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Organic Reactions between Therapeutic Compounds and Pathogenic Microorganism Cellular Chemical Components

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Abstract

The biological properties of therapeutic compounds to degrade pathogenic microorganisms have been explained from organic chemistry perspective. Precisely, this paper has properly shown the mode of action concerning acidic therapeutic compounds possessing hydroxyl or carboxylic groups including alkaloid therapeutic compounds bearing a nitrogen atom or an amine group. In other words, this conceptual research has disclosed detailed reaction mechanisms, which demonstrate how an adequate medicine can neutralize or degrade one of the essential microbial cellular chemical constituents, such as the deoxyribonucleic acid (DNA).

Keywords

Therapeutic Compounds, Reaction Mechanisms, Glycoproteins, Glucolipids, Phospholipids, Deoxyribonucleic Acid (DNA)

1. Introduction

1.1. Glycoprotein

A glycoprotein is an organic compound bearing a glycosyl motif bound to a proteinic portion. It is found in all cells of the leaving organisms such as animals, bacteria, virus, and plants [1]. It reacts with nucleophilic extracellular substances such as medicines to assist them to penetrate the intracellular environment (Figure 1).

1.2. Glycolipid

A glycolipid is one of the cellular membrane organic compounds functioning as a

receptor of the adequate extracellular chemical substances, which intend to pass through the cellular membrane [1]. It is a combination of a glycosyl residue connected to a third carbon atom of a glycerol moisty, at which the first and the second carbon atoms are respectively attached to a fatty acid molecule (**Figure 2**).

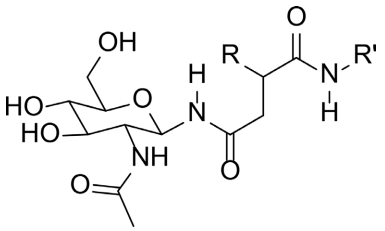


Figure 1. Glycoprotein definition.

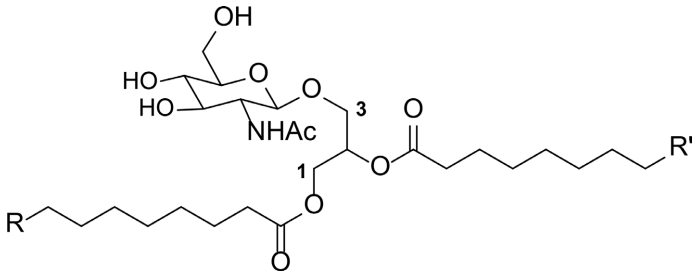


Figure 2. Glycolipid definition.

1.3. Phospholipid

A phospholipid is a cellular membrane component, which is an organic substance formed by a glycerol residue, a phosphate group and two fatty acids [1]. Indeed, the two fatty acids are bound to the first and to the second glycerol carbon atom, while the third glycerol carbon atom bears the phosphate group. It is also an appropriate cellular receptor for nucleophilic extracellular substances, which cross the cellular membrane in order to reach the cellular cytoplasm (**Figure 3**).

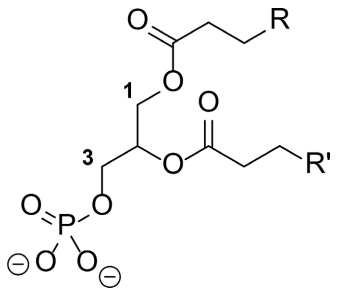


Figure 3. Phospholipid definition.

1.4. Therapeutic Organic Compounds

Therapeutic organic compounds are natural or synthetic organic substances susceptible to destroy or inhibit pathogen microorganisms by preventing their development. Indeed, I have read the literature regarding therapeutic organic com-

pounds towards pathogenic microorganisms, and I have found that their action mechanisms do not properly taking account the organic reactions. To alleviate this type of insufficient, I have proposed organic reaction mechanisms, which constitute an added value to enhance the comprehension of the biological activities of pharmaceutical substances in comparison to those already published that do not disclose the advanced organic reactions involving healing compounds et pathogenic microorganism cellular constituents such as glycoproteins, glycolipids as well as phospholipids [2]-[7]. It is important to herein remind that this paper aims to demonstrate detailed reaction mechanism in order to elucidate the neutralization of the essential microbial cellular chemical constituents, such as proteins, phospholipids, glucolipids or deoxyribonucleic acid (DNA). In the same perspective, therapeutic compounds studied into this conceptual paper have been selected in respect to their categories containing functional groups such as hydroxyl, amine and carboxylic group.

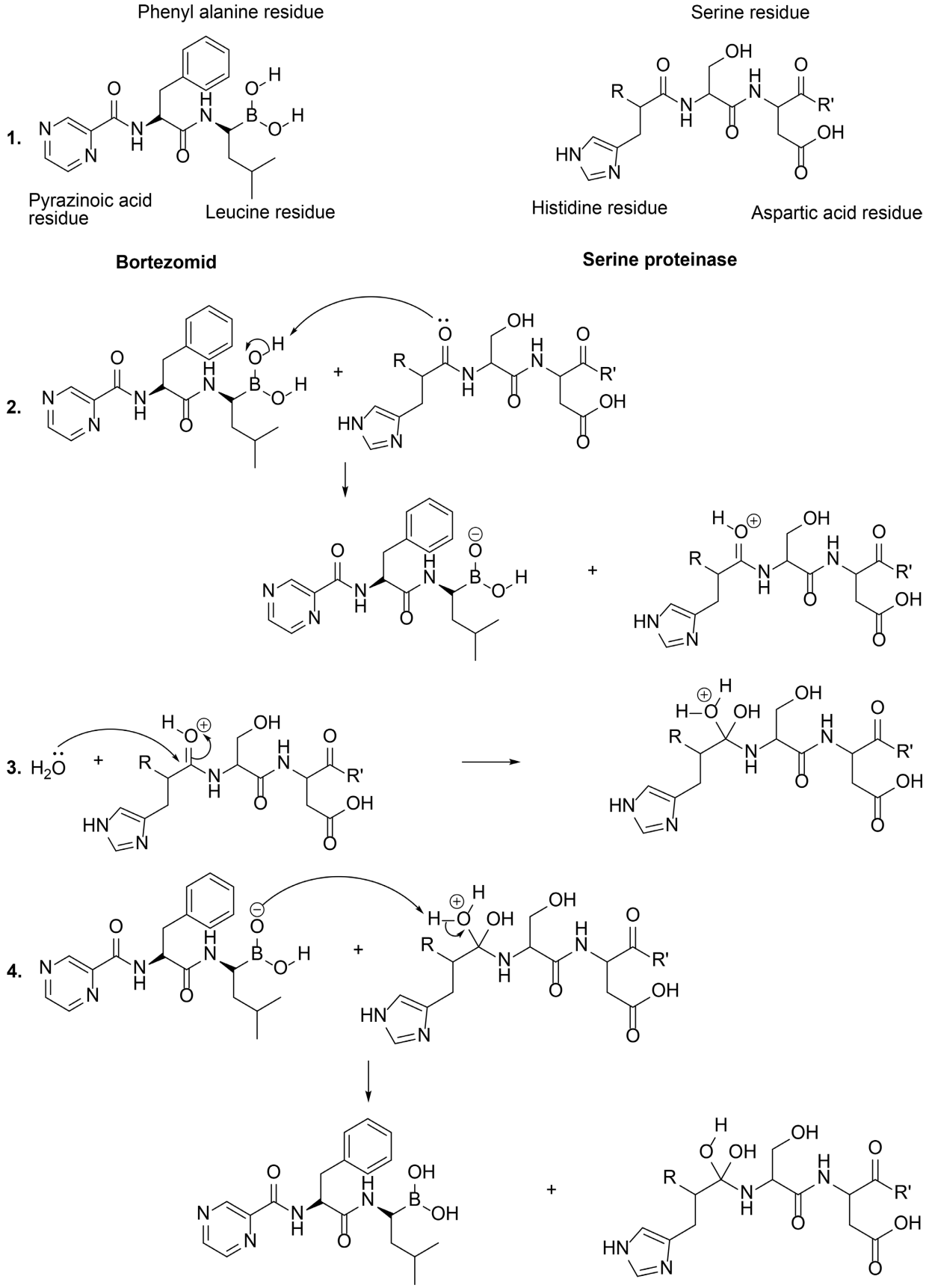
2. Dipeptide Boronic Acid

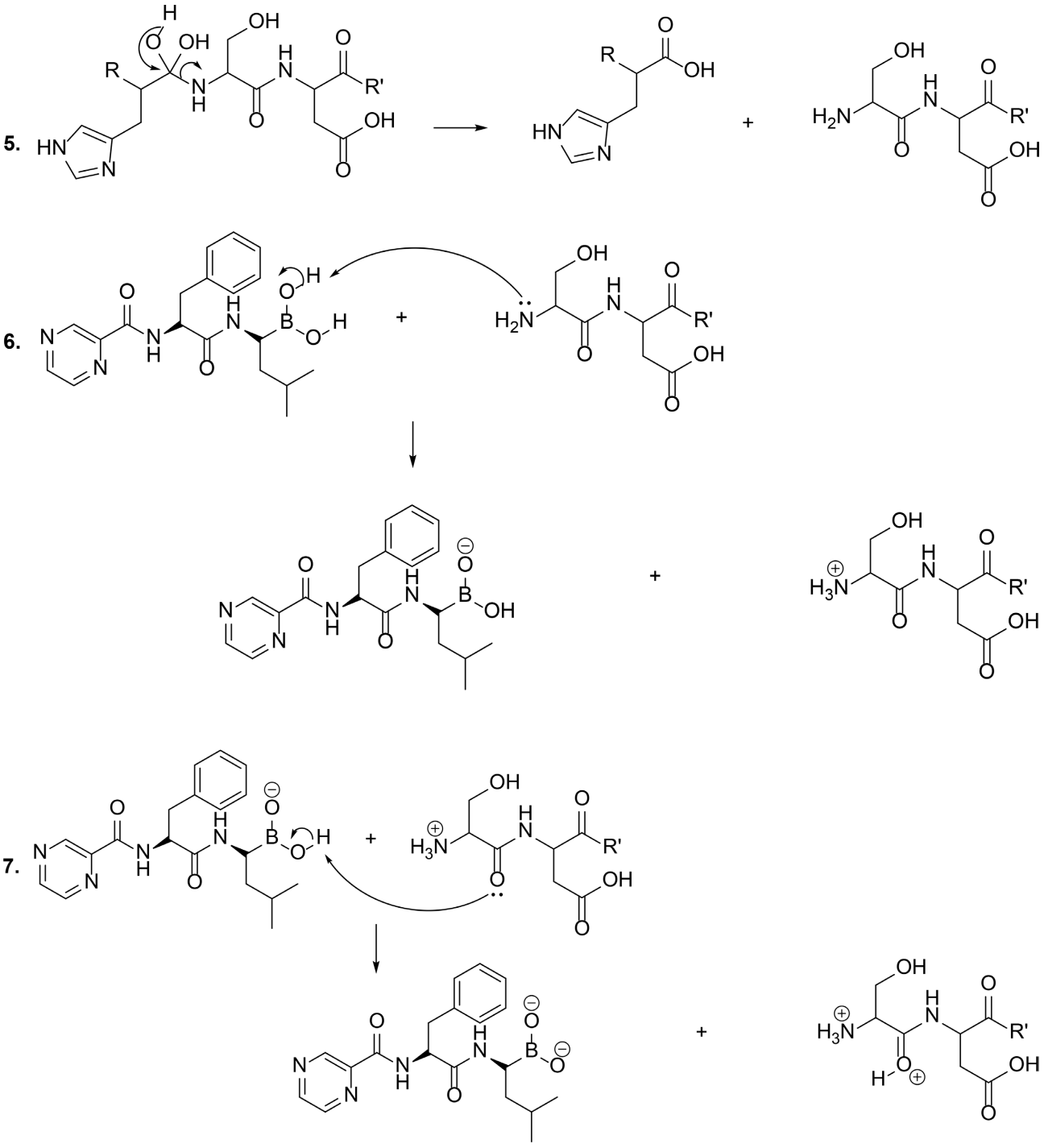
Dipeptide boronic acid, known as bortezomib, is an organic therapeutic compound containing functional groups that are capable to donate protons to nucleophiles or bases [8]-[11]. This therapeutic compound neutralizes pathogen organisms because it has been designed to react mainly with microbial proteinase or an enzyme which accelerates the destruction of microbial proteins, which are essential for the pathogen organism development [8]-[11]. Regarding the mechanism of action of the therapeutic compound, this later will donate protons to proteinase to yield the corresponding products (**Scheme 1**, reaction 2). Then a molecule of water will finalize the deterioration of the targeted proteinase (**Scheme 1**, reaction 3). It has been reported that histidine, serine and aspartic acid residues constitute a catalytic region of serine proteinase [12].

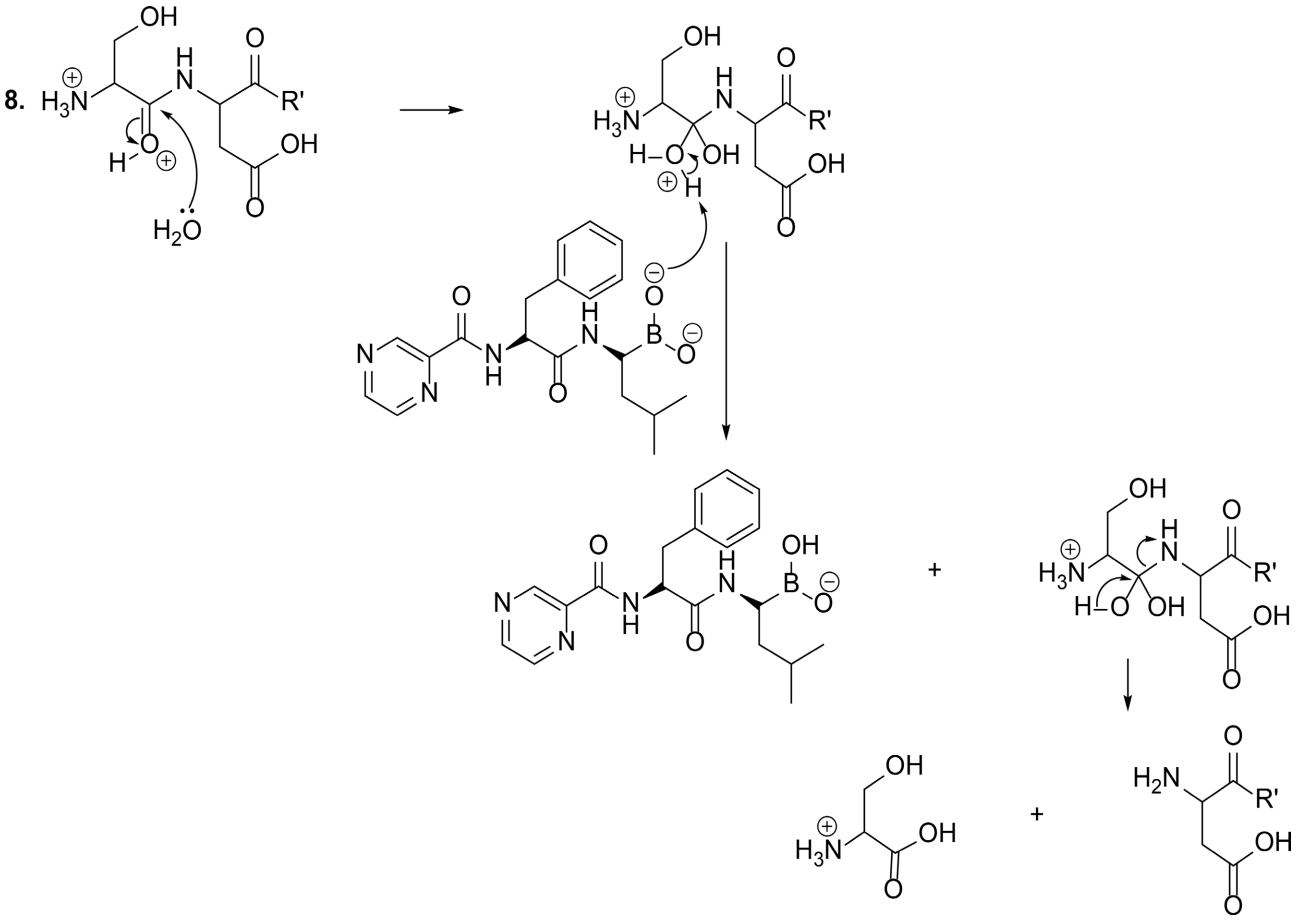
3. Berberine

This therapeutic compound has medicinal properties against diarrhea, microbial pathogens as well as gastrointestinal suffering [13]. The structure of this compound shows functional groups where organic reactions can take place. Indeed, the proposed reaction mechanism, plausibly explains the reaction between berberine hydroxyl group and the phospholipid of the pathogenic organism cellular membrane, is that a phosphate group of phospholipid accepts a proton of the berberine hydroxyl group to generate a conjugate base of berberine and the protonated phospholipid (**Scheme 2**, reaction 1). This step is followed by two substitution reactions due to nucleophilic addition of two equivalents (eq.) of berberine conjugate base towards two fatty acid substituents of the protonated phospholipid, and the same conjugate base will reaction with the resulting phosphoglycerate to displace a corresponding leaving group (**Scheme 2**, reaction 2, reaction 3).

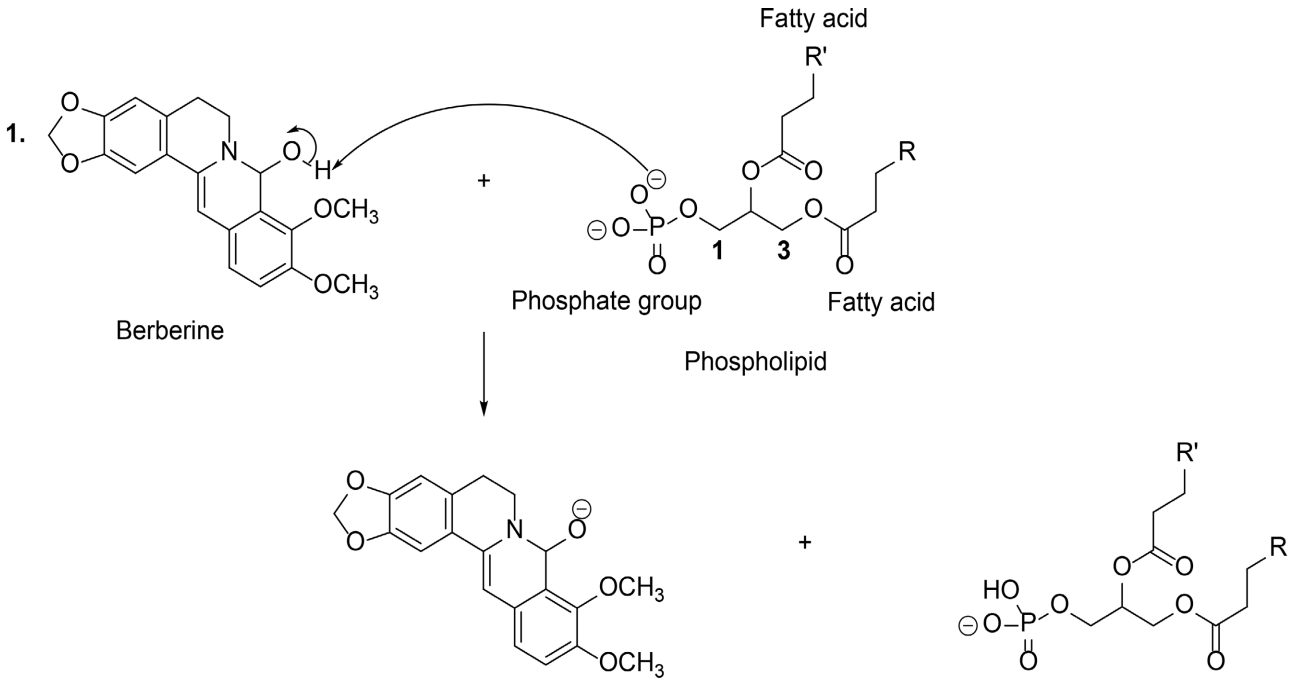
When the berberine analogue has reached the cellular cytoplasm, the parent

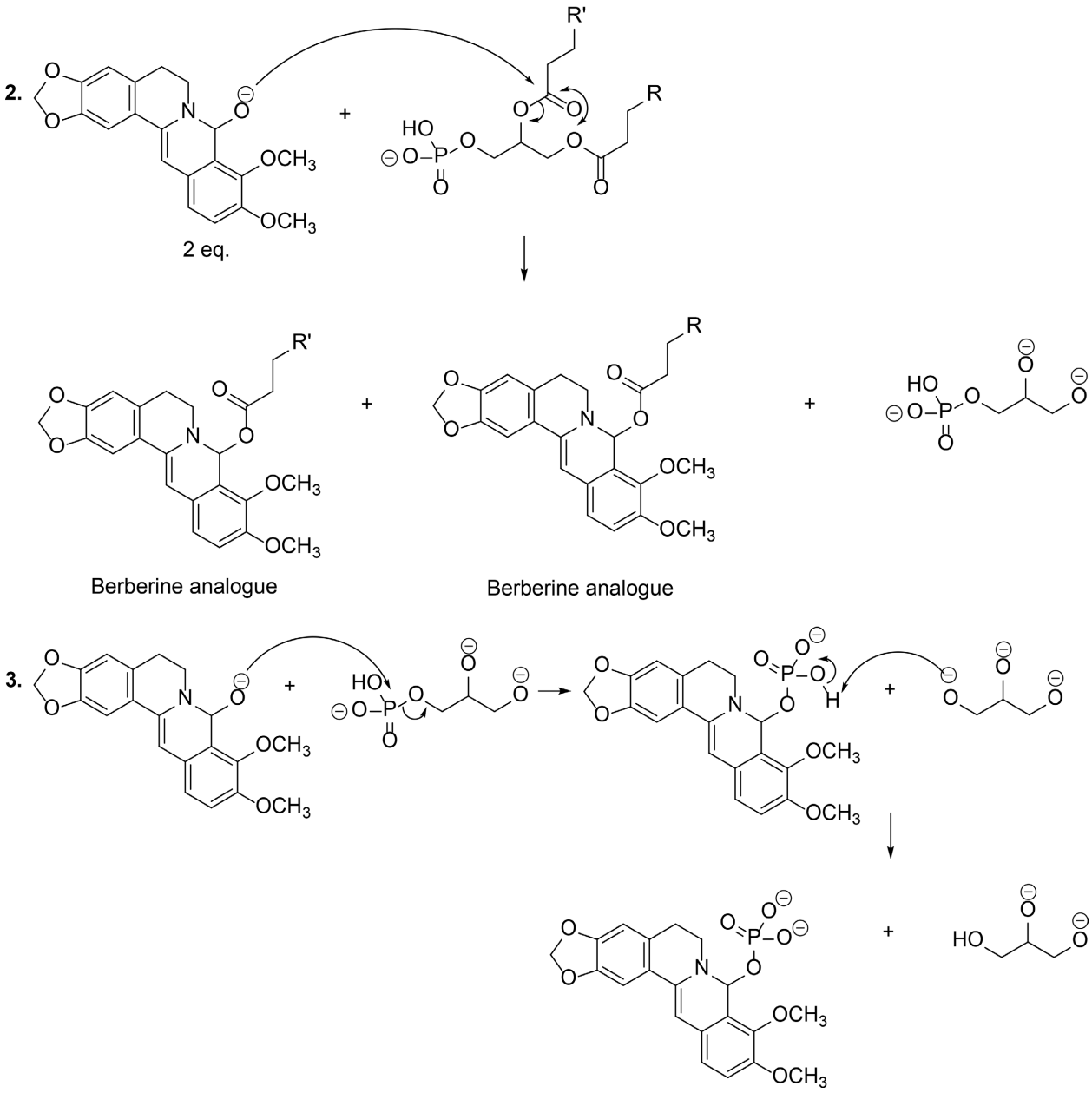

Phenyl alanine residue
Serine residue
1.
Pyrazinoic acid residue
Leucine residue
Histidine residue
Aspartic acid residue
Bortezomid
Serine proteinase
2.
3.
4.





Scheme 1. Dipeptide boronic acid chemistry.



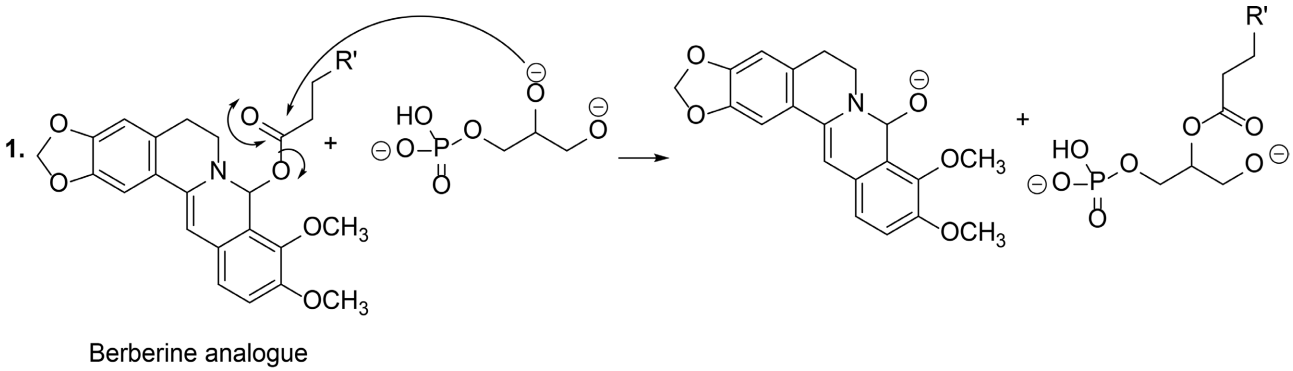


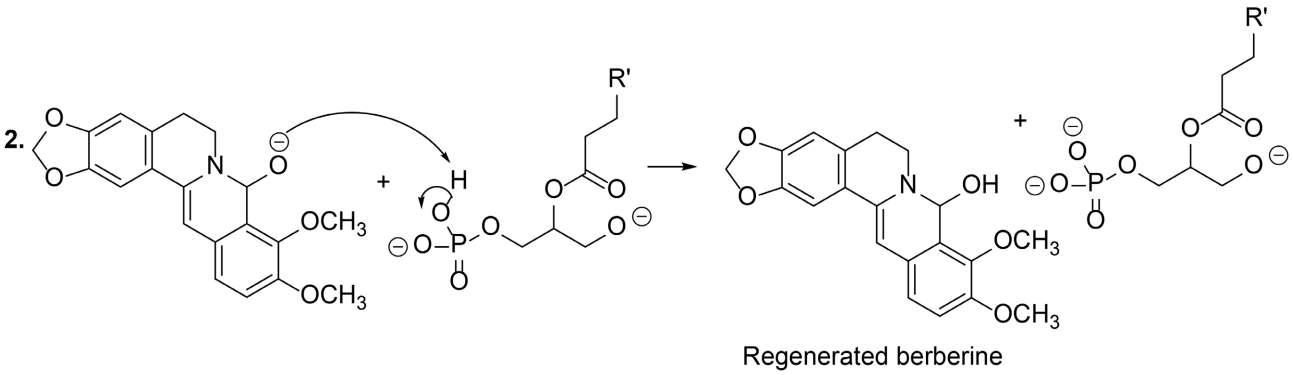
Scheme 2. Berberine chemistry.

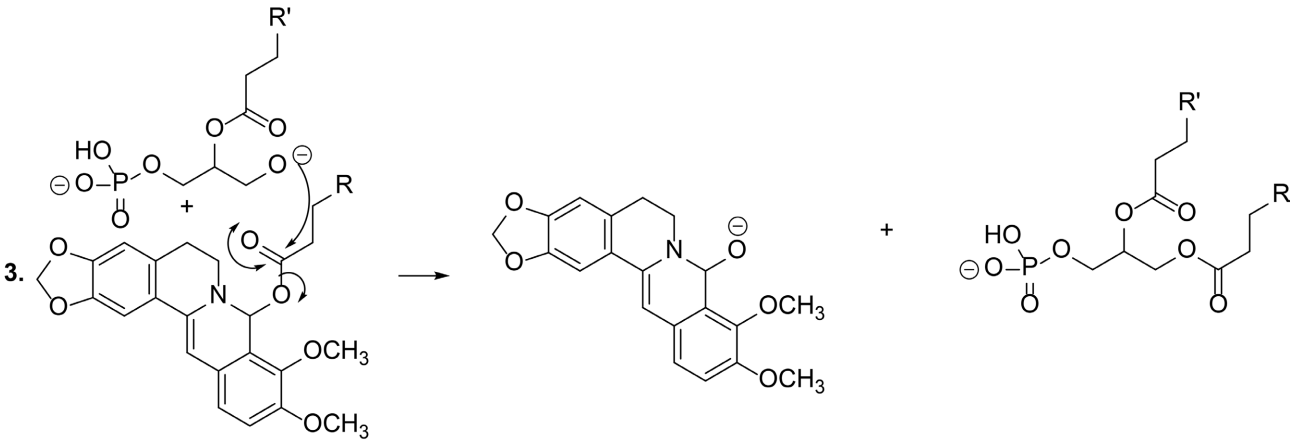
compound or the original one will be regenerated as well as the phospholipid to allow berberine to destroy or react with deoxyribonucleic acid (DNA). Consequently, the targeted pathogenic organism can no longer survive (Scheme 3, reaction 5).

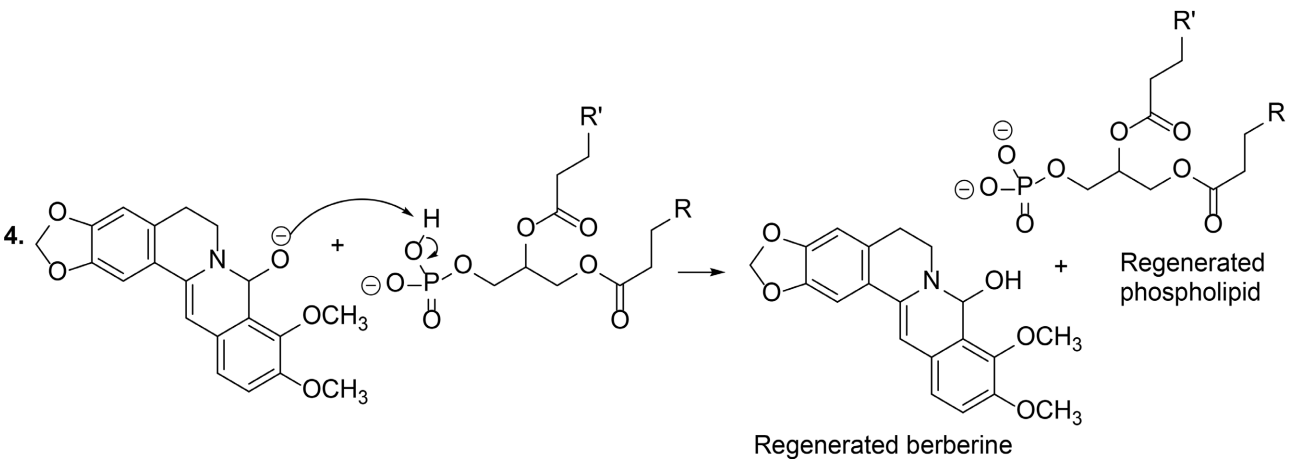
Berberine can be regenerated through the reaction between the intermediate berberine phosphate and the appropriate nucleophile (Scheme 4).

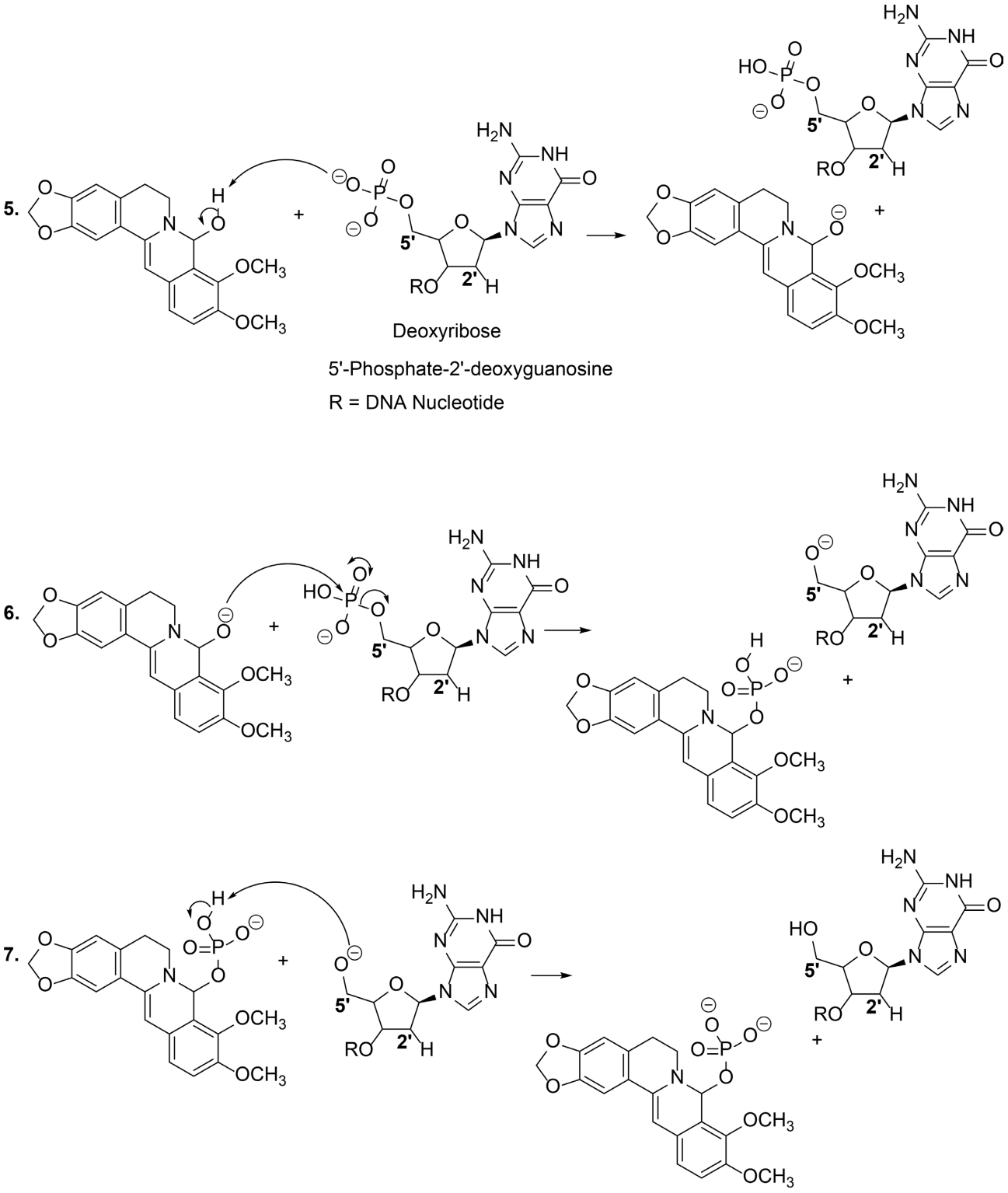
Experimental studies have revealed that in acid environment, the structure of berberine possesses quaternary nitrogen resulting from the displacement of a molecule of water because berberine reacts with mineral acids such as hydrogen chloride or sulfuric acid (Scheme 5) [14].


1.
R'
HO
OCH3
OCH3
Berberine analogue

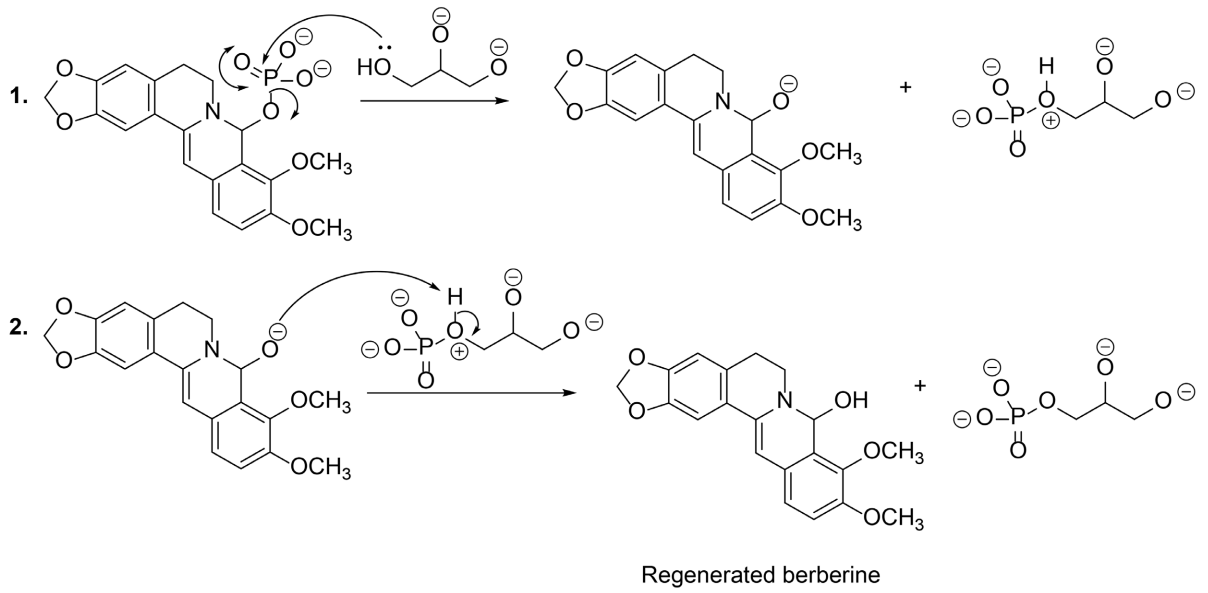

2.
R'
H
OH
OCH3
OCH3
Regenerated berberine


3.
R'
R
HO
OCH3
OCH3

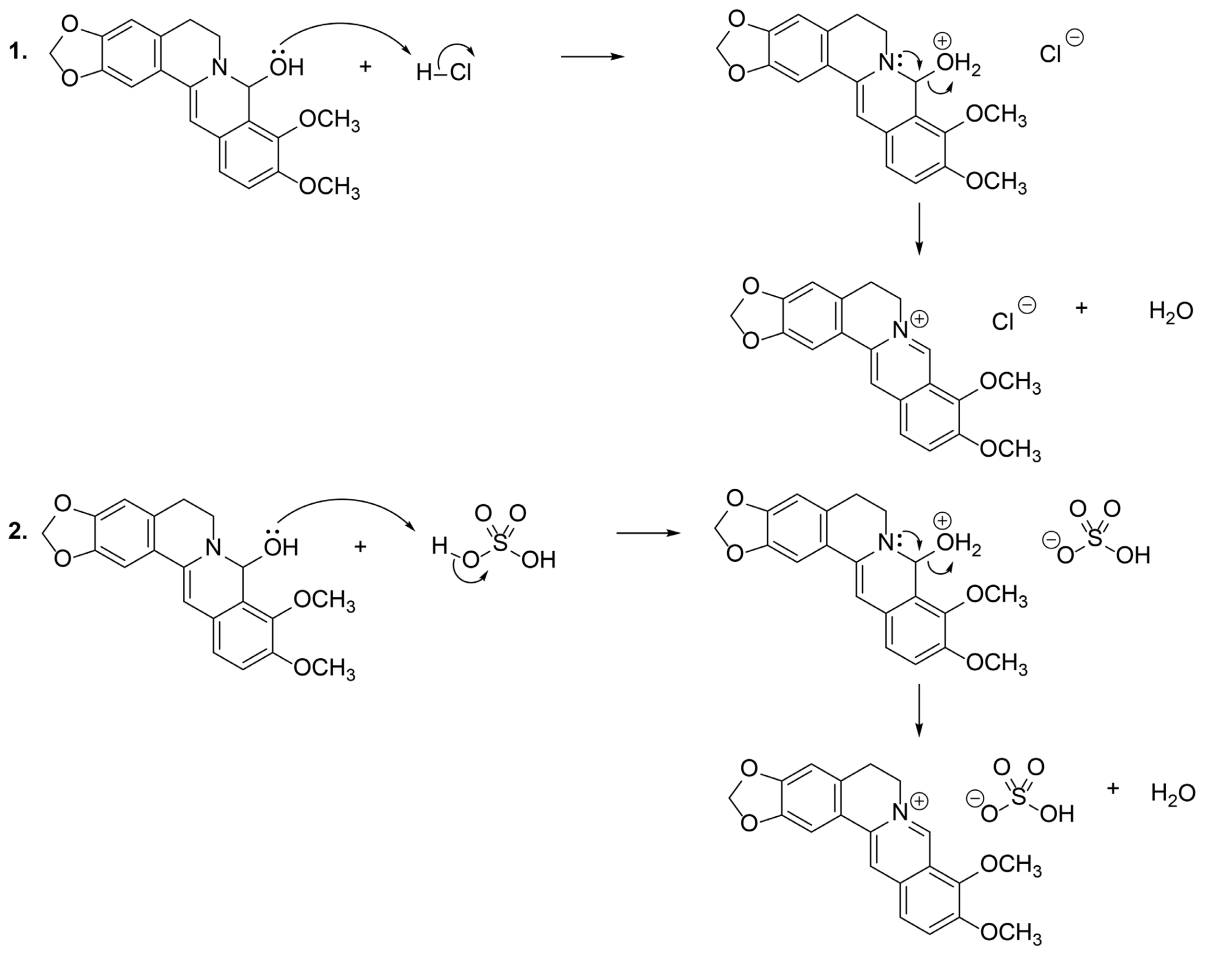

4.
R'
R
H
OH
OCH3
OCH3
Regenerated phospholipid
Regenerated berberine



Scheme 3. Berberine analogue chemistry.



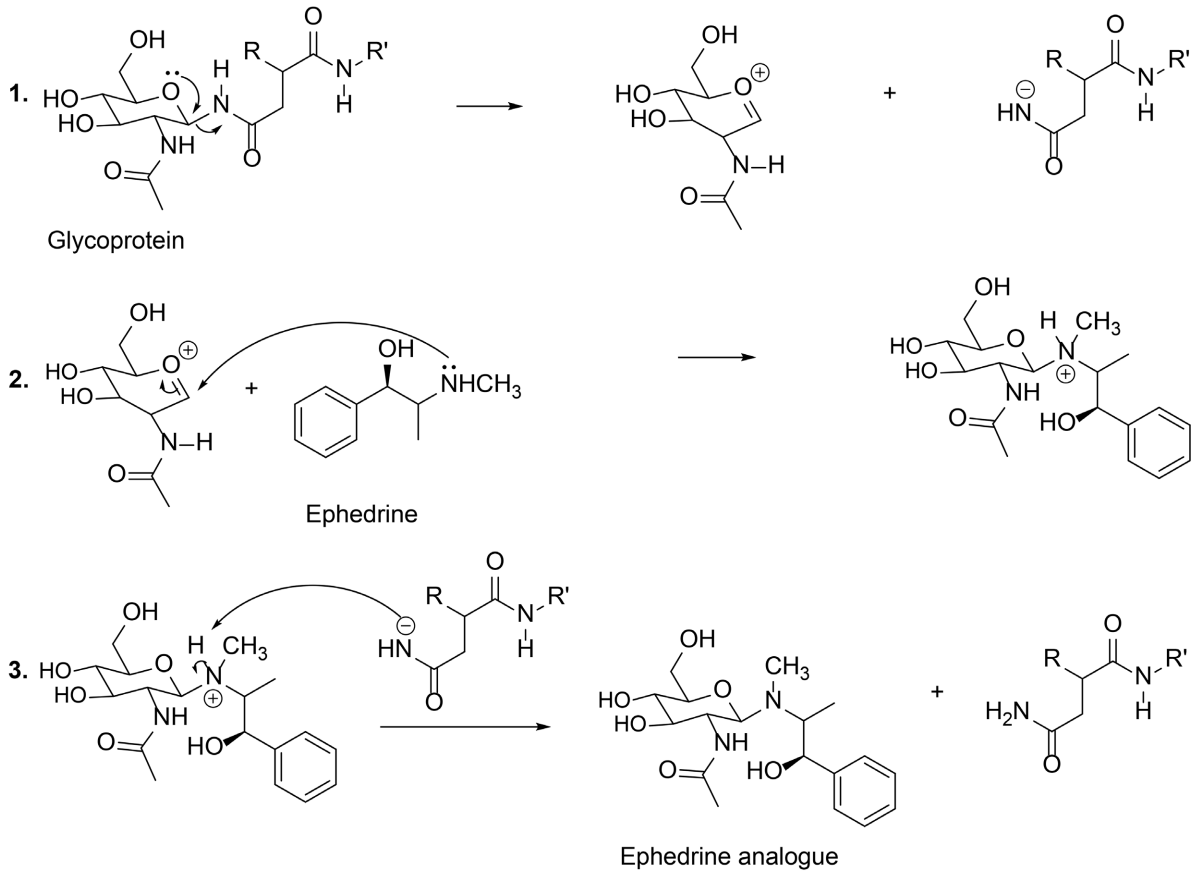
Scheme 4. Berberine regeneration.



Scheme 5. Acid environment berberine chemistry.

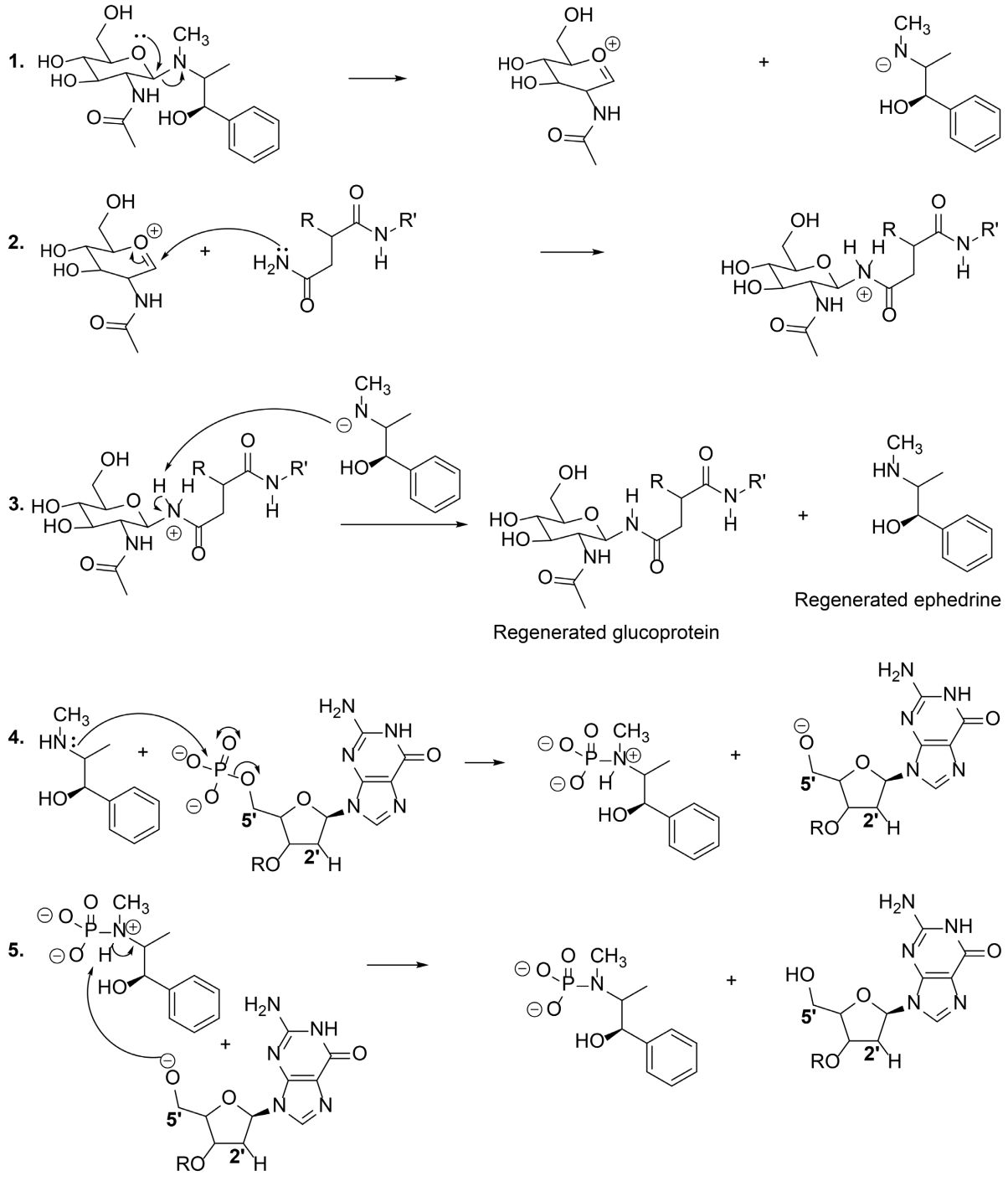
4. Ephedrine

This pharmaceutical compound is an alkaloid containing an amine functional group where an interesting reaction towards the targeted microbe occurs. This therapeutic compound utilizes the electron density around an intrinsic nitrogen atom to react with the cellular membrane glycoproteins. The plausible reaction mechanism starts with the disintegration of the glycoprotein due to an electron lone pair, on the intra oxygen, to generate a cyclic oxocarbonium ion, which is an excellent electrophile or more reactive than the entire glycoprotein due to its chair-like conformation. This conformation des not align the electron lone pair on the intra oxygen antiperiplanarly to the leaving group (Scheme 6, reaction 1) [15]. This step is followed by the nucleophilic addition of an electron lone pair of the nitrogen atom to produce the corresponding ammonium compound, which gives a proton to an adequate nucleophile to furnish the glycoprotein analogue, which can diffuse or migrate through the cellular membrane up to the cellular cytoplasm (Scheme 6, reaction 2, reaction 3).



Scheme 6. Ephedrine alkaloid chemical properties.

Within the cellular cytoplasm, the ephedrine analogue will react with the nucleophilic reactive entity to regenerate glycoprotein and the therapeutic original ephedrine that will now react with the deoxyribonucleic acid (DNA) (Scheme 7).



Scheme 7. Ephedrine analogue chemistry.

5. Aspirin

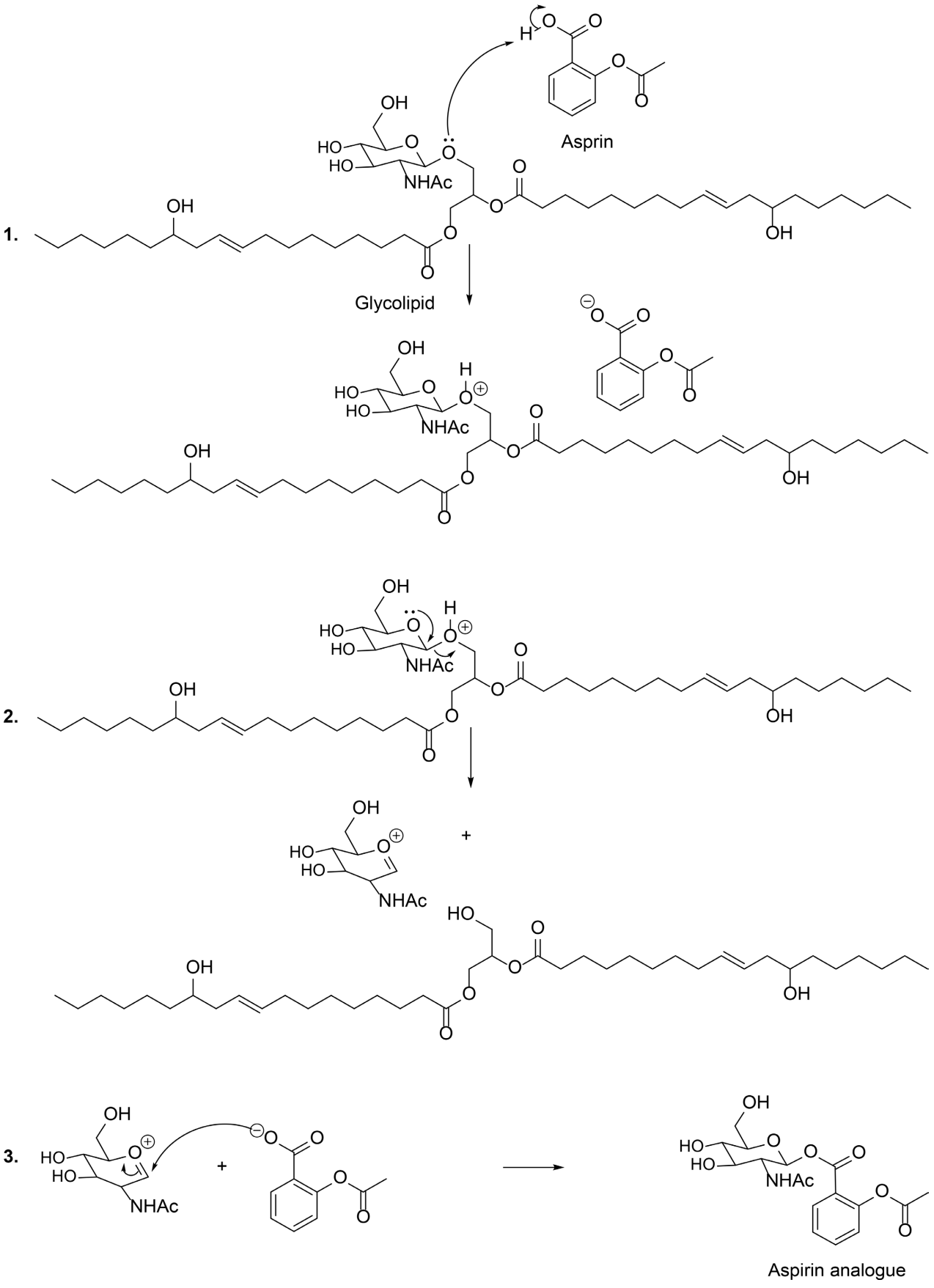
Experimental observations have disclosed that aspirin has diverse medicinal properties particularly anticancer, anti cholesterol, antibacterial dissemination including anti-inflammatory properties [16]-[19]. The structure of this therapeutic compound shows a carboxylic function group, which can react with glycolipid to fur-

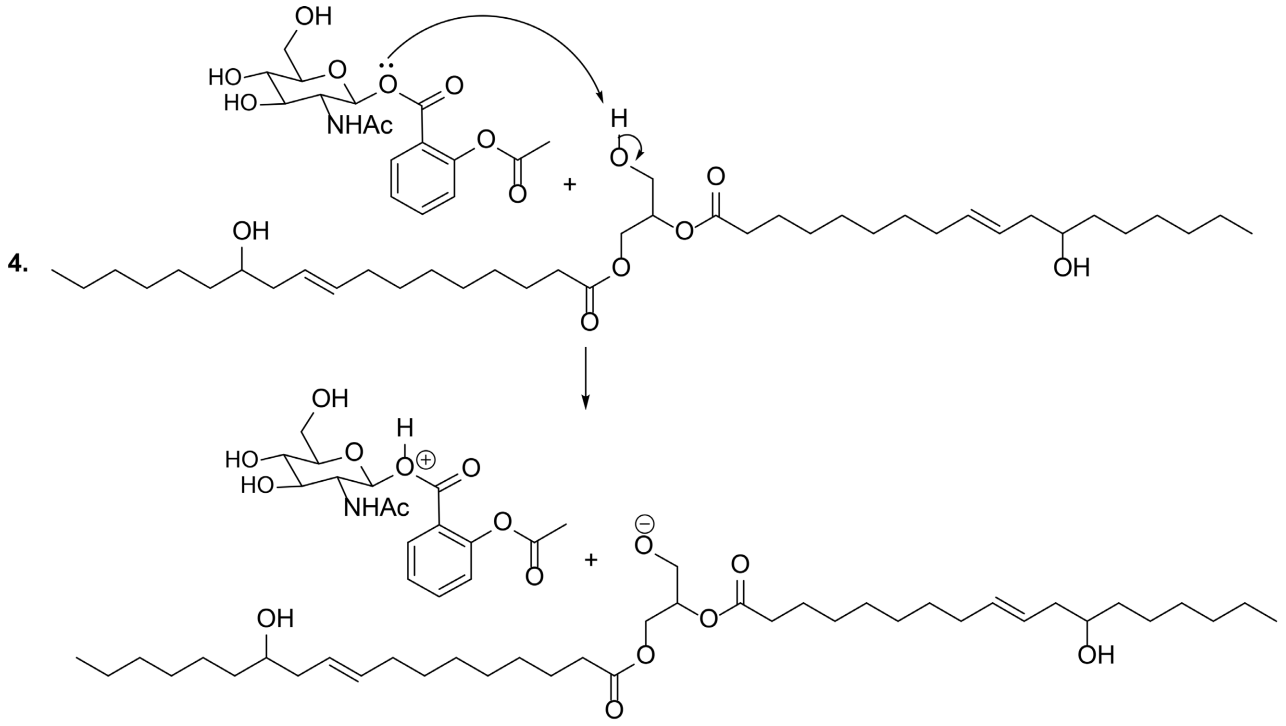
nish the corresponding products. This kind of reaction implies the protonation of the glycolipid to generate the corresponding products (**Scheme 8**, reaction 1). This step is followed by the electron lone pair delocalisation in order to displace the leaving group along with the carbo-oxonium ion (**Scheme 8**, reaction 2). In the third step, the aspirin conjugate base will combine with the carbo-oxonium ion to produce the corresponding aspirin analogue or glycolipid analogue because of a glycoside group bearing amide moisture is attached to aspirin (**Scheme 8**, reaction 3). Consequently, the aspirin analogue can now easily move through the cellular membrane to reach the pathogenic microorganism cytoplasm where the aspirin will be regenerated before reacting or neutralizing the pathogenic microorganism DNA in order to inhibit its development (**Scheme 8**, reaction 7). The glycolipid will be regenerated as well (**Scheme 8**, reaction 6).

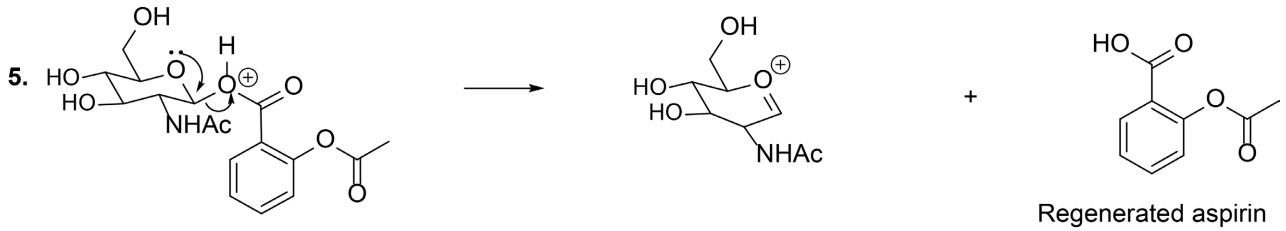
6. Scientific Literature

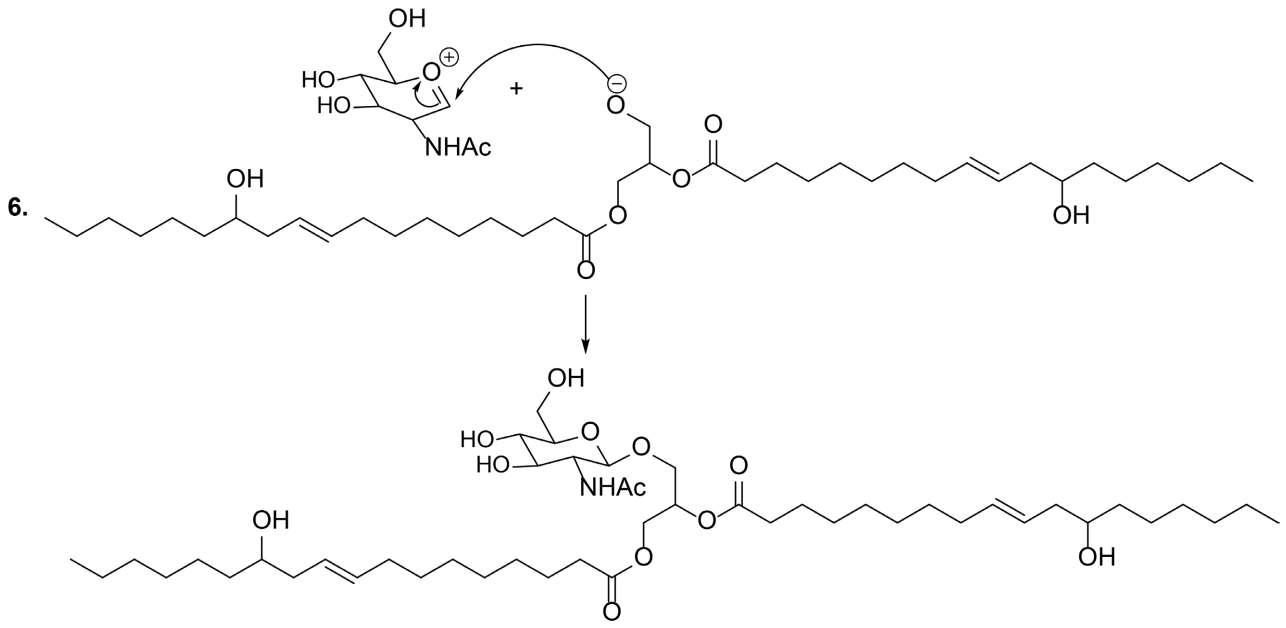
The proposed explanation model regarding the interactions between therapeutic compounds, and the pathogenic microorganism principal chemical components is essentially supported by experimental observations found in the scientific literature. Indeed, il has been reported that the medicines interact straight with the cellular membrane, and this kind of interaction is still disregarded [20] [21]. In the same perspective, it has also been reported that healing organic compounds react with cellular membrane chemical constituents so as they can pass through the cellular membrane to reach the cytoplasm [20] [21]. In other words, the capability of therapeutic compounds to react with the cellular membrane chemical constituents remains one of the significant therapeutic characteristic, which plays a fundamental role into their bioactive properties [20] [21]. In the same situation, Authors have found specifically during the experimental observations that rifampicin and levofloxacin react with the lipid phosphate group (**Scheme 9**, reaction 2) as well as the carbonyl group of lipids (**Scheme 9**, reaction 4) [22].

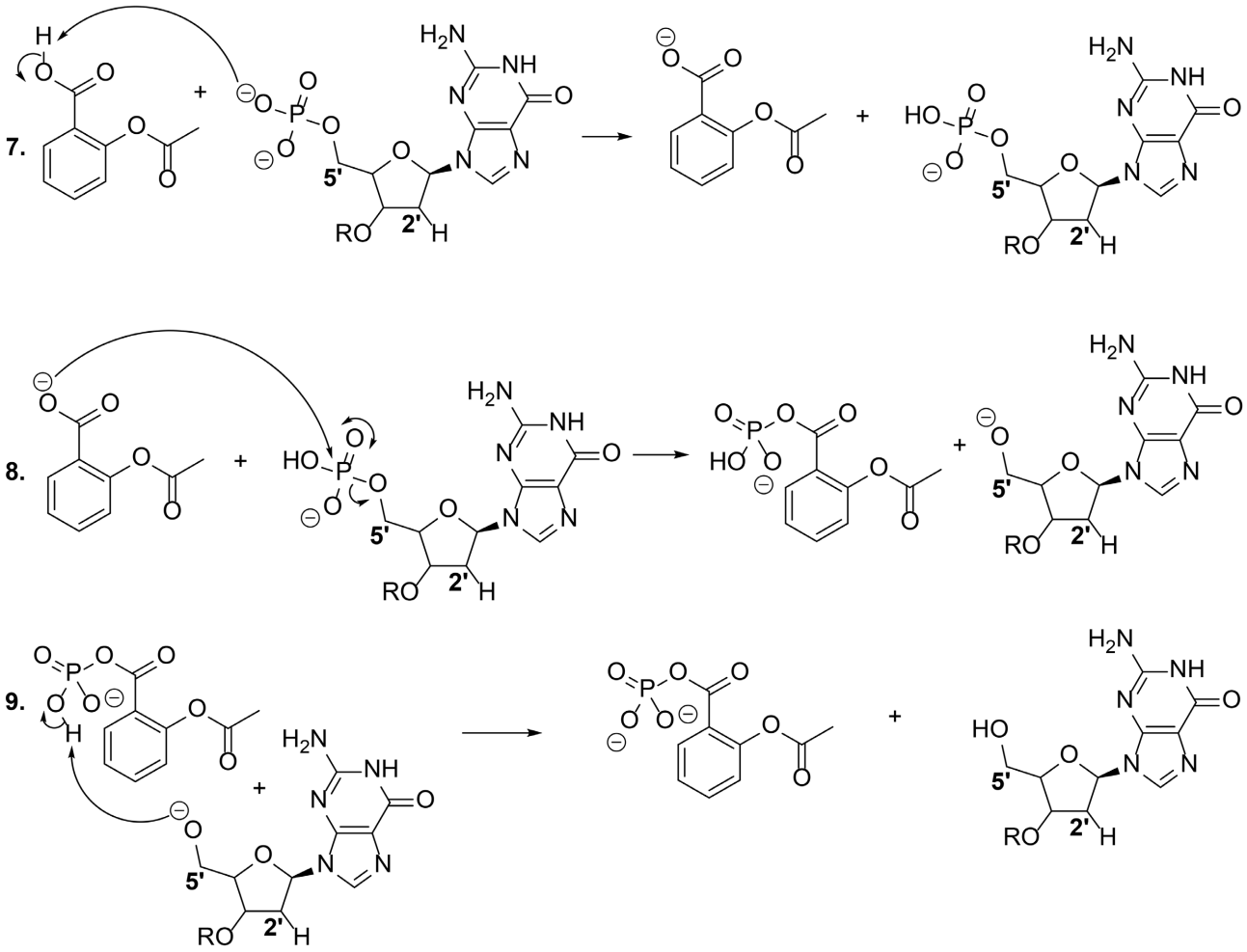
The plausible reaction mechanism for rifampicin and levofloxacin is due to their structure because the structure of an organic compound determines its reactivity. Accordingly, rifampicin can use its functional polar groups to interact with phosphate group (**Scheme 9**, reaction 2) or carbonyl group (**Scheme 9**, reaction 4) of the cellular membrane lipids, as nucleophiles in order to cross the cellular membrane and so to reach the cytoplasm [23]. Indeed, according to the electronegativity, the hydroxyl group oxygen atom is more electronegative than the nitrogen atom into the piperazine group, which is present into these two therapeutic compounds (**Scheme 9**). Therefore, the nitrogen atom behaves better as nucleophile than the oxygen atom because the nitrogen atom can easily concede its free double electrons to an electrophile such as a phosphate group to generate an analogue to phospholipid or the rifampicin bearing a phosphate group (**Scheme 9**, reaction 2). In the same perspective, it has been reported that glutamate, and phenylalanine play a fundamental role into the active site of ATPase (adenosine triphosphate enzyme) (**Scheme 9**, reaction 1), an enzyme which facilitates the


1.
Asprin
Glycolipid
2.
3.
Aspirin analogue

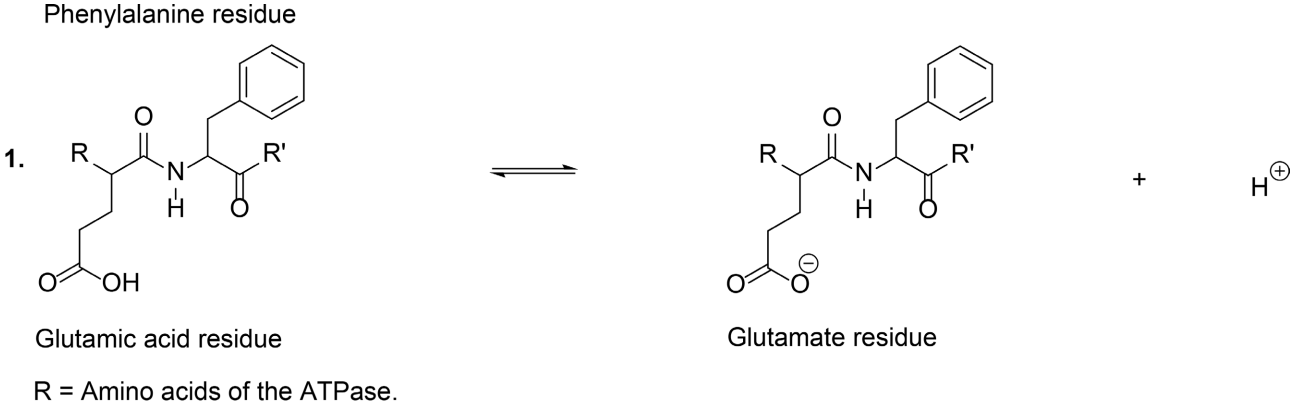

4.
OH
HO
HO
NHAc
O
H
+
OH
OH
HO
HO
NHAc
H
+
OH
OH

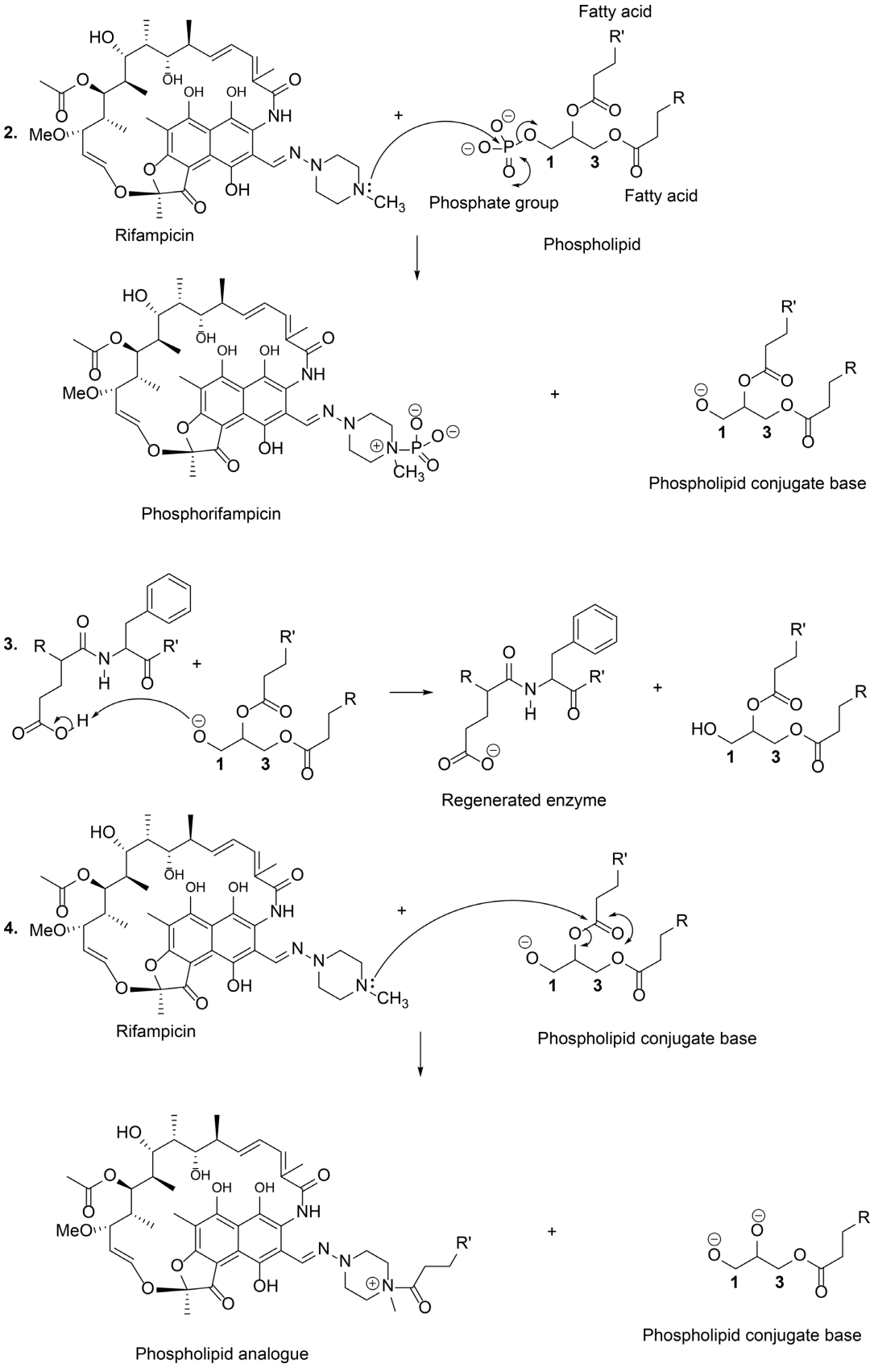

5.
OH
HO
HO
NHAc
H
OH
HO
HO
NHAc
+
HO
O
Regenerated aspirin


6.
OH
HO
HO
NHAc
+
OH
OH
OH
HO
HO
NHAc
OH
OH
Regenerated glycolipid



Scheme 8. Aspirin chemistry.

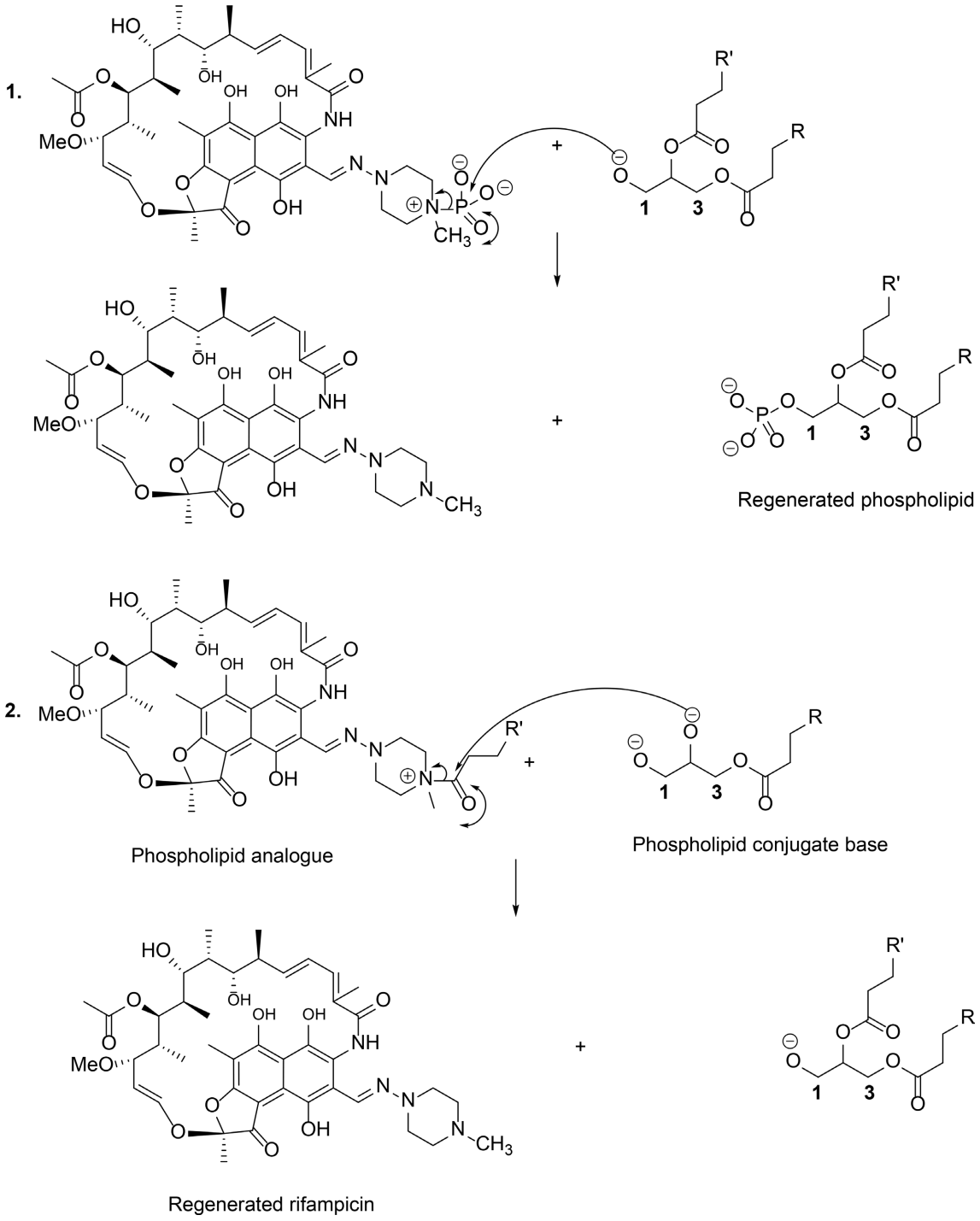


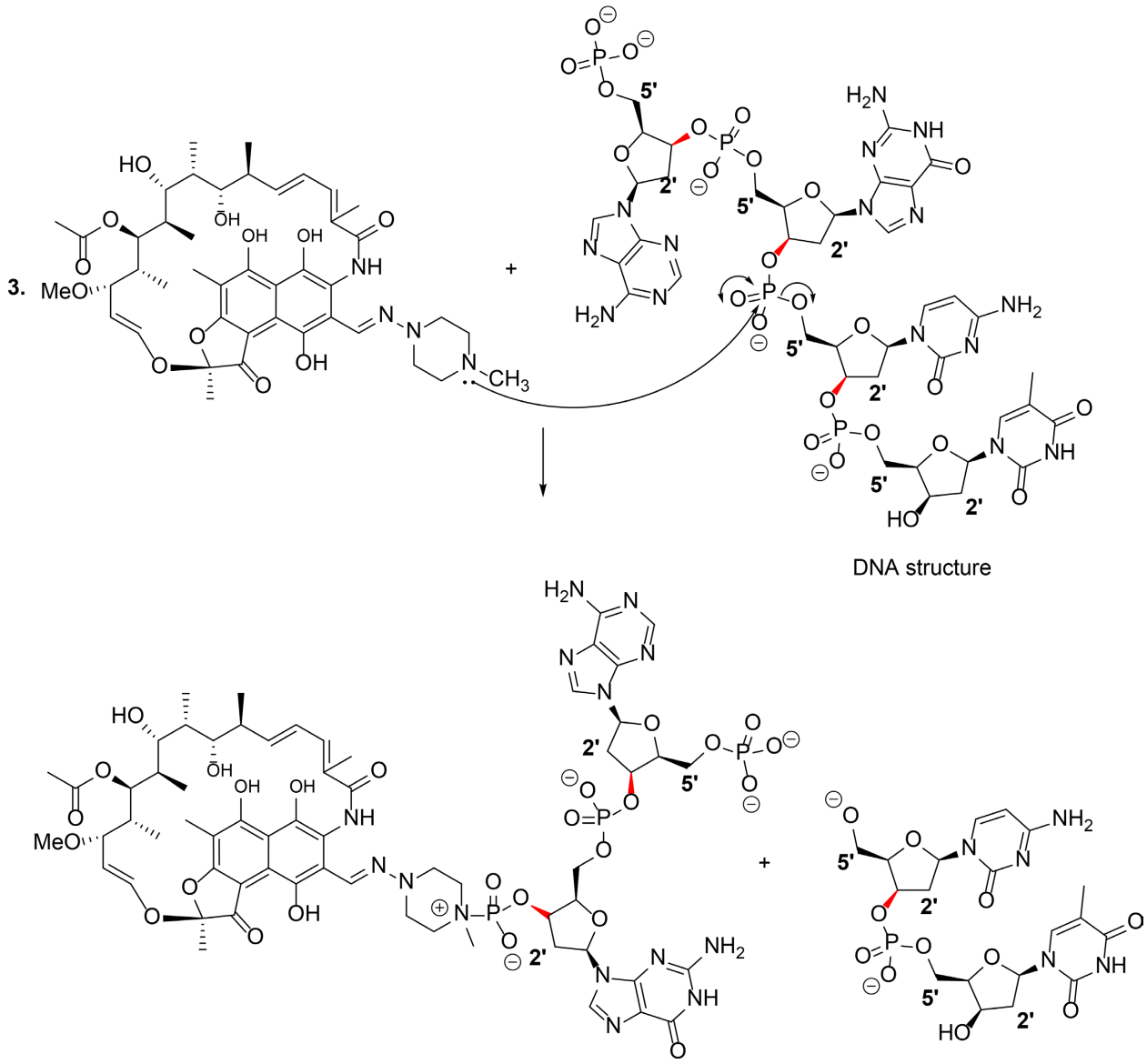


Scheme 9. Rifampicin reaction mechanism.

extracellular therapeutic substances to cross the cellular membrane [23]-[26].

Due to the presence of a phosphate group or a fatty acid group, rifampicin can easily cross the cellular membrane to penetrate the cytoplasm area of a cell, where it will be released by reacting with the nucleophilic conjugate base of the phospholipid because these biochemical reactions are reversible (Scheme 10, reactions 1-2). Then the free rifampicin will react with the microorganism deoxyribonucleic acid (DNA) to destroy the pathogenic agent (Scheme 10, reaction 3).

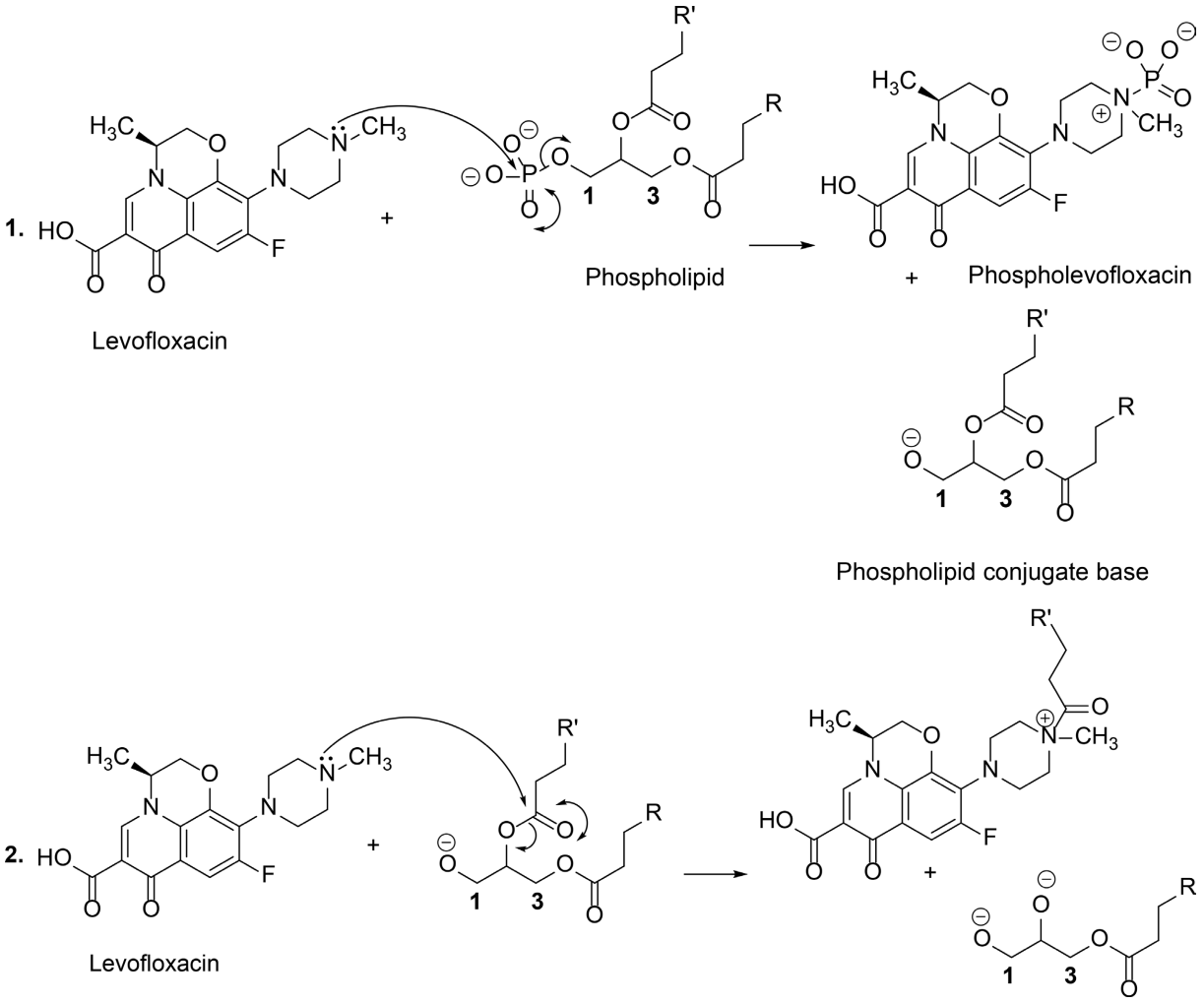




Scheme 10. Rifampicin reaction mechanism of DNA degradation.

The interaction of levofloxacin with the phospholipid phosphate group or with the carbonyl group of the phospholipid follows the same pathway as the stated above for rifampicin. In other words, levofloxacin will use its nitrogen atom into the piperazine group to react with the electrophilic phosphate group as well as the electrophilic carbonyl group of the appropriate phospholipid to produce the corresponding intermediate derivative, and its penetration into the cellular cytoplasm will be guided by the presence on it of the phosphate group (Scheme 11, reaction 1) as well as the presence of the acyl group (Scheme 11, reaction 2).

It is important to mention that organic compounds, which constitute the cellular membrane and organic compounds into the cellular cytoplasm are both into the same cellular reaction medium. Consequently, the levofloxacin bearing a phosphate group will react with the phospholipid conjugate base to regenerate

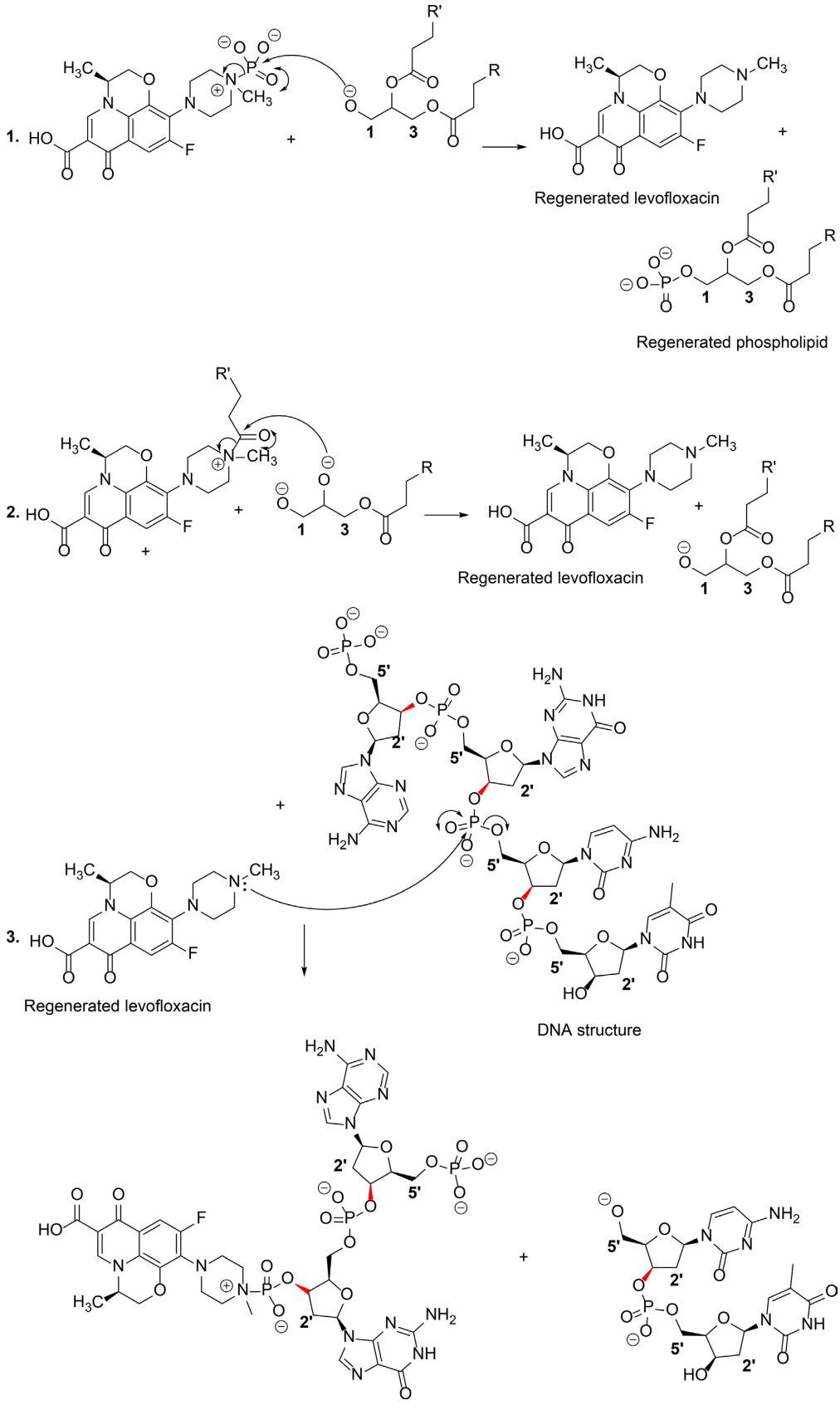


Scheme 11. Levofloxacin reaction mechanism.

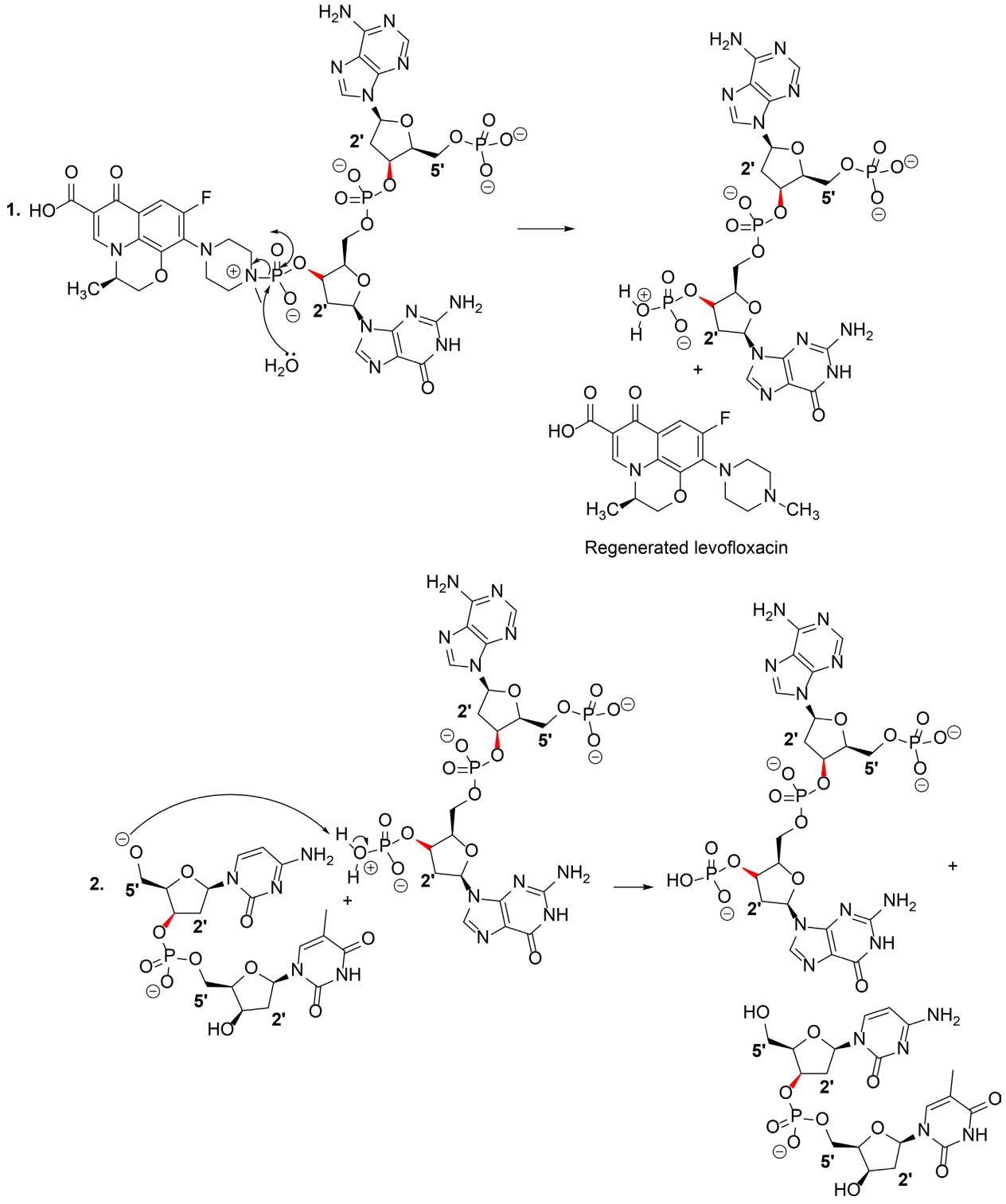
the phospholipid and the levofloxacin (Scheme 12, reaction 1). In the same context, the levofloxacin bearing an acyl group will react with the second phospholipid conjugate base to regenerate phospholipid and the levofloxacin (Scheme 12, reaction 2). Being free or regenerated into the cellular cytoplasm, the levofloxacin medicine can now neutralize the microorganism DNA by utilizing its nucleophilic character to donate electrons to the microorganism DNA electrophile (Scheme 12, reaction 3).

Being that the cellular cytoplasm is an aqueous reaction medium, a molecule of water can hydrolyze the resulting products to regenerate an entire therapeutic compound (Scheme 13).

It has also been reported that levodopa is a promedicine of dopamine, and it is used to treat Parkinson disease. Being very polar organic compound, the dopamine is not administrated as such, but it is administrated as levodopa because this later is an amino acid susceptible to be recognized by a specific cellular receptor such as glycoprotein, which will facilitate it to pass through the cellular membrane [27] [28]. When levodopa is now into the cytoplasm, decarboxylase enzyme

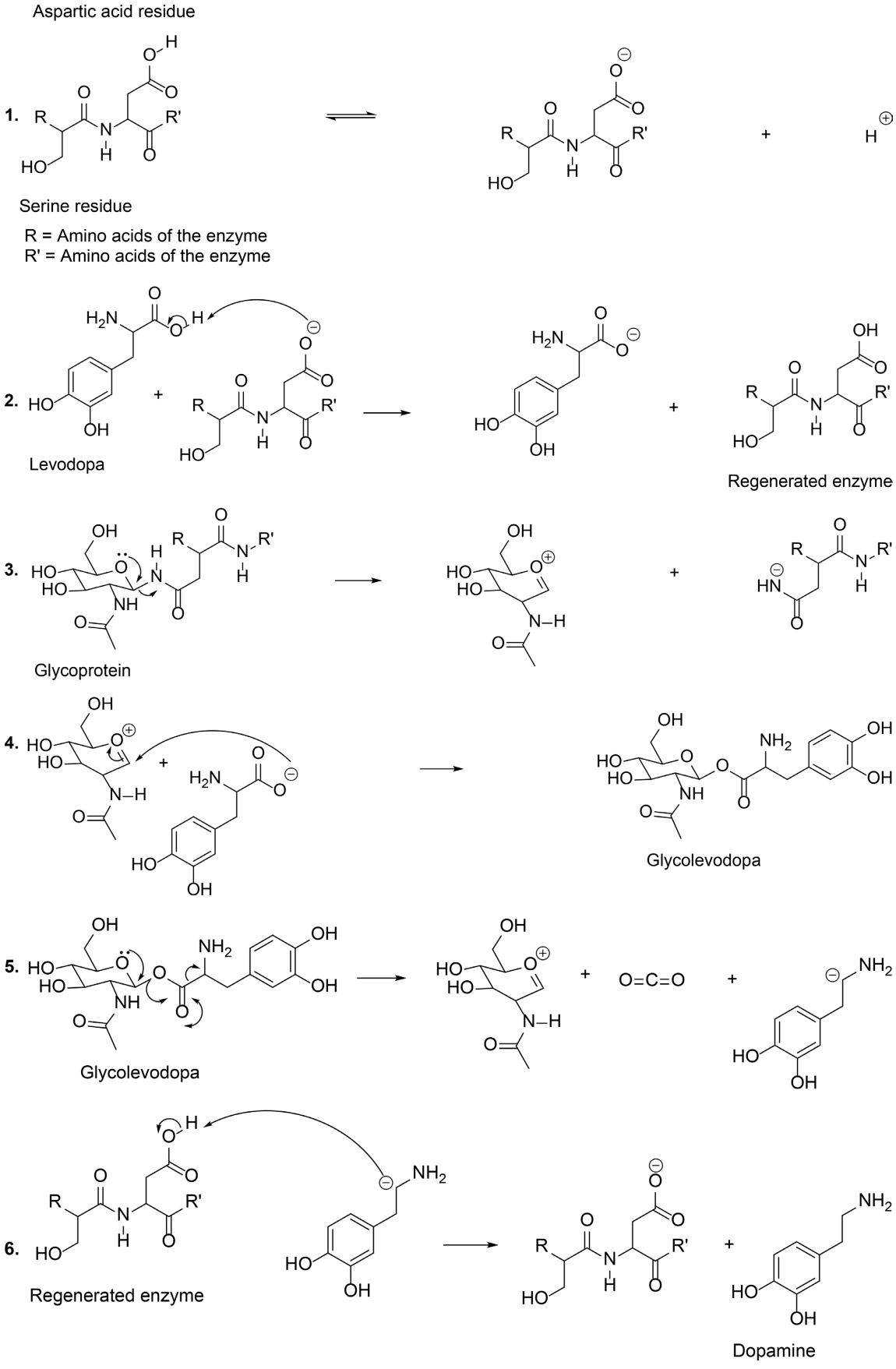


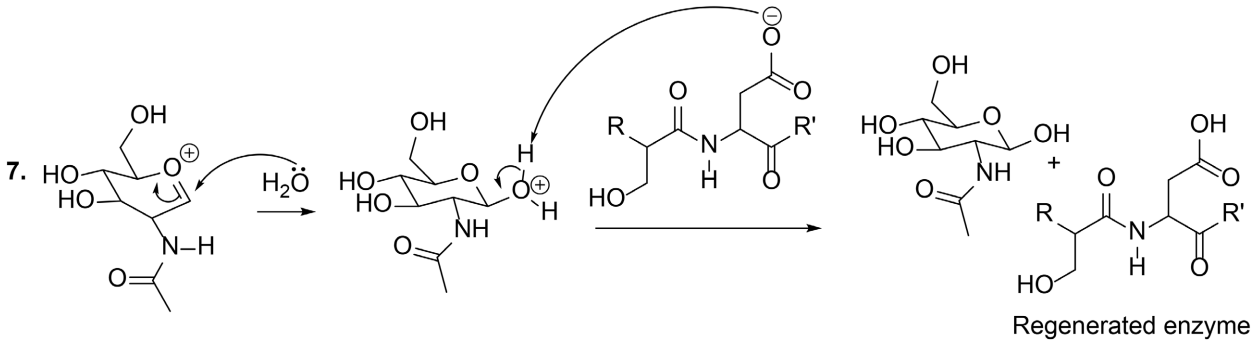
Scheme 12. Levofloxacin reaction mechanism of DNA degradation.



Scheme 13. Levofloxacin regeneration.

will catalyze the cleavage of its carboxylic group, and the dopamine is thus released within the cell. It is important to mention that serine and aspartate residues play a fundamental catalytic role into the decarboxylase enzyme active site (Scheme 14, reaction 1).


Aspartic acid residue
1.
Serine residue
R = Amino acids of the enzyme
R' = Amino acids of the enzyme
2.
Levodopa
Regenerated enzyme
3.
Glycoprotein
4.
Glycolevodopa
5.
Glycolevodopa
6.
Regenerated enzyme
Dopamine



Scheme 14. Pro medicine chemistry.

7. Conclusion

I have comprehensibly explained the degradation of pathogenic microorganisms by adequate therapeutic compounds using advanced organic reactions. Indeed, therapeutic compounds chosen into this conceptual study have been selected according to their chemical behavior due to their characteristic functional groups where organic reactions take place. It is noteworthy to mention that the selected therapeutic compounds can react as acids, bases, nucleophiles or electrophiles with microorganism glycoproteins, phospholipids, glycolipids or deoxyribonucleic acid (DNA). In other words, the biological activity of this kind of therapeutic organic compounds has been demonstrated in detailed fashion using reaction mechanism stage by stage up to the neutralization of the targeted pathogenic microorganisms and in that context, to support this conceptual study, scientific experimental observations regarding the reactivity of some medicines such as rifampicin and levofloxacin have been found that they react with the cellular membrane constituents before entering the intracellular environment.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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