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Analysis of Bacteria Resistance Using the Resonant Recognition Model

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Abstract

Antibiotics have been used extensively to treat practically all known bacterial infections, but the great potential of antibiotics has been jeopardized by the emergence of antimicrobial resistance with some bacteria even becoming resistant to most antibiotics in use. Here, the Resonant Recognition Model (RRM), which is biophysical model capable of analyzing bimolecular interactions, has been utilized to find out the specificity of resistance within resistant bacteria. The definite biophysical characteristic that distinguishes between ordinary and resistant bacteria has been identified. This result is used here to propose approaches to neutralize and possibly eliminate problem of resistant bacteria.

Keywords Resistant Bacteria; Resonant Recognition Model; Molecular Modelling

1. Introduction

Antibiotics have been used for about 80 years and are powerful drugs, which can threat practically all known bacterial infections [1]. Unfortunately, the great potential of antibiotics has been negatively affected by the emergence and rise of antimicrobial resistance with some bacteria even becoming resistant to most antibiotics in use [2]. The main action of antibiotics is by preventing synthesis of bacteria cell wall, which is made of polymer mesh layer called peptidoglycan formed from linear chains of two alternating amino sugars, so called NAG and NAM, which are cross linked by 4-5 amino acid chains [3], as presented in Figure 1.

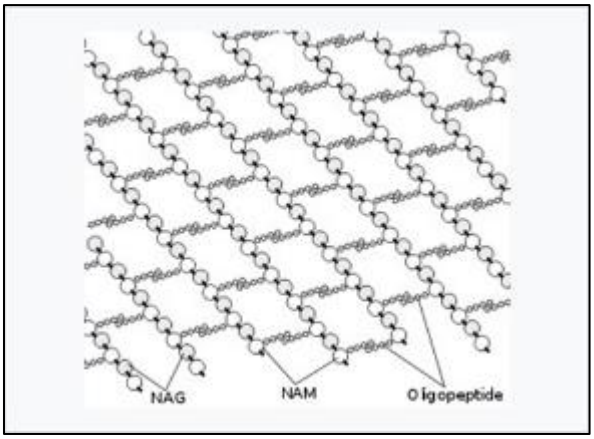

Figure 1 The structure of peptidoglycan. NAG = N-acetylglucosamine (also called GlcNAc or NAGA), NAM = N-acetylmuramic acid (also called MurNAc or NAMA)

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Some antibacterial drugs, like beta-lactam antibiotics, are selective inhibitors for peptidoglycan synthesis and are active only during active growth of bacteria [4-5]. These antibacterial drugs interfere with the production of peptidoglycan by binding to bacterial enzymes, known as penicillin binding proteins, which are crucial for formation of peptidoglycan [6-7].

There are various mechanisms by which bacteria become resistant to antibiotics, where the main and the most specific mechanisms are production of enzyme, so called beta-lactamase. This enzyme provides antibiotic resistance by breaking antibiotic structure. The second important mechanism of bacterial resistance is modification of antibiotic's receptor, penicillin binding proteins, to prevent/reduce binding of beta-lactam antibiotics [4-7]. The other mechanisms, like efflux pumps [8] or ppGpp [9] signalling is either less specific or more complicated for practical application.

We have concentrated here on two main mechanisms of bacterial antibiotic resistance: production of beta-lactamase and changes in penicillin binding proteins. With the aim of preventing bacterial antibiotic resistance, we are here proposing the following approaches:

- To inhibit beta-lactamase activity.
- To mimic activity of antibiotics with peptides that will bind to penicillin binding proteins and to prevent mesh formation.
- To identify the characteristic frequency of the peptidoglycan mesh to resonantly interfere with its activity.

To achieve this, we firstly have analyzed all the participants in the processes listed above. For that purpose, we have utilized the Resonant Recognition Model (RRM), to identify characteristics of beta-lactamase, to identify characteristics of penicillin binding proteins, as well as to identify characteristics of peptidoglycan mesh. The RRM model, which proposes that molecular biological function is characterized by certain periodicities (frequencies) within distribution of free electron energies along macromolecule [10-16], is capable to identify these characteristics.

2. Methods and Materials

Here, we have utilized the Resonant Recognition Model (RRM) which is innovative approach using knowledge from quantum physics, biochemistry and mathematics to analyze interactions between proteins and their targets, which could be other proteins, DNA, RNA, or small molecules. The RRM model has been extensively previously published in detail within the number of publications [10-17]. The RRM model is based on the findings that certain periodicities (frequencies) within the distribution of energy of delocalized electrons along protein backbone are critical for macromolecule biological function and/or interaction with their targets. The distribution of delocalized electrons energies is calculated by assigning each amino acid specific physical parameter representing the energy of delocalized electrons of each residue. Consequently, the spectral characteristics of such energy distribution (signal) are calculated using Fourier Transform. This means that the linear numerical signal representing the distribution of energies along the macromolecule is transformed into the frequency domain and is characterized by number of different frequencies containing all information from the original signal. Comparing such spectra using cross-spectral function for macromolecules, which are sharing the same biological function/interaction, it has been shown that they share the same frequency within the spectrum of free energy distribution along the macromolecule [10-17]. Peak frequencies in such multiple cross-spectral function present common frequency components for all macromolecular sequences compared. The comprehensive analysis done so far confirms that all macromolecular sequences, with the common biological function and/or interaction, have common frequency component, which is specific feature for the observed biological function/interaction [10-17]. Thus, each specific macromolecular biological function/interaction within macromolecule is characterized by specific RRM frequency.

Generally, biological functions are driven by proteins that selectively interact with other proteins, DNA/RNA regulatory segments or small molecules. Through extensive use of RRM model, it has been shown that proteins and their targets share the same matching RRM characteristic frequency [10-17]. The matching of periodicities within the distribution of energies of free electrons along the interacting proteins can be regarded as the resonant recognition and as such is highly selective. Thus, RRM frequencies characterize not only protein function, but also recognition and interaction between protein and its targets: proteins (receptors, binding proteins, and inhibitors), DNA/RNA regulatory segments or small molecules. In addition, it has been also shown that interacting macromolecules have opposite phases at their characteristic RRM recognition frequency [10-17].

Once the characteristic frequency for biological function and/or interaction of the macromolecule is identified, it is possible to design new peptides/proteins with the desired RRM frequency components [10-13,15] and consequently have been experimentally tested to have indeed desired biological functions and/or interactions [18-22].

As 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, it could be considered as resonant recognition. This resonance could be achieved with resonant energy transfer between the interacting macromolecules through oscillations of a physical field, which could be electromagnetic in nature. Since there is evidence that proteins and DNA have certain 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 model proposes that the charge is travelling through the macromolecular backbone at the estimated velocity of $7.87x10^5$m/s [10-13]. 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 far infra-red, infra-red, visible and part of ultra-violet light. To support this idea, we compared our computational predictions with number of published experimental results [10-13]. These comparisons have shown a strong linear correlation between frequencies, as calculated using the RRM model and experimentally measured characteristic frequencies, with the slope factor of K=201 [10-13]. This frequency range is overlapping the frequency range previously associated with the RRM numerical frequency spectrum that has been calculated from the charge velocities through the protein backbone [10-13]. This correlation can be presented as follows:

$$\lambda = K / f_{rrm}$$

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

The initially established RRM approach cannot be applied for interactions between proteins and small molecules, as small molecules are not linear sequential molecules. To be able to analyze interaction between small molecules and proteins, we have proposed that small molecules also recognize proteins on the distance and interact with proteins through electromagnetic resonant energy transfer enabling specific biological activity [23]. To expand the idea of electromagnetic resonant recognition to small molecules and their interaction with proteins, RRM model has been extended by calculating electromagnetic frequencies of free electron energies within the small molecule and comparing these frequencies with RRM characteristic frequencies for relevant interacting proteins [24-26].

The RRM resonant frequency of small molecules can be calculated as follows [24-26]:

$$f_{sm} = (K \times E) / (h \times (c / n))$$

where: f_{sm} is numerical RRM frequency corresponding to electromagnetic radiation resonances due to energies of free electrons within small molecules, K=201 is coefficient in nm previously semi empirically identified to characterize the relationship between RRM frequencies and related electromagnetic radiation frequencies, E is energy of free electrons within small molecules, h is Planck constant, c is speed of light, n is refraction index in biological materials.

The hypothesis that frequencies produced by energies of free electrons within small molecules are critical for small molecules' biological functions and their recognition and interaction with proteins, has been tested for a number of small molecules interacting with their receptors [24-26].

All protein sequences used within this research are from UniProt database and are listed in Appendix A.

3. Results

3.1. Previous Results: Analysis of beta-lactamases

To survive the effects of antibiotics, bacteria are constantly finding new defense strategies, called "resistance mechanisms". For example, some Enterobacteriaceae can produce enzymes called extended spectrum beta-lactamases. Beta-lactamases are enzymes that open the beta-lactam ring, inactivating the antibiotic. Extended spectrum beta-lactamases are enzymes that produce resistance to most beta-lactam antibiotics, including penicillin's, cephalosporins, and the monobactam aztreonam. Infections caused by bacteria producing beta-lactamases have been associated with poor antibiotic treatment outcomes.

We have analyzed beta-lactamase proteins from different bacterial strains, as listed in Appendix A, using the RRM model, and the common RRM characteristic frequency has been identified to be at f1=0.0352+0.0041, as presented in Figure 2.

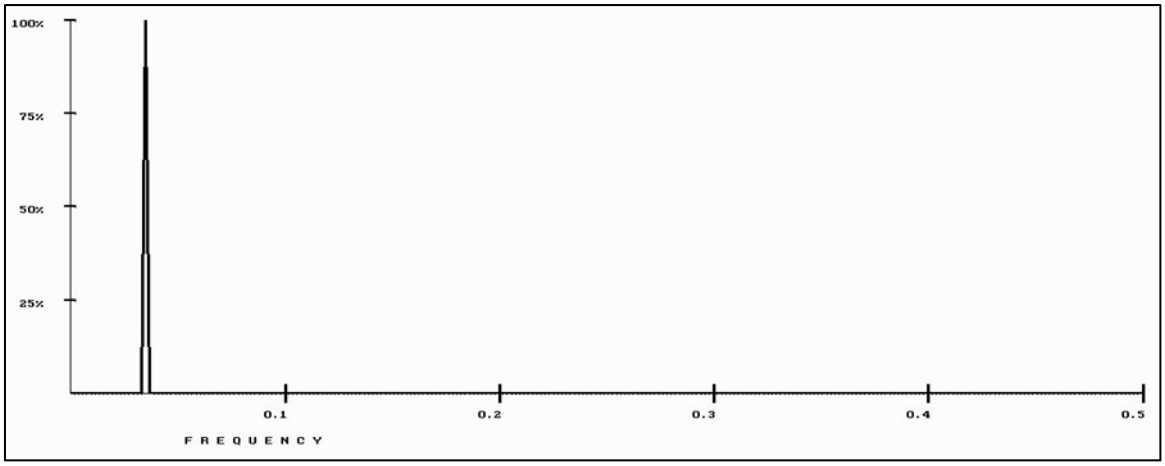

Figure 2 RRM cross-spectrum of 22 beta-lactamase proteins with common characteristic frequency is at f1=0.0352+0.0041

It is interesting to note that there is one common characteristic for all analyzed beta-lactamase proteins from many different bacterial strains. This would mean that all beta-lactamases have the same common RRM characteristic frequency f1 characterizing common beta-lactamase function, i.e., opening the beta-lactam ring and inactivating antibiotic. Thus, we have already designed, using the RRM frequency f1, peptide capable of interfering with the process of antibiotic inactivation by beta-lactamase, which have been experimentally tested [18].

3.2. Analysis of penicillin binding proteins

Penicillin binding proteins are a group of proteins that are characterized by their affinity for and binding penicillin. All beta-lactam antibiotics (except for tabtoxinine beta-lactam, which inhibits glutamine synthetase) bind to penicillin binding proteins, which are essential for bacterial cell wall synthesis.

Here, the aim has been to understand activity of antibiotics and their binding to penicillin binding proteins, which is critical for blocking synthesis of bacterial walls. The first step was to analyze penicillin binding proteins from different bacterial strains to find out if there is common RRM characteristic, which can be related to penicillin recognition and binding to penicillin binding proteins.

When we have analyzed penicillin binding proteins from different bacterial strains, as listed in Appendix A, using the RRM, two prominent common RRM frequencies have been identified at f2=0.2529+0.0017 and f3=0.2412+0.0017, as presented in Figure 3.

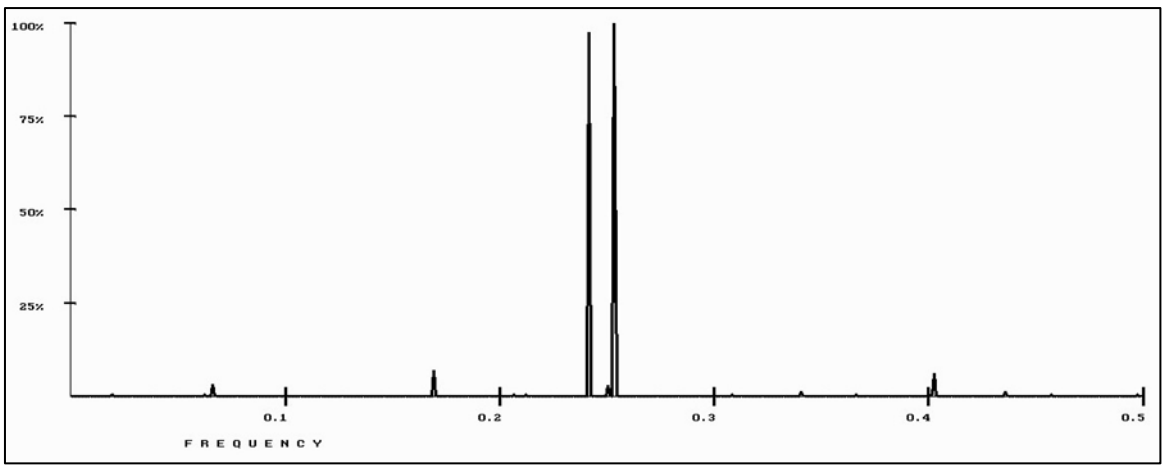

Figure 3 RRM cross-spectrum of 14 penicillin binding proteins with two equally prominent common characteristic frequencies, which are at f2=0.2529+0.0017 and f3=0.2412+0.0017

It is interesting to note that those two RRM characteristic frequencies are close to each other opening the possibility of wider range of frequencies, which are characterizing antibiotic recognition and binding penicillin binding proteins.

Furthermore, we have analyzed interaction between penicillin binding proteins and antibiotics as small molecules, using the extended RRM model for small molecules as described above. Most antibiotics, such as penicillin, are beta-lactam antibiotics. Beta-lactam antibiotics are antibiotics that contain a beta-lactam ring in their molecular structure, as presented in Figure 4. This includes penicillin derivatives (penams), cephalosporins (cephems), monobactams, carbapenems [27] and carbacephems [28]. Most beta-lactam antibiotics work by inhibiting cell wall biosynthesis through binding to penicillin binding proteins in the bacterial organism and are the most widely used group of antibiotics.

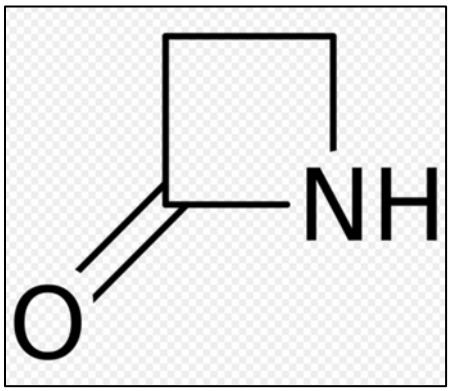

Figure 4 Molecular structure of beta-lactam ring

Using the chemical formula of beta-lactam ring as molecular structure characterizing beta-lactam molecules (C_3NHO), we have calculated, as described above, energy of free electrons within beta-lactam ring to be at Ebl=0.0767Ry. For this energy and refraction index for proteins, which is between 1.36-1.55 [29], we have calculated the corresponding RRM frequency for beta-lactam ring to be between 0.23-0.26, as highlighted in yellow in Figure 5.

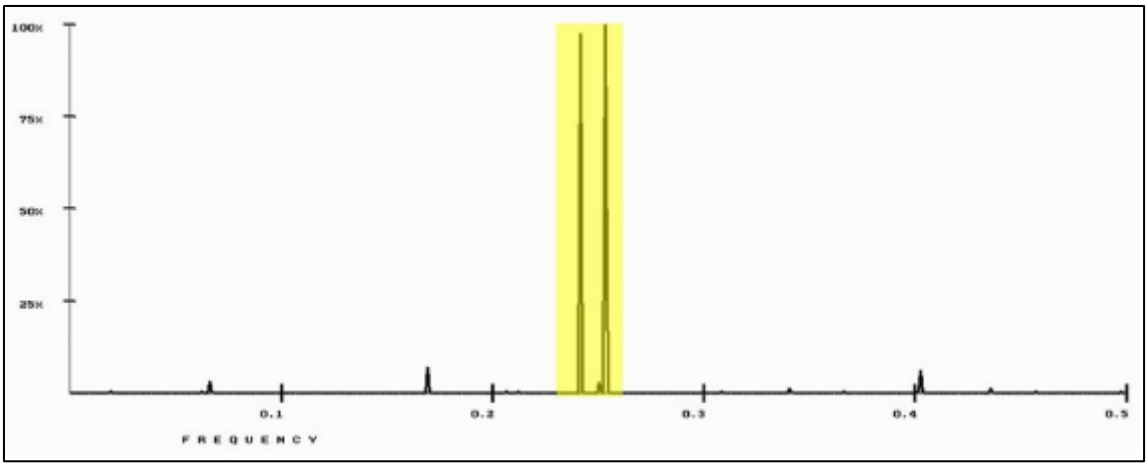

Figure 5 RRM cross-spectrum of 14 penicillin binding proteins with two equally prominent common characteristic frequencies, which are at f2=0.2529+0.0017 and f3=0.2412+0.0017. RRM frequency range for beta-lactam rings between 0.23-0.26 highlighted in yellow

It is important to notice that this RRM frequency range for beta-lactam ring overlaps both characteristic RRM frequencies for penicillin binding proteins, which are at f2=0.2529+0.0017 and f3=0.2412+0.0017. As there is an overlap between characteristic RRM frequencies for penicillin binding proteins and RRM frequency for beta-lactam ring, we propose that this frequency range is critical for recognition and interaction between beta-lactam antibiotics and penicillin binding proteins and could be used in further design of peptides that can mimic antibiotic activity and could bypass antibiotic bacterial resistance.

3.3. Peptidoglycan mesh

The main action of antibiotics is to prevent synthesis of peptidoglycan mesh by binding to penicillin binding proteins that are crucial for formation of peptidoglycan. Thus, antibiotics are active only during peptidoglycan synthesis, which is happening only during the active growth of bacteria. However, it would be extremely beneficial to have agent capable of destroying peptidoglycan mesh, within the bacteria's cell wall, also during the passive non growth phase. For that purpose, we have initially analyzed peptidoglycan deacetylase, the enzymes which are potentially important targets for the design of new drugs [30]. In pathogenic bacteria, peptidoglycan deacetylase modifies cell-wall peptidoglycan by removing the acetyl group, which makes the bacteria more resistant to the host's immune response and other forms of attack, such as degradation by lysozyme [30]. When we have analyzed peptidoglycan deacetylase proteins from different bacterial strains, as listed in Appendix A, using the RRM, the most prominent common RRM frequency has been identified at f4=0.2422+0.0047, as presented in Figure 6.

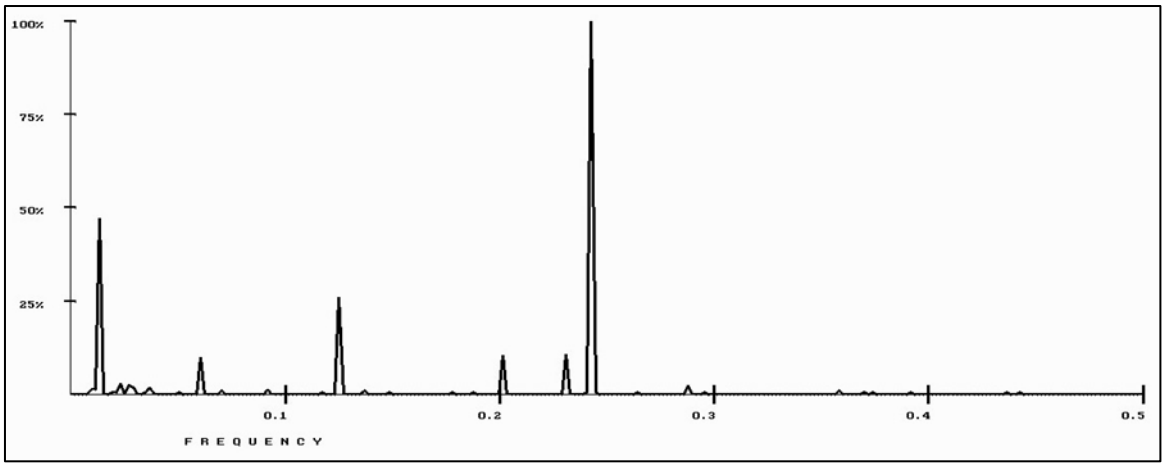

Figure 6 RRM cross-spectrum of 9 peptidoglycan deacetylase proteins with the most prominent common characteristic frequency at f4=0.2422+0.0047

It is interesting to note that frequency f4 is between previously identified frequencies f2 and f3 that are characterizing penicillin binding proteins. In addition, frequency f4 is within the range of frequencies characterizing antibiotic beta-lactam ring as well. This indicates that frequency range between 0.23-0.26, which characterize penicillin binding proteins, peptidoglycan deacetylase proteins and antibiotics as small molecules, is critical for formation and sustainability of peptidoglycan mesh. To investigate this possibility, we have also analyzed resonant frequencies of peptidoglycan mesh.

The peptidoglycan mesh is a polymer consisting of sugars and amino acids that forms a mesh-like layer outside of plasma membrane of most bacteria, forming the cell wall. The sugar component consists of alternating residues NAG and NAM, while short peptides (4-5 amino acids) are cross-linking sugar polymers (NAG/NAM), as presented in Figure 1. Peptidoglycan serves as structural role in the bacterial cell wall, giving structural strength, as well as counteracting the osmotic pressure of the cytoplasm.

Based on RRM approach, it would be possible to influence such bio macromolecular structure through the resonance at its periodicity (characteristic frequency) with the aim to destabilize the peptidoglycan structure. When periodicity of NAM/NAG structure is numerically calculated, using Fourier Transform with assumption that distance between NAM and NAG is arbitrary value of 1, it will show only one peak at arbitrary numerical frequency at 0.5. However, to be able to compare this frequency with RRM characteristic frequency of proteins, it is required to adjust for difference in distances between NAM and NAG residues along sugar backbone to distance between amino acids along the protein backbone. It has been experimentally measured that the length of 2-repeat disaccharide units (NAM-NAG-NAM-NAG) is 30Å, meaning that distance between two neighboring residues is 7.5Å [30]. Thus, the previously calculated frequency of 0.5 for NAM/NAG polymer can be translated into RRM frequencies relevant for amino acids chains by adjusting distance of 7.5Å between NAM and NAG residues to distance of 3.8Å between amino acids along protein backbone. With this adjustment the periodicity (characteristic frequency) of NAM/NAG polymer is calculated to be at f5=0.2533 (0.5x3.8/7.5).

It is interesting to note that this frequency f5 is also within the range of antibiotics such as small molecules, penicillin binding proteins, as well as peptidoglycan deacetylase proteins. As all these different types of molecules are related to

peptidoglycan mesh in some way as mentioned above, we propose that this frequency range is critical for formation and sustainability of peptidoglycan mesh and as such could be the best target for inhibiting bacterial activity and avoiding bacterial antibiotic resistance.

4. Discussion

Here, we have utilized the RRM model to analyze important aspects of bacterial resistance, as well as possibility for inhibition of bacterial growth and activity.

Previously we have analyzed group of beta-lactamase proteins, which are produced by bacteria to fight against antibiotic activity, and we have found one common RRM characteristic frequency at f1=0.0352+0.0041, as presented in Figure 2. Consequently, we have already designed, using the RRM frequency f1, peptide capable of interfering with the process of antibiotic inactivation by beta-lactamase, which have been experimentally tested [18].

Here, we have investigated the possibility for inhibition of bacterial growth to mimic antibiotic activity through inhibition of penicillin binding proteins, which are crucial for development of bacterial peptidoglycan mesh. When we analyzed penicillin binding proteins from different bacterial strains two prominent common RRM frequencies have been identified at f2=0.2529+0.0017 and f3=0.2412+0.0017, as presented in Figure 3. It is interesting that the RRM characteristic frequency for beta-lactam ring of antibiotics, as small molecules, which is in the frequency range of 0.23-0.26, overlaps both RRM characteristic frequencies of penicillin binding proteins, indicating that this frequency range is crucial for interaction between penicillin binding proteins and antibiotics. In addition, we have also identified that same frequency range is characterizing peptidoglycan deacetylase, as well as sugar periodicity within peptidoglycan mesh.

All these results indicate that frequency range of 0.23-0.26 is related to peptidoglycan mesh formation and sustainability. Having identified RRM frequency range related to peptidoglycan mesh formation and sustainability, it would be possible to design peptides that can directly act on peptidoglycan mesh formation and sustainability and somehow mimic antibiotic activity. Such approach would give completely new direction to design novel peptide-based antibiotics and possibly eliminate problem of resistance bacteria.

Compliance with ethical standards

Disclosure of conflict of interest

Authors declare they have no competing interests.

Authors contribution

Conceptualization, I.C., D.C. and I.L.; Methodology, I.C. and D.C.; Software, D.C.; Resources, I.L.; Writing—Original Draft Preparation—Review and Editing, I.C., D.C. and I.L.

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Appendix A

Twenty-two active beta-lactamase proteins:

- >active 24-286 sp|P62593|BLAT_ECOLX Beta-lactamase TEM OS=*Escherichia coli* OX=562 GN=bla PE=1 SV=1
- >active 35-307 sp|P9WKD3|BLAC_MYCTU Beta-lactamase OS=*Mycobacterium tu-berculosis* (strain ATCC 25618 / H37Rv) OX=83332 GN=blaC PE=1 SV=1
- >active 35-307 sp|A5U493|BLAC_MYCTA Beta-lactamase OS=*Mycobacterium tu-berculosis* (strain ATCC 25177 / H37Ra) OX=419947 GN=blaC PE=1 SV=1
- >active 28-292 sp|P52663|BLAN_ENTCL Imipenem-hydrolyzing beta-lactamase OS=Enterobacter cloacae OX=550 GN=nmcA PE=1 SV=1
- >active 22-286 sp|Q9S169|BLA24_ECOLX Beta-lactamase SHV-24 OS=*Escherichia coli* OX=562 GN=bla PE=1 SV=1
- >active 30-291 sp|Q47066|BLT1_ECOLX Beta-lactamase Toho-1 OS=*Escherichia coli* OX=562 GN=bla PE=1 SV=1
- >active 35-307 sp|P9WKD2|BLAC_MYCTO Beta-lactamase OS=*Mycobacterium tu-berculosis* (strain CDC 1551 / Oshkosh) OX=83331 GN=blaC PE=3 SV=1
- >active 35-307 sp|P0A5I7|BLAC_MYCBO Beta-lactamase OS=Mycobacterium bovis (strain ATCC BAA-935 / AF2122/97) OX=233413 GN=blaC PE=3 SV=1
- >active 34-275 sp|O07293|BLO18_PSEAI Beta-lactamase OXA-18 OS=*Pseudomonas aeruginosa* OX=287 GN=bla PE=1 SV=1
- >active 28-290 sp|P23954|BLO2_KLEOX Beta-lactamase OXY-2 OS=*Klebsiella oxy-toca* OX=571 GN=bla PE=1 SV=2
- >active 22-286 sp|Q9S424|BLA13_KLEPN Beta-lactamase SHV-13 OS=*Klebsiella pneumoniae* OX=573 GN=bla PE=3 SV=1
- >active 29-291 sp|P28585|BLC1_ECOLX Beta-lactamase CTX-M-1 OS=*Escherichia coli* OX=562 GN=bla PE=1 SV=2
- >active 25-291 sp|P22391|BLO1_KLEOX Beta-lactamase OXY-1 OS=*Klebsiella oxy-toca* OX=571 GN=bla PE=1 SV=1
- >active 21-266 sp|Q06778|BLO11_PSEAI Beta-lactamase OXA-11 OS=*Pseudomonas aeruginosa* OX=287 GN=bla PE=3 SV=1
- >active 22-275 sp|Q51574|BLO15_PSEAI Beta-lactamase OXA-15 OS=*Pseudomonas aeruginosa* OX=287 GN=bla PE=1 SV=1
- >active 22-286 sp|O08337|BLA8_ECOLX Beta-lactamase SHV-8 OS=*Escherichia coli* OX=562 GN=bla PE=3 SV=1
- >active 22-286 sp|P0A3M2|BLA5_PSEAI Beta-lactamase SHV-5 OS=*Pseudomonas aeruginosa* OX=287 GN=bla PE=3 SV=1
- >active 12-260 sp|P96348|BLA6_KLEPN Beta-lactamase SHV-6 (Fragment) OS=*Klebsiella pneumoniae* OX=573 GN=bla PE=3 SV=1
- >active 24-286 sp|Q48406|BLAT_KLEOX Beta-lactamase TEM-12 OS=*Klebsiella ox-ytoca* OX=571 GN=blaT-12b PE=1 SV=1
- >active 21-266 sp|Q9R976|BLO19_PSEAI Beta-lactamase OXA-19 OS=*Pseudomonas aeruginosa* OX=287 GN=bla PE=3 SV=1
- >active 22-286 sp|Q93LM8|BLA34_ECOLX Beta-lactamase SHV-34 OS=*Escherichia coli* OX=562 GN=bla PE=3 SV=1
- >active 27-308 sp|P37321|BLE1_PSEAI Extended-spectrum beta-lactamase PER-1 OS=*Pseudomonas aeruginosa* OX=287 GN=per1 PE=1 SV=1

Fourteen penicillin binding proteins (PBPs):

- >sp|P02918|PBPA_ECOLI Penicillin-binding protein 1A OS=*Escherichia coli* (strain K12) OX=83333 GN=mrcA PE=1 SV=1
- >sp|P76577|PBPC_ECOLI Penicillin-binding protein 1C OS=*Escherichia coli* (strain K12) OX=83333 GN=pbpC PE=1 SV=1

- >sp|P02919|PBPB_ECOLI Penicillin-binding protein 1B OS=*Escherichia coli* (strain K12) OX=83333 GN=mrcB PE=1 SV=2
- >sp|Q07868|PBP2B_BACSU Penicillin-binding protein 2B OS=*Bacillus subtilis* (strain 168) OX=224308 GN=pbpB PE=1 SV=3
- >sp|P70997|PBPG_BACSU Penicillin-binding protein 2D OS=*Bacillus subtilis* (strain 168) OX=224308 GN=pbpG PE=2 SV=3
- >sp|P42971|PBPC_BACSU Penicillin-binding protein 3 OS=*Bacillus subtilis* (strain 168) OX=224308 GN=pbpC PE=1 SV=1
- >sp|P38050|PBPF_BACSU Penicillin-binding protein 1F OS=*Bacillus subtilis* (strain 168) OX=224308 GN=pbpF PE=2 SV=2
- >sp|P54488|PBP2A_BACSU Penicillin-binding protein 2A OS=*Bacillus subtilis* (strain 168) OX=224308 GN=pbpA PE=1 SV=1
- >sp|O32032|PBPI_BACSU Penicillin-binding protein 4B OS=*Bacillus subtilis* (strain 168) OX=224308 GN=pbpI PE=2 SV=1
- >sp|Q8DR59|PBPA_STRR6 Penicillin-binding protein 1A OS=*Streptococcus pneu-moniae* (strain ATCC BAA-255 / R6) OX=171101 GN=pbpA PE=1 SV=1
- >sp|Q796K8|PBPH_BACSU Penicillin-binding protein H OS=*Bacillus subtilis* (strain 168) OX=224308 GN=pbpH PE=1 SV=3
- >sp|Q00573|PBPA_STROR Penicillin-binding protein 1A (Fragment) OS=*Streptococcus oralis* OX=1303 GN=ponA PE=3 SV=1
- >sp|P59676|PBPX_STRR6 Penicillin-binding protein 2X OS=*Streptococcus pneu-moniae* (strain ATCC BAA-255 / R6) OX=171101 GN=pbpX PE=1 SV=1
- >sp|P14677|PBPX_STRPN Penicillin-binding protein 2x OS=*Streptococcus pneu-moniae* serotype 4 (strain ATCC BAA-334 / TIGR4) OX=170187 GN=pbpX PE=1 SV=2

Nine active peptidoglycan deacetylase proteins:

- >active sp|A0A0H3GDH9|PGDA_LISM4 Peptidoglycan-N-acetylglucosamine deacetylase PgdA OS=Listeria monocytogenes serotype 1/2a (strain 10403S) OX=393133 GN=pgdA PE=2 SV=1
- >active sp|A0A3Q0NBH7|PGDA_LISMG Peptidoglycan-N-acetylglucosamine deacetylase PgdA OS=Listeria monocytogenes serotype 1/2a (strain EGD / Mackaness) OX=1334565 GN=pgdA PE=1 SV=1
- >active sp|Q8Y9V5|PGDA_LISMO Peptidoglycan-N-acetylglucosamine deacetylase PgdA OS=Listeria monocytogenes serovar 1/2a (strain ATCC BAA-679 / EGD-e) OX=169963 GN=pgdA PE=1 SV=1
- >active sp|Q8DP63|PGDA_STRR6 Peptidoglycan-N-acetylglucosamine deacetylase OS=Streptococcus pneumoniae (strain ATCC BAA-255 / R6) OX=171101 GN=pgdA PE=1 SV=1
- >active sp|Q81EK9|PGDA1_BACCR Peptidoglycan-N-acetylglucosamine deacety-lase BC_1960 OS=Bacillus cereus (strain ATCC 14579 / DSM 31 / JCM 2152 / NBRC 15305 / NCIMB 9373 / NRRL B-3711) OX=226900 GN=BC_1960 PE=1 SV=1
- >active sp|Q81EJ6|PGDA2_BACCR Peptidoglycan-N-acetylglucosamine deacety-lase BC_1974 OS=Bacillus cereus (strain ATCC 14579 / DSM 31 / JCM 2152 / NBRC 15305 / NCIMB 9373 / NRRL B-3711) OX=226900 GN=BC_1974 PE=1 SV=1
- >active sp|Q81AF4|PGDA3_BACCR Peptidoglycan-N-acetylglucosamine deacety-lase BC_3618 OS=Bacillus cereus (strain ATCC 14579 / DSM 31 / JCM 2152 / NBRC 15305 / NCIMB 9373 / NRRL B-3711) OX=226900 GN=BC_3618 PE=1 SV=1
- >active 2-293 sp|O25080|PGDAE_HELPY Peptidoglycan deacetylase OS=Helicobacter pylori (strain ATCC 700392 / 26695) OX=85962 GN=pgdA PE=1 SV=1
- >active 2-293 sp|B5ZA76|PGDAE_HELPG Peptidoglycan deacetylase OS=Helicobacter pylori (strain G27) OX=563041 GN=pgdA PE=1 SV=1