Original Article

# Molecular Detection of ZapA Gene and Multidrugs Resistance Among *Proteus Mirabilis* Isolated From Different Clinical Samples in Al-Najaf Province

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## Abstract

**Background:** *Proteus mirabilis* is regarded as a significant causal agent in both community and hospital-acquired illnesses. P. mirabilis exhibits factors implicated in both cell-associated and extracellular pathogenicity. Included among the expressed factors are virulence factors that can induce pathogenicity in infected hosts, such as lipopolysaccharides, iron-sensitive proteases, urease, haemolysin systems, and swarming fimbriae. **Methods:** The strain was extracted from clinical specimens obtained from patients in prominent hospitals in Al-Najaf province. Traditional identification using microscopic morphology, colony characteristics on selective and differential culture media, and biochemical reactions revealed that only 20 out of 140 clinical samples tested positive for P. mirabilis. **Results:** The distribution of these 20 samples resulted in positive growth of P. mirabilis. The gender distribution consisted of 13 females (65%) and 7 males (35%). This study encompassed the phenotyping of swimming and swarming motilities, in addition to blood haemolysis and proteolytic enzymes. The results indicated that the isolates hemolyze blood and that 90% of the isolates generate protease. **Conclusion:** All clinical isolates of P. mirabilis possess the ability to generate a substantial biofilm. The disc diffusion antibiotic susceptibility assay of the isolates for frequently utilised antibiotics against P. mirabilis demonstrated a broad spectrum of resistance rates. The greatest resistance percentage was recorded in Nitrofurans (Nitrofurantoin) at 100%, followed by Macrolides (Azithromycin) and Tetracyclines (Tetracycline and Doxycycline) at 90%, Ciprofloxacin at 85%, and Amoxicillin at 80%. The lowest resistance percentages were noted for Levofloxacin (35%) and Aminoglycosides (Gentamicin) (15%).

**Keywords:** *Proteus Mirabilis, ZapA* Gene, Biofilm, Antibiotic Resistance.

## Introduction

*Morganella morganii* It is included under the Proteobacteria phylum of the Enterobacteriaceae family and exhibits a close relationship with other proteobacterial species.[1] All are rod-shaped bacteria; hence, the genus is assumed to possess the typical properties of facultative anaerobic Gram-negative rods.[2] The organism possesses peritrichous flagella, exhibits vigorous motility, and does not produce spores or capsules; it is oxidase-negative but catalase- and nitrate-positive.[3] The colony differentiation of the enteric bacterium, Proteus mirabilis, exhibits polymorphism, manifesting as vegetative and multiflagellated colony cells.[4] Both swimming and swarming represent the primary behaviours of this organism; however, the capacity to swarm forcefully on surfaces in a directed manner is among the most recognised phenomena in microbiology.[5] P. mirabilis is frequently the causative agent of urinary tract infections (UTI), often triggered by diseases including kidney infections with calculi, catheter-associated UTI, intermittent catheterization-associated UTI, and catheter condoms. The species of the genus Proteus, particularly P. mirabilis, appear to be the most commonly observed in urinary tract infections.[6] Nosocomial secondary infective agents are most

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commonly isolated from severe urinary tract infections, accounting for around 12% of all such cases, and from catheter-induced bacteriuria. They may also induce simple lower urinary tract infections.[7] The pathogenic agent mentioned is capable of inducing infections beyond those specified above. It may opportunistically infect the human respiratory tract, wounds, burns, skin, eyes, ears, nose, and throat, resulting in diseases such as bacteremia and sepsis. The aforementioned bacterium also induces gastroenteritis when food is contaminated.[8]

Potential virulence factors exhibited by P. mirabilis, a quintessential motile bacterium, including those implicated in both the infectious process and the disease. These encompass clustered pili and fimbriae, flagella, haemolysins, ureases, extracellular proteases, amino acid deaminases, and endotoxins including lipopolysaccharides and capsules. Additional variables of Proteus pertain to the evolution of resistance to normal human serum.[9]

A prominent virulence component of P. mirabilis is its distinctive bristling surface motility, which often leads to catheter encrustation, subsequent obstruction, and complex catheter-associated urinary tract infections. Two other virulence factors shown to facilitate crystalline biofilm formation in P. mirabilis are the urease enzyme and capsular polysaccharide. Recent evidence demonstrates that the efflux pump system significantly contributes to the formation of crystalline biofilms in P. mirabilis. The upregulation of the bcr gene, which is part of the efflux pump genes within the major facilitator superfamily (MFS), contributes to the gradual onset of bicycline resistance.

The quorum sensing system in P. mirabilis may contain peptide-encoding genes; hence, their expulsion obstructs signal transduction during biofilm development.[10] The persistent increase in antibiotic resistance in clinical samples of P. mirabilis among hospitalised patients has been a significant worry during recent decades.[11] ESBLs or strains harbouring AmpC-type cephalosporinases are prevalent in certain states. This group exhibits general resistance to additional anti-infective agents apart from the initial group, namely penicillins and cephalosporins (including oximido compounds).[12] Consequently, phenotypic and molecular expressions were associated with antibiotic resistance, virulence factors, and biofilm formation in disease-specific clinical isolates of P. mirabilis.

## Materials and Methods

### Samples Collection and Identification

A total of 140 isolates obtained from patients with infections (urethral, pressure ulcer, colon, otitis media, wound, burn, bacteremia) were collected between April 2024 and February 2025. Ninety-two samples were collected from the midstream urine flow, fifteen from the ear, twelve from the blood, ten from the stool, eight from the burn area, and three from the wound; patients were classified by sex and age. The isolates were inoculated and maintained in oxygenated media (37°C, 24-48 hours) on MacConkey agar and blood agar for chemical testing to classify the bacteria.[13] The bacteria possessing Urease were identified by puncturing and extracting from an Urease-containing agar slant, followed by incubation at 37°C for 24 to 48 hours.

### Detection of Biofilm Formation Tissue Culture Plate Method

The trypticase soy broth medium (TCBM) has a proven track record in inhibiting biofilm formation. Recently isolated organisms were grown in 10 ml of trypticase broth (1% glucose, 37°C, 24 hours). The bacterial suspensions were prepared at a ratio of 100 to 1:60 in the medium. To achieve an optical density at 600nm. Ninety-six flat-bottom wells were filled with 200 µl of the bacterial suspension together with the control microorganisms. The sterile broth was utilised to assess the sterility of the bacteria and the specificity of their connection with the plate (Table 1).

Following incubation at 37°C for 24 hours, the plate was gently tapped to extract the contents of the wells, subsequently undergoing four washes with 200 µl of phosphate buffer. Subsequently, 2% sodium acetate was introduced to the wells for 30 minutes; the adhered bacteria were then eliminated by rinsing the plate. The biofilms produced from the treatment were subjected to crystal violet (0.1% CV) for 30 minutes, after which the surplus stain was eliminated using enough water. Subsequently, the biofilms were dried, and the optical density of the stains was measured at 570 nm using a micro-ELISA reader.

The studies were performed in triplicate, resulting in the recording of the average of three distinct optical densities. Optical density values indicated the extent of bacterial adherence to the wells and their biofilm formation. The calculation of OD values was facilitated by classifying biofilm formation as strong, moderate, or non/weak according to the standard tables from the research of Panda and Bakir.

### Congo Red Agar Method

The selective medium for Congo Red consists of 37 g/l of BHI broth and 50 g/l of sucrose, both produced from Agar No. 1 at 10 g/l, together with 0.8 g/l of Congo Red. A concentrated solution of Congo Red in water was prepared and subsequently incorporated into BHI agar, which had been autoclaved at 55°C. The previously isolated uropathogens were grown on pre-prepared CRA plates and incubated in air at 37°C for 24 hours. The black, dry, crystalline colonies that developed on the CRA plates were identified as biofilm.[14] Other people established colonies exhibiting various hues of pink to red, which were deemed deficient in protein synthesis.[15]

**Table 1: Classification of Bacterial Biofilm Formation by Tissue Culture Plate Method (TCP)[16].**

| Biofilm Formation | Adherence | Mean OD Values |
|---|---|---|
| Non/Weak | Non/Weak | < 0.120 |
| Moderate | Moderately | 0.120-0.240 |
| High | Strong | > 0.240 |

### Antibiotic Susceptibility Test of P. mirabilis: The Test of Culture-sensitivity

The susceptibility of P. mirabilis isolates was assessed using Bauer-Kirby disc diffusion methodology.[17] Mueller-Hinton plates were inoculated with a 0.5 McFarland suspension of P. mirabilis, followed by the placement of discs. The inhibition zones were measured in millilitres following overnight incubation at 37°C, utilising various antibiotics, including Amoxicillin, Azithromycin, Doxycycline, Tetracycline, Ceftriaxone, Gentamicin, Ceftazidime, Nitrofurantoin, Imipenem, Levofloxacin, Ciprofloxacin, Ampicillin, Tobramycin, Amikacin, and Meropenem.

### DNA Extraction of Bacteria

In accordance with the kit manufacturer's instructions (G-Biosciences, USA), the DNA was extracted and subsequently detected by electrophoresis utilising a UV transilluminator. The Polymerase Chain Reaction (PCR) method was employed to detect the zapA gene in P. mirabilis. Primers developed by Alpha DNA, a Canadian company, are designated as forward and reverse primers in Table 1. The objective of the conditions enumerated in Table 2 is to enhance the circumstances of zapA. To ascertain the dimensions of the PCR components, legitimate products were subjected to electrophoresis following the addition of a 1% agarose gel. The gel underwent electrophoresis (1.5 hours; 85 volts; 5 mg/ml Ethidium Bromide). A UV transilluminator's designated position was employed to document a singular, distinct light band using a gel methodology. A 1,000-bp ladder (Bionaer, Korea) was employed to ascertain the molecular weights of additional components.

### Molecular Identifications

The zapA gene was utilised for the identification of P. mirabilis. The primers were produced by Canadian Alpha DNA, as indicated in Table 2. To enhance the results for ascertaining the size of the PCR, 1% agarose gel electrophoresis was employed. The gel was stained for approximately 1.5 hours at 80 volts with 4 mL of ethidium bromide. The molecular weight of the amplified products was assessed utilising a 100bp ladder (Bionaer, Korea).

**Table 2: The Recorded Standard PCR Cycles of the Primer that Apply in the Thermocycler.**

| Steps | Temp, Time |
|---|---|
| Primary denaturation | 95°C for 1 min |
| No. of cycles | 35 |
| Denaturation | 94°C for 30 min |
| Annealing | 53°C for 1 min |
| Extension | 72°C for 1 min. |
| Final extension | 72°C for 5 min |
| *zapA* gene (540bp) | F: ACCGCAGGAAAACATATAGCCC<br>R: GCGACTATCTTCCGCATAATCA |

## Results and Discussion

### Isolation and Identification of P. mirabilis

The study included 140 clinical specimens obtained from patients undergoing treatment for infections, including urinary tract infections, colitis, otitis media, wounds, burns, and bacteremia, at three hospitals in Al-Najaf (Al-Sadder Medical City, Hospital, and Al-Zahra Hospital for Maternity and Children) from April 2024 to February 2025. The samples were identified following cultivation on several types of media. Assessments utilising the VITEK-2 Compact System, with biochemical and morphological investigations.

The initial diagnosis indicated that merely 20 of the bacterial isolates were identified as P. mirabilis, determined through microscopic examination and the characteristics of bacterial colonies under selective and differential growth circumstances. The bacteria appeared as small, rod-shaped, non-capsulated bacilli, with a pinkish-red hue and lacking spore production. The isolates exhibited swarming behaviour on blood agar, characterised by concentric growth rings emanating from a central location. Moreover, when cultivated on MacConkey agar, they failed to generate any lactose-fermenting colonies and exhibited a colouration that was either translucent or pale yellow. Biochemical diagnostic: Subsequently, biochemical testing confirmed the preliminary diagnosis. All P. mirabilis isolates tested negative for urease, oxidase, indole, Voges-Proskauer, and citrate utilisation tests, whereas the methyl red, citrate utilisation, and catalase tests yielded positive results. Alk/A exhibited favourable outcomes for gas production and hydrogen sulphide (H2S) in the triple sugar iron (TSI) assay.

### Antibiotic Susceptibility Profile

The disc diffusion method employed for the culture sensitivity test of P. mirabilis isolates against 16 antibiotics from 12 classes, as per CLSI (2020), demonstrated a broad spectrum of antibiotic resistance rates. Nitrofurans (Nitrofurantoin) exhibited a 100% resistance rate, Macrolides (Azithromycin) and Tetracyclines (Tetracycline) shown a 90% resistance rate, Ciprofloxacin showed an 85% resistance rate, and Amoxicillin presented an 80% resistance rate. Conversely, Levofloxacin had a 35% resistance rate, whereas Aminoglycosides (Gentamicin) demonstrated a 15% resistance rate. All isolates exhibited susceptibility (Table 3). Antibiotic resistance is notably widespread among P. mirabilis bacterial isolates from various clinical samples, as indicated by numerous studies.[18]

**Table 3: Antibiotics Susceptibility test of *p. mirabilis* Isolates.**

| Antibiotics Classes | Antibiotics | Resistant N (%) | Intermediate N(%) | Sensitive N(%) |
|---|---|---|---|---|
| B-lactam agents | Amoxicillin/ clavulanic | 16 (80%) | 1 (5%) | 3 (32 %) |
| Quinolones | Ciprofloxacin | 17 (85%) | 0 (0%) | 3 (15%) |
| | Levofloxacin | 7 (35%) | 9 (45%) | 4 (20%) |
| Macrolides | Azithromycin | 20 (100%) | 0 (0%) | 0(0%) |
| Tetracyclines | Doxycycline | 18 (90%) | 0 (0%) | 2 (10%) |
| | Tetracycline | 20 (100%) | 0 (0%) | 0 (0%) |
| Penicillins | Ampicillin | 17 (85%) | 1 (5%) | 2 (10%) |
| Cephems | Ceftriaxone | 15 (75%) | 0 (0%) | 5 (25%) |
| Monobactams | Imipenem | 13(65%) | 0 (0%) | 7 (35%) |
| Aminoglycosides | Amikacin | 12 (60%) | 3 (15%) | 5 (25%) |
| | Gentamicin | 3 (15%) | 0 (0%) | 17 (85) |
| Nitrofurans | Nitrofurantoin | 20 (100%) | 0 (0%) | 0 (0%) |

## Phenotypic Detection of Some Virulence Factors

P. mirabilis is acknowledged as pathogenic and is linked to many virulence factors. Some were detected via conventional phenotypic approaches in this study, while others were identified through phenotypic and later genotypic methods utilising monoplex PCR. This experiment examined the biofilm-forming potential of P. mirabilis isolates. All twenty isolates (100%) demonstrated well-developed biofilms, as per the results. Subsequently, Table (4) and Figure (1) indicated that no isolates were unable to form the biofilm. The results of this study indicated that all clinical isolates of P. mirabilis can produce biofilm effectively. The results aligned with the findings of Ali and Yousif[5], which indicated that 45 (93.75%) of the 48 P. mirabilis isolates demonstrated significant biofilm development, whereas 3 (6.25%) did not.

**Table 4: The Rate at which *P. mirabilis* Forms Biofilms.**

| Type of Biofilm Formation | *P. mirabilis* N (%) n=20 |
|---|---|
| High | 20 (100) |
| Moderate | 0 (0) |
| Weak/Non | 0 (0) |

Bacteria within biofilms are protected from a variety of stresses, such as immune responses and antimicrobial agents, by their capacity to produce biofilm.

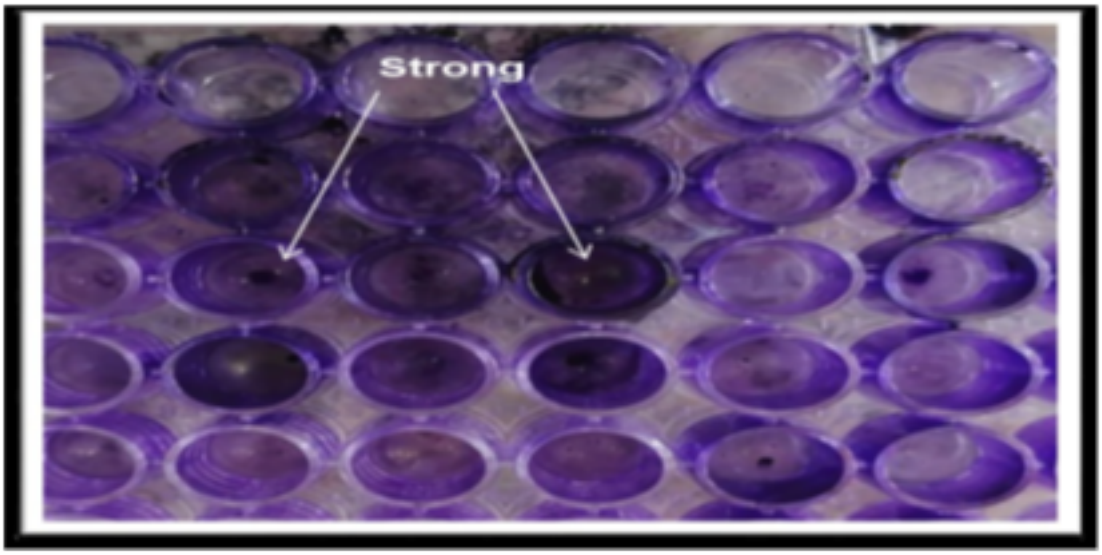


Figure 1: Biofilm Development of P. mirabilis Detected Phenotypically Using the Plate Technique.

## Detection of Protease Enzyme

Bacterial isolate samples were chosen to examine their capacity for protease enzyme production. Proteases, determined by their genetic results, indicated the existence of genes responsible for encoding this enzyme. The findings indicated that 18 P. mirabilis isolates (90%) produced protease enzymes (Figure 2). A disparity was noted in the isolates' capacity to synthesise protease. The enzyme protease hydrolyses peptide bonds between the amino acids constituting a protein.

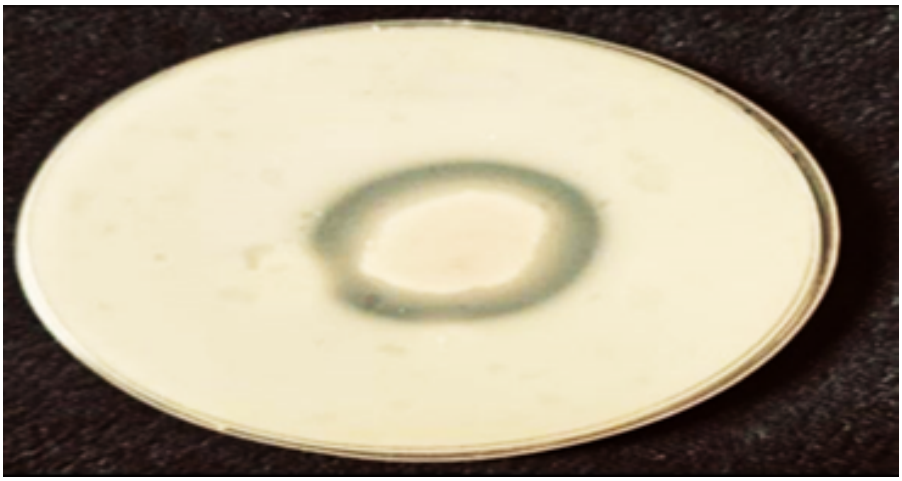

Figure 2: Production of Protease of *P. mirabilis* Incubation on Skim Milk Agar on 37°C for 24hr.

### Molecular Detection of ZapA Gene

This analysis identified the zapA gene in 6 (30%) P. mirabilis isolates, while 14 isolates tested negative, as illustrated in Figure 3. Numerous investigations[18,19] indicating the extensive distribution of zapA among P. mirabilis demonstrated that both phenotypic and molecular detection of urease activity were congruent, and that all P. mirabilis isolates from clinical material exhibited a significant production of urease relative to other bacteria.

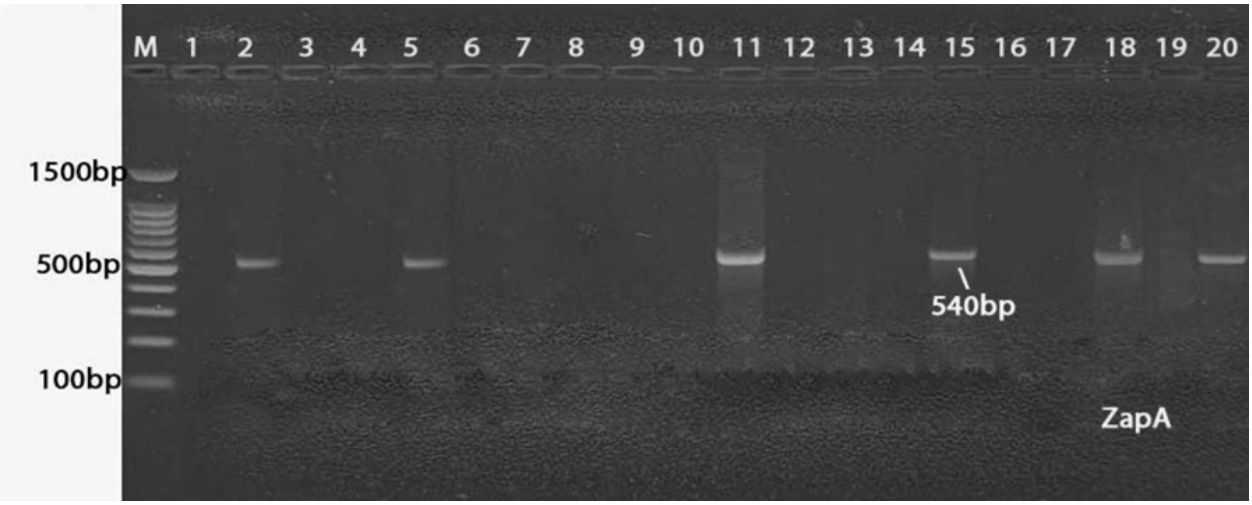


Figure 3: Electrophoretic Analysis of 540 kb ZapA Gene Amplification Products in P. mirabilis Isolates was Conducted Using a 1% Agarose gel at 1.5H and 85V. Column L: DNA size specification - 250-9000 base pairs. Isolates harbouring the ZapA Gene are Located In Columns 2, 5, 11, 15, 18, and 20. Minutes Lack the ZapA Gene in Columns 1, 3, 4, 6, 7, 8, 9, 10, 12, 13, 14, 16, 17, and 19.

Serralysin degrades secretory IgA, sIgA2, and IgA or IgG antibodies, which are essential components for initiating an immune response on mucosal surfaces. In urinary infections, zapA from P. mirabilis contributes to pathogenesis and appears to be activated alongside various other virulence genes and gene products, including the swarmer cell differentiation gene. The IgA proteases of P. mirabilis, associated with zapA, represent a critical virulence component, as mutations in zapA significantly impede both bacterial colonisation and proliferation in the bladder and kidneys.[20] Previous research suggested that P. mirabilis IgA-Protease may cleave the heavy chain of serum IgA at sites analogous to those targeted by the IgA proteinases of pathogenic Haemophilus and Neisseria species, as it acts on the α chain of serum IgA1, sequentially cleaving the tail piece, followed by CH3, and ultimately CH2. Unlike other proteinases, P. mirabilis proteinases can facilitate the cleavage of the secretory component. It distinguishes itself from other proteinases by exhibiting a more rapid cleavage of the α1 chain in serum IgA1 compared to secretory IgA1. [21] Similar to other toxins generated by Proteus, swarmer cells will enhance zapA expression. Infection activates zapA, leading to IgA breakdown in vivo. Nielubowicz and Mobley[22] established that the ascending UTI infection paradigm necessitates zapA for infection. This corroborates previous studies indicating that P. mirabilis isolates from UTI patients exhibited a comparable frequency of the gene.[5] The study is unicentric and hence may not accurately represent the broader epidemiological patterns of P. mirabilis infections in Iraq or other regions, therefore limiting generalisability. The quantity of positive clinical isolates may be inadequate to accurately represent the entire epidemiology based on patient demographics, lacking representation of seasonal fluctuations due to samples being isolated during a certain timeframe. The study also lacks the inclusion of medication combinations for resistance reduction, utilising a narrow antibacterial panel.

## Conclusion

P. mirabilis is a pathogenic microorganism that induces many illnesses, including urinary tract infections and others. Positive proliferation of P. mirabilis was seen in 20 samples categorised by gender, comprising 13 (65%) female and 7 (35%) male samples. Bacterial isolates have the ability to produce protease enzymes, with 90% demonstrating the capacity to create a biofilm. The zapA gene was identified in 6 (30%) P. mirabilis isolates in this study, excluding 14 isolates that had negative findings.

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