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EXPLORATION OF *AVERRHOA CARAMBOLA* EXTRACT AS AN *E. COLI* BIOFILM MODULATING AGENT

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ABSTRACT

*Escherichia coli*is a gram-negative bacillus associated with intestinal illness in humans. *E. coli* biofilm consists of a bacterial colony embedded in a matrix of extracellular polymeric substances (EPS), which protects the microbes from adverse environmental conditions and results in infection. Biofilm physiology is characterized by increased tolerance to stress, antibiotics, and host immunological defence. *Averrhoa carambolaL.*, commonly known as carambola or star fruit, is a perennial tree in the family Oxalidaceae. It contains oxalic acid and caramboxin. It is a rich source of minerals and vitamins, including magnesium, potassium, and vitamin C. In this study, the antibacterial properties of *Averrhoa carambola* fruit water extract (AW) and *Averrhoa carambola* ethanolic extract (AE) against *E. coli* were evaluated, and their effects on biofilm formation and dispersion were determined. The broth microdilution method was used to determine the minimum inhibitory concentration (MIC) at concentrations rangingfrom 1 µl/ml to 25 µl/ml, and absorbance was measured at 600 nm. An agar diffusion assay was performed to determine the zone of inhibition for AW and AE, with the zones7.97mm and 9.21mm,respectively, observed at 6 µl/ml and 13 µl/ml concentrations in comparison with the antibiotic gentamicin. Biofilm dispersion assay was performed, and before and after staining, the optical density was recorded. Both AW and AE extracts have exhibited a zone of inhibition against *E. coli*, and optical density measurements indicated inhibition of bacterial growth at 75 µg/ml. Biofilm dispersion assay values recorded before and after staining demonstrated antibiofilm activity. The findings indicate that AW and AE extracts possess antibacterial activity and inhibit *E. coli* biofilm formation.

KEYWORDS: *Averrhoa carambola*, Extract, Biofilm, Exopolysaccharide, MIC, AW, AE.

1. INTRODUCTION

Escherichia coli are comma-shaped bacteria, and gram-negative bacillus are known to be a part of normal intestinal flora but can also be the cause of intestinal and extra-intestinal illness in humans. There are hundreds of identified *E. coli* strains, resulting in a spectrum of disease from mild, self-limited gastroenteritis to renal failure and septic shock. Its virulence contributes to *E. coli's* ability to evade host defenses and develop resistance to common antibiotics.[1] The review will divide *E. coli* infections into those causing intestinal illness and those causing extra-intestinal illness. Intestinal illnesses will be described by the causative *Escherichia coli* subtypes, including entero-toxigenic *Escherichia coli* (ETEC), entero-hemorrhagic *Escherichia coli* (EHEC), which is also known as shiga toxin-producing *Escherichia coli* (STEC) and will be referred to as EHEC/STEC, entero-invasive *Escherichia*

coli (EIEC), enteropathogenic *Escherichia coli* (EPEC), and entero-aggregative *Escherichia coli* (EAEC).[2] Extraintestinal illnesses will be described based on clinical disease. Intestinal illness can be caused by a variety of organisms. Watery diarrheal illness is most commonly caused by viruses, including norovirus and rotavirus, but can also be caused by bacteria, including *Staphylococcus aureus*, *Bacillus cereus*, and *Vibrio cholerae*, among others. For patients presenting with inflammatory or bloody diarrheal symptoms, it's important to consider etiologic causes, including *Shigella spp.*, *Salmonella spp.*, *Campylobacter jejuni*, and *Yersinia*, among others. Extra-intestinal infections previously discussed can be caused by a variety of viruses and bacteria and are dependent on the specific illness.[3]

Pseudomonas aeruginosa and *E. coli*are the most prevalent gram-negative biofilm- forming medical device-associated pathogens, particularly with respect to catheter- associated urinary tract infections. In a similar manner to gram-positive bacteria, Gram- negative biofilm formation is fundamentally determined by a series of steps, namely adhesion, cellular aggregation, and the production of an extracellular polymeric matrix. The majority of Gram-negative biofilm consists of polysaccharides of a simple sugar structure that provide an optimum environment for the survival and maturation of bacteria, allowing them to display increased resistance to antibiotics and predation.[4] *E. coli* biofilm consists of a bacterial colony embedded in a matrix of extracellular polymeric substances (EPS), which protects the microbes from adverse environmental conditions and results in infection. Besides being the major causative agent for recurrent urinary tract infections, biofilm physiology is characterized by increased tolerance to stress, biocides, including antibiotics, and host immunological defenses.The description of a widespread association between bacteria and surfaces dates back to the seventeenth century, when Antonie van Leeuwenhoek observed animalcules on the dental plaque surface. Bacteria have most often been studied in artificial but controlled conditions using agitated single-celled planktonic cultures, whereas bacterial interactions with a surface promote behaviours leading to the development of structured and heterogeneous matrix- encased bacterial communities known as biofilms. Over the past decade, the adverse effects of biofilm development on human activities have spurred research to develop strategies to combat detrimental biofilms.[5]

In a liquid environment, bacteria are subjected to hydrodynamic forces when approaching surfaces. In Gram-negative bacteria such as *E. coli*, active motility is dependent on a flagellar apparatus that is necessary for them to swim in liquid or semi-liquid medium. Studies using *E. coli* as a model system showed that motility itself is required to form a biofilm and allows bacteria to spread along the surface. Although this suggests that the requirement for force-generating cell surface organelles is a common theme in biofilm formation, nonmotile bacteria can still form biofilms under certain conditions.Initial bacterial adhesion to abiotic surfaces is highly dependent on physicochemical and electrostatic interactions between the bacterial envelope and the substrate. Attractive and repulsive forces between the bacteria and the surface lead to reversible attachment of bacteria to the surface.[6] Environmental conditions such as pH, ionic force of the medium, and temperature influence this reversible attachment, as well as the nature of the surface itself. Hydrophobic surfaces, such as plastic, are more likely to be colonized by bacteria than hydrophilic surfaces, such as glass and metal.Irreversible adhesion to surfaces is strengthened by adhesive organelles of the fimbrial family, including type 1 fimbriae, curli, and conjugative pili. Biofilm maturation corresponds to the three- dimensional growth of the biofilm after initial attachment to the surface, leading to the formation of a heterogeneous physicochemical environment in which biofilm bacteria display characteristic physiological traits that distinguish them from planktonic counterparts.

One of the most distinctive features that distinguishes biofilms from planktonic populations is the presence of an extracellular matrix embedding the biofilm bacteria and determines mature biofilm architecture. The biofilm matrix is composed mainly of water but also includes exopolysaccharide polymers, proteins, nucleic acids, lipids, absorbed nutrients, and metabolites.[7] The matrix offers a hydrated viscous layer protecting embedded bacteria from desiccation and host defenses, and may also act as a diffusion barrier for antimicrobial compounds. The adhesive properties of the matrix enable bacteria to remain in proximity to the surface and adhere to each other, contributing to the three- dimensional growth of the biofilm structure. Several exopolysaccharides found in the *E. coli* biofilm matrix, such as cellulose, PGA, and colonic acid, are key components of the biofilm matrix.[8]

On that background, in the present work, we have extensively cultured *E. coli* in various media and culture conditions, and determined its growth inhibition under the stress of various compounds. The present work will play a pivotal role in extending the work towards understanding *E. coli* biofilm physiology, biofilm phenotypes, and associated virulence in the host system.

2. MATERIALS AND METHODS

2.1 Demonstration of *E. coli*

First, remove the *E. coli* culture from the incubator and place the mother culture in a laminar airflow unit. Then aliquot 50 µl of culture onto one end of a clean,grease-free slide. Using a clean slide, prepare a smear on the slide. Afterward, hold the slide over the burner flame for 5–7 sec. Flood the slide with crystal violet for 30 sec. Subsequently, rinse the slide with distilled water. Flood the slide with iodine solution for

5–10 seconds. Then wash gently again with water. Allow the slide to dry at room temperature. At last, observe the slide under the microscope.

2.2 Preparation of Culture Media

Nutrient broth has the same formulation as nutrient agar, only agar has been omitted. It is made from a mixture of tryptone/peptone and beef or yeast extract and NaCl. Sodium chloride helps maintain osmotic pressure.

Nutrient agar is made with peptone, beef extract, and agar. An agar slant tube is a screw- capped culture tube partly filled with an agar mix.

2.3 Determination of minimum inhibitory concentration of unknown compound by the broth microdilution method

First, we will take 10 test tubes; then, we will add 10 µl of egg albumin to each test tube, using a micropipette for each 10 µl. After that, add distilled water to the same test tube 10µl (1 to 10 test tubes). We will then mix it thoroughly and add it to 1 to 10 test tubes. Then, check the absorbance of each test tube through the colorimeter machine, we have to bring the zero with the water-filled test tube into the colorimeter machine. One unknown sample is taken, and using the same technique, I have determined the absorbance of the unknown sample.[9]

2.4 Agar diffusion assay

Paper discs soaked with the compounds were placed on agar plates inoculated with *E. coli*. Plates were incubated at 37°C for 48 hrs and the extent of bacterial inhibition was measured from the diameter of the clear zone around the disc.

2.5 Antibiotic sensitivity assay

Suspend 1.5 of nutrient broth powder in 100ml of distilled water with 3 g of agar agar powder. Stir this mixture until all components fully dissolve. Autoclave the dissolved mixture at 1210 C at 15 psi pressure for 15-20 minutes. Once the nutrient agar is put into each plate, leave the plate on the sterile surface until the agar has solidified.After that, mark 1, 2, 3, 4 in each plate and add gentamicin according to the required concentrations and incubate it for 48 hours. After that, take the OD.[10]

2.6 Motility test

First, take a test tube, add *E. coli* cells and the compound, and incubate for 24 hours. Now, take agar powder, 5% dextrose, and 1% agarose, prepare an agar medium, and pour it into Petri plates. Then, add the previously incubated cells by injecting them into the center of the agar plate using a pipette.Incubate at 35°-37°C and examine daily for up to 7 days. Observe for a diffuse zone of growth flaring out from the line of inoculation.

2.7 Evaluation of biofilm formation of selected E. coli

*E. coli*were incubated at 37 °C for 48h in test tubes containing LB media. Following incubation, cells were washed three times with sterile phosphate buffer saline (PBS). Test tubes were then stained with 0.1% (v/v) safranin, and the excess stain was removed by washing again with sterile PBS and dried at 37 °C. Safranin present in the test tube surface adhered *E. coli* were then dissolved in 30% (v/v) glacial acetic acid. Absorbance of dissolved safranin was measured at 492 nm. Assay was performed in triplicate in three different sets.[11]

2.8 Biofilm dispersion assay

Take a test tube, add bacterial cells, and incubate for 48 hours. After 48 hours, add the compound to the test tube and incubate for an additional 48 hours. After that, stain with crystal violet and leave for 1 minute, then check the OD.

2.9 Studies of antibiofilm activity of test compounds

To determine the antibiofilm activity of test compounds, *E. coli*were separately inoculated with 1-25µg/ml of each compound and incubated at 37 °C for 48 h. Test tubes were then washed, stained, kept for 10 mins, and absorbance was measured as in the biofilm evaluation assay described above. Percentages of biofilm attenuation by each compound against *E. coli* were calculated with respect to the untreated control.[11]

3. RESULTS

3.1 Antibacterial activity of *Averrhoa carambola* extracts against Escherichia coli

The antibacterial activity of *Averrhoa carambola* fruit extracts was evaluated against *Escherichia coli* (*E. coli*) using agar diffusion and broth microdilution assays. The minimum inhibitory concentration (MIC) represents the lowest concentration of an antimicrobial agent capable of inhibiting visible bacterial growth under in vitro conditions. In the present study, the MIC of the extracts was determined by the broth microdilution method using concentrations ranging from 1 µl/ml to 25 µl/ml.

The antibacterial effect of the extracts was further examined using the agar diffusion assay. The *Averrhoa carambola* fruit water extract (AW) showed bacterial inhibition at a concentration of 6 µl/ml, producing approximately 20% inhibition and forming a clear zone of inhibition measuring 7.97 mm. Similarly, the *Averrhoa carambola* ethanolic extract (AE) demonstrated stronger antibacterial activity, showing 68% inhibition at a concentration of 13 µl/ml and producing a clear inhibition zone measuring 9.21 mm.

The antibiotic gentamicin, used as the positive control, exhibited a larger zone of inhibition measuring 10 mm at a concentration of 2 µl/ml, confirming the susceptibility of the tested bacterial strain and validating the experimental conditions. The comparative antibacterial activities of the extracts and the control antibiotic are summarized in Table 1, while the inhibition zones observed in the agar diffusion assay are illustrated in

Figure 1.

Table 1: Antibacterial activity of *Averrhoa carambola* extracts against *E. coli.*

Compound	Concentration	Zone of inhibition
Averrhoa carambola fruit water extract (AW)	6µl/mL	7.97mm
Averrhoa carambola ethanolic extract (AE)	13µl/mL	9.21mm
Control (Gentamicin)	2µl/mL	10mm

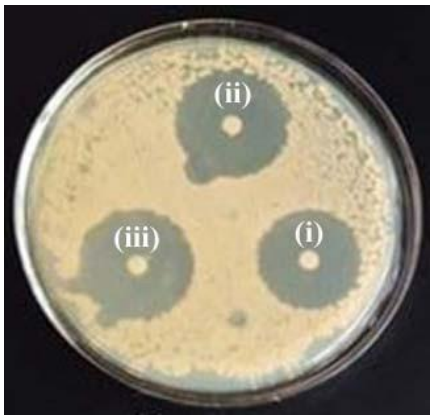

Figure 1: Zone of inhibition produced by *Averrhoa carambola* fruit extracts and gentamicin against *E. coli* in the agar diffusion assay. Agar diffusion assay showing antibacterial activity of samples: (i) AW 6µl/mL, (ii) AE 13µl/mL, (iii) Gentamicin 2µl/mL. Clear zones indicate bacterial growth inhibition.

3.2 Biofilm dispersion assay

Biofilm formation and dispersion represent important stages in the biofilm life cycle, where bacterial cells transition between surface-associated communities and planktonic forms. The antibiofilm activity of *Averrhoa carambola* extracts against *E. coli* was evaluated using a biofilm dispersion assay.

The results demonstrated that the fruit water extract inhibited biofilm formation by approximately 65% at a concentration of 5 µl/ml. In comparison, the ethanolic extract exhibited slightly higher antibiofilm activity, showing 71% inhibition at a concentration of 6 µl/ml. These observations indicate that both extracts are capable of disrupting biofilm development and stability in *E. coli*, with the ethanolic extract showing comparatively stronger antibiofilm activity. The quantitative results of the biofilm dispersion assay are presented in Figure 2.

Figure 2: Biofilm dispersion assay showing inhibition of *E. coli* biofilm formation by *Averrhoa carambola* extracts.

3.3 Catalase test

Biochemical characterization of the bacterial isolate was performed using the catalase test. When hydrogen peroxide was added to the bacterial smear, immediate formation of oxygen bubbles was observed. The appearance of bubbles indicates the decomposition of hydrogen peroxide by the catalase enzyme.

This positive catalase reaction confirms that the bacterial isolate used in the study is catalase-positive, which is consistent with the known biochemical characteristics of *Escherichia coli*. The catalase test results are shown in Figure 3.

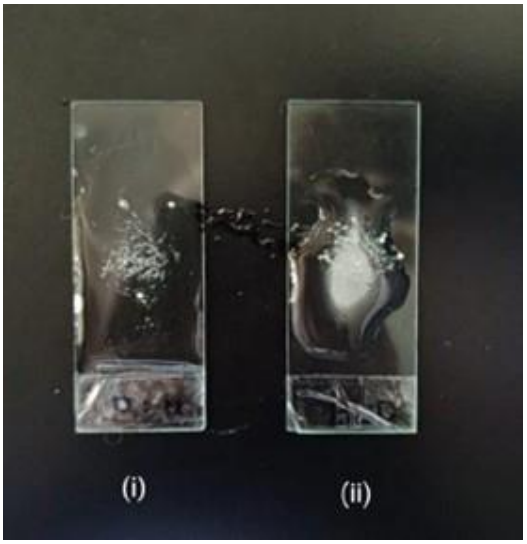

Figure 3: Catalase test demonstrating catalase activity of *E. coli.* The formation of visible bubbles indicates a positive catalase reaction.

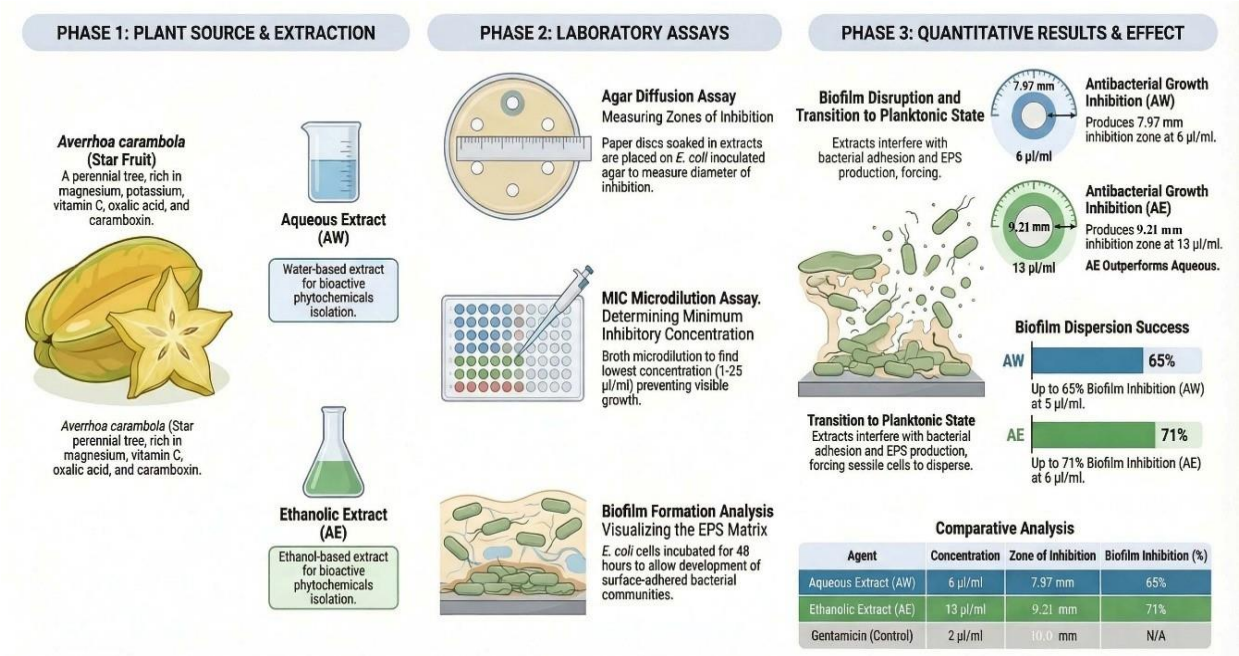

Figure 4: Extracts of *Averrhoa carambola* (star fruit) were prepared using aqueous and ethanolic solvents and evaluated against E. coli through agar diffusion, MIC, and biofilm assays. Both extracts inhibited bacterial growth and disrupted biofilm formation, with the ethanolic extract showing stronger antibacterial activity (12 mm zone; 71% biofilm inhibition) compared to the aqueous extract (7.97 mm; 65%). Overview of the exploration of *Averrhoa carambola* extract as an *E. coli* biofilm modulating agent.

4. DISCUSSION

Biofilm formation is one of the most important survival strategies employed by pathogenic bacteria, enabling them to persist in hostile environments and evade antimicrobial agents. Biofilms formed by *Escherichia coli* (*E. coli*) are particularly significant in clinical settings, especially in catheter-associated infections and recurrent urinary tract infections.[12] The extracellular polymeric substances (EPS) matrix surrounding bacterial cells protects them from antibiotics and host immune responses, thereby increasing bacterial tolerance and persistence. Consequently, the discovery of natural compounds capable of disrupting bacterial growth and biofilm formation has become an important area of antimicrobial research.[13]

In the present study, the antibacterial and antibiofilm activities of *Averrhoa carambola* (*A. carambola*) fruit extracts were investigated against *E. coli*. The agar diffusion assay demonstrated that both aqueous extract (AW) and ethanolic extract (AE) inhibited bacterial growth. However, AE exhibited comparatively stronger antibacterial activity, producing a larger zone of inhibition than AW. This difference in activity may

be attributed to the higher solubility of bioactive phytochemicals in organic solvents such as ethanol, which allows more efficient extraction of phenolic compounds, flavonoids, and other antimicrobial constituents present in medicinal plants. Previous studies have also reported that ethanol-based plant extracts often show stronger antimicrobial activity compared to aqueous extracts due to improved extraction of bioactive secondary metabolites.[14]

The broth microdilution assay further confirmed the inhibitory potential of the extracts, demonstrating a concentration-dependent reduction in bacterial growth. The antibacterial activity observed in this study suggests that phytochemicals present in *A. carambola* may interfere with essential cellular processes such as membrane permeability, enzyme activity, or protein synthesis in *E. coli.*[14] Gentamicin, used as a positive control in this study, exhibited significantly higher antibacterial activity, which is consistent with its well-established mechanism of action involving inhibition of bacterial protein synthesis through binding to the 30S ribosomal subunit.[15]

Apart from planktonic growth inhibition, the ability of plant-derived compounds to disrupt bacterial biofilms is of considerable importance. Biofilm-associated bacterial cells exhibit significantly greater resistance to antimicrobial agents than planktonic cells. In this study, both AW and AE demonstrated notable antibiofilm activity in the biofilm dispersion assay, with AE showing slightly higher inhibition compared to AW. The observed reduction in biofilm formation suggests that phytochemical components of *A. carambola* may interfere with bacterial adhesion, quorum-sensing pathways, or EPS production, which are critical steps in biofilm development.[16]

Several previous studies have reported antibiofilm properties of plant-derived compounds against pathogenic bacteria. For instance, vitexin has been shown to alter the surface hydrophobicity of *Staphylococcus aureus* and inhibit biofilm formation.[11] Similarly, natural phytochemicals and small bioactive molecules have demonstrated significant antibiofilm activity against *Pseudomonas aeruginosa* and other bacterial pathogens. These findings support the hypothesis that plant secondary metabolites can act as potential antibiofilm agents and may serve as alternatives or complementary agents to conventional antibiotics.[11]

The biochemical characterization of the bacterial isolate using the catalase test confirmed the presence of catalase enzyme activity. The formation of visible bubbles following the addition of hydrogen peroxide indicates the decomposition of hydrogen peroxide into water and oxygen, which is a characteristic feature of catalase-positive bacteria such as *E. coli*. This observation further supports the correct identification and metabolic activity of the bacterial strain used in the present study.

Overall, the findings of this study demonstrate that *A. carambola* fruit extracts possess both antibacterial and antibiofilm activity against *E. coli*. The comparatively stronger activity observed in AE suggests that organic solvent extraction may enhance the recovery of bioactive phytochemicals responsible for antimicrobial activity. However, further investigations, including phytochemical characterization, identification of active compounds, and mechanistic studies, are necessary to elucidate the molecular basis of the antibacterial and antibiofilm effects of *A. carambola* extracts.

5. CONCLUSION

The present study demonstrates that fruit extracts of *Averrhoa carambola* possess notable antibacterial and antibiofilm activity against *Escherichia coli* (*E. coli*). Both aqueous extract (AW) and ethanolic extract (AE) exhibited inhibitory effects on bacterial growth, as confirmed by agar diffusion and broth microdilution assays. The aqueous extract produced a zone of inhibition of 7.97 mm at a concentration of 6µl/ml, while the ethanolic extract showed comparatively stronger antibacterial activity with a 9.21 mm inhibition zone at 13µl/ml. The antibiotic gentamicin, used as the positive control, exhibited a larger inhibition zone, confirming the susceptibility of the tested bacterial strain.

In addition to antibacterial activity, both extracts demonstrated significant antibiofilm potential against *E. coli.* The biofilm dispersion assay revealed that AW inhibited biofilm formation by 65% at 5µl/ml, whereas AE exhibited 71% inhibition at 6µl/ml, indicating comparatively higher antibiofilm efficacy. These findings suggest that bioactive phytochemicals present in *Averrhoa carambola* may interfere with bacterial growth and biofilm development.

Overall, the results indicate that *Averrhoa carambola* fruit extracts possess promising antibacterial and antibiofilm properties against *E. coli*. Further studies involving phytochemical characterization and mechanistic investigations are required to identify the active compounds responsible for these effects and to explore their potential application as natural antimicrobial agents.

AUTHOR CONTRIBUTION

MCD and SA have developed the idea and conceptualized the idea. RS, GD, and PD have performed all experiments and collected data. MCD, SA, and MG have analysed all observed data. RS and GD have written the manuscript, MCD, SA, and MG have edited the manuscript and finalised the present version of the manuscript.

Competing Interest

All authors declare that they have no competing interests.

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