

# Comparison of Linear and Quadratic Modelling of the Human Auditory System Using a System Identification Approach

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**Abstract**— Engineering analysis has been utilized with great success over the past few decades to characterize physiological systems. For example, system identification approaches have been developed to describe the linear and nonlinear properties of such systems in a very general way, allowing for new insights to be made into physiological function. Recent work has seen the application of these techniques to the analysis of human sensory systems using the electroencephalogram (EEG). This includes studies that estimate and use linear impulse response estimates of visual function and, more recently, auditory function. These responses are known as the VESPA and AESPA respectively (Visual/Auditory Evoked Spread Spectrum Analysis). While a nonlinear extension of the VESPA has been previously described, no examination of any such extension has yet been put forward for the AESPA. This paper investigates such an extension and quantifies the relative contribution of linear and quadratic processes to the EEG in response to novel auditory stimuli. While the ability to predict novel EEG is poor, it is significant ( $p < 0.01$ ) for greater than 80% of the tests conducted. Further, the prediction ability of the quadratic model ( $r=0.0418$ ) was slightly greater than that of the linear model ( $r=0.0361$ ).

**Keywords** – EEG, system identification, neural.

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## I INTRODUCTION

A considerable amount of research has been done on the modelling of nonlinear time-invariant systems, most of which has been based on the general mathematical foundation of the Volterra-Wiener approach. The Volterra series was first studied by Vito Volterra around 1880 as a generalization of the Taylor series of a function and was used by Wiener (1958) to model the input-output relationship of a nonlinear system [1].

Several successful examples of the use of nonlinear modelling have been reported across different physiological domains. One of the first successful applications on visual neuronal pathways was performed on the catfish retina [2]. In this case, the input signal was a band-limited Gaussian white noise (GWN) stimulus of current injected into the horizontal cell layer and the recorded output signal was the sequence of action potentials generated by a ganglion cell. A later study reported a model of the photoreceptor of the eye using actual quasi-white light stimuli [3].

The Volterra-Wiener approach has also been applied to EEG and in particular the visual evoked potential [4]. In that study, the visual system was

stimulated by a light source, the luminance of which was modulated using a band-limited GWN signal of 25 s duration. After averaging over a number of presentations of this stimulus, the first and second order Wiener kernels were calculated and used to estimate the visual evoked potential to a standard flash stimulus. Furthermore, the amount of variance in the EEG that is accounted for by the model during stimulation by the GWN signal was calculated both when just the linear component was used as well as when both first and second orders were used. The study reported that a 1<sup>st</sup> order impulse response accounted for 16.5% of the variance of a 20 s long interval of EEG, but that extension to a 2<sup>nd</sup> order model resulted in a large improvement to 70% of explained variance [4]. While this improvement suggested that the addition of a 2<sup>nd</sup> order to the model was extremely important in capturing information processing as indexed by the EEG, it must be noted that the data used in the model consisted of only 10 s of the recorded EEG which was averaged over as many as 60 artifact-free trials. The short duration of these artifact free trials combined with the fact that so many of them were included in the average would result in a huge reduction in signal variance. In addition, it is

important to note that the data used to generate the model was the same data that was subsequently tested by the model. As such, it is possible that the huge improvement in model performance with the 2<sup>nd</sup> order extension is a result of overfitting.

In an effort to test this possibility in more detail, we recently examined the advantage of using a quadratic model over a linear model using the VESPA method (Visual Evoked Spread Spectrum Analysis)[5]. This method utilizes the least squares algorithm to fit a response model of the how the human visual system – as measured by EEG – responds to contrast changes. This is typically carried out by stochastically modulating the luminance or contrast of a checkerboard stimulus on a computer monitor while recording EEG [5], which has a number of advantages over standard methods which typically assess the visual system by averaging EEG responses over repeated presentation of simple, discrete events. Because the VESPA in its linear form had been shown to have useful application across a range of fields, including cognitive neuroscience [6], clinical research [7] and fundamental research on vision [8], it was important to assess the ability of the linear method to accurately model the visual system and to assess the potential advantages of incorporating second order modelling terms. We wished to avoid possible overfitting by refraining from averaging EEG data across multiple presentations and by only testing the model on novel data that were not used to fit the model. The results of that study suggested that both the linear and quadratic VESPA models could predict novel EEG with only low accuracy, but in a highly significant way [9]. Furthermore, no statistical difference in the ability to predict was found between the linear and quadratic models.

More recently, we have introduced a similar modelling approach to auditory processing, known as the AESPA (Auditory Evoked Spread Spectrum Analysis)[10]. This method, which despite producing responses with relatively poor signal-to-noise ratios compared with the more standard averaged auditory evoked response paradigms, has a number of advantages over those standard methods. Utilizing these advantages the linear AESPA has yielded important findings on the neural processing of natural speech [11] and on the cognitive neuroscience of attention [12, 13]. As with the VESPA, it is important to a fuller understanding of these attention results to determine the ability of a linear AESPA to model auditory processing and to assess the potential advantages of modelling higher orders. As such, we aim in this paper to quantify how well the linear AESPA impulse response can predict the EEG in response to an unknown stimulus and by how much, if at all, our system identification approach is improved through the inclusion of second order terms in the AESPA analysis.

## II METHODS

### *a) Subjects*

Participating in the study were 11 subjects (one female; aged 22–35 yr), all of whom had normal hearing. The experiment was undertaken in accordance with the Declaration of Helsinki. The Ethics Committee of St. Vincent’s University Hospital in Dublin approved the experimental procedures and each subject provided written informed consent. These data were presented previously using only a linear analysis [10].

### *b) Stimuli*

A Gaussian broadband noise (BBN) waveform, with energy limited to a bandwidth of 0–22.05 kHz was used as a carrier signal. The amplitude of this carrier stimulus was modulated using Gaussian noise signals with uniform power in the range 0–30 Hz, i.e., at a rate of 60 modulations/s. This rate was chosen based on the fact that EEG power below 30 Hz is typically very low. Modulating signals with the desired statistical properties were precomputed and stored. Taking into account the logarithmic nature of auditory stimulus intensity perception, the values of these modulating signals ( $x$ ) were then mapped to the amplitude of the audio stimulus  $x'$ , using the following exponential relationship

$$x' = 10^{2x} \quad (1)$$

and normalized to between 0 and 1. It was expected that this would result in a more linear perception of audio intensity modulation. The modulating noise signal was then interpolated to give a smooth transition from one modulation amplitude to the next and stored. An example of the amplitude-modulated BBN and signal can be seen at the top of Fig. 1.

The audio stimuli were generated with a SoundBlaster Extigy soundcard and presented to subjects using high-fidelity Sennheiser HD650 headphones.

### *c) Experimental Procedure*

All subjects underwent ten presentations of the amplitude-modulated BBN stimulus, each of 120-s duration. Subjects were instructed to keep their eyes open and to keep eye movements and blinks to a minimum for the duration of each run. Subjects were also instructed to limit all other types of motor activity during each run.

### *d) EEG acquisition*

EEG data were recorded from 130 electrode positions, filtered over the range 0–134 Hz, and digitized at the rate of 512 Hz using a BioSemi Active Two system. Synchronization between the audio stimuli and the recorded EEG data was ensured by including the signal on the parallel port of the presentation computer, indicating the onset and offset of the stimuli, among the recorded signals.

EEG data were digitally filtered with a high-pass filter, where the passband was  $> 2$  Hz and with a  $-60$  dB response at 1 Hz and a low-pass filter with passband  $< 35$  Hz and a  $-50$  dB response at 45 Hz. The data at each channel were rereferenced to the average of the two mastoids.

*e) Signal Processing*

The estimation of the AESPA is based on the assumption that the output EEG,  $y(t)$ , consists of a convolution of the audio amplitude modulation signal,  $x(t)$ , with an unknown impulse response  $w(\tau)$ , plus noise (see Figure 2), i.e.,

$$y(t) = w(\tau) * x(t) + \text{noise}. \quad (2)$$

Given the known audio amplitude modulation signal and the measured EEG, we obtain  $w(\tau)$ , i.e., the AESPA, by performing linear least squares estimation. This can be carried out analytically using the following equation,

$$w = \langle x_t x_t^T \rangle^{-1} \langle x_t y_t \rangle, \quad (3)$$

where  $x_t$  is a column vector of input stimulus contrast values in a certain window of time around  $t$ ,  $y_t$  is the EEG value at time  $t$  and  $\langle \rangle$  denotes mean over  $t$ . This involves inversion of the input signal's autocorrelation matrix  $\langle x_t x_t^T \rangle$  (see appendix A – [5] for details). By adding a regularization matrix with terms along or adjacent to the diagonal of the input signal's autocorrelation matrix before inversion one can increase the bias of the estimate, but reduce overall estimation error by greatly reducing the variance (see appendix B – [5]). As in that study, the regularization parameter,  $\lambda$ , used in the current study was empirically chosen to be  $4.4 \times 10^{-3}$ . This resulted in good reduction of off-sample error without affecting the height or the latency of the response peaks.

It should be noted that for the estimation step, the values of  $x(t)$ , were assumed to be constant across each 16.67 ms modulation period. Furthermore the initial modulation values, i.e., the linear values obtained prior to the exponential mapping, were used. This seemed reasonable under the assumption that the exponential mapping would actually result in a more linear intensity perception.

The AESPA was estimated using a sliding window from 200 ms pre-stimulus to 400 ms post-stimulus that was advanced sample by sample. This window was chosen in order to present the AESPA using an interval similar to that typically used for plotting the average Auditory Evoked Potential (AEP). However, the meaning of the interval is slightly different, as the AESPA, unlike the AEP, does not correspond to a specific discrete event occurring at time 0. Instead, each time-point on the time axis can be interpreted as being the relative time between the EEG and the input intensity signal. Therefore, the AESPA at  $-100$  ms, for example,

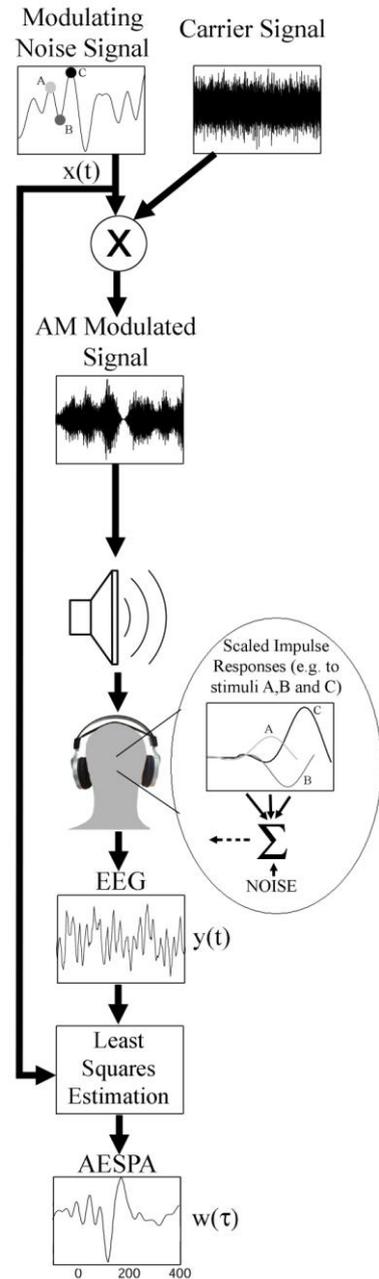


Figure 1. Flow diagram of auditory-evoked spread spectrum analysis (AESPA) acquisition. A coloured noise signal is used to modulate the amplitude of a carrier signal. The electroencephalogram (EEG) is modelled as a sum of overlapping scaled impulses in response to the amplitude modulations of the stimulus, plus noise. Three such scaled impulse responses are shown, corresponding to stimulus values A, B, and C.

indexes the relationship between the input intensity signal and the EEG 100 ms earlier; obviously this should be zero. As another example, the AESPA at  $+100$  ms indexes how the input intensity signal affects the EEG 100 ms later. The steps involved in generating the stimuli and the estimation of the AESPA are illustrated in Figure 1. It should be noted that we restricted our analysis to just the fronto-central electrode site Fz, given the topographic

distributions observed in previous AESPA studies [10].

*f) Quantification of Model Performance*

In order to quantify the AESPA method’s ability to accurately model the auditory system, we carried out the following analysis. Firstly, for each subject, we determined the linear AESPA and quadratic AESPA models by averaging the corresponding AESPAs over 9 of the 10 runs undertaken by that subject. We then used the stimulus waveform from the one remaining run as input to these models in order to predict the output EEG for that run. We then computed correlation coefficients between the predicted output EEG with the actual recorded EEG according to the following formula:

$$r = \frac{\text{cov}(y, y_p)}{\sqrt{\text{var}(y) \text{var}(y_p)}}, \quad (4)$$

where  $y$  is the recorded EEG and  $y_p$  is the predicted EEG. This process was repeated 10 times for each subject by rotating the run to be tested each time and the correlation coefficients were averaged within each subject, excluding those runs where the resulting correlation was not found to be significant at the  $p < 0.01$  level.

III RESULTS

Fig. 2 plots both the linear and quadratic AESPA responses averaged across all subjects at fronto-central electrode location Fz, referenced to the average of the two mastoids. The nonlinear AESPA is plotted on a color scale with two time axes. The value of the AESPA at any point on this 2-D plot represents the strength of the relationship between the EEG at any given time point and the interaction (product) of the two inputs at the preceding times denoted by the  $x$  and  $y$  axes. Qualitatively the diagonal of the quadratic responses seems to be quite similar to the trajectory of the linear model, which is not surprising. However, it is interesting to note that there are some non-zero clusters off the diagonal. These include negativities indicating a relationship between stimuli at 80 and 120 ms preceding the EEG and some positive ridges between about 80 and 120 ms just off the diagonal.

Table 1 lists the percentage of test trials that were predicted significantly for both linear and nonlinear models for each subject, where we determined significance using a threshold of  $p < 0.01$ . A considerable number of trials were found to be significant with an average of ~80% for the linear model and an average of ~90% for the quadratic

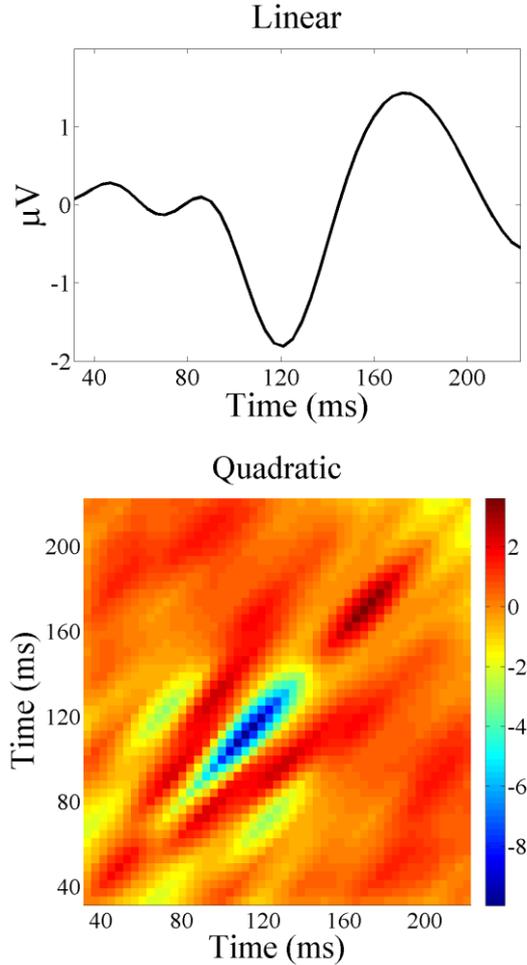


Figure 2. Linear (upper panel) and quadratic (lower panel) AESPA responses at electrode location Fz, averaged across all subjects.

TABLE I. Percentage of trials where predicted EEG (using a model fit from nine trials) and recorded EEG (from the tenth trial) were significantly correlated. Pearson correlation coefficients ( $r$ ) for only those runs where a significant ( $p < 0.01$ ) result was found are also shown.

Subject	% of significant linear runs	% of significant nonlinear runs	$r_{\text{linear}}$	$r_{\text{nonlinear}}$
1	100%	100%	.0495	.0536
2	70%	80%	.0341	.0380
3	80%	90%	.0322	.0365
4	90%	90%	.0470	.0508
5	100%	100%	.0519	.0602
6	40%	60%	.0182	.0259
7	60%	90%	.0236	.0288
8	90%	100%	.0272	.0350
9	100%	100%	.0304	.0315
10	70%	80%	.0273	.0305
11	90%	90%	.0444	.0619
<b>MEAN</b>	<b>80.1%</b>	<b>89.1%</b>	<b>.0361</b>	<b>.0418</b>

model. While the number of significantly predicted trials was high, the strength of the correlations was low. Table 1 also shows the average Pearson correlation coefficients between the recorded EEG from one of the ten trials for each subject and the predicted EEG based on the other nine trials. This average was carried out averaging only those test runs where a significant result was found. The average across all subjects was  $r=0.0361$  for the linear case and  $r=0.0418$  for the nonlinear case. It should be noted that even for those subjects for whom the same numbers of trials were found to be significant using the linear and nonlinear models, the correlation coefficients were increased when using the nonlinear model. A paired t-test revealed that correlation coefficients using the nonlinear model were higher than those using the nonlinear model ( $p = 0.0019$ ).

Fig. 3 shows one second's worth of recorded EEG and the corresponding linear and quadratic model predictions for the best performing subject (subject 5). The EEG plotted is from run 1 for that subject, while the model predictions are based on the AESPAs obtained using runs 2-10 for that subject.

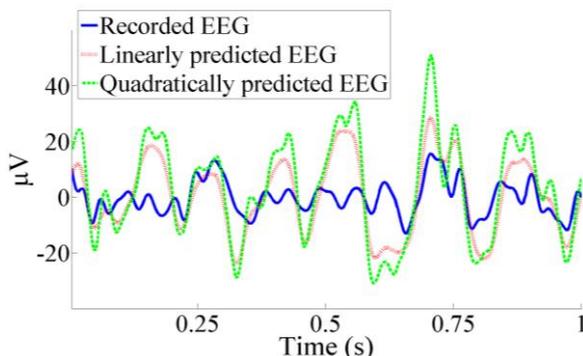


Figure 3. One second of recorded EEG from the first 120s run of subject 5 at electrode location Fz and the corresponding model predictions for that run from the linear and quadratic models. The models were fit using data from runs 2-10 of subject 5.

#### IV DISCUSSION

We have obtained linear and quadratic models of the auditory system based on spread spectrum stimulation of that system while recording EEG. The acquired models have been shown to have significant predictive power by comparing their output in response to novel stimuli with actual recorded EEG to those novel stimuli.

Despite the fact that such a large percentage of correlation tests carried out were found to be statistically significant, the correlation values obtained were not remarkably high. This was not terribly surprising given the notoriously noisy nature of the EEG signal. Because of the lack of spatial

resolution on the scalp as a result of volume conduction and the extremely deleterious effects of any muscle movements (including blinks), one would not expect a signal originating from, presumably, just auditory cortex to be completely predictable. However, Fig. 3 gives a sense of how, in some instances, the AESPA method can significantly predict previously unseen EEG.

Furthermore, unlike in the case of the VESPA, we have found a small, but significant improvement with the addition of a second order to our modelling approach. This suggests that there are meaningful interactions between stimuli at different time points that affect the subsequent EEG activity. The fact that evidence for nonlinear processing exists is not at all surprising given the necessarily nonlinear nature of real world objects such as the brain. In fact, the fact that the auditory system responds in a highly nonlinear way to auditory intensity has been well documented [14]. We attempted to correct for some of this nonlinearity using our mapping (1), however many other types of nonlinearity exist in the brain, such as saturation and burst firing.

Having made this point, it is interesting to contrast our results with those obtained using the VESPA stimulus where no quadratic advantage was found [9]. One possible reason for this is that that study utilized Gaussian visual contrast modulations such that stimulus spent less than 2% of its time below 15% contrast. This would likely result in the obtained VESPA being dominated by the activity of the parvo (P) cells of the visual system, which represent ~80% of cells in the retina and which have been found to respond approximately linear to contrast change [15,16,17]. An additional speculation is that the saccadic movement of the eyes may make it important not to integrate visual information over lengthy timescales, whereas for auditory information, such integration and interaction between auditory stimuli at different timescales may be important.

In addition it should be noted that the average ability to model the VESPA did improve slightly with the addition of a 2<sup>nd</sup> order, but the result was not significant. That study used fewer subjects (seven), and as such, it the lack of an effect may have been due to the lack of statistical power.

In general the ability to predict novel EEG in response to auditory stimuli was poorer than previously found in vision [9]. This likely stems from the aforementioned low SNR of the AESPA response compared with that of the VESPA. Previous work has suggested that this may be due to the fact that many cells in auditory cortex appear to be specialized for processing discrete events [10]. We speculate that this may be an evolutionary adaptation due to the importance of discrete events in the auditory domain, where such events are less common in vision.

There are a number of other potential improvements to the work carried out in this paper that may result in better modelling performance. Firstly, the lack of any artifact rejection in the present study has undoubtedly had a negative effect on our correlation values. The use of a technique such as independent component analysis or, more simply, the use of shorter trials might serve to improve the estimate, by reducing the impact of artifacts such as eye-blinks. Also, the time window over which both the linear and quadratic AESPA were estimated was somewhat narrow in this paper, which was done in order to minimize compute time. While it is with some confidence that we assumed that the linear AESPA coefficients outside this range would be very close to zero, it is not clear that the same would necessarily be true for the quadratic AESPA. Broadening this time window might elucidate important 2nd order effects at longer time lags. Finally, expansion of the model to even higher orders might also result in improvement. This could be accomplished using machine learning techniques such as support vector regression to avoid the curse of dimensionality.

#### V CONCLUSION

Use of the linear AESPA method and its quadratic extension has enabled us to model how the auditory system responds to novel amplitude-modulated stimuli. When modelling single trial EEG, a small, but significant improvement was observed when using the quadratic model compared with the linear model. Some explanations for this have been offered and some suggested improvements to the model have been suggested. This work suggests that incorporating higher order terms into the AESPA model could be important when investigating the characteristics of the auditory system, both in the healthy brain and in the case of neurological or psychiatric disorder.

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