

DETECTION OF DNA REPAIR SYSTEMS IN *ACINETOBACTER BAUMANNI* ISOLATED FROM PATHOLOGICAL SPECIMEN

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ABSTRACT : Microorganisms, especially small ones, such as bacteria that represent the simplest and smallest neighborhoods, must have very efficient repair systems for their genetic material because the prokaryotic cells possess a unique genome and thus one copy of each gene. Most of its materials or codes are effective, so any damage to the genome will have catastrophic consequences for the cells, if not deadly, and therefore the neighborhoods appear to be normal to continue life. This is why our study included DNA repair systems for *Acinetobacter* bacteria to include a study of the optical repair system where the bacteria were exposed for different time periods 100, 150, 200 and 200 seconds. For ultraviolet rays, after which they were isolated into two parts, in the dark and in the light, then the optical repair system was studied. To investigate the eradication system, the lowest inhibitory concentration was measured using different concentrations of caffeine 15, 20, 25, 30, 35mM. After that, different periods of radiation were shown and the presence of the Excision repair system was established.

Key words : DNA repair systems, *Acinetobacter baumannii*, pathologica.

INTRODUCTION

Just as there are mechanisms for mutation or damage in DNA, there are mechanisms in place to fix these glitches. It is worth noting the SOS self-response system as this system stimulates any type of repair mechanisms (Zhou *et al*, 2020; Su *et al*, 2020). The most important of which is the Nucleotide Excision Repair Mechanism. Stimulation of the production of SOS proteins occurs through (Peterson *et al*, 2020; Bai *et al*, 2019). The DNA aggregation and DNA polymerase is inhibited and the RecA enzyme creates strands around the ssDNA, which stimulate the RecA and inhibit the LexA repressor, as this inhibitor prevents the coding of the genes of the SOS self-response system and when it stimulates RecA (Butala *et al*, 2020; Jiang *et al*, 2020). It works to remove the inhibition and the SOS genetics are cloned and encoded and thus leads to stimulation of the repair mechanisms, the first of which is the nucleotide shear repair mechanism (Corral *et al*, 2020; Geisinger *et al*, 2020). Mismatch Repair (MMR) this mechanism is used for the incorrect correlation between incompatible rules and especially the wrong association between Adenine and other inconsistent rules. Normally, an enzyme called Dam methylase adds a methyl group to the adenine in the GATC sequence (when it is bound to thymine). If the

adenine is wrongly associated with one of the other bases, then the dam methylase does not add the methyl group to the adenine (Daniel *et al*, 2020). This mechanism is used to repair the wrong association that occurs as a result of both types of point mutations (Transition and Transversion) and Frameshift mutations, meaning that this mechanism is used to fix simple lesions and Bulky lesion (Shin *et al*, 2019; Thöming *et al*, 2020). Nucleotide Excision Repair Mechanism, This mechanism is used to repair Bulky lesion, which occurs in more than one base and leads to defective double helix, for example (Olivares *et al*, 2020). Thymine Dimer. Thymine Dimer Recognition is distinguished by the binding of UvrA and UvrB enzymes where the UvrAB complex performs a double-DNA scan and stands at the site of Thymine dimer after which the UvrA is separated (Martegani *et al*, 2020). The removal of the Thymine dimer is done through the linking of the UvrC, where the UvrBC complex works to make a nick cut on both sides of the Thymine dimer. Then the UvrD that works to disengage is then linked to the piece containing the Thymine dimer (Anderson *et al*, 2019).

MATERIALS AND METHODS

Detection of photo DNA repair system

Bacteriology *Acinetobacter* exhibited 254 nm for Ultraviolet Radiation For a period of 200, 150, 100, 250

seconds, then take 2 ml of each A treatment of 0 leaves 1 mL of them in the dark and 1 mL of light for an hour then I made a series of relief for her and planted it on feeding nests and covering the dishes with aluminum foil and incubated 37 for 24 hours (Abdel-Hamid *et al*, 2020).

Detection of the excision repair system

The bacteria were shown to have a different concentration of 20, 25, 30, 35 Mm of caffeine different from UV rays and planted on the feeder containing the concentration below the inhibitor of the lowest substance. The plates were covered with aluminum foil and incubated with a caffeine degree 37°C for 24 h (Soldano *et al*, 2020).

RESULTS AND DISCUSSION

This type of repair is specialized in the type of damage without affecting the structure of the DNA tape.

Table 1 : shows the photo repair system in DNA of *Acintobacter baumannii* after exposure to UV radiation.

| Time (sec) | Survival bacteria (Number of colonies) | | | |
|------------|--|-------|------|------|
| | Light | Light | Dark | Dark |
| 100 | 20 | 25 | 5 | 2 |
| 150 | 15 | 15 | 2 | 1 |
| 200 | 10 | 9 | 2 | 1 |
| 250 | 6 | 5 | 1 | 0 |

The resulting thymine diodes can be repaired by ultraviolet radiation by a mechanism called photo reactivation, which works directly against the damage through the activity of an enzyme called photolyase, which is activated by the energy absorbed along the wavelength of 300-500 nanometers, after which it removes the damage. Bacteriology *Acinetobacter* exhibited 254 nm for Ultraviolet Radiation For a period of 200, 150, 100, 250 seconds, then take 2 ml of each A treatment of 0 leaves 1 mL of them in the dark and 1 mL of light for an hour then I made a series of relief for her and planted it on feeding nests and covering the dishes with aluminum foil and incubated 37 for 24 hours. The results showed that the bacteria that were exposed to ultraviolet radiation had a damage, this damage led to the lack of growth of these bacteria and this is very clear in the dishes that were placed in the dark has a little opposite growth that was put in the light grew, but the greater the exposure time the rate of growth of the bacteria decreased until Existing in the light is indicative of significant damage.

Table 2 shows a detailed description of the effect of exposure time with ultraviolet radiation on damage to DNA on the one hand and the effect of exposure to light on the growth of bacteria, where the results showed that the bacteria that were exposed to radiation were in the light

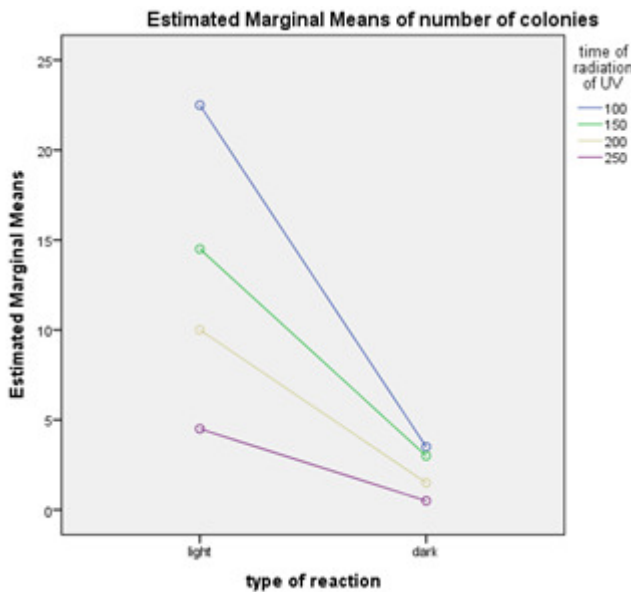


Fig. 1 : The effect of UV exposure time on bacterial growth.

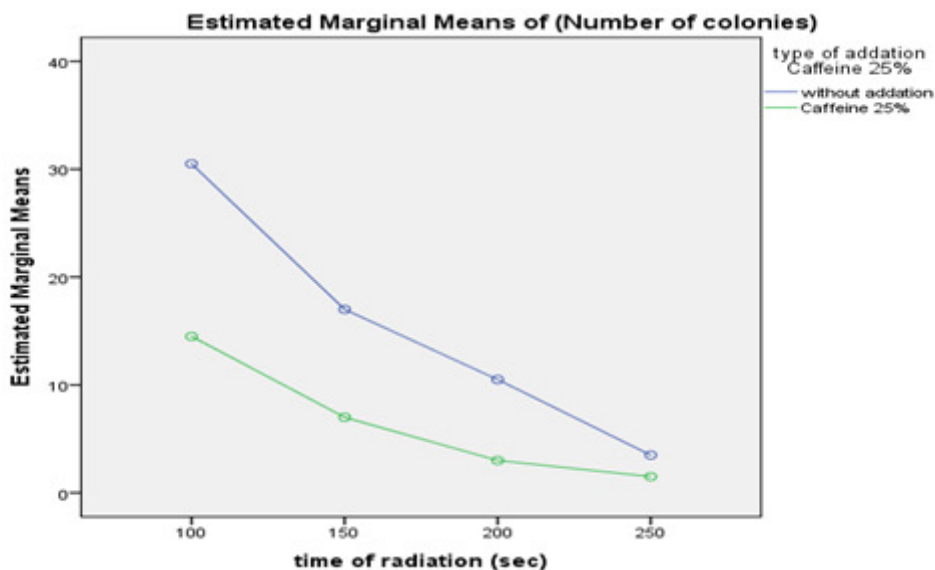


Fig. 2 : shows a 50% reduction in bacterial growth after exposure to UV rays.

Table 2 : showing the mean and Std. Error of bacteria that received a DNA repair after exposure to UV radiation.
Time of radiation of UV * type of reaction
Dependent Variable: number of colonies

| Time of radiation of UV | type of reaction | Mean | Std. Error | 95% Confidence Interval | |
|-------------------------|------------------|--------|------------|-------------------------|-------------|
| | | | | Lower Bound | Upper Bound |
| 100 | light | 22.500 | 1.199 | 19.735 | 25.265 |
| | dark | 3.500 | 1.199 | .735 | 6.265 |
| 150 | light | 14.500 | 1.199 | 11.735 | 17.265 |
| | dark | 3.000 | 1.199 | .235 | 5.765 |
| 200 | light | 10.000 | 1.199 | 7.235 | 12.765 |
| | dark | 1.500 | 1.199 | -1.265 | 4.265 |
| 250 | light | 4.500 | 1.199 | 1.735 | 7.265 |
| | dark | .500 | 1.199 | -2.265 | 3.265 |

Table 3 : The Excision repair system in DNA of *Acintobacter baumannii* after exposure to UV radiation.

| Time (sec) | Survival bacteria (Number of colonies) | | | |
|------------|--|---------|--------------|--------------|
| | Without | Without | Caffeine 25% | Caffeine 25% |
| 100 | 30 | 32 | 15 | 14 |
| 150 | 18 | 16 | 8 | 6 |
| 200 | 11 | 10 | 4 | 2 |
| 250 | 4 | 3 | 2 | 1 |

the highest growth rate of bacteria that grew in the dark and had given the exposure time For 100 second scans, the best results are compared to 150, 200 and 250, respectively. We also notice that at the time of exposure 250 seconds, the rate of growth decreased dramatically, indicating significant damage to DNA. The optical repair system does not have the ability to repair it.

Table 3 shows the DNA repair system, the type of eradication, where we observe in the period of exposure of bacteria to ultraviolet rays for a period of 100 seconds. The number of colonies decreased to 50% in the added dishes of 25% of caffeine. We note that the greater the time periods for exposure of bacteria to ultraviolet rays, the lower the growth, and this is normal for a killing of these bacteria a direct repair system.

Table 4 shows the arithmetic mean of the growing colonies in the presence and absence of caffeine, as we note a decrease in the arithmetic mean of the number of developing colonies in the presence of caffeine by 50% compared to others that have grown without adding caffeine. This indicates the existence of a DNA repair system. This is evident in Fig. 2. The *uvrA* protein binds with ATP to the DNA at the distorted or distorted region, then the *uvrB* protein binds to the formed complex, which increases the UVrA-ATP complex specialty of the irradiated DNA. As for the *uvrC* protein, it cuts the DNA

Table 4 : shows the Mean and Std. Deviation of *Acintobacterbaumanni* after exposure to UV radiation.
Descriptive Statistics
Dependent Variable: (Number of colonies)

| Time of radiation (sec) | Type of addation Caffeine 25% | Mean | Std. Deviation | N |
|-------------------------|-------------------------------|-------|----------------|----|
| 100 | without addition | 30.50 | .707 | 2 |
| | Caffeine 25% | 14.50 | .707 | 2 |
| | Total | 22.50 | 9.256 | 4 |
| 150 | without addition | 17.00 | 1.414 | 2 |
| | Caffeine 25% | 7.00 | 1.414 | 2 |
| | Total | 12.00 | 5.888 | 4 |
| 200 | without addition | 10.50 | .707 | 2 |
| | Caffeine 25% | 3.00 | 1.414 | 2 |
| | Total | 6.75 | 4.425 | 4 |
| 250 | without addition | 3.50 | .707 | 2 |
| | Caffeine 25% | 1.50 | .707 | 2 |
| | Total | 2.50 | 1.291 | 4 |
| Total | without addition | 15.38 | 10.663 | 8 |
| | Caffeine 25% | 6.50 | 5.451 | 8 |
| | Total | 10.94 | 9.377 | 16 |

strip in an area about 8 bases to the left of the (Upstream) dual Brimidine region, and also cuts the strip at a distance of 4-5 bases from the right (Downstream). As for the UVRD protein, which is the DNA helicase D (which is similar to the DNA used when starting to multiply), it separates the strips and loosens their spiral structure to release the segment consisting of about 12-13 base pairs. Then DNAPI and other enzymes fill the gap in direction 5' to 3' and then weld it. In the event of failure to repair, DNAPIII begins to multiply the DNA strips after the damage region, leaving an affected area that may reach about 800 pairs of bases and is called a translesion injury.

Then the injury or gap area is covered by RecA proteins that cover the individual DNA strips until other systems such as repair recombination repair that occurs after replication so that the recA gene-free mutations are very sensitive to ultraviolet radiation and this correction or repair is part of the SOS system.

CONCLUSION

Through the results obtained in our study of DNA repair systems in *Acinetobacter baumannii* bacteria it was found that they have photo DNA repair system and Excision repair system.

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