Modelling Large-scale Brain Dynamics of Healthy Infants

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https://github.com/daniel-lyon/Infant-Brain-Modelling
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Abstract. Measuring empirical electroencephalograms (EEG) of infants is particularly challenging due to limited attention spans and reduced behavioural control. As a result, empirical EEG data becomes increasingly scarce as age decreases. As infants mature into childhood, brain activity undergoes rapid developmental changes, but the underlying neurophysiological aspects are poorly understood. Here, a model of large-scale electrical brain activity focusing on infants and young children is investigated. Expanding upon previous research, physiological parameters were extrapolated backwards through childhood from 10 years of age down to birth and preterm periods. Through the extrapolation process, it was found that the intrathalamic loop left the realm of physiological plausibility. A slower maturation of this parameter is theorised and ensuing thalamocortical dynamics are analysed. Modelled absolute and relative delta and theta frequency power decreased, absolute and relative alpha increased, and relative beta increased. There is good agreement in empirical literature for delta, theta, and alpha frequencies, but the beta frequency literature was inconclusive. This work has extended previous research to modelling infant brain activity, laying groundwork for future study on how abnormal brain activity arises.

Keywords: EEG · Power Spectrum · Infant · Brain Activity · Model

1 Introduction

Electroencephalograms (EEGs) have been in use for almost a hundred years now to record electrical signals from a patient's brain activity. Indeed, the first recorded EEG spectra was taken by Hans Berger in 1924 (Berger, 1929). Since then, electroencephalography has matured into one of the most popular methodologies to measure brain activity today. Bulk electrical activity is recorded from the scalp as large populations of interconnected neurons pulse information around the brain. Neural field theory as a mean-field theory describes the interactions and origin of this activity very well. Through spectral analysis, the summation of different frequencies can be used to determine the corresponding spectral power.

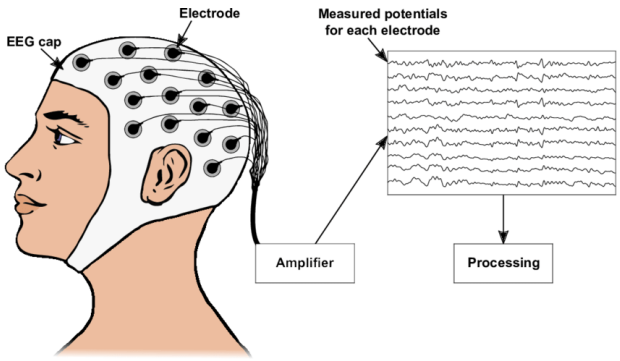


Fig. 1: Diagram of EEG apparatus (Nagel, 2019)

Using convention as from van Albada et al. (2010), the spectrum is split into four key frequency bands defined as the delta band $(1 - 2.5Hz)$, theta band $(2.5 - 7.5Hz)$, alpha band $(7.5 - 12.5Hz)$, and beta band $(12.5 - 25Hz)$. The spectral power in these frequency bands will be critically analysed in section 3 to determine model accuracy.

In the past, models of EEG power spectrum's have been produced, but these mainly pertain to seizure detection (Celka and Colditz, 2002b,a; Hassanpour et al., 2004; Rankine et al., 2006). Some physiology-based modelling research has been conducted on the link between neurophysiology and EEG (van Albada et al., 2010; Robinson et al., 2002; Rowe et al., 2004) in large-scale brain dynamics, which as it currently stands, is poorly understood, but has been successfully modelled regardless. Models of large-scale brain activity focus on deriving key neurophysiological parameter values that are consistent with empirical results. These parameters are then fitted to large-scale functions of corticothalamic dynamics to reproduce empirical EEG results artificially. However, these models do not include infants and mostly focus on adults and older children. In infants and children, The end goal is to improve understanding of the origin

of brain activity, specifically developing infant brains, by extending the work of van Albada et al. (2010) to infants.

EEG has many applications, the most important of which, is diagnosing many prevalent brain disorders such as epilepsy, Alzheimer's, seizures, autism, anxiety, and many more (Soufineyestani et al., 2020). EEG is a noninvasive method that records a patient's brain activity with electrodes, usually attached to a cap (Figure 1). Besides EEG, there are many other technologies in use such as Near Infrared Spectroscopy (NIRS), Functional Magnetic Resonance Imaging (fMRI), Magnetoencephalograpy (MEG), and Electrocorticography (ECoG) to name a few. These are all methods of empirically measuring brain activity.

Apart from being noninvasive, EEG holds many advantages over the other techniques. For example, EEG has high temporal resolution and is a safe & easy method to use whereas fMRI is very costly and slow; NIRS has low temporal resolution and is less performative; and MEG is also very expensive and physically large (Lakshmi et al., 2014). A key paradigm of data acquisition when recording EEGs is measuring the brain's response to sensory, cognitive, and motor stimuli (Sur and Sinha, 2009). This is known as an Event-Related Brain Potential (ERP) and is the reason high temporal resolution is desirable.

Significant challenges in data acquisition begin to arise when recording infant EEG spectra - challenges that do not exist for adults. As reported in Hervé et al. (2022) on the difficulties faced in infant EEG studies, the attention span of one year old infants is limited to a maximum of only a couple minutes (Atkinson and Braddick, 2012), but the average is less than one and a half minutes (Tamis-LeMonda and Bornstein, 1990). The limited attention span of infants and children along with difficulties in understanding, processing, acting, and responding in a controlled manor makes it extremely difficult to accurately record empirical EEG measurements.

Recently, steps have been taken that attempt to solve or improve limitations of some of these challenges (Noreika et al., 2020). By using time-frequency transformation during resting states, for example, can be a sound alternative (Hervé et al., 2022). Moreover, there are many more challenges faced when attempting to record an infant's EEG than there are solutions. Unrelated to the cognitive ability of an infant is it's movement: even small movements such as blinking will introduce undesirable artifacts into the data that need to be accounted for. EEG can be recorded in eyes-open (EO) and eyes-closed (EC) conditions. These conditions are exactly as they sound; there are marked differences in measured brain activity when the patients eyes are open verse when closed (Teplan et al., 2002; Barry et al., 2009). These artifacts can and will appear commonly due to the nature of such a young patient, leading to large data attrition.

With these issues mentioned and knowing a great deal more exist, it is easy to understand why empirical infant EEG data is scarce when compared to the adult counterpart: it is extremely difficult to take accurate EEG measurements for infants empirically. This research endeavours to extend the research conducted by van Albada et al. (2010) to infants and younger children to better understand the origin of developing brain activity. In the following sections, it will be shown how the model was formulated (2), followed by a discussion of results (3) in terms of the literature and available empirical data.

2 Materials & Methods

The developed model has 13 physiological input parameters (Table 1), of which only 9 change with age. These parameters are: α (decay rate of the cell-body potential), β (rise rate of cell-body potential), γ_e (range of pyramidal axons), t_0 (corticothalamic axonal latency), G_{ee} (excitatory cortical gain), G_{ei} (inhibitory cortical gain), G_{ese} (gain for indirect corticothalamic loop), G_{esre} (gain for indirect corticothalamic loop passing through the thalamic reticular nucleus), and G_{srs} (gain for intrathalamic loop).

By taking these parameters as a function of age, this allows for the extension of work done by Robinson et al. (2002) to be extrapolated to younger ages using van Albada et al. (2010) data to plot the signal spectrum for each corresponding age. The variables G_{es} & G_{sn} only appear as a multiplicative factor that scale the spectrum's power without changing shape. The function $\phi_n(\omega)$ is a Fourier transform of the noise amplitude (that is assumed to be white noise) which has a constant, flat power spectrum independent of frequency. As a result, G_{es}, G_{sn}, and $\phi_n(\omega)$ all set equal to 1. Finally, β is simply a multiple of α which was set to $\beta = 4\alpha$.

The EEG power spectrum is then plotted by taking the square of the modulus of equation 1. To find the aforementioned physiological input parameters as a function of age, data from van Albada et al. (2010) who finds these equations is used. This allows for the EEG power spectrum to be plotted for each age which will then be critically analysed in section 3.

Equation 2 is a complex equation with real and imaginary components. As such, the only time Equation 2 is equal to 0 is when the absolute value of the complex number is 0. If Equation 2 approaches

0, Equation 1 can result in a divide by zero error, causing the spectrum to become unstable. Stability

Table 1: Modelled functions and parameter values used to plot equation 1 from ages -0.25 to 10.

Variable	Value/Function	Unit
α	-12[age]$+180$	s^{-1}
γ_e	1.4[age]+43	s^{-1}
t_0	$(-4$[age]$+104)/1000$	s
G_{ee}	-0.14[age]$+5.4$	-
G_{ei}	0.17[age]-10.1	-
G_{ese}	0.01[age]$+10.9$	-
G_{esre}	0.2[age]-9	-
G_{srs}	-0.005[age]-0.21	-
G_{es}	1	-
G_{sn}	1	-
$\phi_n(\omega)$	1	-
$\phi_e(\omega)$	Equation 1	$\mu V^2 Hz^{-1}$
ω	[0,1,2 ... 649, 650]	Hz
β	4α	s^{-1}

plots of the signal spectrum with real x-axis and imaginary y-axis are taken from Equation 2's complex components. Table 1 shows these functions and variable parameters, along with units.

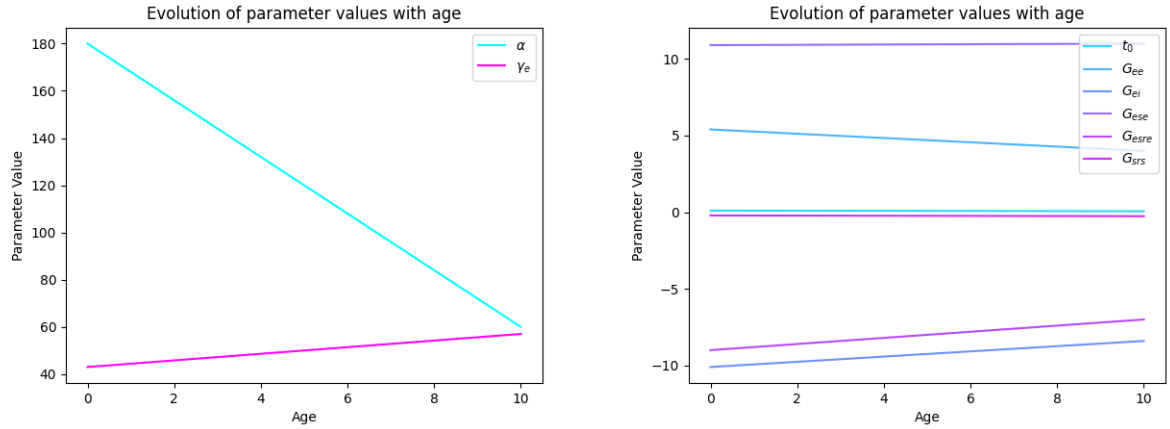
We model the spectral power using the transfer function equations derived by Robinson et al. (2002), given as:

$$\phi_e(\omega) = \frac{G_{es}L}{1-G_{ei}L}\frac{G_{sn}Le^{i\omega t_0/2}}{1-G_{srs}L^2}\frac{\phi_n(\omega)}{q^2r_e^2}, \quad (1)$$

$$q^2r_e^2 = \left(1-\frac{i\omega}{\gamma_e}\right)^2 - \frac{L}{1-G_{ei}L}\left[G_{ee}+\frac{(G_{ese}+G_{esre}L)L}{1-G_{srs}L^2}\right]e^{i\omega t_0}, \quad (2)$$

$$L = \left(1-\frac{i\omega}{\alpha}\right)^{-1}\left(1-\frac{i\omega}{\beta}\right)^{-1}. \quad (3)$$

Using the data in Table 1, equation 1 could then be plotted as a function of angular frequency corresponding to each age. It should be noted that the physiological parameters as a function of age are only valid until about age ≈ 15. After this point, van Albada et al. (2010) has listed more functions from age 15 onwards that are better representing of the brain's physiology at older ages. To plot the equations, the Python programming language was used with matplotlib and numpy libraries. The code of which is



(a) α and γ_e parameter evolution

(b) Evolution of remaining parameters

Fig. 2: Graphical evolution of physiological parameter trends with increasing age. A) shows the α and γ_e together as the magnitude of these two parameters matured more rapidly. B) Shows the other six parameters, whose magnitude does not evolve as fast as A).

publicly available on GitHub (the link to the code repository is on the title page). Another set of empirical data was used to plot an average 18-28 age signal spectrum, given in Table 2. This data, which also comes from van Albada et al. (2010), is all empirical data to create a comparison of the model, albeit it at older ages.

The extrapolation done by van Albada et al. (2010) of the physiological parameter G_{srs} must be different from the true maturation of G_{srs}. This was found by running a sensitivity analysis which found G_{srs} to be the largest contributor. Essentially, G_{srs} changed polarity at young ages, causing Equation 2 to approach 0 - the region of instability. The maturation rate at which this physiological parameter changes is not constant and agrees with early infancy and childhood being a key period of rapid growth. The listed equation for G_{srs} is taken from the older age parameters in van Albada et al. (2010) Table 4 - postulating a slower maturation of G_{srs} in the thalamus.

Table 2: Empirical parameter values for the mean 18-28 age range.

Variable	Value	Unit
α	50	s^{-1}
γ_e	100	s^{-1}
t_0	0.08	ms
G_{ee}	3.2	-
G_{ei}	-7.7	-
G_{ese}	11.3	-
G_{esre}	-5.7	-
G_{srs}	-0.32	-

Variables that appear in Table 1 but are absent in Table 2 have the same values/functions. The data presented in both Tables 1, 2, and 3 is all that is needed to completely reconstruct the results and figures present in section 3. The resulting graphs and data produced by plotting the equations will then be critically analysed and compared to similar figures from the literature.

Table 3 shows the upper and lower bound errors for the functions in Table 1. These functions will be used for the error analysis in section 3. These errors are also taken from van Albada et al. (2010), who suprisingly, does not provide errors for the mean 18-28 range.

Table 3: Upper and Lower bound errors of the modelled functions and parameter values used to plot equation 1 from ages -0.25 to 10.

Variable	Lower Function	Upper Function
α	-16[age]$+180$	-8[age]$+200$
γ_e	[age]$+39$	1.8[age]$+47$
t_0	$(-5$[age]$+100)/1000$	$(-3$[age]$+108)/1000$
G_{ee}	-0.17[age]$+4.9$	-0.11[age]$+5.9$
G_{ei}	0.10[age]-10.8	0.24[age]-9.4
G_{ese}	-0.01[age]$+10.3$	0.03[age]$+11.5$
G_{esre}	0.1[age]-11	0.3[age]-7
G_{srs}	-0.006[age]-0.26	-0.004[age]-0.16

3 Results & Discussion

Using the equations and parameters mentioned in section 2, the signal power spectrum was first plotted (Figure 3) on the y-axis with frequency on the x-axis. The frequency bands are the delta band from 1-2.5 Hz, theta band 2.5-7.5 Hz, alpha band 7.5-12.5 Hz, and beta band 12.5-30 Hz. These frequency bands are from van Albada et al. (2010), but it should be noted that the frequency bands differ slightly from author to author as they're not entirely agreed upon. For example, in Cragg et al. (2011), the delta band is from 1-4 Hz, theta 4-7 Hz, alpha, 8-12 Hz, and beta 12-25 Hz. It's important to mention these differences in frequency range as it could change where key trends are identified with comparisons to previous empirical research.

In the delta range, as the age increases, it appears to show that the wave spectra initially increases before declining. Empirical research agrees that the absolute EEG power in the delta band compared with the rest of the wave decreases with age. The sources also agree that the relative delta power with age also decreases. (Cragg et al., 2011; Somsen et al., 1997; Benninger et al., 1984; Niemarkt et al., 2011). The model replicates the empirical research results well for the absolute power, but does not do well for the relative power. In the model, relative delta power between ages does increase. Although, these studies use participants where the majority are greater than the age of

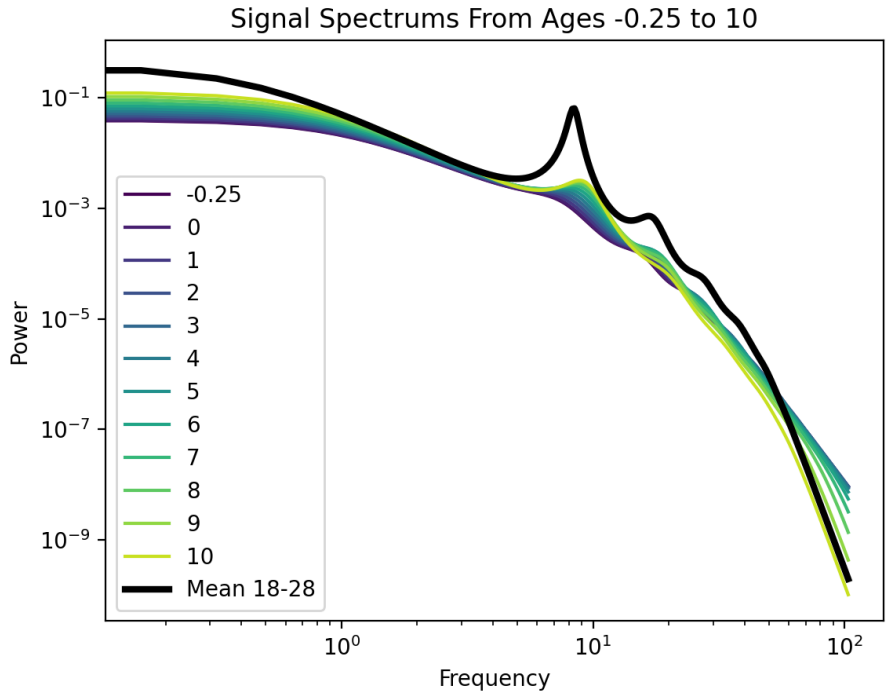


Fig. 3: Modelled log-log plot of signal power spectrums from ages -0.25 (dark blue) to 10 (yellow-olive) with empirical data represented as the mean 18-28 (thick black) line to judge signal progression with age.

6, with the average being about 10-12 years old. This age is higher than what is modelled, which could be the reason the model doesn't agree with these sources, but further empirical research with infants and young children is required here.

In the theta band, the signals actually increase from age 0 until about 7 (Figure 3) before decreasing slightly to our final age of 10. But, the mean 18-28 line shows that after the decrease occurs from 7-10 that it starts to increase again at an age larger than what was plotted. In future research, it would be interesting to find when the spectrum begins to increase again. Empirical research finds that the absolute EEG power in the theta band decreases with age Cragg et al. (2011); Somsen et al. (1997); Benninger et al. (1984); Sankupellay et al. (2011); Niemarkt et al. (2011). All these sources except Niemarkt et al. (2011) agree that the relative theta power also decreases with age. In the model, absolute theta power is seen to decrease, but at younger ages, the relative power is seen to increase until age 7 where it starts to decrease (Figures 3,4). Again, the model performs well showing that absolute theta power does decrease with age, but at first glance, *appears* to fall short on the relative power to older ages.

Interestingly, this is more easily accounted for compared to the delta band: the sources that find the relative theta power decreases are mostly for ages greater than 10, but Niemarkt et al. (2011) is for preterm infants, lending credibility back the model for relative theta power as a slighty decrease in relative theta power was found for ages 7-10. Finding empirical results for the power spectrum on infants is hard work as the EEG data for younger ages becomes increasingly scarce. More empirical infant EEG research is needed to verify the model at the theta band.

At the tail end of the theta band, the power begins to increase into the alpha band. The reason for this tail increase could occur for a couple reasons. Firstly, it could be an increase in the alpha band "dragging" the theta band up with it. Secondly, the differences in alpha band frequency range could be the culprit (Marshall et al., 2002), or finally as explained by Somsen et al. (1997), the absolute power decrease of delta and theta likely produces a relative increase in alpha and beta.

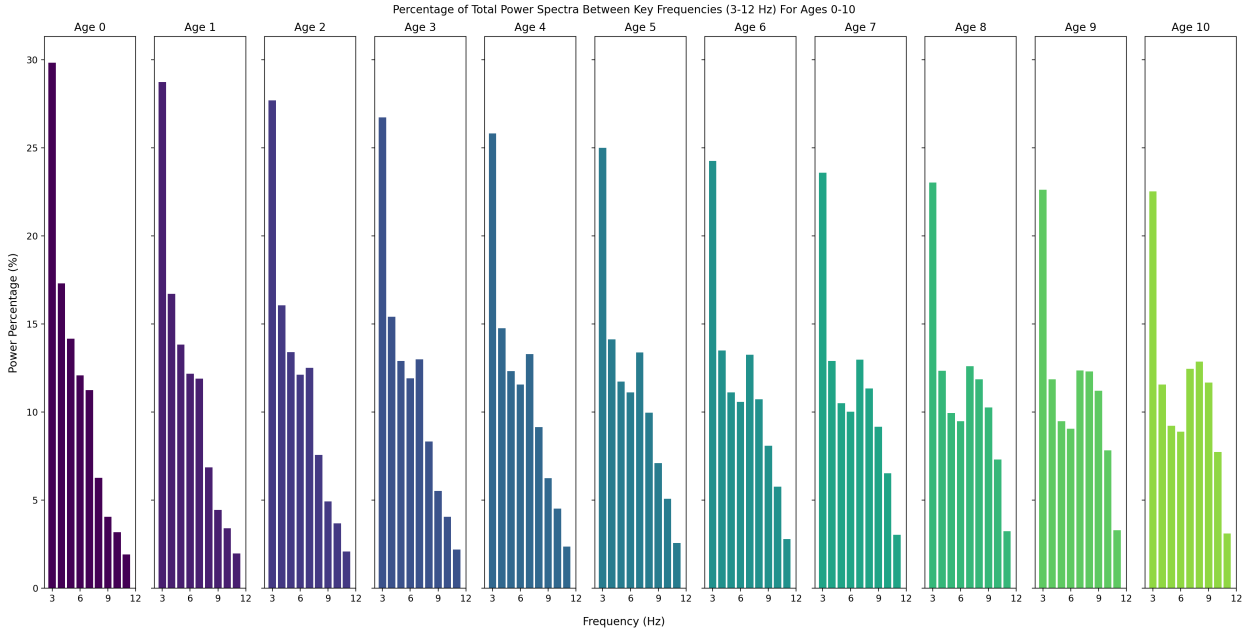


Fig. 4: Modelled absolute theta and alpha power spectrum from ages 0 to 10 not including absolute slow, delta, or beta power. The age -0.25 was dropped as it was too similar to age 0.

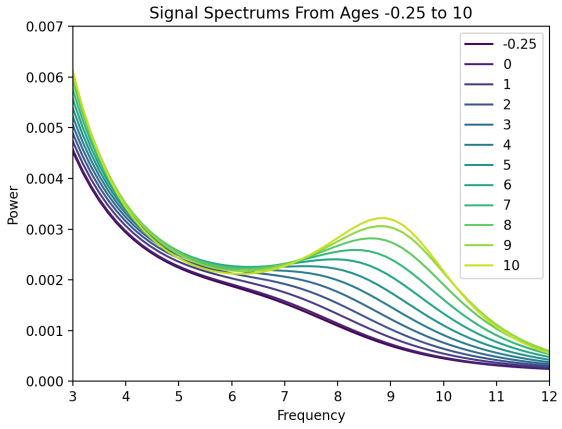


Fig. 5: Zoomed alpha band region and peaks

The alpha peak shows a clear increasing trend in absolute and relative power as seen in Figures 3 and 4. It appears that if ages greater than 10 were plotted, the spectrum could closely approach the mean 18-28 range. Figure 5 shows a zoomed version of figure 3, but focused on the theta and alpha region peak of each age. An identifiable increase in relative alpha frequency is seen. Empirical research agrees that the absolute and relatve alpha power increases with age (Cragg et al., 2011; Marshall et al., 2002; Niemarkt et al., 2011), although Somsen et al. (1997) disagrees that the absolute power increases are significant. But this is likely due to the differences in the chosen alpha band range.

Another key finding from Somsen et al. (1997) is that the peak of the alpha band itself shifts with age. They find that the peak moves away from the theta range into the alpha range for ages 5 to 12. This correlates to a shift of the peak alpha frequency from about 8 to 9 Hz. This is represented in the model as well as the peak alpha frequency does indeed shift from 8-9 Hz. This is very interesting, as the peak alpha frequency of the mean 18-28 line actually shifts backwards to around 8 Hz again. Although, the figures all stop at age 10, so it would be interesting to find when the alpha peak shifts backward again. Future research can delve into this as a possible avenue to explore.

Finally, in the beta range, the signal spectrum increases up until age 6 and then decreases more and more until the final plotted age of 10. This is fascinating because at some point it must then increase, as shown by the mean 18-28 having a significantly greater power. Empirically, some sources find that relative beta power increases with age (Cragg et al., 2011; Niemarkt et al., 2011), but others like Somsen et al. (1997) report that there was was no significant or only a very small change in relative beta power with increasing age. Isler et al. (2022) finds there is a relative decrease in ages <8, which is contrary to the results of the model.

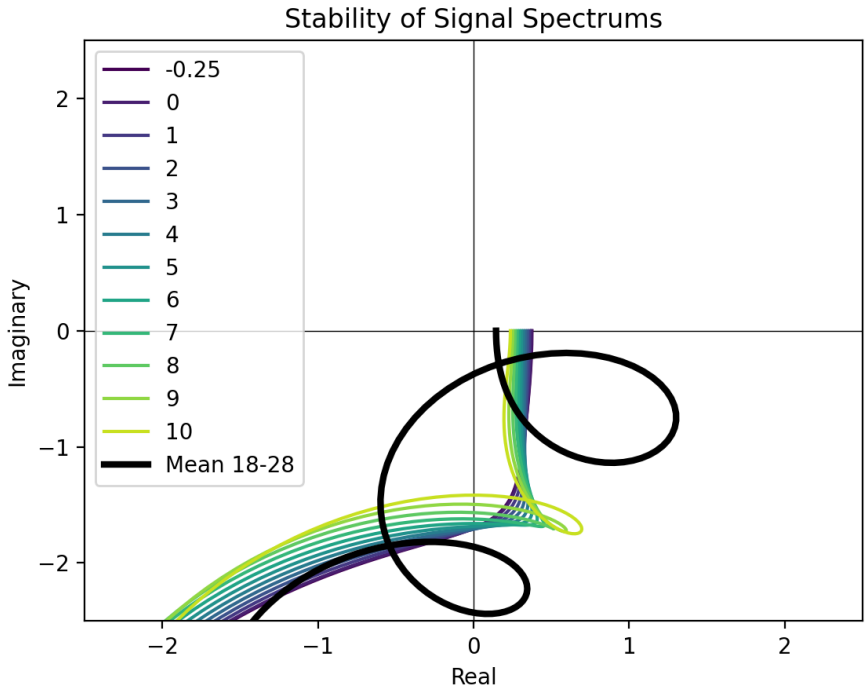


Fig. 6: Stability of the signal spectrum. Starting on the imaginary axis, lines that *start* to the right of the real axis are in the region of stability. If any ages were on the left, they would be unstable.

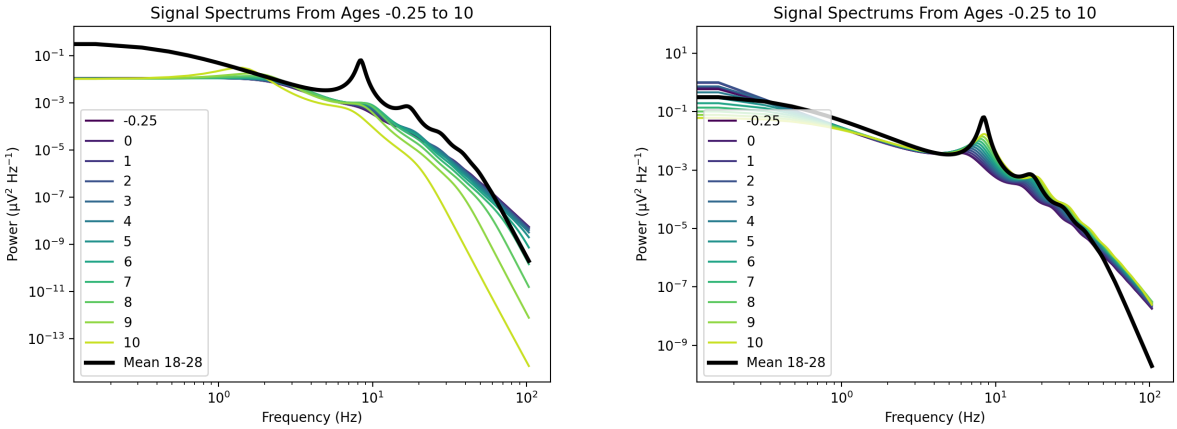
Overall, in the beta band there is no consensus on findings by empirical data. This could be due to a variety of reasons similar to the theta band. The ages reported in Cragg et al. (2011) are for ages greater than 10, but then again Niemarkt et al. (2011) is for infants and they find a similar trend. It could also again be due to the differences in frequency bands, where in this paper van Albada et al. (2010)'s ranges is used, Cragg et al. (2011) uses a slightly different range, and Niemarkt et al. (2011) ages uses another differing range. Despite the differences in beta range, they are only very slight, so this cannot be the entire reason for the discrepancies seen between the model and empirical results.

Another potential reason for the variability in beta band findings is due to the parts of the brain that each source focuses on. As mentioned in section 1, there are many key brain areas to study, and some even concentrate on specific locations of individual electrodes. Cragg et al. (2011) focuses on Cz, Somsen et al. (1997) on a wide range F7, Fz, F8, T3, C3, Cz, C4, 4, T5, P3, Pz, P4, T6, 01 and 02, Benninger et al. (1984) at C3, C4, 01, and 02, Isler et al. (2022) at Cz, Marshall et al. (2002) at F3, F4, F7, F8, C3, C4, P3, P4, 01, and 02, Sankupellay et al. (2011) at C3, C4, A1, A2, 01, 02, and Niemarkt et al. (2011) at Fp1, Fp2, C3, C4, 01, 02, T3, and T4.

Most of these sources have a lot of overlapping electrode locations, but some are also unique and separate from the other sources. This inevitably would have caused some differences to appear in the model as some electrodes have increased "sway" over the results, but it is impossible to know for sure. The model itself is to be interpreted as an average over all brain spheres, electrode locations, health levels, and eyes-closed vs eyes-open conditions. The subjects in all the studies cited in section 3 used healthy patients.

Regarding absolute beta power, there was no data available that could be found. This contributes to a general lack of empirical infant data, especially in the beta band. More empirical research on infant's brain activity is simply needed to make a better justified result of the model's performance in this region. Finding when the beta peak signal begins to increase again after decreasing so much would be an interesting lead to probe in followup research, but it remains to be seen if this is even an a true reflection of empirical analyses.

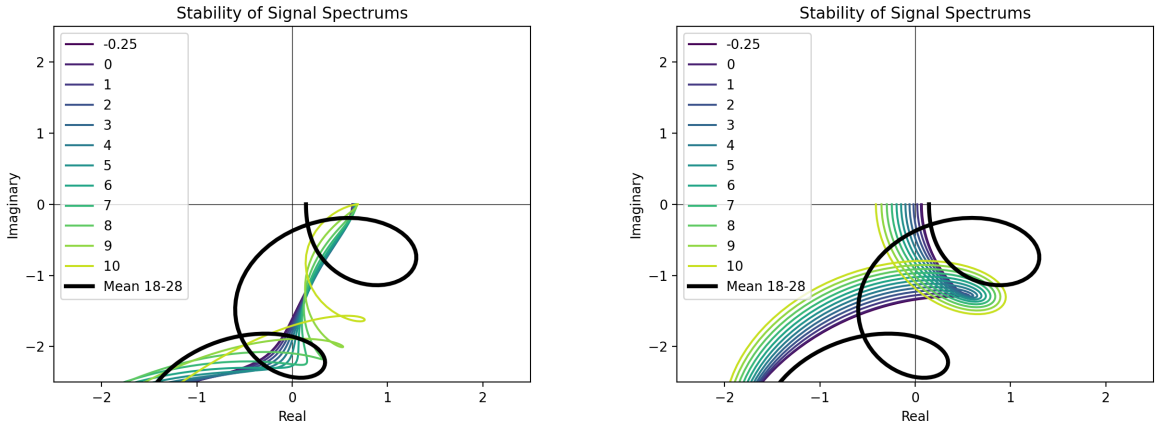
Figure 6 shows Equation 2 plotted in the complex plane to inspect spectrum stability. All age lines are in the stable region, that is, none of the lines cross



(a) Lower signal spectrum error

(b) Upper signal spectrum error

Fig. 7: Using the table of errors (table 3), Figure 3 was re-plotted using the new functions. A) shows the lower bound of the signal and B) represents the limit of the upper bound on the error.



(a) Lower error bound stability

(b) Upper error bound stability

Fig. 8: Using table 3, Figure 6 upper and lower bound error values are graphically represented. A) shows a stable set of points with no logicial or natural evolution. B) shows an unstable dataset but with a natural progression with age.

the origin where the absolute magnitude of the complex number would be equal to 0. For example, if age 3 was stable, 4 unstable, and 5 stable again, then at some point between ages 3 and 4 the line would need to cross the origin. If that happens, then the equation for $q^2 r_e^2$ becomes very closely equal to 0, and therefore $\phi_e(\omega)$ results in extreme and unnatural physiological changes. By ensuring the stability, Figure 6 shows a natural progression as age increases towards the mean 18-28 line. A point for future research could look into how ages 10-18 approach the mean line.

Originally, as noted in section 2, van Albada et al. (2010)'s equation for G_{srs} had to be changed from the original $-0.22[age] + 1.5$ at younger ages to $-0.005[age] - 0.21$ for older ages because the younger equation changed sign and caused the mentioned error. G_{srs} was the only modelled physiological parameter to change sign. This is a notable point to raise because it means that maturation trend reported by van Albada et al. (2010) is different to the true evolution. Through the analysis of the model, improvements were found on older research, and could be expanded upon futher in future.

Figure 7 shows the first set of error analyses performed. Using the upper and lower equations from table 3, a graphical representation of the upper and

lower error bounds of van Albada et al. (2010)'s physiological equations was produced. No discernible or natural progression towards the mean 18-28 empirical data is observed. The upper bound of the signal spectrum error on the other hand, shows a clear, natural progression of the signal with age towards the mean.

Figure 8 shows a graphical representation of the upper and lower error bounds seen on Figure 6. Again, the lower error bound shows no discernible or natural trend towards the mean 18, but notably, all the ages are stable. In stark contrast to the lower bound, the upper bound error shows a very clear trend with age, but unfortunately form age 2 onwards the signals are all increasingly unstable. Certainly, the lower and upper bound errors do not perform as well as the derived parameters - they represent parameters that leave the realm of plausible physiological values. Nevertheless, these plots are useful to define the region of possible parameter values.

4 Conclusion

To conclude, the proposed model and physiological parameters as a function of aged used to model the brain activity of infants appears to work quite well. The delta wave $(1 - 2.5Hz)$ progression is backed up by empirical research for absolute delta power, but the model shows an increase in relative power which is not backed up by those same sources.

Theta $(2.5-7.5Hz)$ shows a relative increase until age 7 where it begins to drop again until the final age of 10. This trends negatively compared to the mean 18-28, and so after age 10, it must begin to rise again at some unknown age. This is backed up by all sources which agree that; absolute theta power decreases, relative theta increases at younger ages, and relative theta decreases at older ages. This is all present in the model, but more empirical sources for younger ages are needed.

The alpha band region $(7.5-12.5)$ presents a clear, positive trend towards the mean. Empirical research finds that absolute and relative alpha wave power increases with age and agrees nicely with the model. Another key aspect identified in empirical research is the shift of the alpha peak itself from $8-9Hz$ which is also present in the model. Although, the mean 18-28 appears to shift back to $8Hz$.

Unfortunately, empirical data in the beta band $(12.5 - 30Hz)$ is inconclusive, and so it is hard to justify the performance of the model in this region. The model shows a relative power increase up until age 6, where it drops until the last plotted age of 10. The relative power should then need to increase at older ages to approach the mean 18-28 range. Empirical sources here disagree, with some finding that relative beta power does increase and some that find no significant changes. One source even finds a relative decrease at younger ages which is the opposite of what the model predicts. Finally, no data was found regarding absolute beta power, which leaves some questions unanswered.

Finally, the stability of the modelled signal spectrum's are all stable, showing a correlated trend towards the mean. The model works very well up until the beta region where more research is needed. Future research can focus on the beta wave and a few small points of interest on plotting of older ages to see how, or if, they approach the mean. Additionally, future research could try to extend the model fitting of van Albada et al. (2010) to infant and child EEG data as was done for Robinson et al. (2002) in this paper. Doing so could determine if the results seen here are realistically possible in real life circumstances.

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