Report on Possible Mechanisms of Tuning Element BEST Patches Remediation of Autism Spectrum Disorders and Attention Deficit Hyperactivity Disorder Control as Predicted by the Resonant Recognition Model

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Introduction

Autism Spectrum Disorders (ASDs) are a heterogeneous group of neurodevelopmental disorders characterised by social communication deficits and stereotyped behaviours with restricted interests. It is diagnosed in early childhood and there is not known cure, although some children recover after extensive psychotherapy. The disorder is hereditary and is associated with malfunction of development and functioning of neuro synapses due to mutations within proteins responsible for proper functioning of nerve cells synapses. On the other hand, Attention Deficit Hyperactivity Disorder (ADHD) is characterised by hyperactivity and attention deficit in young children, but can extend throughout adulthood. One of the attempts in treating these disorders is Tuning Element L.L.C. BEST patches, which belong to new class of health-related products that utilize electromagnetic frequencies. The frequencies in BEST patches are produced with Titanium Salt infused imprints and are passively transmitted through the skin contact. This type of imprinting technology is not new and is used often in electronics by imprinting microchips with different frequencies.

BEST patches should be applied along the spine close to neck or scull. Lasting from few days to about a week, they do not fall off, cannot be felt by wearer and can be worn in water. BEST patches have been used at Green Pediatrics and Dr Brasovan Clinics with great success for threating children with behaviour problems including ASDs and ADHD. Phase 1 clinical study was done in 2015 at Missouri State University (Center for Biomedical and Life Science, Prof. P. L. Durham) and has concluded that BEST patches are harmless and that they may enhance natural micro biome potential. Current studies at the same university are focused on issues of autism and other attention disorders in children. So far there are anecdotal data that BEST patches can improve ASDs symptoms and control ADHD, which is mental disorder of the neurodevelopmental type and other behaviour problems in children as well, suggesting that BEST can offer safe and cost-effective behaviour modification management.

Aim

This work is aimed to find out possible mechanisms of Tuning Element BEST patches involvement in treatment of ASDs and possibly in ADHD control.

ASDs and possibly other behavioural disorders like ADHD are characterised by malfunctioning of proteins involved in nerve synapses. Thus here, we concentrate on the activity of synaptic proteins and receptors associated with ASDs and their role in the pathogenesis of ASDs via synaptic pathways. Within this project, we utilise our own Resonant Recognition Model (RRM) to analyse proteins involved in proper functioning of nerve synapses. We concentrate on role of Neuroligin and Neurexin proteins as their interactions are crucial in functioning of synapses. The aim is to find out if they can resonate with Titanium Salt infused imprints and particles within BEST patches. These resonances could explain the mechanisms of how BEST patches are helping in treatment of ASDs and possibly ADHD control.

Behavioural Disorders

Autism Spectrum Disorders (ASDs) is a neurodevelopmental disorder characterised by impaired social interaction, verbal and non-verbal communication and restricted and repetitive behaviour [1-3]. ASDs are caused by a combination of genetic and environmental factors [2]. Some cases are strongly associated with certain infections during pregnancy including rubella and use of alcohol or cocaine [2]. Autism affects information processing in the brain by altering how nerve cells and their synapses connect and organize, but how this occurs is not well understood [3,4].

It is proposed that ASDs appear mostly due to malfunction of nerve synapses [4]. Synapses are defined as complex process of transferring information (signal) from one nerve to another. This process involves specific interactions of number of proteins both from pre- and post-synaptic nerve cells. There are two main different pathways in synapses: excitatory including developmental and inhibitory pathways [4]. Recent studies have found that synaptic related proteins such as Neuroligins (NLGNs) and Neurexins (NRXN) are associated with the development of ASDs [4]. Here we primarily focus on the activity of synaptic proteins and receptors (Neuroligin and Neurexin) which are crucial for development and pathogenesis of ASDs.

“Neuroligin is cell surface protein involved in cell-cell-interactions via its interactions with Neurexin family members. Neuroligins play a role in synapse function and synaptic signal transmission, probably mediates its effects by recruiting and clustering other synaptic proteins, promote the initial formation of synapses and is required to maintain wakefulness quality and normal synchrony of cerebral cortex activity during wakefulness and sleep” [5].

Neuroligins are responsible for development of synapses (NLGN-3). their excitation (NLGN-1,4) and inhibition (NLGN-2), as presented in Figure 1. Neurexins (NRXN) are a family of synaptic adhesion proteins that are located on the presynaptic membrane and bind to their postsynaptic counterpart NLGNs, as presented in Figure 1. The NRXN family consists of three genes (NRXN1, NRXN2, and NRXN3), each of them generating a long mRNA encoding α-NRXN and a short mRNA encoding β-NRXN. The intracellular domains of α-NRXNs and β-NRXNs are identical, whereas the extracellular domains are different [4].

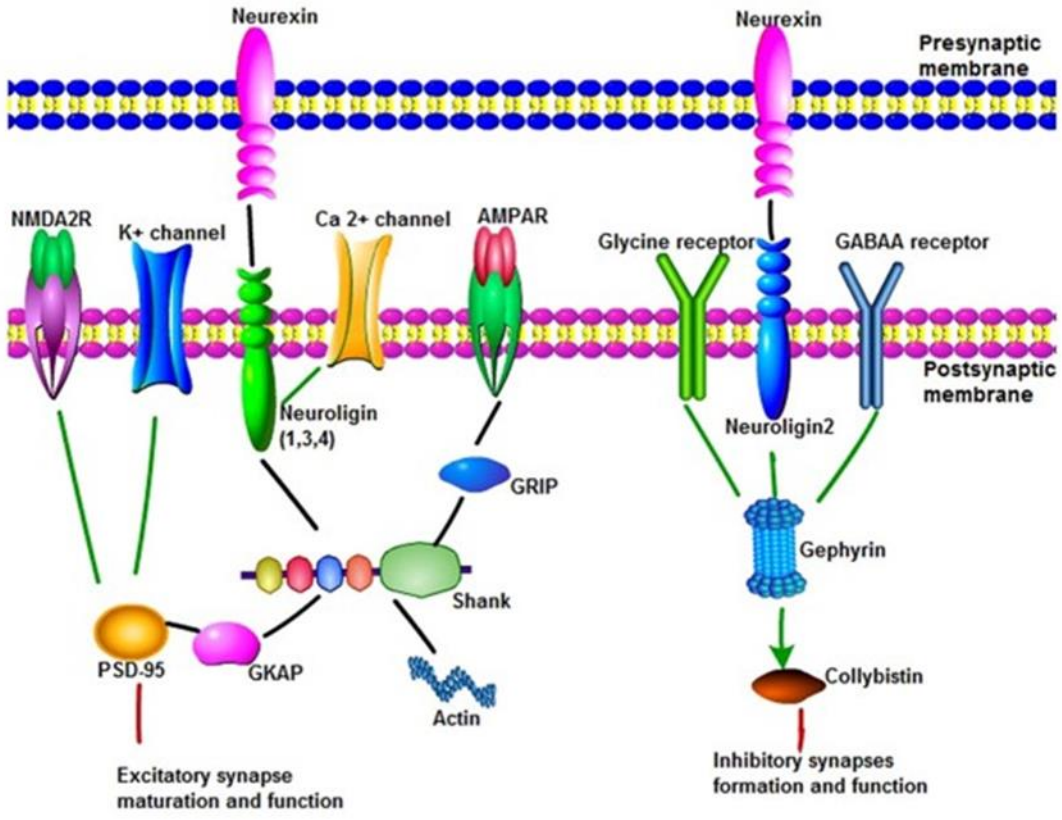

Figure 1. The main synaptic proteins and receptors involved in development, excitatory and inhibitory synapse pathways (taken from reference 4.)

As interaction between Neuroligins and Neurexins is the most critical for synaptic functioning and as the most mutations relevant to ASDs are found in these two groups of proteins, we concentrated our analysis to these two groups of proteins and their interactions. In addition Attention Deficit Hyperactivity Disorder (ADHD) is also neurodevelopmental disorder with unknown cause, but proposed to be related to autism and thus we suppose that some sort of malfunction of neuro synapses could be also cause of ADHD. We have analysed both Neuroligins and Neurexins and their interaction, using our Resonant Recognition Model (RRM) to find out characteristic resonant frequencies of their activity and interaction and to propose that these frequencies can resonate with Titanium Salt infused imprints within BEST patches.

Resonant Recognition Model (RRM)

The RRM is based on the findings that certain periodicities within the distribution of energy of delocalized electrons along protein/DNA molecules are critical for protein/DNA biological functions and/or interactions with their targets [6-8]. If charge transfer through these macromolecules is introduced, then charge moving through macromolecular backbone can produce electromagnetic radiation, absorption and resonance with spectral characteristics corresponding to the energy distribution and charge velocity [6-11].

The RRM enables the calculation of these spectral characteristics, by assigning each amino acid a physical parameter representing the energy of delocalized electrons of each amino acid. Comparing Fourier spectra for this energy distributions by using cross-spectral function, it has been found that proteins sharing the same biological function/interaction share the same

periodicity (frequency) within energy distribution along the macromolecule [6,7]. Furthermore, it has been shown that interacting proteins and their targets share the same characteristic frequency, but have opposite phase at characteristic frequency [6-8]. Thus, it has been proposed that the RRM frequencies characterize, not only a general function, but also a recognition and interaction between the macromolecule and its target, which then can be considered as resonant recognition. This could be achieved with resonant energy transfer between the interacting macromolecules through oscillations of a physical field, which is electromagnetic in nature. Since there is evidence that proteins and DNA have certain conducting or semi-conducting properties, a charge moving through the macromolecular backbone and passing different energy stages, caused by different amino acid or nucleotide side groups, can produce sufficient conditions for a specific electromagnetic radiation or absorption. The frequency ranges of this field depend on the charge velocity. The RRM proposes that the charge is travelling through the macromolecular backbone at the estimated velocity of 7.87×10^5m/s [6,7]. For this velocity and with the distance between amino acids in a protein molecule of 3.8Å, the frequency of protein interactions was estimated to be in the range between 10^{13}Hz and 10^{15}Hz. Therefore, the estimated frequency range for both amino acid and nucleotide macromolecules includes infra-red, visible and ultra-violet light. To support this idea, we compared our computational predictions with number of published experimental results [6,7]:

- Laser light growth promotion of cells, by using the particular frequencies of light to produce the similar effect to that of growth factor proteins;

- Chymotrypsin activation (increase of enzyme activity) achieved by laser light radiation in a range of 850-860nm;

- Activation of highly homologous plant photoreceptors which, although being very homologous, absorb different wavelengths of light;

- Photo activated proteins, e.g. rhodopsin, flavodoxin, etc.

These comparisons have shown a strong linear correlation between frequencies, as calculated using the RRM method and experimentally measured characteristic frequencies, with the slope factor of K=201 [6,7,11]. This finding parallel with the frequency range previously associated with the RRM numerical frequency spectrum that has been calculated from the charge velocities through the protein backbone. This correlation can be represented as following:

$$\lambda = K / frrm$$

where λ is the wavelength of light irradiation in nm, which can influence a particular biological process, frrm is a RRM numerical frequency and K is coefficient of this linear correlation.

We applied this concept on number of proteins and DNA examples [6-12]. The concept has been also experimentally tested by predicting the electromagnetic frequencies for L-Lactate Dehydrogenase [13], where by radiating L-Lactate Dehydrogenase with predicted calculated electromagnetic frequencies the significant change in enzyme activity was achieved. The concept has also been tested independently on experimental measurements of photon emission from dying melanoma cells [14], on photon emission from lethal and non-lethal Ebola strains [15], as well as on classic signalling pathway, JAK-STAT, traditionally composed of nine sequential protein interactions [16].

Keeping all this in mind, we propose that the RRM concept is excellent predictor for proteins and DNA selective interactions, biological processes and pathways in living cells. In our

previous work, we have calculated a large number of specific frequencies for different protein and DNA biological functions and interactions, as presented in Figure 2.

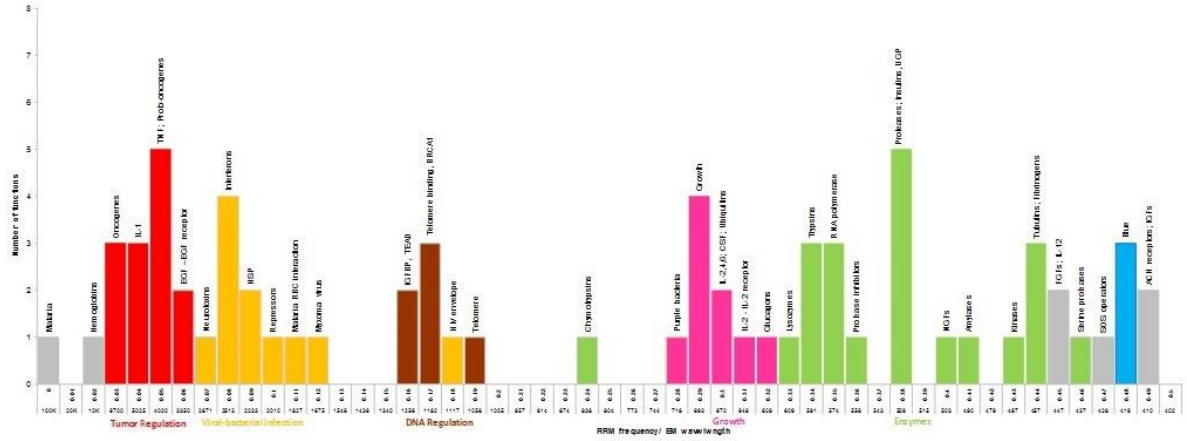

Figure 2. Number of functional groups within each RRM frequency range of 0.01. X axis represent RRM frequency in steps of 0.01, as well as corresponding electromagnetic frequency in nm. Y axis represent number of functional groups. Names of functional groups are written on the top of each bar. Functional super families are differently coloured and labelled below X axis.

Frequencies calculated using the RRM, as described above, have been found to be related to biological function of the proteins [6-13]. However, if we consider protein and DNA complex structures and alpha helices the charge transfer is also possible to occur through these structures in form of solitons [17] (Davydov [18,19], Hayman [20], Sinkala [21]), excitons (Davydov [18,19], Pang [22], Sinkala [21], Yomosa [23]) and phonons (Pang [22], Yomosa [23], Ichinose [24]). These other forms of charge transfers are at velocities different than initially used by the RRM and are ranging from 10^5m/s for solitons and some excitons all the way down to speed of sound and small fractions of speed of sound for phonons. Thus, with the same periodicities within proteins sequences, as determined by the RRM, different modalities of charge transfer can produce different resonant frequencies which not necessarily are related to the protein biological function, but could be related to protein and DNA resonances in general.

In our previous work, we have applied these charges moving modalities to tubulin and microtubule macromolecules and identified number of possible electromagnetic resonance frequencies in these macromolecule structures. These results have been experimentally confirmed in research by Bandyopadhyay [25]. Here, we applied this approach to proteins involved in nerve synapses with the aim to find out if there are possible resonances with these modalities that can resonate with BEST patches imprint frequencies and consequently can help in treatment of behavioural disorders.

Findings

As it has been proposed that the main cause of ASDs and possibly ADHD is malfunctioning of neural synapses and interaction between Neuroligins and Neurexins, we have concentrated on analyses of Neuroligins, Neurexins and their interactions using our own RRM model with the aim to find out the characteristic frequencies of such interactions. Once

when such characteristic frequencies have been found, it can be proposed that Titanium Salt infused imprints within BEST patches can resonate with these frequencies, improve neural synapses normal functioning and consequently alleviate symptoms of ASDs and control ADHD.

Having in mind that ASDs appears in early childhood and thus could be considered as developmental disorder, we have initially analysed activity of Neuroligin3, which was found to be critical in development of synapses [1,2]. When the RRM model is applied to four mammalian Neuroligin3 proteins the common characteristic frequency appears at 0.4155, as presented in Figure 3.

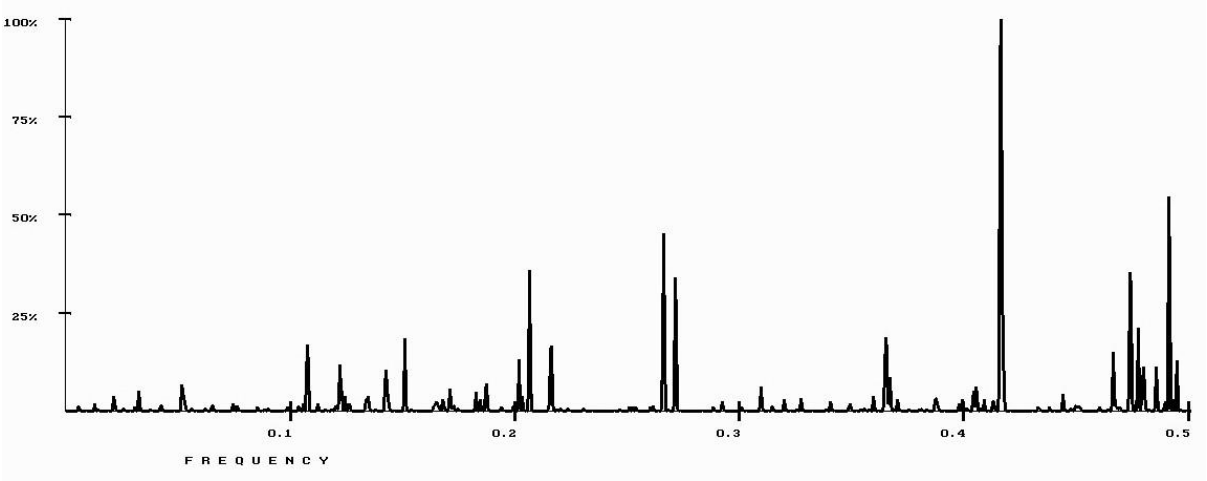

Figure 3. RRM cross-spectrum of four Neuroligin3 proteins.

To make sure that this frequency is characterising not only Neuroligin3 proteins, but also interaction between Neuroligin3 and corresponding Neurexin's, which is critical for proper functioning of synapses, we have compared Neuroligin3 proteins with corresponding Neurexin proteins. The frequency of 0.4155 became more prominent, as presented in Figure 4. According to RRM principles, this frequency is characterising interaction between Neuroligin3 proteins and corresponding α-Neurexin3 proteins.

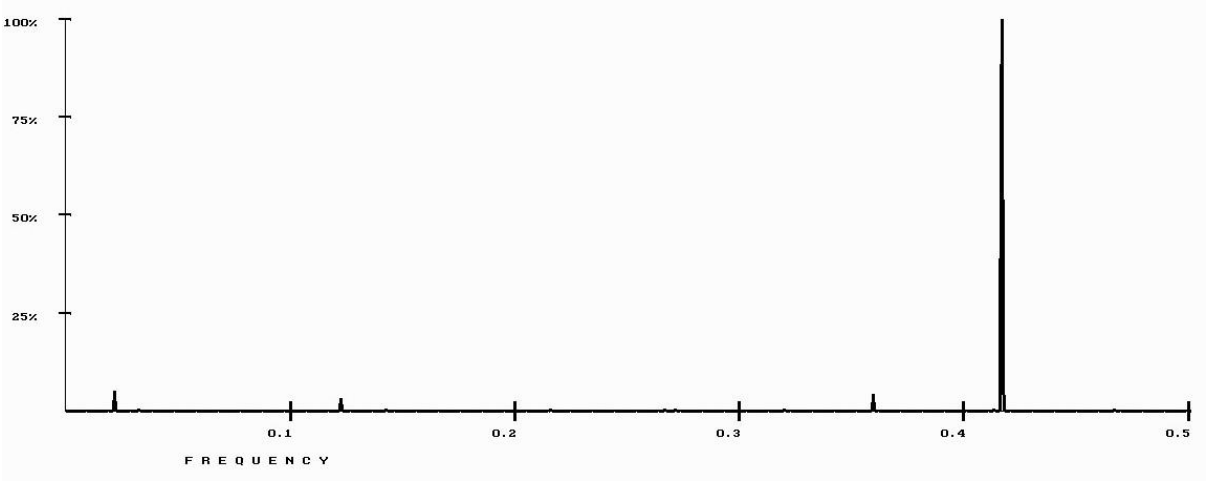

Figure 4. RRM cross-spectrum of four Neuroligin3 proteins and three α-Neurexin3 proteins.

The next step was to find out what would be the characteristic RRM frequency for not only development of synapses, but also for their normal functioning. For that purpose, we have initially compared Neuroligin1, 3 and 4 proteins, which are all involved in excitatory synapse maturation and function, as presented in Figure 1. Interestingly the same RRM characteristic frequency of fe=0.4155 appeared common to all analysed Neuroligin proteins, as presented in Figure 5. These results are pointing out that the same RRM characteristic frequency is characterising both development and normal functioning of excitatory synapse.

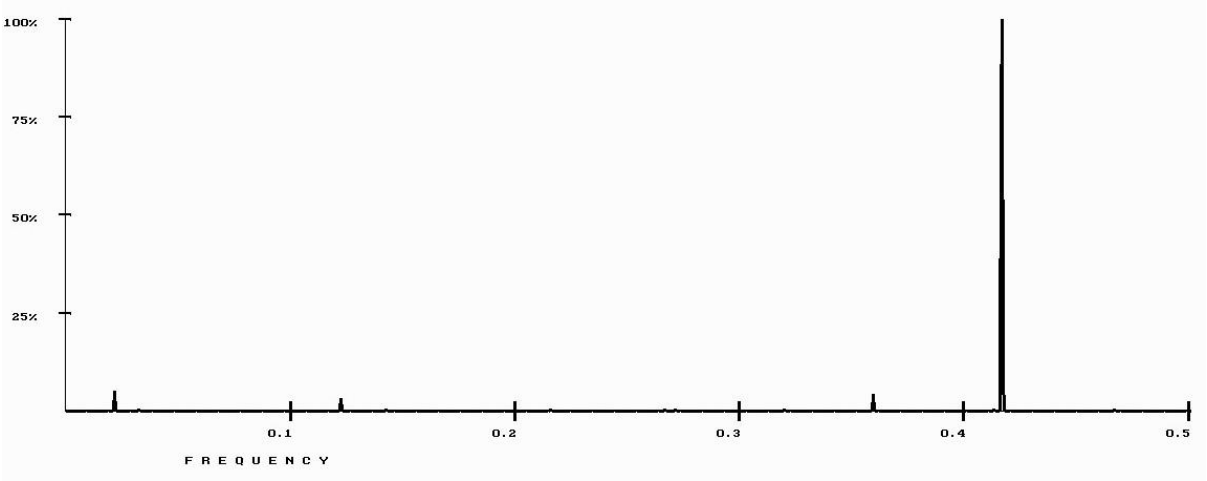

Figure 5. RRM cross-spectrum of nine Neuroligin proteins, including Neuroligins1, 3 and 4.

Once when this characteristic RRM frequency was identified, we can calculate relevant wavelength of related electromagnetic radiation using the formula: $\lambda = K / frrm$. The wavelength related to the frequency relevant for development and functioning of nerve synapses is then λ=484nm. Thus, Titanium Salt or any other conductive particles in the BEST patches, that are in diameter of about Dλ=484nm, Dλ/2=242nm and Dλ/4=121nm, can resonate with synaptic proteins, influence development and normal functioning of nerve synapses and consequently remediate development and symptoms of ASDs.

On the other hand, inhibitory synapses formation and function goes through completely different pathway than excitatory synapse function and it involves Neuroligin2 and its corresponding Neurexins, as presented in Figure 1. When Neuroligin2 proteins were compared with corresponding Neurexin proteins completely different common characteristic RRM frequency appeared at fi=0.0015, as presented in Figure 6. This is very interesting result showing that excitatory and inhibitory synapses pathways have completely different RRM characteristic frequencies.

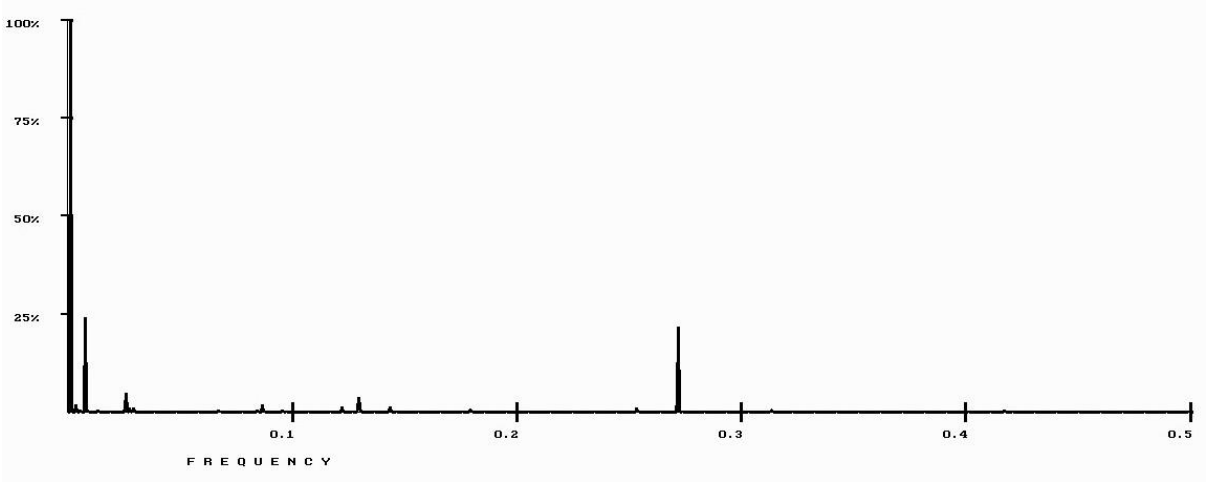

Figure 6. RRM cross-spectrum of two Neuroligin2 proteins and two α- and two β- Neuroxin2 proteins.

Once when this characteristic RRM frequency was identified, we can calculate relevant wavelength of related electromagnetic radiation using the formula: $\lambda = K / frrm$. The wavelength related to the frequency relevant for inhibition of nerve synapses is then λ=64000nm (0.064mm). Thus, Titanium Salt or any other conductive imprints in the BEST patches, that are in length of about Dλ=64000nm, Dλ/2=32000nm and Dλ/4=16000nm, can resonate with inhibitory pathway of synaptic functioning.

It is well known that ASDs are neurodevelopmental disorder, which is the most probably related to the functioning of developmental and excitatory synaptic pathway. Hence, we propose that characteristic RRM frequency of fe=0.4155 for developmental and excitatory synaptic pathway is the most relevant to be targeted for any proposed remediation of ASDs.

Having in mind that ASDs is characterised by lack of communication and restricted interest, while ADHD is characterised by hyperactivity, we propose that ASDs is related to malfunctioning of developmental and excitatory synaptic pathway, while on the contrary ADHD is related to malfunctioning of inhibitory synaptic pathway. Thus, RRM frequency of fi=0.0015 for inhibitory synaptic pathway is the most relevant to be targeted for any proposed remediation of ADHD.

To find out if there are other resonant electromagnetic frequencies that can influence development and excitatory functioning of synapses, we have introduced other modalities of charge transfer through proteins (solitons, excitons and phonons), as described above. When different modalities of charge transfer were applied to RRM characteristic frequencies of fe=0.4155, identified to be related to development and excitatory functioning of nerve synapse and fi=0.0015, identified to be related to inhibitory functioning of nerve synapse, the following resonant frequencies have been identified for each modality, as presented in Table 1.

RRM Frequency	velocity as per RRM 7.87×10^5 m/s	velocity as per Yomosa 3.2m/s	velocity as per Yomosa 1.2×10^5m/s	velocity as per Pang 68m/s	velocity as per Davydov 170m/s	velocity as per Ichinose 0.34m/s	velocity as per Ichinose 5×10^{-4}m/s
Excitatory: 0.4155	426-436TH	1731-1772MH	65-66TH	37-38GH	92-94GH	184-188MH	270-277KH
Inhibitory: 0.0015	1.5-3.1TH	0.2-0.4MH	0.3-0.7TH	0.1-0.3GH	6-13GH	0.7-1.3MH	1-2KH

Table 1. Electromagnetic frequencies for different modalities.

These frequencies are proposed to be able to resonate and may influence with either developmental and excitatory or inhibitory pathway of nerve synapses. Thus, we propose that if these frequencies are imprinted in BEST patches then such BEST's can resonate with either developmental and excitatory or inhibitory pathway of nerve synapses. According to RRM principles all these results could explain the mechanisms how BEST patches remediate the ASDs disorders and could control ADHD.

Conclusion

Within this work, we have analysed synaptic proteins, using the RRM model, with the aim to find the characteristic resonant frequencies for development, excitation and inhibition of synapses and to investigate possibility of these frequencies to resonate with frequencies imprinted within BEST patches and consequently to propose mechanism of ASDs remediation and control of ADHD with BEST patches.

We found that:

- Characteristic frequency for development and excitation of synaptic pathway is fe=0.4155. This numerical RRM frequency relates to electromagnetic wavelength λ=484nm. Thus, Titanium Salt or any other conductive particles in the BEST patches, that are in diameter of about Dλ=484nm, Dλ/2=242nm and Dλ/4=121nm, can resonate with synaptic proteins, influence development and normal functioning of nerve synapses and consequently remediate development and symptoms of ASDs.

- Characteristic frequency for inhibition of synaptic pathway is fi=0.0015. This numerical RRM frequency relates to electromagnetic wavelength λ=64000nm (0.064mm). Thus, Titanium Salt or any other conductive imprints in the BEST patches, that are in length of about Dλ=64000nm, Dλ/2=32000nm and Dλ/4=16000nm, can resonate with inhibitory pathway of synaptic functioning. Having in mind that ASDs is characterised by lack of communication and restricted interest, while ADHD is characterised by hyperactivity, we propose that ASDs is related to malfunctioning of developmental and excitatory synaptic pathway, while on the contrary ADHD is related to malfunctioning of inhibitory synaptic pathway. Thus, RRM frequency of fi=0.0015 for inhibitory synaptic pathway is the most relevant to be targeted for any proposed remediation of ADHD.

- When different modalities of charge transfer through protein backbone is introduced, the resonant frequencies for development and excitation, as well as inhibition of synaptic pathway could then be in different frequency ranges including THz, GHz, MHz and KHz, as presented in Table 1. These frequencies could also resonate with frequency imprinted within BEST patches.

All these findings can explain mechanisms of BEST patches remediating ASDs disorders, as well as control of ADHD through resonances with synaptic pathways. This would mean that BEST patches could help ASDs and ADHD patients to improve their condition without using drugs and their negative side effects.

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Appendix

Three Neuroligin1 proteins:

>sp|Q8N2Q7|NLGN1_HUMAN Neuroligin-1 OS=Homo sapiens GN=NLGN1 PE=1 SV=2

>sp|Q99K10|NLGN1_MOUSE Neuroligin-1 OS=Mus musculus GN=Nlgn1 PE=1 SV=2

>sp|Q62765|NLGN1_RAT Neuroligin-1 OS=Rattus norvegicus GN=Nlgn1 PE=1 SV=1

Three Neuroligin2 proteins:

>sp|Q8NFZ4|NLGN2_HUMAN Neuroligin-2 OS=Homo sapiens GN=NLGN2 PE=1 SV=1

>sp|Q69ZK9|NLGN2_MOUSE Neuroligin-2 OS=Mus musculus GN=Nlgn2 PE=1 SV=2

>sp|Q62888|NLGN2_RAT Neuroligin-2 OS=Rattus norvegicus GN=Nlgn2 PE=1 SV=1

Four Neuroligin3 proteins:

>sp|Q9NZ94|NLGN3_HUMAN Neuroligin-3 OS=Homo sapiens GN=NLGN3 PE=1 SV=2

>sp|Q8WMH2|NLGN3_MACMU Neuroligin-3 (Fragment) OS=Macaca mulatta GN=NLGN3 PE=2 SV=1

>sp|Q8BYM5|NLGN3_MOUSE Neuroligin-3 OS=Mus musculus GN=Nlgn3 PE=1 SV=2

>sp|Q62889|NLGN3_RAT Neuroligin-3 OS=Rattus norvegicus GN=Nlgn3 PE=1 SV=1

Two Neuroligin4 proteins:

>sp|Q8N0W4|NLGNX_HUMAN Neuroligin-4, X-linked OS=Homo sapiens GN=NLGN4X PE=1 SV=1

>sp|Q8NFZ3|NLGNY_HUMAN Neuroligin-4, Y-linked OS=Homo sapiens GN=NLGN4Y PE=2 SV=1

Two α-Neurexin2 proteins:

>sp|Q9P2S2|NRX2A_HUMAN Neurexin-2 OS=Homo sapiens GN=NRXN2 PE=2 SV=1

>sp|Q63374|NRX2A_RAT Neurexin-2 OS=Rattus norvegicus GN=Nrxn2 PE=1 SV=3

Two β-Neurexin2 proteins:

>sp|P58401|NRX2B_HUMAN Neurexin-2-beta OS=Homo sapiens GN=NRXN2 PE=1 SV=1

>sp|Q63376|NRX2B_RAT Neurexin-2-beta OS=Rattus norvegicus GN=Nrxn2 PE=1 SV=1

Three α-Neurexin3 proteins:

>sp|Q9Y4C0|NRX3A_HUMAN Neurexin-3 OS=Homo sapiens GN=NRXN3 PE=1 SV=4

>sp|Q6P9K9|NRX3A_MOUSE Neurexin-3 OS=Mus musculus GN=Nrxn3 PE=1 SV=2

>sp|Q07310|NRX3A_RAT Neurexin-3 OS=Rattus norvegicus GN=Nrxn3 PE=1 SV=1