175$) were significant. These are related to the fact that the topographic distribution of the amplitude in this frequency band was different between the groups, mainly due to the higher amplitude in the occipital site in the 12-month-old group.

At 5.5–7.0 Hz, the main effect of AGE was significant ($F_{1,23} = 12.891$, P = 0.002, $\eta_p^2 = 0.359$), because the overall ampli-

tude in the younger group was smaller than in the older group. Furthermore, the interaction STIM × ELECTRODE × AGE was again significant here ($F_{4,92} = 2.986$, P = 0.023, $\eta_p^2 = 0.115$), as was the main effect STIM ($F_{1,23} = 13.902$, P = 0.001, $\eta_p^2 = 0.377$). These data confirm that the suppression in the older group (P < 0.05) was absent in the younger group. The main effect of ELECTRODE ($F_{4,92} = 25.102$, P < 0.001, $\eta_p^2 = 0.522$) was caused by the topography consistently showing a smaller amplitude in the central than the right temporal sites across the two groups. However, the significant interaction ELECTRODE × AGE ($F_{4,92} = 3.568$, P = 0.009, $\eta_p^2 = 0.134$) was due to a larger amplitude not only in the right but also in the left temporal compared with the central sites only in the older group (P < 0.001).

For the higher frequency bands (7.0–8.5 and 8.5–10.0 Hz), no interactions with STIM were significant. A significant AGE effect was observed (7.0–8.5 Hz: $F_{1,23} = 43.186$, P < 0.001, $\eta_p^2 = 0.652$; 8.5–10 Hz: $F_{1,23} = 11.803$, P = 0.002, $\eta_p^2 = 0.339$), due to a smaller amplitude in the younger group. The main effect of ELECTRODE (7.0–8.5 Hz: $F_{4,92} = 4.721$, P = 0.015, $\eta_p^2 = 0.170$; 8.5–10 Hz: $F_{4,92} = 38.485$, P < 0.001, $\eta_p^2 = 0.626$) was produced by a larger amplitude in the bilateral temporal sites than in the central and frontal sites. The interaction ELECTRODE × AGE was significant in both bands (7.0–8.5 Hz: $F_{4,92} = 12.669$, P < 0.001, $\eta_p^2 = 0.355$; 8.5–10 Hz: $F_{4,92} = 7.819$, P < 0.001, $\eta_p^2 = 0.254$). This reflects that the topography in both bands shows an age-related difference with the 12-month-old group showing a larger amplitude in the central than the frontal sites.

Group-based model of EEG rhythmic components

We used Gaussian fit modeling to examine how many rhythmic peak components could account for the PSD obtained across the analysed frequency range (1-10 Hz) in both age groups. Figure 3 illustrates that the lowest error was obtained using a model of two Gaussian pulses in the 4-month-old group, and three for the 12-month-old group. As shown in Fig. 3B, the 4-month-old group had two overlapping rhythmic components. The first occupies a broad frequency range around 2-8 Hz (as indicated by the green curve) and the second (indicated by the red curve) occupies a narrow range around 4-5 Hz. This means that even though the spatial information is not taken into account, the amplitude characteristics of rhythmic activity are clearly separate for these two components, suggesting their discrete generation mechanisms. On the other hand, the model obtained for the 12-month-old data is more complex. As shown in Fig. 3E, there are three components, two broad-band rhythms covering a lower frequency range (3-6 Hz) and a higher frequency range (6-9 Hz), and a narrow-band rhythm at 6-7 Hz.

Individual EEG rhythmic components

Based on the group-based models obtained as above, we analysed individual PSDs to extract the amplitude of each Gaussian component in the silent and sound conditions separately at each electrode site. The topographic maps constructed were used to examine whether these rhythmic components (i) were spatially distinct, (ii) were functionally distinct in response to the sound stimulation and (iii) consistently existed in individual infants, or, alternatively, the combination of components varied across individuals.

As explained above, in the 4-month-old group, a narrow- and a broad-band Gaussian pulse were identified in the group model. The



FIG. 5. Top: the model with the three rhythmic components applied for 12 month-old individuals. (A) Topography of an individual whose maps show a 'bilateral suppression' pattern for the narrow-band rhythm at 6-7 Hz (Gaussian 2) in the sound condition compared with the silent condition (A contrast indicated by the magenta rectangle). (B) Another individual whose maps show a partial suppression in the broad-band 3-6 Hz rhythm (Gaussian 1) in addition to the primary suppression in the narrow-band 6-7 Hz rhythm (Gaussian 2) at the temporofrontal electrodes (indicated by the red and magenta rectangles, respectively). Note that the topographies of the two rhythms in the sound condition were not similar to each other.

topographic maps of the two amplitude parameters varied somewhat across individuals, but for most individuals the suppression by sound was restricted to the narrow-band pulse. The topographic maps associated with the narrow-band rhythm in individuals can be visually classified into three different categories. The first shows 'bilateral suppression'. This group consists of seven subjects out of 12, and the topographic maps of a representative subject for this group is shown in Fig. 4A. As shown in these maps, in the silent condition, the temporal electrodes bilaterally contribute selectively to the narrow-band rhythm at 4-5 Hz, which is suppressed by the stimulating sound. The second group consists of two subjects who show only 'right-hemisphere suppression'. The topographic maps of a representative subject for this group are shown in Fig. 4B. In this subject, the power contribution to the narrow-band rhythm is present bilaterally in the silent condition but only in the right hemisphere is its power suppressed, whereas, in

the left hemisphere, the topography is slightly more parietal than in the silent condition. In both subgroups, the two rhythms were well separated in topography in that the narrow-band rhythm shows bilateral power concentration, whereas the broad-band rhythm shows more midline-centered power (Fig. 4A and B). The third category includes two subjects who show bilateral suppression of the amplitude associated with the broad-band rhythm and an increase in the amplitude associated with the narrow-band rhythm. The remaining subject showed no systematic topographical features in either component.

In the 12-month-old group, the model identified three Gaussian pulses, two broad-band and one narrow-band (Fig. 5). Although interindividual variance appears to be larger than in the 4-month-old group, all but two subjects out of 13 showed suppression of the amplitude associated with the narrow-band rhythm at 6–7 Hz. A representative subject who shows suppression at this component only (bilateral: 3, right: 2) is illustrated in Fig. 5A. Three subjects showed an additional suppression in the slower rhythm at 3-6 Hz (one of them is depicted in Fig. 5B). Another three showed an additional suppression in the broad-band rhythm at 6-9 Hz. Only one subject showed an overall increase in all the components, and in the remaining subject, there was no systematic change visible in any of the components.

To summarize, this analysis revealed that most individuals at 4 months show sound-induced suppression only for the narrow-band rhythm at 4–5 Hz. In contrast, at 12 months the majority are consistent in showing suppression for the narrow-band rhythm at 6–7 Hz while different infants may be at slightly different developmental stages, as indicated by the presence of suppression of spectrally adjacent components in some cases. Across the two age groups, the right hemisphere appears to be more robustly associated with the suppression.

Discussion

We recorded EEG from awake infants during silence and presentation of piano tones and demonstrated that more than three-quarters of the infants in the two age groups showed sound-induced suppression in a specific frequency component of the EEG. The scalp topography of this rhythm showed maxima at temporal electrodes at 4 months and temporofrontal electrodes at 12 months of age. These topographies were clearly different from the occipital or central topography seen within visual attention (Stroganova et al., 1999) and movement (Orekhova et al., 2006), respectively, where similar functional suppression has been observed in similar frequency ranges (4-7 Hz) between 8 and 12 months of age. Thus, these results provide evidence that the infant-equivalent temporal-tau rhythm exists already at 4 and 12 months of age. This implies that by 4 months of age the thalamocortico-cortico-thalamic loop, which is thought to be the origin of neural oscillatory activities observed as EEG rhythms (Steriade et al., 1990), is in operation for the auditory system. Previously, soundinduced suppression of EEG activities in newborns was observed (Ellingson, 1958), suggesting that the tau rhythm may be present even earlier than 4 months. Our next questions will include when the tau rhythm matures beyond 1 year and whether the precursor of this rhythm is present in the fetal stage. Using fetal MEG, auditory evoked responses reflecting brain activity phase-locked to sound stimuli are already visible (Draganova et al., 2005; Huotilainen et al., 2005) as well as spontaneous rhythmic activities (Eswaran et al., 2007; Vairavan et al., 2009). However, functional reactivity of spontaneous rhythmic activity has not yet been examined at this age. We speculate that sound-induced desynchronization is a robust phenomenon that develops early and matures rapidly at early developmental stages.

Our data show a spatio-spectral shift of the tau rhythm from temporal to temporofrontal electrodes between 4 and 12 months of age. Using auditory evoked responses recorded by MEG, additional frontal activity at 12 months has been shown compared with only temporal sources at 6 months (Imada *et al.*, 2006). As well, an additional contribution to the auditory evoked response from outside the auditory cortex was found when infants are older than 2 months of age using MEG (Lutter *et al.*, 2006). This agrees with the broader EEG topography for the auditory evoked response obtained from older infants across 7, 11 and 20 months of age (Rivera-Gaxiola *et al.*, 2007). Although the tau rhythm generators are not necessarily the same as those for auditory evoked responses, in adults the locations of event-related desynchronization are found in cortical areas specific to each modality – tau suppression in the tempoal lobe, visual alpha suppression in the occipital lobe and mu suppression in the

sensorimotor cortex (Tiihonen *et al.*, 1989; Pfurtscheller *et al.*, 1994; Salmelin & Hari, 1994). Therefore, we suggest that the observed frontal shift in auditory tau in our data may indicate a gradual increase in the involvement of frontal areas, or alternatively, non-primary auditory cortex in the anterior temporal lobe in neural networks for auditory processing.

As for the spectral shift, the frequency of the tau rhythm was narrowly tuned to 4-5 Hz at 4 months, and to 6-7 Hz at 12 months. This corresponds to the overall speeding up of rhythmic activity during this age window (Mizuno et al., 1970; Marshall et al., 2002). Although there was a peak at 4-5 Hz in the spectrum at both ages, functional suppression by sound at this frequency only occurred at 4 months of age. At 12 months, although the peak at 4-5 Hz remained, only the additional peak at 6-7 Hz showed suppression by sound. Moreover, the topography of the peak at 4-5 Hz changed with age, and this difference in topography can be seen in one 12-month-old who showed sound suppression at both peaks (Fig. 5B). Therefore, rather than auditory oscillatory development simply consisting of a gradual increase in the frequency of the tau rhythm with age, our data indicate that with development the network of spatially and functionally distinct rhythmic components becomes more complex, and that modality-specific functions can be transferred to newly developed higher order networks during the first year after birth.

Interestingly, we observed that a number of rhythmic components other than tau increased between 4 and 12 months of age. These broad-band rhythms, which overlapped in frequency with the tau rhythm, did not react to sound presentation. The topography of these components was widely spread across the scalp, with a slight concentration at the midline (e.g. Fig. 4 and 5). This is in sharp contrast to the tau rhythm, which was spectrally confined to a narrow band, and was spatially confined in bilateral temporal or temporo-frontal electrodes. Suppression of the occipital alpha rhythm and the central mu rhythm in infants also appears to be confined to relatively narrow frequency ranges (Stroganova *et al.*, 1999; Orekhova *et al.*, 2006). We suggest that functionally distinct oscillatory networks are in place in similar frequency ranges in parallel, while the local network involving sensory-specific thalamo-cortical neurons is sharply tuned at a certain frequency.

Because we tested infants only in sound and silent conditions, and both involved visual attentive states without movements, we do not know how the occipital alpha and central mu rhythms are related to the observed tau rhythm. Our data do show that spontaneous EEG activity at the same frequency as the tau rhythm was present in other regions, particularly in the occipital electrodes in both groups, but this activity was present both during sound and silent conditions and therefore was not related to sound suppression (Fig. 2B). Previous research found that during visual attention the central mu increased coincident with the occipital alpha suppression, while during movements the occipital alpha increased coincident with the central mu suppression (Pfurtscheller, 1992). This suggests that localized sensory-specific alpha-band rhythms interact with each other at a global scale according to task demands. Future research needs to examine whether these rhythms can be simultaneously identified using multi-modal task conditions and, if so, how they overlap and segregate spectrally and spatially at different stages of maturation. Stroganova et al. (1999) found that the frequency of mu rhythm was affected by both extra- and intra-uterine ages, while the frequency of the occipital alpha rhythm was affected only by the former. They also found that the mu rhythm has a slightly higher frequency compared with the alpha rhythm (7.42 and 6.78 Hz, respectively, at 12 months of age), and speculate that this represents a more advanced maturational stage of the sensorimotor compared with visual functional network, as the former receives sensory inputs earlier

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in utero compared with the latter. In our data, the peak of the tau rhythm occurred at around 6.5 Hz, slightly lower than the two peaks in the Stroganova report. It would be interesting to see whether the auditory system, which receives a wide range of inputs from the environment passively during the last prenatal trimester, shows a somewhat different developmental trajectory from either the visual alpha or the mu rhythms.

Evidence of the tau rhythm was originally found in recordings using depth electrodes over a wide area of the temporal lobe including the medial temporal lobe, while evidence in scalp-EEG recordings was noted to be inconsistent and weak (Niedermeyer, 1997). In contrast, MEG was successful in localizing the tau rhythm in the auditory cortex in adults (Tiihonen et al., 1991; Lehtela et al., 1997). Given the larger amplitude of spontaneous EEG in infants than in adults, together with our noise reduction approaches, it is not particularly surprising that we were successful in identifying the tau rhythm. Furthermore, the initial observation of Niedermeyer that the tau rhythm is spread across the temporal lobe is in line with current theories of the generation of neuronal oscillation, namely that it is mediated by the interaction between inhibitory and excitatory connections between neurons in thalamo-cortical loops rather than reflecting only spontaneous oscillation in a particular group of neurons (Steriade et al., 1990). Thus, it is reasonable to assume that the tau rhythm is generated in a collective neural network that involves many different cortical sites in adults. Consequently, we speculate that our observed spatial shift with age reflects gradual expansion of neural networks contributing to the tau rhythm.

In conclusion, we observed that the auditory-specific tau rhythm exists in infancy as early as 4 months, and develops in its spatial and spectral configuration with increasing age independently of other rhythmic components. We propose that maturation of distinct rhythmic components occurs in parallel, and that oscillatory networks reorganize themselves such that specific functions are transferred to newly developed higher order networks by 12 months, when culture-specific auditory perceptual learning is taking place. This suggests that bridging auditory and cognitive functions such as access to long-term memory is probably accompanied by development of hierarchical neural oscillatory mechanisms.

Acknowledgements

We thank Elaine Whiskin for assistance in recruiting and testing the infants. This research was supported by grants to L.J.T. from the Natural Sciences and Engineering Research Council of Canada and the Canadian Institutes of Health Research.

Abbreviations

EEG, electroencephalogram; MEG, magnetoencephalogram; PSD, power spectral density.

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