International Journal of Pathogen Research

4(1): 17-27, 2020; Article no.IJPR.55979
ISSN: 2582-3876

Effect of Clinical X-rays on Methicillin-resistant *Staphylococcus aureus* and Other Bacterial Isolates from Radiology Equipment

K. S. Adebiyi[1], F. O. Ehigiamusoe[2] and C. E. Oshoma[1*]

[1]Department of Microbiology, University of Benin, Benin City, Edo State, Nigeria.
[2]Department of Radiology, University of Benin Teaching Hospital, Benin City, Edo State, Nigeria.

Authors' contributions

This work was carried out in collaboration among all authors. Authors KSA, FOE and CEO designed the study. Authors KSA and CEO performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors KSA and CEO managed the analyses of the study. Author FOE managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJPR/2020/v4i130102
Editor(s):
(1) Ohanu, Ekechukwu Martin, University of Nigeria, Nsukka, Nigeria.
Reviewers:
(1) Syed Umer Jan, University of Balochistan, Pakistan.
(2) Renata Freire Alves Pereira, Federal Fluminense University, Brazil.
Complete Peer review History: http://www.sdiarticle4.com/review-history/55979

Original Research Article

Received 05 February 2020
Accepted 10 April 2020
Published 17 April 2020

ABSTRACT

Aim: Methicillin-resistant *Staphylococcus aureus* (MRSA) and other nosocomial pathogens are frequently isolated from the Radiology Department of hospitals. These pathogens are repeatedly exposed to refracted electromagnetic radiations during diagnostic clinical X-ray imaging sessions. This study thus investigated the effect of these refracted radiations on the nosocomial bacteria found within Radiology units and their possible survival mechanism.
Methodology: Swab samples were collected from three (3) Radiology units; plain radiography machine, ultrasound machine and mammography machine. The samples were cultured and identified following standard microbiology procedures. The identified bacterial isolates were exposed to X-rays at different *KVp* and *MAs* factors, cell counts and catalase activities of the isolates were determined using standard procedures.
Results: The bacterial isolates identified were *Staphylococcus aureus, Pseudomonas aeruginosa, Bacillus subtilis, Escherichia coli* and *Enterococcus faecalis* where *Staphylococcus aureus* had the highest prevalence (29.41%) and *Enterococcus faecalis* (11.76%) was least. The highest survival level to *KVp* and *MAs* exposure was *S. aureus* with values $11.16 \pm 0.86 \times 10^3$ and $19.50 \pm 0.16 \times 10^3$

**Corresponding author: Email: cyprian.oshoma@uniben.edu, oshomacy@yahoo.com;*

cfu/ml respectively while *Enterococcus faecalis* was least with negative value. There was a positive correlation between catalase activity and cell survival with R-value of 0.2512 and 0.6925 for *KVp* and *MAs* exposure factors respectively.

Conclusion: The study revealed that clinical X-ray does not completely eliminate nosocomial pathogens within Radiology Department and MRSA is a formidable bacterium in the units.

Keywords: Catalase activity; bacterial survival; kilovoltage peak; milliamperage per seconds; X-ray.

1. INTRODUCTION

The Radiology Department of every hospital plays a major role in medical diagnosis, it regularly receives large number of patients resulting in high human traffic which results in the transmission of nosocomial pathogens amongst patients and Radiology Staff [1]. Some of the commonly found pathogens are *Staphylococcus aureus*, *Staphylococcus epidermidis, Candida albicans, Bacillus substilis, Escherichia coli* and *Pseudomonas aeruginosa* [2,3,4]. Among these microbes, *Staphylococcus aureus* poses a serious threat to hospitals due to its ability to acquire *mecA* gene conferring resistance to the antibiotic methicillin. These strains are known as Methicillin-resistant *Staphylococcus aureus* (MRSA) [5]. These pathogens attach to fomites within the Radiology Department [4,6].

Most clinical Radiology units employ X-rays for diagnostic imaging purposes such plain radiography and Mammography units at human tolerable exposure limits. In most clinical X-rays' sections, patients are mainly exposed to two (2) X-ray exposure factors Kilovoltage Peak (KVp) and Milliamperage-seconds (MAs) which are within human tolerable limits [7]. These exposure factors (KVp and MAs) produces radiations which scatter within the imaging room as a result of refraction hence, radiographers take X-ray shots from behind a dense lead shield-chamber for protection against the scattered rays which are dangerous to biological systems resulting from uncontrolled repeated exposures [8]. However, pathogens in these x-ray imaging rooms are not shielded from these radiations. They are continually exposed to varying exposure doses. Surprisingly, some of these pathogens are not killed by these refracted beams of radiation, instead they gradually buildup résistance and adapts to the environmental and oxidative stress build up due to these radiation [9].

Resistance and adaptability enable bacteria to survive human tolerable clinical x-ray radiations especially at low doses. In some organisms however, there is a tolerable exposure limit and excessive genotoxic stress such as high-dose radiation, may cause severe DNA lesions and lead to genome instability which leads to mutation [10]. Ionizing radiations generate intracellular radicals (O_2^-) (which are redox-cycling agents such as menadione and paraquat) which cause oxidative stress, through the increased concentration of active oxygen to a level that exceeds the cell's defense capacity [11]. The cellular component target for these highly reactive oxygen species (ROS) are DNA, RNA, proteins and lipids. Much of the damage is caused by hydroxyl radicals generated from hydrogen peroxide molecules (H_2O_2) [12]. Microbial cells have developed enzymatic components including catalase and superoxide dismutase which help to protect the cells' proteome from oxidative inactivation [13]. Catalase for example, is a very important enzyme found in all aerobic organisms. Microorganisms exposed to oxygen strive against reactive oxygen species such as hydrogen peroxide in their environment. Catalase catalyzes the decomposition of hydrogen peroxide to water and oxygen thereby protecting the cell from oxidative damage due to reactive oxygen species (ROS) [14,15,16]. This study investigated the effect of refracted electromagnetic radiations on bacteria isolated from radiology equipment and their possible survival mechanism.

2. MATERIALS AND METHODS

2.1 Sample Collection

A total of 75 swab samples, 25 each, were collected from 3 radiology units (X-ray, Ultrasound and Mammogram) of a private radiology center in, Benin City, Edo State. Swabbing was carried out aseptically using sterile swab sticks moistened with sterile peptone water spanning 5cm (length) by 2 cm (breadth) in area. The swabs were taken to the laboratory for immediate culturing and incubation within 2hoursaccording to the method described by Public Health England [17].

2.2 Bacterial Enumeration and Characterization

The swab sticks were dipped into 10 ml of sterile distilled water in test tubes and allowed to stand for 30 mins after being slightly whisked. From this stock, a 10-fold dilution was carried out, there after 1.0 ml was transferred using spread plated method, onto plates containing Nutrient Agar and Mannitol Salt Agar respectively. All the plates were incubated at temperature of 37^{o}C for 24h. The method described by Public Health England [17] for estimating bacterial counts was used to enumerate the total viable counts of the isolates. Colonial growths were sub-cultured for purification and purified bacterial isolates were preserved in Nutrient agar (NA) slants. The isolates were characterized by morphology, Gram's reaction and biochemical test using the scheme in Bergey's manual of determinative bacteriology [18,19].

2.3 Antibiotic Susceptibility Test

Antibiotic sensitivity of identified bacterial isolates were carried out using Kirby-Bauer disk diffusion susceptibility test with the following antibiotics: septrin (10µg), chloramphenicol (10µg), sparfloxacin (25µg), ciprofloxacin (10µg), amoxillin (30µg), augmentin (25µg), gentamycin (10µg), pefloxacin (10µg), oflaxicin (30µg) and streptomycin (25µg). The test organisms were inoculated into sterile nutrient broth in a test tube and incubated at 37^{o}C for 24 h. From the pure culture, 0.1 ml was transferred into solidified Nutrient agar (NA) in a petri-dish and a sterile spreader was used to ensure even distribution on the agar plate. The plates were allowed to air dry for 5 min thereafter the standard antibiotics discs were laid on the inoculated agar. The plates were incubated at 37^{o}C for 24 h. Clear zones around the discs were measured and interpreted as either susceptible, intermediate or resistant. Zones of inhibition ≤14 mm were recorded as Resistant, 15 mm – 17 mm as Intermediate and ≥18mm as Susceptible [20].

2.4 Determination of Methicillin Resistant *Staphylococcus aureus* (MRSA)

Sensitivity to Oxacillin was determined using Oxacillin disk placed on a standardized inoculum of *S. aureus* spread plated on Mannitol salt agar then incubated at 37± 2^{o}C for 24h. Clear zones around the discs were measured and interpreted as either susceptible or resistant for the test organisms to the particular antibiotic. Zones of inhibition ≤14 mm were recorded as Resistant, 15 mm – 17 mm as Intermediate and ≥18 mm as Susceptible [20].

2.5 Detection of *mecA* Gene from *S. aureus* Isolates

Strains of *S. aureus* which showed resistance to Oxacillin were analyzed using molecular techniques to detect the presence of the *mecA* gene using specific MRSA gene primers. The primer sequences (5'-CCA ATT CCA CAT TGT TTC GGT CAT A- 3') for *mecA* gene was used which matches with the sequence of *mecA* genes published by Zhang et al. [21]. PCR assay was carried out for the detection of mecA (associated with methicillin resistance) using 1X iQ™ Multiplex Power mix (Bio-Rad Laboratories, Hercules, CA, USA) following the manufacturer's recommendations Electrophoresis of amplicons was performed with 1% agarose gel containing ethidium bromide (EtBr) 0.5 mg·L−1, for 1 h at 100 V in 0.5× TAE buffer (40 mM Tris-HCl, 20 mM Na-acetate, 1 mM EDTA, pH 8.5). A molecular weight marker ladder was included on each gel. Bands were visualized using an UV trans-illuminator [21].

2.6 Exposure of Isolates to X-rays

Pure cultures of the isolates were sub-cultured from a slant into sterile nutrient broth and incubated at 37^{o}C for 24h. A cell suspension was formed using distilled water. An aliquot of2.0ml was spread plated into petri dishes and incubated at 37^{o}C for 24h. The colonial growth after 24h incubation was counted and recorded as pre-exposure count [22]. At this point, some plates were then kept aside for pre-exposure catalase activity test. The other plates were then exposed to different x-ray radiation factors (at High KVP value of 133.00, Medium KVP value of 86.50, Low KVP value of 40.00 and High MAS value of 180.00, Medium MAs value of 100.00 and Low MAS value of 20.00) [23]. After laboratory exposure, the plates surface was carefully scoped, dissolved in 2.0 ml of distilled water and spread plated into new petri dishes. The plates were incubated at 37^{o}C for 24h. The colonial growth after 24h incubation was counted and recorded as post-exposure count. The mean values from each isolate triplicate plates were calculated and recorded [24]. Likewise, some plates were then kept aside for post-exposure catalase activity test.

2.7 Catalase Activity Test

The plates kept aside for Catalase test were used to study the catalase activities of the isolates before and after laboratory exposure to X-ray. A 0.5 McFarland turbidity standard cell suspension of the isolates was made after carefully scoping the plate contents. The suspensions were transferred into test tubes and 5ml of freshly prepared hydrogen peroxide was introduced into the test tube and incubated in a dark laboratory cupboard for 10mins allowing the isolates to find time to synthesis catalase enzyme to dissociate and dissolve the hydrogen peroxide introduced [25]. To investigate the extent of the isolate's hydrogen peroxide dissociation ability (catalase test), a known volume (recorded as initial volume) of iron (II) sulphate was titrated from a burette into the presumably remaining (un-dissociated) hydrogen peroxide solution after the cells' catalyzing activity on it. The titre value of the burette on completion of the left-over (un-dissociated) hydrogen peroxide is used to determine the cell catalase activity of the isolates [26].

2.8 Cellular Catalase Activity of the Isolates

Cellular catalase activity of the isolates was determined by employing Walkley-Black Titration Derivative [27]:

$$\text{Cat. Activity (umol/min)} = ((Bk-T) \times M \times 10^6 / L \times I_t)$$

Where:

Bk = Blank (Volume of Hydrogen Peroxide)
T = Titre Value
M = Molarity of Iron (II) sulphate
L = Cell Load (McFarland Standard)
I_t = Incubation Time
10^6 = Conversion Factor (mol/mins to µmol/min)

The catalase activities of the isolates before and after laboratory exposure to X-ray were calculated and recorded [27].

2.9 Statistical Analysis

Descriptive statistics, analysis of variance and pearson correlation coefficient were used for statistical analysis of obtained data using statistical package for social sciences version 21. Data presentation was done using Microsoft Excel version 2010 [28].

3. RESULTS AND DISCUSSION

Total heterotrophic bacterial count and *S. aureus* counts isolated from the different radiology units are shown in Table 1. The highest number of *Staphylococcus aureus* ($3.61 \pm 0.19 \times 10^3$ *cfu/cm*^{3}) and heterotrophic bacterial ($8.48 \pm 0.2 \times 10^3$ *cfu/cm*^{3}) were isolated from the Ultrasound unit, followed by Mammogram unit and least was X-ray unit. The high bacterial count in the ultrasound unit is probably due to the absence of ionizing radiation for imaging in ultrasound rooms which might have less harmful effect to the isolates [29].

The identified bacterial isolates and their frequency of occurrence from all the units is shown in Table 2. The highest frequency of occurrence was *Staphylococcus aureus* while the least was *Enterococcus faecalis* with values 29.41 and 11.76% respectively. In this study the presence of *Staphylococcus aureus, Pseudomonas aeruginosa, Bacillus substilis, Escherichia coli* and *Enterococcus faecalis* were recorded within the sampled radiology units. These microbes are known to be nosocomial due to their colonization of clinical fomites [4,6]. As such, the presence of these microbes might be due to poor sterilization and sanitization procedures carried out on radiology equipment [1]. The resistance to common cleaning agents and antibiotics has made *Staphylococcus aureus* renown in radiology units [3,4] hence, its high frequency of occurrence than other bacterial isolates.

The antibiotic susceptibility test of the isolates is shown in Table 3. *S. aureus* isolates were highly resistant to most of the antibiotics used including Oxacillin. Other isolates showed a relatively low antibiotics resistance. MRSA is a known multidrug resistant strain of *S. aureus* capable of high-level antibiotic resistance [30] as confirmed in this study using Oxacillin disk test [31] and analysis of data from molecular examination which detected the presence of *mecA* genes which encodes for resistance to methicillin and other β-lactam antibiotics [30]. All the strains of *S auresus* from the various units showed the presence of *mecA* gene as exemplified in Plate 1.

Table 1. Pre-radiation total heterotrophic bacterial (THBC) and *S. aureus* counts from the radiology units

Isolates	X-ray unit ($x10^3 cfu/cm^3$)	Ultrasound unit ($x10^3 cfu/cm^3$)	Mammogram unit ($x10^3 cfu/cm^3$)
THBC	6.28 ± 0.49	8.48 ± 0.32	7.54 ± 0.35
S. aureus	2.47 ± 0.22	3.61 ± 0.19	1.48 ± 0.17

Table 2. Frequency of occurrence of bacterial isolates from the radiology units

Isolates	Frequency (%)
Staphylococcus aureus	29.41
Bacillus subtilis	23.53
Escherichia coli	17.65
Enterococcus faecalis	11.76
Pseudomonas aeruginosa	17.65

However, the high antibiotic susceptibility pattern of the isolates in addition to MRSA isolates goes to show that there is no direct relationship between the effect of antibiotics and the effect of X-ray exposure on the isolates [4]. Therefore, the resistance of *S. aureus* to various antibiotic drugs seems not to be a good indicator for evaluating its possible resistance to X-ray radiation [24]. The possible explanation for the non-relativeness between an isolates' antibiotics resistance and its X-ray irradiation resistance can be attributed to their mode of attack. Antibiotics are tailored to be target specific on sites/organelles of organism or group of organisms while clinical X-ray radiations are not but result to oxidative stress [9,32].

The difference in cell count of the bacterial isolates before and after laboratory exposure to KVp *exposure factor* is shown in Fig. 1. There was an increase in cell count difference for *S. aureus*, *P. aeruginosa*, *B. subtilis* and *E. coli* indicating cellular survival. The highest value of 11.16 ± 0.86x10^3 cfu/ml was for *S. aureus* isolated from X-ray unit followed by *S. aureus* isolated from mammogram unit (1.50 ± 0.80 x10^3 cfu/ml.). The least value-38.50 ± 0.61 x10^3 cfu/ml was from *E. faecalis* indicating cell death at this exposure factor.

The difference in cell count of the bacterial isolates before and after laboratory exposure to *MAs exposure factor* is shown in Fig. 2. There was an increase in cell count difference for *S. aureus*, *P. aeruginosa*, *B. subtilis* and *E. coli* indicating cellular survival. The highest value of 19.50 ± 0.16x10^3 cfu/ml was for *S. aureus* isolated from x-ray unit followed by *S. aureus* isolated from mammogram unit (13.16 ± 0.12x10^3 cfu/ml). The least value-30.00 ± 0.14x10^3 cfu/ml was from *E. faecalis* indicating cell death at this exposure factor.

The difference in catalase activity of the bacterial isolates before and after laboratory exposure to *KVp exposure factor* is shown in Fig. 3. There was an increase in cell catalase activities difference for all *S. aureus* isolates indicating cellular survival. The highest value of 0.73 ± 0.50 x10^6 umol/min was for *S. aureus* isolated from X-ray unit followed by *S. aureus* isolated from

Table 3. Antibiotic resistance pattern of bacterial isolates from the radiology units

Antibiotics	*S. aureus* (N=5)	*B. subtilis* (N=4)	*E. coli* (N=3)	*E. faecalis* (N=2)	*P. aeruginosa* (N=3)
	%R	%R	%R	%R	%R
SXT	100	0	0	50	66.9
CHL	100	50	0	50	33.3
SPA	100	25	66.7	100	0
CPX	80	0	0	0	0
AM	100	0	0	0	0
AU	80	0	0	0	0
CN	100	0	0	0	33.3
PEF	100	25	33.3	0	33.3
OFX	100	0	0	50	0
S	100	0	33.3	0	66.9
OXA	100	ND	ND	ND	ND

Key: N = No. of Isolates tested, %R = Percentage Resistance of Isolates, ND = Not Determined, SXT = Septrin, PEF = Pefloxacin, CHL = Chloramphenicol, OFX = Oflaxacin, SPA = Sparfloxacin, S = Streptomycin, CPX = Ciprofloxacin, Am = Amoxicillin, Au = Augmentin, CN = Gentamycin, OXA = Oxacillin

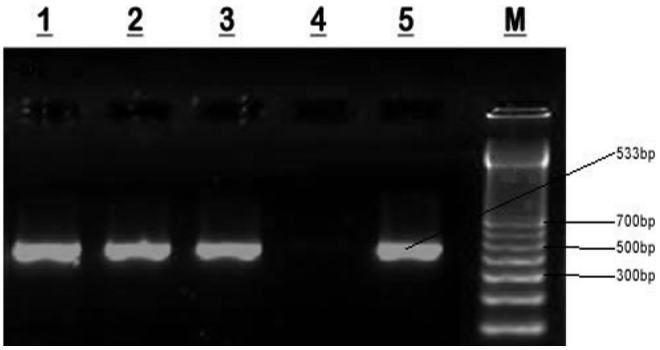

Plate 1. Gel electrophoresis of *mecA* gene of *Staphylococcus aureus* from the radiology units

Lane 1: S. aureus isolated from X-ray Unit, Lane 2: S. aureus isolated from Ultrasound Unit. Lane 3: S. aureus isolated from Mammogram Unit, Lane 4: Shows negative control, Lane 5: S. aureus positive control (533 bp), M = DNA Molecular Weight Marker

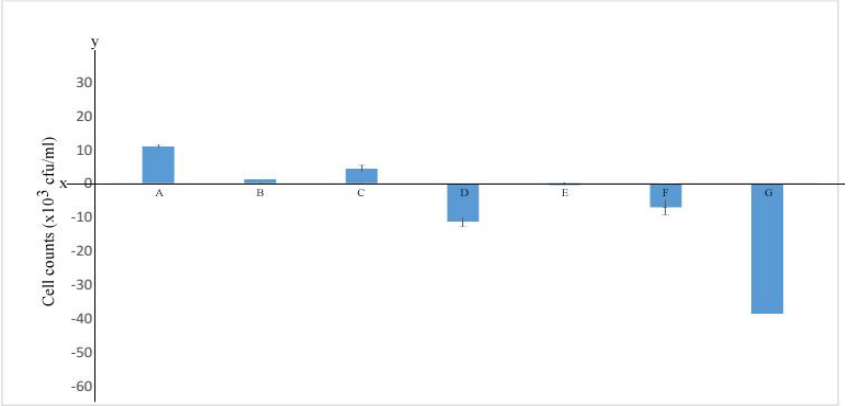

Fig. 1. Difference in cell count of isolates before and after laboratory exposure to KVp X-ray factor

A= S. aureus (X-ray unit isolate), B = S. aureus (Ultrasound unit isolate), C = S. aureus (Mammogram unit isolate), D = P. aeruginosa, E = B. Subtilis, F = E. coli, G = E. faecalis

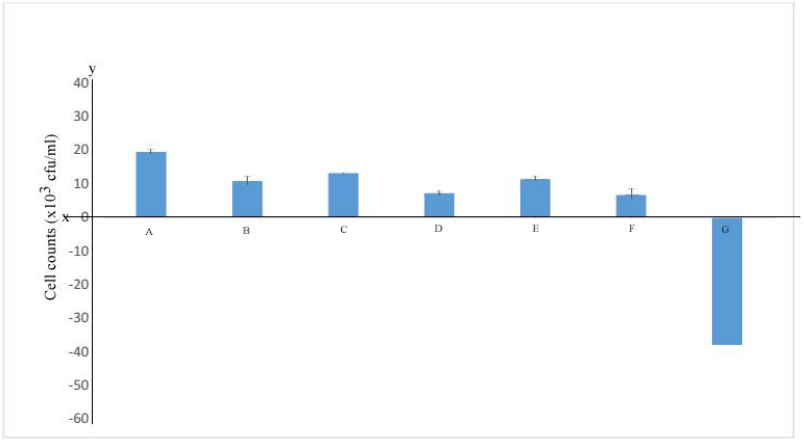

Fig. 2. Difference in cell count of isolates after laboratory exposure to MAs X-ray factor

A= S. aureus (X-ray unit isolate), B = S. aureus (Ultrasound unit isolate), C = S. aureus (Mammogram unit isolate), D = P. aeruginosa, E = B. Subtilis, F = E. coli, G = E. faecalis

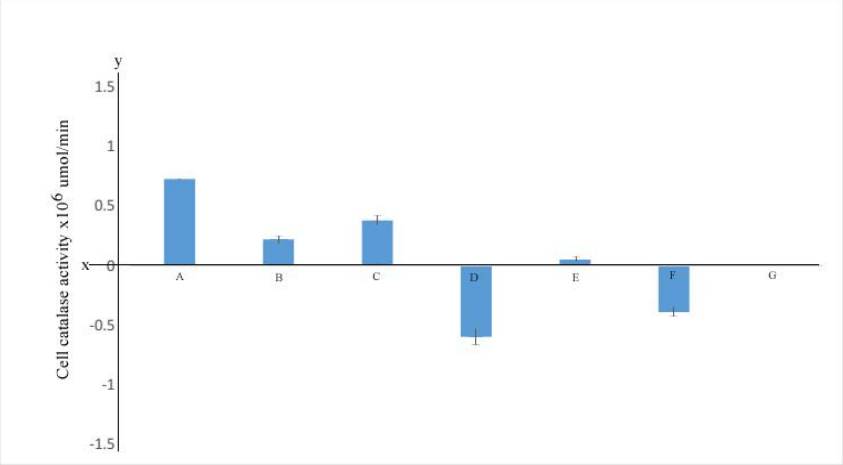

Fig. 3. Difference in catalase activities of isolates after laboratory exposure to KVp X-ray factor
A= S. aureus (X-ray unit isolate), B = S. aureus (Ultrasound unit isolate), C = S. aureus (Mammogram unit isolate), D = P. aeruginosa, E = B. Subtilis, F = E. coli, G = E. faecalis

ultrasound unit (0.22 ± 0.60x10^6 umol/min). The least value -0.39 ± 0.61x10^6 umol/min was from *E. coli* indicating cell death at this exposure factor and *E. faecalis* recorded no value being catalase negative.

The difference in catalase activities of the bacterial isolates before and after laboratory exposure to *MAs exposure factor* is shown in Fig. 4. There was an increase in catalase activities difference for *S. aureus*, *P. aeruginosa*, *B. subtilis* and *E. coli* indicating cellular survival. The highest value of 1.39 ± 0.60x10^{6}umol/min was for *S. aureus* isolated from X-ray unit followed by *S. aureus* isolated from mammogram unit (0.61 ± 0.70x10^{6}umol/min). The least value 0.57 ± 0.90x10^{6}umol/min was for *E. coli* and *E. faecalis* recorded zero (0) being catalase negative.

The relationship between catalase activity and bacteria survival following exposure to KVp and MAs *exposure factors* is shown in Fig. 5. The results how that there is a positive correlation between catalase activity and cell survival with R-value of 0.2512 and 0.695 for KVp and MAs exposure factors respectively.

The bacteria X-ray exposure analysis shows cellular survival of isolates after exposure to various x-ray factors. This is because clinical x-rays which are designated human-tolerable are usually technologically re-engineered to attenuate the adverse biological effects of their radiation on living cells [4,33]. Secondly, prokaryotes are known to exhibit much higher radiation tolerance than animals and humans [9]. As a result, the genotype profile of the cells may have been preserved using some form of reversion mechanism (such as base and nucleotide excision repair) [10,12] or defense mechanisms (catalase and superoxide dismutase synthesis) [13] thereby protecting the cells.

The *Staphylococcus aureus* isolate from X-ray unit had the highest survival level to high X-ray exposure factors followed by *S. aureus* isolate from mammogram unit. This can be attributed to cellular adaptation as both radiology units employ X-ray (ionizing radiation) during imaging unlike ultrasound unit which do not make use of non-ionizing radiation, hence least survival level [34]. Prolong intermittent exposure of the isolate might have resulted to adaptation resulting from the development of adaptive defense mechanism [24] such ability to synthesis catalase enzyme to mitigate the oxidative stress built up by the ionizing radiation [13].

The isolate, *Enterococcus faecalis*, experienced the most lethal effect for all X-ray exposure factors (KVp and MAs) with a negative value on Y-axis indicating cell death. The inability of the isolate to synthesize and secrete catalase enzyme to resist the oxidative stress induced by X-ray exposures may be responsible for the cell death [13]. Unlike the other isolates which were able to withstand the oxidative stress from the

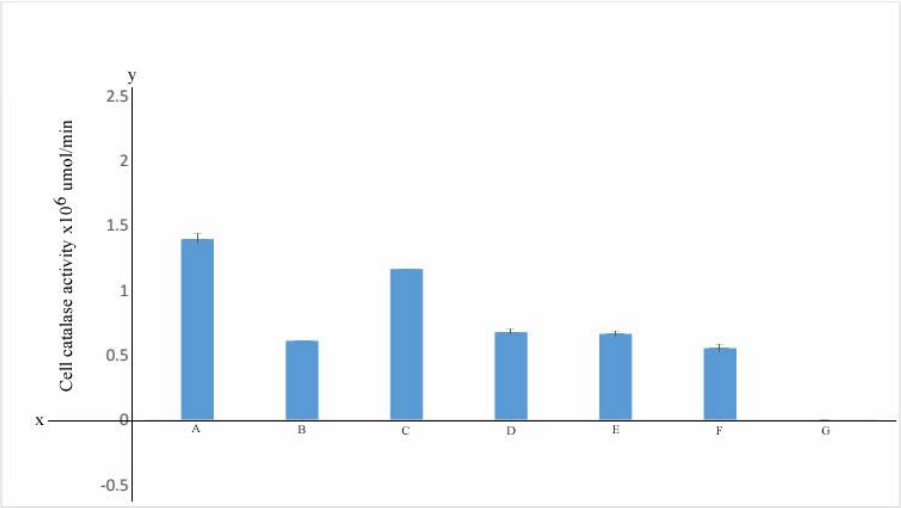

Fig. 4. Difference in catalase activities of isolates after laboratory exposure to MAs X-ray factor
A= S. aureus (X-ray unit isolate), B = S. aureus (Ultrasound unit isolate), C = S. aureus (Mammogram unit isolate), D = P. aeruginosa, E = B. Subtilis, F = E. coli, G = E. faecalis

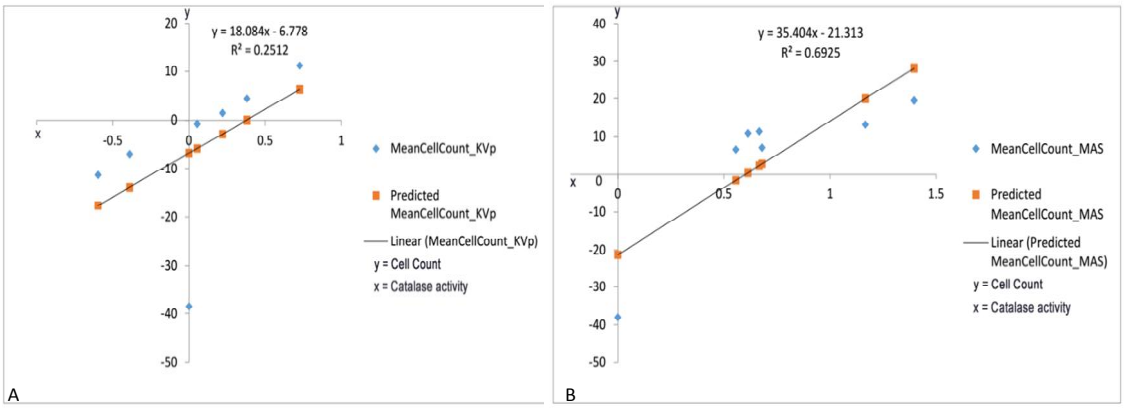

Fig. 5. Relationship between cell count and catalase activities of isolates after laboratory exposure to X-ray of KVp (A) and Mas (B) exposure factor

radiation due to their ability to secrete catalase enzyme.

Statistical analysis shows that there is a positive correlation between catalase activities and cell survival with R value of 0.2512 and 0.6925 for KVp and MAs respectively. This is probable because cellular survival to a large extent was dependent on the ability of the cell to synthesize catalase enzyme which serve as a defense mechanism to the radiation [35]. The difference between the R-value for KVp and MAs exposure factors shows that there is a higher correlation between cell survival and catalase activities of cells exposed to MAs exposure factors hence more cell survival which is probably due to the lower irradiation power of MAs and its extended irradiation time [7] provided for more cellular adaptability to the radiation.

4. CONCLUSION

The application of radiations in clinical X-ray diagnosis promotes the public assumption that X-ray units and equipment may be free from nosocomial microbes. Thereby influencing the level of sanitization practiced by radiographers on X-ray equipment, making them potential fomites for nosocomial pathogen and thus, a

public health threat to patients and Staff. This study reveals the presence of *P. aeruginosa, B. Subtilis, E. coli, E. faecalis and* Methicillin Resistant *Staphylococcus aureus* (MRSA), a public health superbug within the radiology equipment. These isolates expressed cellular adaptiveness when exposed to clinical X-ray irradiation thereby enhancing their ability to survive within the radiology department. This adaptability is aided by the isolates ability to synthesize high level of catalase which helped them to attenuate the harmful effect of the oxidative stress built-up by these clinical radiations.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Ochie K, Ohagwu CC. Contamination of X-ray equipment and accessories with nosocomial bacteria and the effectiveness of common disinfecting agents. African Journal of Basic & Applied Sciences. 2019; 1(2):31-35
2. Hayashi S, Koibuchi H, Taniguchi N, Hirai, Y. Evaluation of procedures for decontaminating ultrasound probes. Journal of Medical Ultrasonics. 2012;39: 11-14.
   DOI: 10.1007/s10396-011-0332-9
3. Hayashi K, Hirayama J, Goldsmith, CE, Coulter WA, Millar BC. Exposure to clinical X-ray radiation does not alter antibiotic susceptibility or genotype profile in Gram-negative and Gram-positive clinical pathogens. British Journal of Biomedical Science, London. 2012;63(3):119-122.
   DOI: 10.1080/09674845.2012.12069137
4. Akpochafor MO, Adeosun AS, Eze CU, Adeneye S, Aweda MA. Assessment of ultrasound equipment as a possible source of nosocomial Infection in Lagos State Hospitals and Radio Diagnostic Centres. Radiography. 2014;13(1):79-86.
   DOI:10.1016/j.radi.2014.09.008
5. Strommenger B, Kettlitz C, Werner G, Witte W, Strommenger. Multiplex PCR Assay for simultaneous detection of nine clinically relevant antibiotic resistance genes in Staphylococcus aureus. Journal of Clinical Microbiology. 2003;(41)4089–94.
   DOI: 10.1128/JCM.41.9.4089-4094.2003
6. Oshoma CE, Ehigiamusoe FO, Olele NE. Microorganisms associated with usable equipments in the Radiology Unit of University of Benin Teaching Hospital. African Scientist. 2016;17(4):275-283.
7. Ching W, Robinson J, McEntee M. Patient-based radiographic exposure factor selection: a systematic review. Journal of Medical Radiation Sciences. 2014;61(3), 176–190.
   DOI: 10.1002/jmrs.66
8. Wall BF, Kendall GM, Edwards AA, Bouffler S, Muirhead CR, Meara JR. Whats are the risks from medical X-rays and other low dose radiation? The British Journal of Radiology. 2006;79(1):285-294.
   DOI: 10.1259/bjr/55733882
9. Hashimoto T, Kunieda T. DNA Protection Protein, a Novel Mechanism of Radiation Tolerance: Lessons from Tardigrades. Life. 2017;7(2):26-37.
   DOI: 10.3390/life7020026
10. Scharer OD. Chemistry and biology of DNA repair. Angewandte Chemie. 2003;42 (26)2946–2974.
11. Sigler K, Chaloupka J, Brozmanova J, Stadler N, Hofer M. Oxidative Stress in Microorganisms. Folia Microbiology. 1999; 44(6):587-624.
    DOI: 10.1007/BF02825650
12. Han W, Yu KN. Response of Cells to Ionizing radiation. Advances in Biomedical Sciences and Engineering. 2009;59:204-262.
    DOI:10.2174/9781608050406109010102 04.
13. Gaupp R, Ledala N, Somerville AG. Staphylococcal response to oxidative stress. Frontiers in Cellular and Infection Microbiology. 2012;2(1)1-33.
    DOI: 10.3389/fcimb.2012.00033
14. Deisseroth A, Dounce, AL. Catalase: physical and chemical properties, mechanism of catalysis and physiological role. Physiological Reviews. 1970;50(3) 319–375.
    DOI: 10.1152/physrev.1970.50.3.319

15. Ankita N, Liang-Jun Y, Chandan KJ, Nilanjana D. Role of catalase in oxidative stress and age-associated degenerative diseases. Oxidative Medicine and Cellular Longevity. 2019;29(1)1-19.
DOI: 10.1155/2019/9613090

16. Baureder M, Reimann R, Hederstedt L. Contribution of catalase to hydrogen peroxide resistance in *Enterococcus faecalis*. FEMS Microbiology Letters. 2019;331(2)160–164.
DOI: 10.1111/j.1574-6968.2012.02567.x

17. Public Heath England. Detection and enumeration of bacteria swabs and other environmental samples. Microbiology services, food, water and environmental microbiology standard method. Public Health England Publication, London, UK. 2014;21.

18. Cheesbrough M. District laboratory practice in tropical countries. Part 2: Cambridge University Press. Cambridge CBZ ZRU, UK. 2002;64-68:136-142.
DOI: 10.1017/CBO9780511581304

19. Bergeys DH and John G Holt. Bergey's manual of determinative bacteriology. Philadelphia: Lippincott Williams & Wilkins. 2000;102-112.

20. Bauer AW, Kirby WMM, Sherris JC, Turck M. Antibiotic susceptibility testing by a standardized single disk method. American Journal of Clinical Pathology. 1996;45: 493-496.

21. Zhang K, Sparling J, Chow BL, Elsayed S, Hussain Z, Church DL. New quadriplex PCR assay for detection of methicillin and mupirocin resistance and simultaneous discrimination of *Staphylococcus aureus* from coagulase-negative staphylococci. Journal of Clinical Microbiology. 2004; 42(11):4947-4955.
DOI: 10.1128/JCM.42.11.4947-4955.2004

22. Sanders ER. Aseptic laboratory techniques: Plating methods. Journal of Visualized Experiments. Advance online publication. 2012;63:3064-3069.
DOI: 10.3791/3064

23. Focea R, Poiata A, Creanga D, Luchian T. *Staphylococcus aureus* responds to accelerated electrons and low dose X-Rays. Romania Journal of Physics. 2012; 57(7):1167–1176.

24. Lagier JC, Edouard S, Pagnier I, Mediannikov O, Drancourt M, Raoult D. Current and past strategies for bacterial culture in clinical microbiology. Clinical microbiology reviews. 2015;28(1):208–236.
DOI: 10.1128/CMR.00110-14

25. Valentine JS, Wertz DL, Lyons TJ, Iou LL, Goto JJ, Gralla EB. The dark side of dioxygen biochemistry. Current Opinion in Chemical Biology. 1998;2:253-262.

26. Whittenbury R. Hydrogen peroxide formation and catalase activity in the Lactic Acid Bacteria. Journal of General Microbiology. 1963;35:13-26.
DOI: 10.1099/00221287-35-1-13

27. Walkley A, Black IA. A critical examination of rapid method for determining organic carbon in soils. Soil Science. 1947;63: 251-263.

28. Ogbeibu AE. Biostatistics: A practical approach to research and data handling 2nd edition. Mindex Publishing Company Lagos, Nigeria. 2005;264-268.

29. Craig W, Dimitre H, Jeffrey S. Ultrasound imaging in radiation therapy: From Interfractional to Intrafractional Guidance. Cureus. 2015;7(6):e280.
DOI: 10.7759/cureus.280

30. Shrestha B, Pohkrel B, Mohapatra, T. Antibiotic susceptibility pattern of nosocomial species of *Staphylococcus aureus* in a tertiary care hospital in Nepal. Nepal Medical College Journal. 2009;135 (1):234-238.

31. Anand KB, Agrawal P, Kumar S, Kapila K. Comparison of cefoxitin disc diffusion test, oxacillin screen agar, and PCR for mecA gene for detection of MRSA. Indian Journal of Medical Microbiology. 2009;27(1):27-9.

32. Kapoor G, Saigal S, Elongavan A. Action and resistance mechanisms of antibiotics: A guide for clinicians. Journal of Anaesthesiol Clinical Pharmacology. 2017; 33(3):300–305.
DOI: 10.4103/joacp.JOACP_349_15

33. International Commission on Radiological Protection (ICRP). General Principles for the radiation protection of workers. ICRP Pub. 1998;75:3-6.

34. Yasuko H, Tatsuo I. Cellular responses to ionizing radiation change quickly over time during early development in Zebrafish. Cell Biology International. 2019;43:516–527.
DOI: 10.1002/cbin.11117

35. Doctrow SR, Lopez A, Schock AM, Duncan NE, Jourdan MM, Olasz EB, Moulder JE, Fish BL, Mäder M, Lazar J, Lazarova Z. A synthetic superoxide dismutase/catalase mimetic EUK-207 mitigates radiation dermatitis and promotes wound healing in irradiated rat skin. Journal of Investigative Dermatology. 2013;133(4):1088–1096.
DOI: 10.1038/jid.2012.410

Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sdiarticle4.com/review-history/55979