Anti-Bacterial Activity of the Prophet Mohammad (SWS) Drink's against Pathogenic Bacteria

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Abstract

Drink of the Prophet Mohammad (Kefiran) which is acidifying fermented milk is accepted as a good example of a probiotic mixture of bacteria and yeast. In this study colorimetric VITEK-2 Compact system was used to identify isolates and to detect susceptibility test to several antimicrobial agents. The study also investigated the antimicrobial activity of 24, 48, 72 and 96 hours fermented DPM (kefiran) against isolated bacteria from UTI patients. The bacteria isolated were eleven gram negative bacteria, included, Acinetobacter baumannii 8 (8%), Enterobacter cloacae 4 (4%), Escherichia coli 16 (15%), Klebsiella oxytoca 6 (6%), Klebsiella pneumonia 11 (10%), Micrococcus luteus 3 (3%), Morganella morganii 4 (4%), Proteus mirabilis 7 (7%), Proteus vulgaris 4 (4%), Pseudomonas aeruginosa 12 (11%) and Serratia marcescens 4 (4%) and four gram positive bacteria, Enterococcus cloacae 2 (2%), Enterococcus faecalis 2 (2%), Staphylococcus aureus 15 (14%), and Staphylococcus haemolyticus 6 (6%). The results of antimicrobial susceptibility test against gram negative and positive bacteria showed the majority of isolates were resistant to most antimicrobials. The MIC values ranged from ($\leq 0.125 - \leq 32 \mu g/ml$). The inhibition zone of 24 hours incubation of DPM (Kefiran) against all isolates was between (7-8 mm). The effects at 48hours of incubation of DPM (kefiran) against all isolates was between (8-9 mm). The effects at 72 hours of incubation the inhibition zone was between (10-11 mm). The maximum activity of the Prophet Mohammad Drink's was recorded at 96 hours of incubation period against all isolates; the inhibition zone was between (10-12 mm).

Keywords: Urinary tract infections, Vitek-2 compact, Drink of the Prophet Mohammad (Kefiran), antimicrobial activity, pathogenic bacteria.

1. Introduction

Drink of the Prophet Mohammad (Kefiran) originated in the Caucasus Mountains several centuries ago and was traditionally produced with caprine milk primarily by inhabitants closely associated with the herding of goats and sheep. Kefiran (DPM) has a rich history as it pertains to its genesis and spread throughout the regions of the Balkan and Caucasus regions of Eastern Europe; in fact, the origins of kefir predate written records. Because of its ancient and apparently mysterious origin, kefir was known in antiquity as the "Drink of the Prophet Mohammad" and the culture used to prepare it as the "Grains of the Prophet Mohammad"; it was believed that the Prophet of Islam, Mohammad, was given the original kefir grains by the Angel Gabriel to be given to his followers, thus introducing kefir to the Orthodox Christians living in the mountainous regions of modern day Georgia [1, 2, 3]. Historically, kefir grains were considered a gift from Allah among the Muslim people of the northern Caucasian mountains. The tribes of the northern Caucasus have produced kefir for hundreds of years. They jealously guarded both their kefir grains and the method of fermenting the beverage. These tribes believed that the grains were given to them by the Prophet Muhammad, who blessed them with exceptional healthpromoting properties. As a result, the tribes were forbidden to share the grains or the method of preparing kefir with outsiders [4]. The word kefir is derived from the Turkish word keyif, which means "feeling good" after its ingestion [5, 6]. Grains of the Prophet Mohammad (Milk Kefir Grains) play a natural starter culture role during the production of kefir and are recovered after the fermentation process by milk straining [7]. These grains are composed of microorganisms immobilized on a polysaccharide and protein matrix, where several species of bacteria and yeast coexist in symbiotic association. In this ecosystem there is a relatively stable microorganism population, which interacts with and influences other members of the community. This population provides the synthesis of bioactive metabolites, which are essential for grain growth and microorganism inhibition, such as food pathogens and contaminants [8]. Kefir grains vary in size, from 0.3 to 3.0 cm in diameter are characterized by an irregular, multilobular surface, united by a single central section, and their color varies from white to yellowish white. The grains are elastic and have a viscous and firm texture [9, 10, and 11]. Kefir possesses antimicrobial activity in vitro against a wide variety of Gram- positive and Gram-negative bacteria, as well as some fungi [12, 13]. Some coliforms are actively inhited by kefir microorganisms, and pathogenic bacteria such as Shigella and Salmonella do not grow when they are introduced to kefir [2, 14]. Lactobacillus acidophilus isolated from kefir, shows inhibitory activity against several Gram-positive and Gram-negative microorganisms [15, 16, 17, 18]. Of all the kefir starter microbial components, the microphillic homofermentative lactococci and acetic acid bacteria are the most active against coliforms. Van Wyk (2001) showed that kefir possesses an inhibitory activity against Staphylococcus aureus, Bacillus cereus, Escherichia coli, Clostridium tyrobutyricum

and Listeria monocytogenes. Studies have also indicated that yeasts such as Torulaspora, when separated from kefir, possess pronounced antimicrobial activity against coliforms [4, 9]. The exact cause of the inhibition is not known, but may be due to the antagonistic action of various species of lactic acid bacteria (LAB) [9, 10]. Lactic acid bacteria are also capable of preventing the adherence, establishment, replication, and pathogenic action of certain enteropathogens [11]. The precise mechanism of this antagonistic activity is not clear, but may include the activity of lactic acid or volatile acids, hydroge peroxide [12, 13] Carbon dioxide, acetaldehyde, & diacetyl, or bacitracin & bacitracin-like products [2]. Urinary Tract Infections (UTIs) are one of the most prevalent extraintestinal bacterial infections. Nowadays, it represents one of the most common diseases encountered in medical practice affecting people of all ages from the neonate to the geriatric age group [20]. Worldwide, about 150 million people are diagnosed with UTI each year [21]. Most infections are caused by retrograde ascent of bacteria from the faecal flora via the urethra to the bladder and kidney especially in the females who have a shorter and wider urethra and is more readily transfer by microorganisms [22]. The structure of the females urethra and vagina makes it susceptible to trauma during sexual intercourse as well as bacteria been massaged up the urethra and into the bladder during pregnancy and or child birth [23, 24]. Majority of UTIs are not life threatening and do not cause any irreversible damage. However, when the kidneys are involved, there is a risk of irreparable tissue damage with an increased risk of bacteremia [25]. Many different microorganisms can cause UTIs though the most common pathogens causing the simple ones in the community are Escherichia coli and other Enterobacteriacae, which accounts approximately 75% of the isolates. In complicated urinary tract infections and hospitalized patients, organisms such as Enterococcus faecalis and highly resistant Gram-negative rods including *Pseudomonas spp.* are comparatively more common. The relative frequency of the pathogens varies depending upon age, sex, catheterization, and hospitalization [26]. In Iraq the kefir grains are not available commercially, and are culturally donated from person to person. The present study was carried out to investigate the antimicrobial activity of 24, 48, 72 and 96 hours fermented of kefiran (DPM) against bacteria isolated from Urinary tract infections patients.

2. Materials and Methods

2.1. Isolation & Identification of Bacteria

This study was carried out in Al-Numan Hospital in Baghdad province, during Jun to 30th September 2013. One hundred thirty urine samples were collected from the outpatient with signs and symptoms of UTI. Midstream urine samples were collected in sterile containers by using clean and sterile catch method recommended by (Tula, and Iyoha, 2014). Then culture on nutrient agar, blood agar and MacConkey agar plates, using sterile standard loop (1ml) then incubated at 37°C for 24-48 hours. The identification of isolates was based on microscopic morphology, staining characteristics, culture and biochemical properties using ID card (GN card and GP card), Vitek 2 Compact BioMeriux Company [27].

2.2. Antimicrobial susceptibility test

The antimicrobial susceptibility test was performed using several types of AST card, Vitek 2 Compact BioMeriux Company [28].

2.3. Preparation of Drink of the Prophet Mohammad (Kefiran)

Starter culture of the prophet Mohammed grains (milk kefir grains) was imported from Kingdom of Jordan. DPM was prepared by adding1 liter of milk to 100grams of kefir grains and incubated at room temperature for 24, 48, 72 and 96 hrs. Subsequently, and filtered through a sterile plastic sieve. The Drink of the Prophet Mohammad was then stored in glass container at 8°C in refrigerator until used [6].

2.4 Antimicrobial Activity of Drink of the Prophet Mohammed (Kefiran)

Antimicrobial activity was demonstrated by agar diffusion assay. Mueller Hilton agar medium (20 mL) was poured into each Petri dish (90 mm diameter). Suspensions (100 μ L) of target strain cultured for 24 hrs. were spread on the plates uniformly, and a wells of 6 mm diameter were made with a sterile cork porer. (100 μ L) of the Prophet Mohammed Drink (Kefiran) samples were transferred into the wells of agar plates inoculated with target strains. The plates were incubated at 37 °C. The diameter of inhibition zone was measured after 12-15 hrs. DPM (Kefiran) sample was taking after (0, 24, 48, 72 and 96 hrs.) of incubation [13].

3. Results

The bacteria isolated from UTI patient samples are shown in (Table 1). Eleven gram negative bacteria, included, *Acinetobacter baumannii* 8 (8%), *Enterobacter cloacae* 4 (4%), *Escherichia coli* 16 (15%), *Klebsiella oxytoca* 6 (6%), *Klebsiella pneumonia* 11 (10%), *Micrococcus luteus* 3 (3%), *Morganella morganii* 4 (4%), *Proteus mirabilis* 7 (7%), *Proteus vulgaris* 4 (4%), *Pseudomonas aeruginosa* 12 (11%) and *Serratia marcescens* 4 (4%) and four gram positive bacteria, *Enterococcus cloacae* 2 (2%), *Enterococcus faecalis* 2 (2%), *Staphylococcus aureus* 15 (14%), and *Staphylococcus haemolyticus* 6 (6%).

Tuble 1. Ducteria isolated if on c	i mai y ti act miccu	ons patients.
Isolate	No.	Percentage
Acinetobacter baumannii	8	8
Enterobacter cloacae	4	4
Escherichia coli	16	15
Klebsiella oxytoca	6	6
Klebsiella pneumonia	11	10
Micrococcus luteus	3	3
Morganella morganii	4	4
Proteus mirabilis	7	7
Proteus vulgaris	4	4
Pseudomonas aeruginosa	12	11
Serratia marcescens	4	4
Enterococcus cloacae	2	2
Enterococcus faecalis	2	2
Staphylococcus aureus	15	14
Staphylococcus haemolyticus	6	6
Total	104	100

Table 1: Bacteria isolated from Urinary tract infections patients.

The results of antimicrobial susceptibility test against gram negative bacteria shows the all isolates were resistant to Ampicillin, Amoxicillin/Clavulanic Acid, Cefazolin, Ceftriaxone, Aztreonam, Gentamicin, Tetracycline and Ttimethprim / Sulfamethoxazole, and sensitive to Piperacillin/Tazobactam, Cefepime, Ertapenem, Imipenem, Meropenem, Amikacin, Ciprofloxacin and Levofloxacin (Table 2).

Table 2: Susceptibility tests of antimicrobials on	gram negative bacteria isolated from UTI patients.
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Bacterial isolates	Percentage of resistance										
	AM	AUC	PPL	CZ	CI	CEF	AZ	EPM			
Acinetobacter baumannii	88	80	39	81	84	44	77	30			
Enterobacter cloacae	90	81	37	83	82	40	75	31			
Escherichia coli	91	85	45	82	81	42	74	33			
Klebsiella oxytoca	87	83	40	88	85	43	76	36			
Klebsiella pneumonia	90	81	41	80	90	44	77	31			
Micrococcus luteus	85	77	38	82	86	46	71	35			
Morganella morganii	87	79	39	83	94	47	73	37			
Proteus mirabilis	84	81	40	84	81	41	77	31			
Proteus vulgaris	86	76	40	81	81	42	74	33			
Pseudomonas aeruginosa	85	77	38	82	86	46	71	35			
Serratia marcescens	87	79	39	83	94	47	73	37			

AM = Ampicillin, AUC = Amoxicillin/Clavulanic Acid, PPL= Piperacillin /Tazobactam, CZ = Cefazolin, CI = Ceftriaxone, CEF = Cefepime, and AZ = Aztreonam, EPM = Ertapenem.

Bacterial isolates	Percentage of resistance									
	IPM	MPM	AK	GM	CIP	LEV	Т	TRI		
Acinetobacter baumannii	37	33	40	70	33	39	80	88		
Enterobacter cloacae	33	34	39	72	30	37	88	85		
Escherichia coli	39	32	41	73	34	33	82	80		
Klebsiella oxytoca	33	35	40	77	36	31	85	80		
Klebsiella pneumonia	35	34	39	74	33	38	80	84		
Micrococcus luteus	38	33	40	72	38	36	83	82		
Morganella morganii	37	37	38	75	31	36	82	88		
Proteus mirabilis	39	30	33	70	38	38	80	87		
Proteus vulgaris	33	37	36	75	35	32	84	85		
Pseudomonas aeruginosa	37	37	38	75	31	36	82	88		
Serratia marcescens	39	30	33	70	38	38	80	87		

IPM= Imipenem, MPM= Meropenem, AK= Amikacin, GM= Gentamicin, CIP= Ciprofloxacin, LEV=

Levofloxacin, T= Tetracycline, and TRI= Ttimethprim / Sulfamethoxazole.

The antimicrobial susceptibility test against gram positive bacteria shows all isolates were resistance to

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Cefoxitin Screen, Benzylpenicillin, Ampicillin, Oxacillin, Gentamicin, Erythromycin, Quinupristin / Dalfopristin, Vancomycin, Tetracycline, Rifampicin, Trimethoprim / Sulfamethoxazole, and sensitive to Gentamicin High Level, Strepmycin, High Level Ciprofloxacin, Levofloxacin, Moxifloxacin, Inducible Clindamycin Resistance, Clindamycin, and Tigecycline (Table 3).

Table 3: Susceptibility tests of antin	nicrobials on gram positive	e bacteria isolated from UTI ا	oatients.
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Bacterial isolates				I	Percentage	of resista	nce			
	CEF	CEF BEP AM OX GM HL S HL GM CIP L								MOF
Enterococcus cloacae	67	63	68	70	30	33	66	30	31	32
Enterococcus faecalis	77	73	78	80	38	37	76	33	35	33
Staphylococcus aureus	74	77	76	84	39	33	74	30	32	32
Staph. Haemolyticus	76	75	79	83	37	35	77	31	33	34

CEF = Cefoxitin Screen, BEP = Benzylpenicillin, AM = Ampicillin, OX= Oxacillin, GMHL= Gentamicin High Level, SHL= Strepmycin High Level, GM = Gentamicin, CIP = Ciprofloxacin, LEV = Levofloxacin, & MOF Moxifloxacin.

Bacterial isolates				Percenta	ge of re	esistar	ice			
	I CLM R	I CLM R E CLM QUP V T TIG F RIP							TRI	
Enterococcus cloacae	33	80	40	78	57	75	41	8 7	54	67
Enterococcus faecalis	37	90	44	88	55	81	43	90	55	77
Staphylococcus aureus	38	87	43	85	56	80	45	91	70	74
Staph. Haemolyticus	33	88	45	87	60	79	47	92	71	70

ICLMR = Inducible Clindamycin Resistance, E = Erythromycin, CLM = Clindamycin, QUP = Quinupristin / Dalfopristin, V = Vancomycin, T = Tetracycline, TIG = Tigecycline, F = Nitrofurantoin, RIP = Rifampicin, & TRI = Trimethoprim / Sulfamethoxazole.

The result of Minimum Inhibitory Concentration of antimicrobials against gram negative bacteria Isolates shows that the MIC of Ampicillin, Amoxicillin/Clavulanic Acid, Cefazolin, Ceftriaxone, Aztreonam, Gentamicin and Tetracycline were ($\leq 16 - \leq 32 \ \mu g/mL$), the MIC of Ttimethprim/Sulfamethoxazole was ($\leq 160 - \leq 320 \ \mu g/mL$), the MIC of Piperacillin/Tazobactam was ($\leq 4 - \leq 8 \ \mu g/mL$), the MIC of Cefepime was ($\leq 1 - \leq 4 \ \mu g/mL$), the MIC of Ertapenem and Meropenem were ($\leq 0.125 - \leq 1 \ \mu g/mL$), the MIC of Imipenem was ($\leq 1 - \leq 2 \ \mu g/mL$), the MIC of Amikacin was ($\leq 2 - \leq 8 \ \mu g/mL$), the MIC of Cefepime was ($\leq 0.25 - \leq 2 \ \mu g/mL$), the MIC of Levofloxacin was ($\leq 0.5 - \leq 1 \ \mu g/mL$) Table (4).

Table 4: Minimum Inhibitory Concentration of antimicrobials against gram negative bacteria Isolates										
from UTI patients.										
Bacterial isolates MIC (µg/l)										
AM AUC PPL CZ CL CEE AZ EPM										

Dacterial isolates				IVIIV	c (μg/I)			
	AM	AUC	PPL	CZ	CI	CEF	AZ	EPM
Acinetobacter baumannii	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	$\leq 4 - \leq 8$	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Enterobacter cloacae	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Escherichia coli	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Klebsiella oxytoca	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Klebsiella pneumonia	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Micrococcus luteus	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Morganella morganii	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Proteus mirabilis	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Proteus vulgaris	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125- ≤ 1
Pseudomonas aeruginosa	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 8	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1
Serratia marcescens	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	$\leq 4 - \leq 8$	≤32-≤64	≤32-≤64	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.125-≤1

AM= Ampicillin, AUC= Amoxicillin/Clavulanic Acid, PPL= Piperacillin/Tazobactam, CZ= Cefazolin, CI= Ceftriaxone, CEF= Cefepime, AZ= Aztreonam, & EPM= Ertapenem.

Bacterial isolates		MIC (µg/l)									
	IPM	MPM	AK	GM	CIP	LEV	Т	TRI			
Acinetobacter baumannii	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Enterobacter cloacae	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Escherichia coli	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Klebsiella oxytoca	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Klebsiella pneumonia	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Micrococcus luteus	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Morganella morganii	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Proteus mirabilis	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Proteus vulgaris	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Pseudomonas aeruginosa	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			
Serratia marcescens	≤1-≤2	≤0.125 - ≤ 1	$\leq 2 - \leq 8$	$\leq 16 - \leq 32$	$\leq 0.25 - \leq 2$	$\leq 0.5 - \leq 1$	$\leq 16 - \leq 32$	$\leq 160 - \leq 320$			

IPM= Imipenem, MPM= Meropenem, AK= Amikacin, GM= Gentamicin, CIP= Ciprofloxacin, LEV= Levofloxacin, T= Tetracycline, TRI= Ttimethprim/Sulfamethoxazole.

The result of Minimum Inhibitory Concentration of antimicrobials against gram positive bacteria Isolates shows that the MIC of Cefoxitin Screen was ($\leq 8 - \leq 16 \ \mu g/mL$), the MIC of Benzylpenicillin, Ampicillin, Oxacillin, Gentamicin, Quinupristin/Dalfopristin, Vancomycin and Rifampicin were ($\leq 16 - \leq 32 \ \mu g/mL$), the MIC of Gentamicin High Level, Strepmycin High Level and Levofloxacin were ($\leq 1 - \leq 4 \ \mu g/mL$), the MIC of Ciprofloxacin was ($\leq 0.5 - \leq 1 \ \mu g/mL$), the MIC of Moxifloxacin was ($\leq 0.125 - \leq 1 \ \mu g/mL$), the MIC of Inducible Clindamycin Resistance was ($\leq 0.5 - \leq 2 \ \mu g/mL$), the MIC of Erythromycin and Tetracycline were ($\leq 8 - \leq 16 \ \mu g/mL$), the MIC of Clindamycin and Tigecycline were ($\leq 0.25 - \leq 1 \ \mu g/mL$), the MIC of Nitrofurantoin was ($\leq 64 - \leq 256 \ \mu g/mL$), the MIC of Trimethoprim/Sulfamethoxazole was ($\leq 160 - \leq 320 \ \mu g/mL$) (Table 5).

Table 5: Minimum Inhibitory Concentration of antimicrobials against gram positive bacteria Isolates from UTI patients.

I.	MIC (µg/l)											
No.	CEF	BEP	AM	OX	GM HL	S HL	GM	CIP	LEV	MOF		
1	$\leq 8 - \leq 16$	≤1 - ≤ 8	$\leq 16 - \leq 32$	<u>≤</u> 4 - ≤ 16	$\leq 1 - \leq 4$	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.5- ≤1	≤1- ≤4	≤.125- ≤1		
2	$\leq 8 - \leq 16$	≤1 - ≤ 8	$\leq 16 - \leq 32$	$\leq 4 - \leq 16$	$\leq 1 - \leq 4$	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.5- ≤1	≤1- ≤4	≤.125- ≤1		
3	<u>≤</u> 4 - ≤ 8	≤1 - ≤ 8	≤8 - ≤ 16	≤8 - ≤ 16	$\leq 1 - \leq 4$	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.5- ≤1	≤1- ≤4	≤.125- ≤1		
4	$\leq 8 - \leq 16$	≤1-≤4	$\leq 8 - \leq 16$	$\leq 8 - \leq 16$	≤1 - ≤ 4	≤1 - ≤ 4	$\leq 16 - \leq 32$	≤0.5- ≤1	≤1- ≤4	≤.125-≤1		

1= Enterococcus faecalis, 2= Staphylococcus aureus, 3= Staphylococcus haemolyticus, 4= Staphylococcus hominis. I. No. = Isolate Number.

CEF= Cefoxitin Screen, BEP= Benzylpenicillin, AM= Ampicillin, OX= Oxacillin, GMHl= Gentamicin High Level, SHL= Strepmycin High Level, GM= Gentamicin, CIP= Ciprofloxacin, LEV=Levofloxacin, MOF= Moxifloxacin.

I. No.	МІС (µg/l)											
	I CLM	Е	CLM	QUP	V	Т	TIG	F	RIP	TRI		
	R											
1	$\leq 0.5 - \leq 2$	$\leq 8 - \leq 16$	$\leq 0.25 - \leq 1$	$\leq 16 - \leq 32$	$\leq 0.5 - \leq 1$	$\leq 8 - \leq 16$	$\leq 0.25 - \leq 1$	$\leq 64 - \leq 256$	≤0.5- ≤ 1	$\leq 160 - \leq 320$		
2	$\leq 0.5 - \leq 2$	≤8 - ≤ 16	≤0.25 - ≤ 1	$\leq 16 - \leq 32$	$\leq 0.5 - \leq 1$	$\leq 8 - \leq 16$	≤0.25 - ≤ 1	$\leq 64 - \leq 256$	≤0.5-≤1	$\leq 160 - \leq 320$		
3	$\leq 0.5 - \leq 2$	$\leq 8 - \leq 16$	≤0.25 - ≤ 1	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	$\leq 8 - \leq 16$	≤0.25 - ≤ 1	≤64 - ≤ 256	≤16-≤32	$\leq 160 - \leq 320$		
4	$\leq 0.5 - \leq 2$	$\leq 8 - \leq 16$	$\leq 0.25 - \leq 1$	$\leq 16 - \leq 32$	$\leq 16 - \leq 32$	$\leq 8 - \leq 16$	$\leq 0.25 - \leq 1$	≤64 - ≤ 256	≤16-≤32	$\leq 160 - \leq 320$		

¹⁼ Enterococcus faecalis, 2= Staphylococcus aureus, 3= Staphylococcus haemolyticus, 4= Staphylococcus hominis. I. No. = Isolate Number. ICLMR=Inducible Clindamycin Resistance, E= Erythromycin, CLM= Clindamycin, QUP = Quinupristin / Dalfopristin, V = Vancomycin, T = Tetracycline, TIG = Tigecycline, F = Nitrofurantoin, RIP = Rifampicin, & TRI = Trimethoprim / Sulfamethoxazole.

The data in (Table 6) shows that Drink of the Prophet Mohammad (Kefiran) has effective antibacterial activities on the UTIs isolates as indicated by the diameter of their zone of inhibition. The effect of Drink of the Prophet Mohammad (Kefiran) on all isolates was at 24 hours of incubation, the diameter of inhibition zone was 7mm for Acinetobacter baumannii, Enterobacter cloacae, Escherichia coli, Klebsiella oxytoca, Klebsiella pneumonia, Morganella morganii, Proteus vulgaris, Serratia marcescens, and Enterococcus faecalis. 8mm for Micrococcus luteus, Proteus mirabilis, Pseudomonas aeruginosa, Staphylococcus aureus and Staphylococcus haemolyticus. The effect at 48 hours of incubation was 8mm for Acinetobacter baumannii, Enterobacter cloacae, Escherichia coli, Klebsiella oxytoca, Klebsiella pneumonia, Morganella morganii, Proteus vulgaris, Serratia marcescens, Enterococcus cloacae and Enterococcus faecalis. 9mm for Micrococcus luteus, Proteus mirabilis, Pseudomonas aeruginosa, Staphylococcus aureus and Staphylococcus haemolyticus. The diameter of inhibition zone at 72 hours of incubation was 10mm for Acinetobacter baumannii, Klebsiella oxytoca, Micrococcus luteus, Morganella morganii, Proteus mirabilis, Proteus vulgaris, Pseudomonas aeruginosa and Enterococcus cloacae. 11mm for Enterobacter cloacae, Escherichia coli, Klebsiella pneumonia, Enterococcus faecalis, Staphylococcus aureus and Staphylococcus haemolyticus. The maximum activity of the Prophet Mohammad Drink's was recorded at 96 hours of incubation of grains of the Prophet Mohammad (kefir grains) against all isolates; the inhibition zone was between (10-12 mm).

 Table 6: Antimicrobial activities of drink of Prophet Mohammed (kefiran) on bacteria isolated from UTI patients.

	Incubation periods of drink of prophet mohammed				
Bacterial isolates	0 hrs.	24 hrs.	48 hrs.	72 hrs.	96 hrs.
	I.Z(mm)	I.Z.(mm)	I.Z.(mm)	I.Z.(mm)	I.Z.(mm)
Acinetobacter baumannii	0.0	7.0	8.0	10.0	10.0
Enterobacter cloacae	0.0	7.0	8.0	11.0	12.0
Escherichia coli	0.0	7.0	8.0	11.0	12.0
Klebsiella oxytoca	0.0	7.0	8.0	10.0	11.0
Klebsiella pneumonia	0.0	7.0	8.0	11.0	11.0
Micrococcus luteus	0.0	8.0	9.0	10.0	11.0
Morganella morganii	0.0	7.0	8.0	10.0	10.0
Proteus mirabilis	0.0	8.0	9.0	10.0	12.0
Proteus vulgaris	0.0	7.0	8.0	10.0	10.0
Pseudomonas aeruginosa	0.0	8.0	9.0	10.0	12.0
Serratia marcescens	0.0	7.0	8.0	11.0	12.0
Enterococcus cloacae	0.0	8.0	9.0	11.0	12.0
Enterococcus faecalis	0.0	8.0	9.0	11.0	12.0
Staphylococcus aureus	0.0	8.0	9.0	11.0	12.0
Staphylococcus haemolyticus	0.0	8.0	9.0	11.0	12.0

I.Z. = Inhibition Zone.

4. Dissection

Several species of bacteria were isolated from patients with urethral tract infections which included *Acinetobacter baumannii*, *Enterobacter cloacae*, *Enterococcus cloacae*, *Enterococcus faecalis*, *Escherichia coli*, *Klebsiella oxytoca*, *Klebsiella pneumonia*, *Micrococcus luteus*, *Morganella morganii*, *Proteus mirabilis*, *Proteus vulgaris*, *Pseudomonas aeruginosa*, *Serratia marcescens*, *Staphylococcus aureus* and *Staphylococcus haemolyticus*. Similar organisms have been reported [24, 29]. Overall, the evaluation results of the newly redesigned colorimetric VITEK-2 ID was so impressed by the performance because more than 98% of the isolates were correctly identified to the species level without any further additional tests. Also, the present results indicated that the current VITEK-2 has overcome its inherent weakness in IDs of streptococci and glucose-nonfermentative GNR. Until present, API test strips has been long considered as the gold standard Q in ID test [28, 30]. But the accuracy of the VITEK-2 was finally estimated to be 98.3%, compared with 97.5% by the respective API test strips. Our results were highly consistent with a series of evaluation results recently published for GPC [31], GNR [32].

The antimicrobial activities of commercially prepared antibiotics on the bacterial isolates showed, that all isolates were sensitive to quinolones (Ciprofloxacin, Levofloxacin and Ofloxacin) this agree with many references which showed that most bacteria isolated from UTIs patient were sensitive to quinolones compounds [33, 34, 35]. The resistance to other types of antimicrobials differs with different isolates; these are in agreement with [36]. The interpretation of results due to littleness using of Quinolones in Baghdad hospitals compared with other antimicrobials such as Ampicillin, Chloramphenicol, Erythromycin, Gentamicin and Oxacillin. The

maximum antimicrobial effect of Drink of the prophet Mohammed (kefiran) noted at 72 and 96 hours of incubation table (6), in these periods the largest inhibition zones were recorded, these finding were in agreement with several references [36, 37,10]. The antimicrobial activity of (DPM) under different incubation periods studied against a number of pathogenic microorganisms which causes urethral tract infections, DPM had its strongest antimicrobial effects, and this implies the existence of an antimicrobial component other than acetic acid and large proteins. There are numerous reports indicating that a decrease in the pH of Kefir beverage is caused by the accumulation of organic acids, produced as major end-products of carbohydrate metabolism by lactic acid bacteria (LAB). Accumulation of lactic acid and a subsequent decrease in pH results in a broadspectrum inhibitory activity against Gram-negative bacteria [38]. The undissociated forms of lactic and acetic acid penetrate the microbial cell membrane. This results in acidification of the cytoplasm and the formation of inhibition, especially against enzymes, by salt excesses [3]. At a higher intracellular pH these acids dissociate to produce hydrogen ions, which interfere with important metabolic functions such as oxidative phosphorylation and substrate translocation [4]. The antimicrobial effect of lactic or acetic acid depends on the pK_a value of the acid, as well as the pH of the external environment [5, 6]. These acids are known to inhibit E. coli [7] and B. cereus [8]. At a pH 5.0 acetic acid inhibits the growth of Salmonella typhimurium [9]. A synergism between lactic and acetic acid has been reported for the inhibition of *E. coli* and *Salmonella spp.* [10, 11]. Lactic acid (pK_a 3.86) is a stronger acid than acetic acid $(pK_a 4.75)$ [12] and in well-buffered foods with a pH of 4-6, acetate has a stronger antimicrobial effect as a greater portion of the acid is undissociated [13]. The second factor found in DPM which have antimicrobial activity is hydrogen peroxide (H_2O_2) . The production of hydrogen peroxide (H_2O_2) by lactic acid bacteria depends on the strain and the availability of oxygen [1]. In the presence of oxygen, H_2O_2 is produced by lactic acid bacteria through electron transport via flavin enzymes. In the presence of H_2O_2 , superoxide anions from destructive hydroxy radicals (OH), leading to increased membrane permeability [3] and to the peroxidation of membrane lipids [4]. Bactericidal oxygen metabolites cause the destruction of nucleic acids and cell proteins, and have a strong oxidizing effect on the bacterial cell [5, 6, 7]. Hydrogen peroxide accumulates in the growth media and inhibits Pseudomonas spp. [8] and S. aureus [9]. Inhibitory compounds can also be formed from H_2O_2 , such as in raw milk where it reacts with endogenous thiocyanate, catalyzed by lactoperoxidase [10, 11]. The third factor found in DPM is Carbon dioxide; Carbon dioxide contributes to the antimicrobial activity of lactic acid bacteria by replacing the existing molecular oxygen, creating an anaerobic environment [1]. The forth factor found in DPM is Acetaldehyde, at concentration of 10 to 100 ppm has antimicrobial activity against Staphylococcus aureus, E. coli and Salmonella typhimurium [1, 3, 4]. The fifth factor found in DPM is Diacetyl (2, 3-butanedione) is an end-product of pyruvate metabolism [5] of citratefermenting lactic acid bacteria [6, 7] that elicits antimicrobial activity against various spoilage microorganisms and food-borne pathogens [8, 9]. Diacetyl is effective against yeasts, moulds and Gram-negative bacteria. Archer et al., (1996) reported the inhibition of S. typhimurium by sublethal concentrations of diacetyl. The compound reacts with arginine-binding proteins of Gram-negative bacteria and interferes with arginine utilization [9, 10, 11]. High concentrations of diacetyl are required for an antimicrobial effect. Dose-dependent inhibition experiments established that 0.2 mg.ml^{-1} is required for the antimicrobial activity against Gram-negative bacteria and yeasts, while 0.3 mg ml⁻¹ is required for he inhibition of non-lactic Gram-positive bacteria [2, 9]. The sixth factor found in DPM is Bacteriocins are bacterial proteins or peptide with bactericidal or bacteriostatic activity against genetically closely related species [40]. Bacteriocins generally vary with regards to their mode of action, molecular weight, genetic origin, biochemical properties and spectrum of activity. The can be produced spontaneously or induced and the genetic determinants of most bacteriocins are located on plasmids, with only a few exceptions being chromosomal encoded [38]. The release of bacteriocins from producer cells requires the expression and activity of bacteriocin-release proteins, and the presence of detergent resistant phospholipase A in the bacterial outer membrane [38]. The bacteriocins that are released are species specific. The majority of bacteriocins produced by lactic acid bacteria have been characterised according to their activity as a proteinaceous inhibitor, on the estimation of their molecular mass, and on the determination of their spectrum of inhibition [41]. Bacteriocins inhibit a broad spectrum of Gram-positive and Gram-negative bacteria [4]. Antimicrobial activity increased with fermentation time until 96 hours, these finding in agreement with [10]. As seen in almost all cases tested. This also implies that the active antimicrobial components are very likely metabolites produced by the bacteria and/or yeasts responsible for the fermentation of Drink of the Prophet Mohammad.

5. Conclusion

The antibacterial activity of Drink of the Prophet Mohammad (Kefir) increase with increase incubation periods (96 hours).

References

1. Rosell, J. M., (1932). Yoghourt and Kefir in Their Relation to Health and Therapeutics. The Canadian

Medical Association Journal, 3, 341-345.

2. Koroleva, N. S., (1988). Technology of kefir and kumys. Chapter \forall II. Bulletin of the International Dairy Federation, 277, 96-100.

3. Margulis, L., (1996). From kefir to death. In: Brockman, J., Matson, K. (Eds.), *How Things Are*. William Morrow and Co., New York, pp. 69-78.

4. Powell, E., (2006). Bacteriocins and bacteriocin producers present in kefir and kefir grains. (Unpublished Master's thesis), Stellenbosch University, South Africa.

5. Lopitz-Otsoa, F., Rementeria, A., Elguezaba, N., and Garaizar, J., (2006). Kefir: a symbiotic yeasts-bacteria community with alleged healthy capabilities. *Rev. Iberoam Micol*, 23, 67-74.

6. Tamime, A.Y., (2006). Production of Kefir, Koumiss and Other Related

Products. In: Tamime, AY (ed.), Fermented Milk Blackwell Science Ltd, Oxford, UK, p.174-216.

7. Rattray, F.P. and M.J. O'Connell, (2011) Fermented Milks Kefir. In Fukay, J. W. (ed.), Encyclopedia of Dairy Sciences (2th ed. Academic Press, San Diego, USA, p.518-524.

8. Farnworth, E.R., and Mainville, I., (2008). Kefir –A fermented Milk Product In: Farnworth, E. R. (2th ed.), *Handbook of Fermented Functional Foods* (2 ed). CRC Press Taylor & Francis Group, Boca Raton, London, New York, p. 89-127. 9. Garrote, G.L., A.G. Abraham, and G. L. De Antoni, (2010). Microbial Interactions in Kefir: A Natural Probiotic Drink. In F. Mozzi, R. R. Raya & G. M. Vignolo (Eds.), *Biotechnology of Lactic Acid Bacteria – Novel Applications* pp. 327-340. Iowa: Blackwell Publishing.

10. Magalhães, K.T., Pereira, G.V.M., Campos, C.R., Dragone, G., and Schwan, R.F., (2011). Brazilian Kefir: Structure, Microbial Communities and Chemical Composition. *Braz J Microbiol*, 42, 693-702. 11. Rea, M.C., Lennartsson, T., Dillon, P., Drina, F.D., Reville, W.J., Heapes, M., and Cogan, T.M., (1996). Irish kefir-like grains: their structure microbial composition and fermentation kinetics. *J. Appl. Microbiol*, 8, 83-94. 12. Saloff-Coste, C. J., (1996). Kefir. Nutritional and health benefits of yoghurt and fermented milks. *Danone World Newsletter*, 11, 1-7.

13. Garrote, G. L., Abraham, A. G., and De Antoni, G. L., (2000). Inhibitory Power of Kefir: The role of organic acids. *J. Food Prot*, 63(3),364-369.

14. Gibson, G. R., Saavedra, J. M., MacFarlane, S., and MacFarlane, G. T., (1997). Probioticas and intestinal infections. In: probiotics 2: Applications and Practical Aspects (edited by R. Fuller). Pp. 19-39. New York: Chapman and Hall.

15. Gilliland, S. E., and Speck, M. L., (1977). Antagonistic action of *Lactobacillus acidophilus* toward intestinal and foodborne pathogens in associative cultures. *Journal of Food Protection*, 40, 820-823.

16. Apella, M. C., Gonzalez, S. N., Nader de Macias, M. E., Romero, N., and Oliver, G. (1992). In vitro studies on the inhibition of the growth of the *Shigella sonnei* by *Lactobacillus casei* and *Lactobacillus acidophilus*. *Journal of Applied Bacteriology*, 73, 480-483.

17. Gupta, P. K., Mital, B. K., and Garg, S. K., (1996). Inhibitory activity of *Lactobacillus acidophilus* against different pathogens in milk. *Journal of Food Science and Technology*, 33, 1437-149.

18. Helander, I. M., von Wright, A., and Mattila-Sandholm, T. M., (1997). Potential of lactic acid bacteria and novel antimicrobials against Gram-negative bacteria. *Trends in Food Science and Technology*, 8, 146-150.

19. Van Wyk, J., (200). The inhibitory activity and sensory properties of kefir, targeting the low –income African consumer market. M.Sc. Thesis. University of Stellenbosch, South Africa.

20. Kunin, C.M., (1994). Urinary tract infections in females. Clin. Infect .Dis, 18, 1-12.

21. Gupta, P. K., Hooton, T.M., and Stamm, W.E., (2001). Increasing antimicrobial resistance and the management of uncomplicated community-acquired urinary tract infections. *Ann Intern Med*, 135, 41-50.

22. Jones, R.N., Inabo, H.I., and Obanibi, H.B.I., (2006). Antimicrobial susceptibility of some urinary tract clinical isolates to commonly used antibiotics. *Afr. J, Biotechnol*, 5(5), 487-489.

23. El-Sweih, N., Jamal, W., and Rotimi, V. O., (2008). Spectrum and antibiotic resistance of uropathogens isolated from hospital and community patients with urinary tract infections in two large hospitals in Kuwait. *Med. Principl. Pract*, 14, 401-407.

24. Kolawale, A.S., Kolawale, O.M., Kandaki-Olukemi, Y.T., Babatunde, S.K., Durowade, K.A., and Kplawale, C.F., (2009). Prevalence of urinary tract infections among patients attending Dalhatu Araf Specialist Hospital, Lafia, Nasarawa State, Nigeria. *Int. J. Med. Med. Sci*, 1(5), 163-167.

25. Hvidberg, H., Struve, C., Krogfelt, K.A., Christensen, N., Rasmussen, S.N., and N. Frimodt-M ler, (2000).Development of a long-term ascending urinary tract infection mouse model for antibiotic treatment studies. *Antimicrob. Agents Chemother*, 44, 156-163.

26. Sefton, A. M., (2000). The impact of resistance on the management of urinary tract infections. *Int. J. Antimicrob. Agents*, 16,489–491.

27. Wallet, F., Loïez, C., Renaux, E., Lemaitre, N., and Courcol, R. J., (2005),

Performances of VITEK 2 colorimetric cards for identification of gram positive and gram-negative bacteria. *J. Clin.Microbiol*, 43, 4402-4406.

28. Nakasone, I., Kinjo, T., Yamane, N., Kisanuki, K., and Shiohira, C. M., (2007). Laboratory-based evaluation of colorimetric VITEK-2 Compact system for species identification and of the advanced expert system for detection of antimicrobial resistances: VITEK-2 Compact system identification and antimicrobial susceptibility testing. *Diagn. Microbiol. Infect. Dis*, 58, 191-198. 29. Tula, M. Y., and Iyoha, O., (2014). Distribution and Antibiotic Susceptibility Pattern of Bacterial Pathogens Causing Urinary Tract Infection in Mubi General Hospital, Yola-Nigeria. *British Journal of Medicine & Medical Research*, 4(19), 3591-3602

30. Shetty, N., Hill, G., and Ridgway, G. L., (1998). The vitek analyser for routine bacterial identification and susceptibility testing: protocols, problems, and pitfalls. *J. Clin. Pathol*, 51, 316-323.

31. Sellenriek, P., Holmes, J., Ferrett, R., Drury, R., and Storch, G. A., (2005). Comparison of MicroScan Walk-Away®, Phoenix[™] and VITEK-TWO®Microbiology systems used in the identification and susceptibility testing of bacteria. Abstr. 105th General Meeting of the American Society for Microbiology.

32. Sönksen, U. W., Christensen, J.J., Nielsen, L., Hesselbjerg, A., Hansen, D.S., and Bruun, B., (2010). Fastidious Gram-Negatives: Identification by the Vitek 2 Neisseria-Haemophilus Card and by Partial 16S rRNA Gene Sequencing Analysis. *The Open Microbiology Journal*, 4, 123-131.

33. Munoz-Davila, M. J., (2014). Role of Old Antibiotics in the Era of Antibiotic Resistance. Highlighted Nitrofurantoin for the Treatment of Lower Urinary Tract Infections. *Antibiotics*, 3, 39-48.

34. Shariff, A.R., Shenoy, S., Yadav, T., and Radhakrishn, M., (2013). The Antibiotic Susceptibility Patterns of Uropathogenic *Escherichia coli*, With Special Reference to the Fluoroquinolones. *J. Clin. Diag. Rese*, 7(6), 1027-1030.

35. Lee, G. C., and Burgess, D. S., (2012). Treatment of Klebsiella Pneumoniae

Carbapenemase (KPC) infections: a review of published case series and case reports. *Annals of Clinical Microbiology and Antimicrobials*, 11, 32.

36. Taherikalani, M., Keshavarz, B., Emaneini, M., Azadi, N. A., (2013). Increased of resistant to antibiotics among bacteria isolated from burn wounds. *Rev Epidemiol Control Infect*, 3(2), 38-39.

37. Santos, A., San Mauro, M., Sanchez, A., Torres, J. M., and Marquins, D., (2003). The antimicrobial properties of different strains of *Lactobacillus spp*. Isolated from kefir. *Systematic and Applied Microbiology*, 26,434-437.

38. Naidu, A. S., Bidlack, W. R., and Clemens, R.A., (1999). Probiotic spectra of lactic acid bacteria (LAB). *Critical Reviews in Food Science and Nutrition*, 38, 26-34.

39. Archer, M. H., Dillon, V. M., Campbell-Platt, G., and Owens, J. D., (1996). Effect of diacetyl on growth rate of *Salmonella typhimurium* determined from detection times in a micro-well plate photometer. *Food Control*, 7, 63-67.

40. Tagg, J. R., Dajani, A. S., and Wannamaker, L. W., (1976). Bacteriocins of Gram-positive bacteria. *Bacteriology Reviews*, 40, 722-756.

41. Klaenhammer, T.R., (1993). Genetics of bacteriocins produced by lactic acid bacteria. *FEMS Microbiology Reviews*, 12, 38-86.

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