KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY, KUMASI

COLLEGE OF SCIENCE FACULTY OF BIOSCIENCES

DEPARTMENT OF FOOD SCIENCE AND TECHNOLOGY

MICROBIOLOGICAL SAFETY AND ANTIMICROBIAL SUSCEPTIBILITY (RESISTANCE) EVALUATION OF WEANIMIX

BY JESSICA ADDOTEY

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(Btech. Food Technology)

A THESIS SUBMITTED TO THE DFEPARTMENT OF FOOD SCIENCE AND TECHNOLOGY, IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF DEGREE OF

MASTER OF SCIENCE IN FOOD QUALITY MANAGEMENT

JULY, 2018

DECLARATION/ CERTIFICATION

I hereby, declare that this submission is my own work towards the Degree of Master of Science in Food Quality Management (MSc. Food Quality Management) and that, to the best of my knowledge, it contains no material previously published by another person nor

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material which has been accepted for the award of any other degree of the University, except where due acknowledgement has been made in the text

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ABSTRACT

Weaning food is a manufactured food designed and marketed for feeding babies and infants less than 12 months of age. It is usually prepared for bottle-feeding or cup-feeding from powder (mixed with water) or liquid (with or without additional water). The nutritional benefits of complementary foods or weaning foods and the sensitivity period in which they are fed on by infants, it is very important to investigate the microbiological safety of the

weanimix as well as determination of antibiotic resistance of pathogens that may be isolated against commonly used antibiotics for treatment of infections.

An aggregate of 90 samples were gathered and were allocated unique codes and instantly taken to the laboratory for isolation of Pathogens. From the study, weanimix were found to be highly contaminated with *Salmonella*, *S. aureus* and *E. coli*. And in terms of Antimicrobial activities; some isolates from the weanimix were susceptible to some antibiotics while other isolates were resistant.

It is recommended that Regulatory agencies(FDA, Ghana Health Services, Local District Assemblies) should take up the challenge in helping local food manufactures to produce safe foods especially weanimix.

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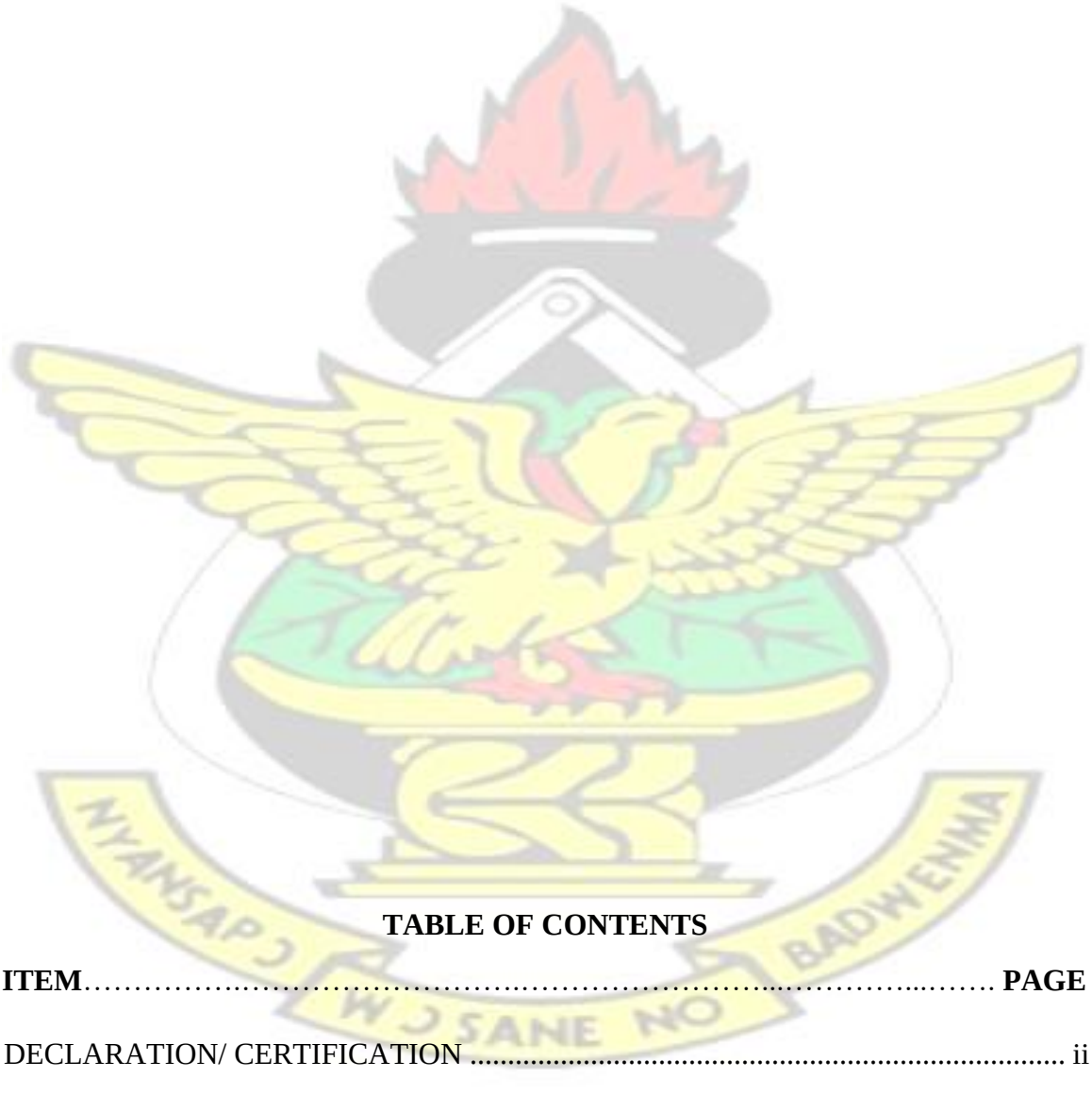

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CHAPTER ONE

1.0. GENERAL INTRODUCTION

1.1. Background

As per Agostoni *et al.* (2008), complementary feeding with infant formula begins when breast milk is no longer sufficient to meet the nutritional requirements of infants.

World Health Organization (WHO,2009) said "target go for corresponding encouraging is for the most part taken to be 6 to 23 months of age". Weaning food is a manufactured food designed and marketed for feeding babies and infants less than 12 months of age. It is usually prepared for bottle-feeding or cup-feeding from powder (mixed with water) or liquid (with or without additional water).

In 2003, Global Strategy for Infant and Young Child Feeding, distributed by the WHO and UNICEF rehashed that "prepared nourishment items for babies and youthful kids should, when sold or generally dispersed, meet relevant guidelines prescribed by the Codex Alimentarius Commission. The use of infant formula in less economically developed countries is linked to poorer health outcomes because of the prevalence of unsanitary preparation conditions, including lack of clean water and lack of sanitizing equipment.

In African nations, selective breastfeeding is typically sufficient up to three to four months of age, however after this period it ends up being dynamically deficient to help the nutritious requests of the developing new-born child. Thus, in a weaning procedure there is always the need to introduce soft, easily swallowed foods to supplement the infant‘s feeding early in life.

According to Pickett-Bernard (2006), new-born child recipe as indicated by U.S. Government Food, Drug and Cosmetic Act (FFDCA, 2006) defines infant formula as "a

food which purports to be or is represented for special dietary use solely as a food for infants by reason of its simulation of human milk or its suitability as a complete or partial substitute for human milk". In Ghana infant formula like weanimix is the product used in supplementing semi solid food in addition to breast milk during weaning. Public Policy on exclusive breast feeding in Ghana is up to six months before infants diet could be supplemented with infant formula which is usually about the time their first teeth appear. Weaning age is a critical period of childcare. Until an infant is introduced to the family food, breast milk needs to be supplemented when infants reach six (6) months. With the introduction of weaning foods which in many countries are prepared under unhygienic conditions, infants who until then have only consumed breast milk may be exposed to infective doses of foodborne pathogens according to Motaijemi *et al.* (1993).

1.2. Statement of Problem

Weanmix is a blend of grains; usually roasted Maize, Soya Beans, Millet, Sorghum, Groundnuts. These grains are treated with agro-chemicals and are likely to cause microbiological resistance in the pathogens that may be present on the grains. Its high protein content makes it an ideal product for weaning infants and also for microbial growth once there is contamination of the product. These products are traditionally prepared by individuals based on the availability of particular grain usually in different combinations and packaged for sale without labelling. Hence improper handling of the foods would eventually result in microbial contamination which may likely result in food borne illnesses of the infants being weaned with these food products.

Therefore, given the nutritional benefits of weanimix and sensitivity period in which the infants are fed, it is very important to investigate the microbiological safety of the weanimix

as well as determination of antibiotic resistance of pathogens that may be isolated against commonly used antibiotics for treatment of child infections. This will inform decisions regarding the regulation of production of the locally manufactured infant formula in order to safeguard infants‘ health safety.

1.3. Research Questions

The investigation was constituted on the accompanying questions:

1. Does the infant formula - weanimix sold in the various hospitals for infants under 1 year conform to food safety regulations?
2. Are these pathogens antimicrobial resistant or have the pathogens developed antibacterial resistance?

1.4. Main Goal

The main objective of the study is to evaluate the microbiological safety of weanimix and its antimicrobial resistance.

1.5. Justification of Project

This study is relevant because the immune system of infants is not fully developed to recover from any infection as a result of microbiological contamination from the weanmix by a resistant strain pathogen. bacteria which could lead to serious health

effects.

The study into the microbiological safety of locally produced infant formula-weanimix and its antimicrobial resistance, will inform decisions regarding the production and regulation of these locally manufactured infant formulas. Moreover, this will help to promote

production of infant formula according to food safety regulations and help to safeguard infants‘ health safety. Also, the study will add up to the store of knowledge in academia regarding the microbiological safety of locally produced infant formulas and its antimicrobial resistance.

CHAPTER TWO

2.0. LITERATURE REVIEW

2.1. Introduction

As per Daelmans *et al.* (2009), infants‘ complementary food or weaning food was designed to be a medical nutritional tool for babies who are unable to breastfeed. The worldwide idea of nourishment and sustenance issue has caused over 33% of all deaths of children under the age of 5 years. New-born child and tyke bolstering works on beginning from birth are significant and can influence prompt and long term nutritious status. WHO and UNICEF in 2008 met a specialized gathering, it was concurred that there was a need to inspect the proof for powerful intercessions to enhance complementary foods (CFs) and encouraging practices. Furthermore, distinguishing activities expected to incorporate these mediations into wellbeing administration conveyance.

Lonnerdal, (2012) shared the view that, " infant‘s complementary food or weaning foods are the predominant source of nutrition for many infants and are fed during a sensitive period of development, having probably short- and long-term consequences for infant health‖.

Lee *et al*. (2013) included that regarding food safety, "new-born children and kids are considered to be a part of the high-risk group of people as their immune systems may have not yet been completely created".

Aggett, *et al.* (2001) additionally opined that, "it is strange that concerns for safety of products used for babies ought to be investigated closer than foods for grown-ups who have built up several mechanisms to face up with supplement insufficiencies and abundance".

Janisiewicz and Korsten, (2002), kept up that, "—most bacteria and fungi that arrive on the developing crop plant either are totally amiable to the harvest's wellbeing or, in many instances, give a characteristic natural boundary to infestation by the subset of microorganisms responsible for crop damage.".

Miedes and Lorences (2004) likewise opined that ―fungi in particular, produce an abundance of extracellular pectinases and hemicellulases that are important factors for fungal spoilage‖ While Tournas and Katsoudas (2005b) shared the view that, "some deterioration organisms are fit for colonizing and making injuries on solid and undamaged plant tissue"

More so, Mandrell, *et al.* (2006) noticed that, "a different group of epiphytic microorganisms present a further aggressive boundary to the deterioration living being likewise regularly colonizes the peripheral natural product surface". It was noticed that defeating these obstructions requires a dazzling arrangement of biochemical apparatuses that enable the decay microorganism to:

(1) distinguish and perceive the plant surface.

(2) utilize at least one procedures to accomplish irreversible connection to the plant surface.

(3) start steps prompting disguise of the tissue.

In addition, Bartz *et al.* (2015) shared the view that ―on plant structures other than the fruit, internalization can be achieved through a number of specialized vessels and surface structures employed by the plant to absorb and release water and to provide CO_2 and O_2 exchange‖.

Saavedra (2007) placed that in resent times, "a few kinds of microscopic organisms have been progressively incorporated into Powdered Infant Formula (PIF) or infant food production for their potential health benefits; in literature these bacteria are referred to as probiotic agents". Joint FAO/WHO (2001) defined Probiotics "as live and indispensable microorganisms ready to profit human wellbeing when consumed in satisfactory sum, as part of a food or a nutritious supplement".

In general, antimicrobial resistance is the capacity of a microorganism to resist the growth inhibitory or killing activity of an antimicrobial beyond the normal susceptibility of the specific bacterial species.

Acar and Röstel (2001) moreover communicated that, "antimicrobials include any substance that has a growth inhibiting of killing effect on microorganisms in a clinical setting or for reducing bacterial loads in materials and surfaces".

Gevers *et al.* (2003a) held the view that, "qualities presenting protection from antimicrobials can be moved in microbial groups by conjugative plasmids, transposons, and integrons, and insertional components, lytic and mild bacteriophages. A few investigations estimated that additionally commensal microscopic organisms may go about as supplies of anti-toxin protection qualities which may add to the spread of protection".

Moreover, probiotic bacteria used as food supplements are not the only microorganisms involved in the dissemination of resistant determinants in the food chain: a recent study, instead, demonstrated the possible role of ready-to-eat salads in the spread of bacteria within kitchen environment and placing salads within the spectrum of food products that may be vehicles for antibiotic resistant bacteria/genes with clinical interest (CamposOrtega and Hartenstein, 2013).

Liu and Latham (2009) in a related report kept up that, "the safety of food products and above all the safeness of commercial strains ought to be assessed before dispatch on the market, not only for potential disease-causing traits, but also for their capability of acquiring and transferring resistance determinants and added that, the accompanying rules are required to guarantee the safety of food products:

i) a base marked grouping of 10^9 CFU of live microorganisms/every day measurements; ii) identification of each probiotic by integrating phenotypic and genotypic characterization, and conforming of microbial species nomenclature to the international Code of Nomenclature; iii) absence of pathogens.

Aureli, *et al.* (2000) additionally uncovered that by and large, "―the content of some products is not always in agreement with the statements on the label‖. The European Food Safety Authority (2008) report suggests that commercial strains should not harbour transferable antibiotic resistance; specifically, assurance of Minimum Inhibitory Concentrations (MICs) of the most critical antimicrobial for each bacterial strain used in food preparations.

Gevers *et al*. (2003b) included that, "―genes conferring resistance to antimicrobials can be transferred in microbial communities by conjugative plasmids, transposons, and integrons, and insertional components, lytic and mild bacteriophages". Some studies speculated that commensal bacteria may act as reservoirs of antibiotic resistance genes which may contribute to the spread of resistance.

2.2 The Relative Risks of Complementary Feeding Versus the Benefits of Breastfeeding

Public health campaigns and medical literature have traditionally described the benefits of breastfeeding, comparing health outcomes among breastfed infants against a reference group of formula-fed infants. Although mathematically synonymous with reporting the ―risk of not breastfeeding,‖ this approach implicitly defines formula feeding as the norm. Cattaneo *et al.* (2011) have noted this subtle distinction impacts public perception of infant

feeding. If ―breast is best,‖ then formula is implicitly ―good‖ or ―normal.‖ This distinction was underscored by national survey data showing that, in 2003, whereas 74.3% of US residents disagreed with the statement: ―Infant formula is as good as breast milk,‖ just 24.4% agreed with the statement: ―Feeding a baby with formula instead of breast milk increases the chance of the baby becoming ill. (Li, *et al.* 2010)

These distinctions appear to influence parents‘ feeding decisions. In 2002, the Ad Council conducted focus groups to develop the National Breastfeeding Awareness Campaign, targeted at reproductive-aged women who would not normally breastfeed.

They found that women who were advised about the ―benefits of breastfeeding‖ viewed lactation as a ―bonus,‖ like a multivitamin, that was helpful but not essential for infant health. Women responded differently when the same data were presented as the ―risk of not breastfeeding,‖ and they were far more likely to say that they would breastfeed their infants.

2.2.1 Infectious Morbidity in infants

Compared with breastfed infants, formula-fed infants face higher risks of infectious morbidity in the first year of life. These differences in health outcomes can be explained, in part, by specific and innate immune factors present in human milk as indicated by Hamosh (2001).

Nathavitharana *et al. (*1995) said that ―plasma cells in the mother‘s bronchial tree and intestine migrate to the mammary epithelium and produce IgA antibodies specific to antigens in the mother infant dyad‘s immediate surroundings, providing specific protection against pathogens in the mother‘s environment‖. In addition, innate immune factors in milk provide protection against infection. Oligosaccharides prevent attachment of common

respiratory pathogens, such as *Haemophilus influenzae* and *Streptococcus pneumoniae*, to respiratory epithelium, and glycoproteins prevent binding of intestinal pathogens such as *Vibrio cholerae*, *Escherichia coli*, and rotavirus (Newburg *et al*. 1998). Glycosaminoglycans in milk prevent binding of HIV gp120 to the CD4 receptor, reducing risk of transmission, and human milk lipids contribute to innate immunity, with activity against *Giardia lamblia, H influenzae*, group B streptococci, *S epidermidis*, respiratory syncytial virus (RSV), and herpes simplex virus type 1 (HSV-1) according to Hamosh (1998)

2.2.2 Otitis Media

According to Ip *et al*. (2009), said ―44% of infants will at least 1 episode of otitis media in the principal year of life". And the risk among formula-fed infants is doubled (95% confidence interval [CI], 1.4–2.8) compared with infants who are exclusively breastfed for more than 3 months. Human milk oligosaccharides and antibodies to common respiratory pathogens in the infant‘s environment are thought to provide protection from infection.

2.2.3 Lower Respiratory Tract Infection

Bachrach *et al.* (2003) found that babies who were not breastfed confronted a 3.6-crease expanded hazard (95% CI, 1.9– 7.1) of hospitalization to bring down respiratory tract contamination in the main year of life, compared with new-born children who were only breastfed for over 4 months.

2.2.4 Gastrointestinal Infections

Multiple studies suggest complementary fed infants face an increased risk of gastroenteritis. Chien and Howie (2001) found that ―infants who were complementary fed or fed a mixture of weaning foods and human milk were 2.8 times (95% CI, 2.4–3.1) more likely to develop gastrointestinal (GI) infection than those who were exclusively breastfed. Data from the Promotion of Breastfeeding Intervention Trial (PROBIT) found that‖ infants were 1.7 times (95% CI, 1.1–2.5) more likely to develop GI illness‖.

2.2.5 Obesity and Metabolic Disease

According to Horta *et al.* (2015), epidemiologic studies ―suggest that children who are complementary fed in infancy are more likely to become obese or develop type 2 diabetes.‖

Complementary fed in infancy is also associated with a 1.6-fold risk (95% CI, 1.2–2.3) of type 2 diabetes, compared with being breastfed according to Ip, *et al.* (2009). Moreover, human milk contains adipokines, which may play a role in regulating energy intake and long-term obesity risk (Aydin *et al.* 2008). Several authors have postulated that longchain polyunsaturated fatty acids in breast milk may affect blood pressure and insulin resistance in later life (Farmer *et al.* 2005).

2.2.6 Neurodevelopment

Anderson *et al.* (1999) have examined associations between infant feeding and cognitive development, with mixed results. Several studies reported ―modestly lower IQ scores in complementary fed children compared with breastfed children, whereas others reported no association between infant feeding and intelligence‖. Dewey and associates randomized mothers in Honduras to introduction of complementary foods at 4 months versus continued

exclusive breastfeeding until 6 months postpartum. Infants in the complementary food group crawled later than those that were exclusively breastfed from 4 to 6 months (*P* = .007) (Stuebe, 2009). Among normal birth weight infants, those who were randomized to complementary foods before 6 months were less likely to be walking at 12 months (39 vs 60%; *P* = .02). Kramer *et al.* (2001) similarly found differences in neurodevelopment with shorter breastfeeding in the PROBIT study. At age 6.5 years, verbal IQ scores were 7.5 points lower (95% CI, −0.8 to −14.3) among children in the usual care group than among children in the breastfeeding support group. Kramer's results suggest that hospital policies that support breastfeeding can impact neurodevelopment at school age. (Kramer *et al.* 2008).

2.2.7 Sudden Infant Death Syndrome (SIDS)

Ip *et al.* (2009) suggest that ―complementary feeding increased odds of SIDS compared with breastfeeding‖. These associations persisted after adjustment for sleeping position, maternal smoking, and socioeconomic status. In reviewing the evidence, the American Academy of Paediatrics Task Force on Sudden Infant Death Syndrome concluded that ―factors associated with breastfeeding, but not breastfeeding per se, were associated with a lower incidence of SIDS‖. (America Academy of Pediatrics, 2005).

2.3 Improving weaning in Infants

Mosha and Svanberg (1983) maintained that, ―germination can improve the nutritional value of weaning foods by reducing the water-binding capacity of cereal flour and that; it allows the porridge to have a free-flowing consistency even with a high proportion of

flour‖.

Brandtzaeg *et al*. (1981) also added that, ―germination also converts insoluble proteins to soluble components and increases the levels of lysine as well as of vitamins B and C‖.

Caplice and Fitzgerald (1999) maintained that, ―living bacteria are daily used for human consumption; bifidobacteria and Lactic Acid Bacteria (LAB) are often used in the production of fermented foods, beverages and dietary supplements‖. Lu and Walker (2001) added that, ―the amount of ingested viable cells seems to be able to influence the probiotic effectiveness‖. So, it is essential that products available on the market are correctly labelled and that the viability and identity of each strain is ensured as stated by Toscano *et al.* (2017).

Gueimonde *et al.* (2013) maintained that, ―the presence of antibiotic resistance in microorganisms introduced in food chain should be avoided even in non-pathogenic bacteria used in food supplementation‖. Toscano, *et al* (2017) observed high frequency of antibiotic resistance among all strains isolated from the tested products and the isolates comprised strains resistant to tetracycline (70%) and erythromycin (10%).

Van Eldere *et al.* (2014) stated ―that, a microorganism can acquire resistance to an antimicrobial to which it was previously sensitive, meaning that the antimicrobial will no longer be able to kill or inhibit the growth of the microorganism at the same level as before‖. Further Van Eldere *et al.* (2014) posited the three types of resistance as described below:

2.3.1 Microbiological resistance

Reduced susceptibility of bacteria to antibiotics above a breakpoint that is defined by the upper limit of normal susceptibility of the concerned species, which is also called epidemiological resistance, the microbiological resistance can often be confirmed genotypically by demonstrating the presence of a certain antimicrobial resistance gene or resistance mechanism via molecular techniques.

2.3.2 Pharmacological resistance

This is based on pharmacokinetic parameters and the normal susceptibility of a bacterial species. If the minimal inhibitory concentration (MIC) of the antibiotic for the bacteria concerned is within the concentration range that can be attained by that antimicrobial, it is susceptible. If the MIC of the antibiotic for the concerned bacteria is higher than the concentration that can be attained at the site of infection, then the bacterium is regarded as resistant.

2.3.3 Clinical resistance

An infection with the concerned bacterium cannot be treated appropriately anymore and treatment failures are evident.

2.4 Food Safety

Bacteria are not visible to the naked eye; they exist in and on the human body, soil and in the air but only a few types can grow in food and causes food poison. Under favourable condition bacteria can multiple very rapidly simply by dividing in two every 20-30 minutes depending on the species so that one cell could produce up to 16 million within 8 hours.

Food does not have to look, smell or taste ―off‖ to be potentially hazardous to health (Interagency Microbiological Risk Assessment Guideline Working Group.2012). The presence of the type of microorganism in food reflects the condition of the food products. However there are a few pathogenic microorganisms which when present causes food borne illness. Infants are very susceptible to food borne diseases and if they consume contaminated food they are likely to contract infections or intoxication leading to illness and often death.

2.4.1 Escherichia coli

This is a faecal coliform which exist in the digestive system of creatures and man. It is an indication poor hygiene practice (Interagency Microbiological Risk Assessment Guideline Working Group, 2012). Right now there are four perceived classes of enterovirulent E. coli (alluded to EEC gathering) that cause gastro enteritis in human.

They are unsafe when found in food (FDA, 2006).

2.4.2 Staphylococcus aureus

This is commonly found on the skin, mucous membrane, and hands. This organism can readily be transferred to food by poor handling. They are gram positive and some strains are capable of producing a highly heat stable protein toxin that causes illness in humans**.** (Interagency Microbiological Risk Assessment Guideline Working Group.2012).

2.4.3 Salmonella species

These organisms account for over 50% of all reported cases of food poisoning (O'Hara and Pirog, 2016). They are gram negative bacteria. There is a wide spread occurrence in

animals especially poultry and swine. Environmental source of these organisms include water, soil, insects, factory surfaces, kitchen surfaces, animal faeces (Interagency Microbiological Risk Assessment Guideline Working Group.2012).

2.5 Total bacteria counts

This reflects the conditions in which the food was produced, stored or abused. The spoilage of many foods may be imminent when the total count reaches 10-100m/g. With experience this can be used to predict the shelf life of the product (Interagency Microbiological Risk Assessment Guideline Working Group.2012).

2.6 Control of Antimicrobial Resistance

Verraes *et al*. (2013) opined that, bacteria can be resistant to antibiotics by using several mechanisms: enzymatic degradation of antibiotics, antibiotic target modification, changing the bacterial cell wall permeability and alternative pathways to escape the activity. Enzymatic degradation or modification of antibiotics is a very common mechanism of resistance. Livermore and Woodford, (2006) sited cases as the β-lactamase compounds hydrolysing the β-lactam ring of β-lactam anti-infection agents, for example, cephalosporins, which are for the most part of worry in Gram-negative microbes.

Wright (1999) added that, another group of antibiotics to whom resistance is mainly mediated by enzymatic degradation are the aminoglycosides, where inactivation is caused by *acetyltransferases, nucleotidyltransferases and phosphotransferases*.

Drlica and Zhao (1997) in a related study opined that, ―resistance by target modification implies a modification of the target molecule of the antibiotic, in general an enzyme, so that the antibiotic loses its binding capacity and hence its activity." he cited that, examples

of this mechanism are mutations in the gyrase and topoisomerase genes that are the targets of the quinolone and fluoroquinolone antibiotics.

CHAPTER THREE

3.0. MATERIALS AND METHODS

3.1 Sample Collection and study area

Samples of weanimix were obtained from six (6) different antenatal health centres in the Accra Metropolis (Kaneshie Polyclinic, Mamprobi Polyclinic, Dansoman Polyclinic, Ussher Polyclinic, Princess Marie Children's Hospital and La General Hospital). A total of ninety (90) samples were randomly collected and were assigned unique code and immediately taken to the laboratory for analysis.

Sample Size drawn from the various health centres

Sample loc.(Health Centres)	KP	PM	MP	LP	UP	DP
Sample size(n)	10	20	10	15	15	20

*KP= Kaneshie Polyclinic *PM = Princess Marie Hospital *MP= Mamprobi Polyclinic
*LP= La General Hospital *UP= Ussher Polyclinic *DP= Dansoman Polyclinic

3.2. Preparation of Samples

All media were prepared according to the manufacturer's instructions. The media were chosen as per the instruction in the ISO 7218 method of microbiology of food and animal feeding stuffs. 25g of each of the sample was weighed and mixed with 225ml of sterile maximum recovery diluents (MRD). This was used as the 10^{-1} dilution for each of the samples. With a sterile pipette, serial dilutions up to 10^{-4} were prepared by taking 1m1 of

the 10^{-1} dilution and transferred into 9 ml of the sterile diluents (MRD) in a universal bottle to give a 10^{-2} dilution. This process was repeated for new bottles until a final dilution of 10^{-4} was obtained for each sample. 25 g of each of the sample was weighed and mixed with 225ml of sterile buffered peptone water (BPW) for Salmonella detection.

3.3 Methods (Isolation of Colonies)

3.3.1 Aerobic Plate Count – ISO 4833-1

Pour plating was done by taking 1m1 aliquot of each dilution and dispensed into sterile petri dishes, about 15ml of molten plate count agar (PCA) was added, swirled gently to ensure homogenized mixture and allowed to solidify. Duplicate plates were prepared for each dilution. All the plates were allowed to dry in an inverted position for a few minutes and then incubated at 30ºC for 72hrs. Colonies were counted using the colony counter.

3.3.2 *Staphylococcus aureus* – ISO 6888-2

Pour plating technique was also used to determine counts of *Staphylococcus aureus* by taking 1ml of each dilution into sterile petri dishes; about 15 ml of molten Baird Parker agar with RPF supplement (BPA + RPF) was poured into it, mixed well and allowed to set. The plates were incubated at 37°C for 48hrs in an inverted position to allow bacterial colonies to be formed. Black colonies with halo surrounding were presumed to be Positive for *Staphylococcus aureus*, colonies were counted using the colony counter.

3.3.3 *E. coli* – ISO 16649-2

One ml of each serial dilution was inoculated into sterile petri dishes, about 15ml of molten Tryptone Bile X-Glucuronide (TBX) selective agar was added, mixed and allowed to set, and the plates were incubated at 44ºC for 24 hrs. Plates showing blue green colonies were presumed to be E. coli positive, colonies were counted using the colony counter.

3.3.4 *Salmonella* – 1SO 6579

25 g of each sample were weighed into 225 ml Buffered Peptone Water (BPW) and incubated at 37ºC for 24 hrs., 0.1 ml and 1 ml from BPW was transferred into two secondary enrichment broths; Muller-Kauffmann Tetrathionate / Novobiocin broth (MKTTn) and Rappaport-Vassiliadis medium with soya (RVS) broth respectively and then incubated at 37ºC for another 24 hrs. A loopful (5µl) from each secondary enrichment broth was streak on two selective media; Xylose lysine deoxycholate agar (XLD) and Brilliance Salmonella agar (BSA). Typical colonies were black with red precipitate surrounding on XLD and black metallic sheen on BSA.

3.3.5 *Bacillus cereus* – ISO 7932

About 0.1 ml of each serial dilution was spread over the surface of B. cereus selective agar thus Mannitol Yolk Polymyxin (MYP) and allowed to stand for 15 min for the inoculum to absorb into the agar. The plates were incubated at 30ºC for 24 hrs.

Presumptive colonies were large pink colonies with a zone of precipitation surrounding it

3.4 Antibacterial resistance testing

The susceptibility pattern of the isolates to antimicrobial agents was determined using the disc diffusion (Kirby-Bauer) methods as described by the National Committee for Clinical Laboratory Standards (now Clinical and Laboratory Standards Institute) (NCCLS Doc. M2-A6; 2009). A loop full (5µl) of each isolate was emulsified in 10 mL sterile nutrient broth in a test tube and the density measured with McFarland densitometer (Grant-bio Den-1 no. 05O102-1109-0368. England) with a total aerobic plate count of 10^6 CFU/ml. A sterile cotton swab was dipped into the standardized suspension of the bacterial culture and used to spread the surface of Mueller-Hinton agar plates evenly (Oxoid, Basingstoke, United Kingdom).

The plates were permitted to dry for a couple of minutes. Anti-infection circles (Oxoid, Basingstoke, United Kingdom) with the accompanying fixations, antibiotic medication Trimethoprim/Sulfamethoxazole, (Co-trimoxazole) (23.75µg/1.25µg), Ciprofloxacin (5µg), Cefuroxime (30µg), Augmentin (20µg/10 µg) and Ceftriaxone (30 µg) were put on the plates**.**

The separation between circles was around 15mm to prevent overlapping of zone of inhibition. The plates were then incubated at 37ºC for 24 hrs, and the zones of inhibition measured with protocol 3 symbiosis (Cambridge UK). Each zone of isolates was compared with the recorded diameters of the control organism *E. coli* ATCC 25922 to determined susceptibility or resistance. For the purpose of analysis, all isolates with intermediate zones of inhibition were classified as resistant.

3.5 Data Analysis

Information from the Six (6) diverse antenatal health centers in the Accra Metropolis was analyzed with SPSS 21. The information was subjected to straight forward graphic measurements.

CHAPTER FOUR

4.0. RESULTS AND DISCUSSION

4.1. Microbiological analysis

From Table 4.1, the results of total viable count (TVC) of all 90 samples range between 1-4.53 log CFU/g. The health centre, DP has the highest range, followed by PM, MP, KP, LP, and UP. The Mean is between 3.50 (± 3.88) log CFU/g and 2.35 (±2.63) log CFU/g. With *Staph. aureus* PM has the highest detection of 4.32 log CFU/g and UP with the lowest of 3.08 log CFU/g.

Table 4.1: The Results of Total Viable Count (TVC) of all the samples.

HEALTH CENTER	Range TVC (log CFU/g)	Mean (log CFU/g)	Staph. (log CFU/g)	Mean (log CFU/g)
UP	1 – 3.08	2.35 (±2.63)	3.08	2.35 (±2.63)
KP	1 – 4.23	3.27 (± 3.73)	4.23	3.37 (±3.73)
MP	1 – 4.04	3.09 (± 5.54)	4.04	3.09 (±3.54)
LP	1 – 3.79	2.74 (± 3.21)	3.79	2.74 (±3.21)
PM	1 – 4.32	3.12 (± 3.67)	4.32	3.12 (±2.67)
DP	1 – 4.53	3.50 (± 3.88)	3.52	2.44 (±2.89)

***Refer to the Regulatory limit of the microbial counts (Appendix I, page 69)**

Table 4.2: Detection of microorganisms

HEALTH CENTRE	E. coli	B. Cereus	Salmonella
DP	Detection (0)	Detection (0)	Detection (2)
UP	Detection (0)	Detection (0)	Detection (1)
KP	Detection (0)	Detection (0)	Detection (2)
MP	Detection (0)	Detection (0)	Detection (0)
LP	Detection (1)	Detection (0)	Detection (4)
PM	Detection (1)	Detection (0)	Detection (4)

*Refer to the Regulatory limit of the microbial counts (Appendix I, page 69)

From table 4.2, among all the health centres, LP and PM have one detection each for *E. coli,* and the rest of the health centres have no detection for *E. coli*. Moreover, all the health centres have no detection for *B. Cereus*.

For *Salmonella* detection, LP, PM have four detection each and KP, DP have two detection each. UP has one detection and MP has no detection for *salmonella*.

Table 4.3: Antimicrobial Activity from All Health Centres

Code (health center)	Cefuroxime 30ug/ml			Ceftriaxone 30ug/ml			ENTRATIONS OF A (MM) Ciprofloxacin 5ug/ml			Augmentin 20ug/10ug/ml			C0-trimoxazole 23.75ug/1.25ug/ml		
	SA	*Sal*	*E.c*	*SA*	*Sal*	*E.c*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*
DC	10/14	13/16	-	12/19	26/27	-	19-21	26/29	-	16-18	13/14	-	15-18	12/12	-
UP	0/9	11	-	0/18	24		20-23	26	-	17-18	11	-	18-19	10	-
PM	0/12	6/7	12	0/11	21-30	18	19-24	24-32	34	0-19	0-12	15	15-19	11-13	35
KP	8-14	0/16	-	11-19	25/28	-	19-21	28/31	-	10-18	13/13	-	15-19	9/14	-
MP	0/10	-	-	0/15	-	-	19/23	-	-	10/17	-	-	15/17	-	-
LP	0-15	0-10	13	0-20	16-22	19	15-24	22-30	32	13-18	11-19	16	12-19	9-12	

The samples collected from this sampling site Usher polyclinic (UP) showed antibiotic susceptibility patterns of the samples which tested positive for *Salmonella* spp. and S. *aureus*. All three samples with *S. aureus* showed resistance to cefuroxime and ceftriaxone with zones of inhibition measured below 14 mm, two samples contaminated with *Salmonella spp* showed resistance to cefuroxime, Augmentin and co-trimoxazole with zones of inhibition measured below 14 mm, 19 mm and 10mm respectively,

Again, samples collected from Princess Marie Children's Hospital (PMCH) the six samples with *S. aureus* showed resistance to cefuroxime and ceftriaxone with zones of inhibition measured below 14 mm; four samples contaminated with *Salmonella spp* showed resistance to Cefuroxime, Ceftriazone and Augmentin with zones of inhibition measured below 14 mm and 19 mm, two shows resistance to Co-trimoxazole with the zone of

inhibition below 10mm; one sample however was contaminated with the *E. coli* which was resistance to cefuroxime with the zone of inhibition below 14mm.

The samples collected from Kaneshie polyclinic (KP) had three samples with *S. aureus* showed resistance to Cefuroxime, two to Ceftriaxone and four to Augmentin with zones of inhibition measured below 14 mm and 19mm, one of the samples which was contaminated with *Salmonella spp* showed resistance to Cefuroxime and Co-trimoxazole with the zone of inhibition below 14mm and 10mm, two showed resistances to Augmentin with zones of inhibition measured below 19 mm.

Also, samples collected from Mamprobi polyclinic (MP) had two samples with *S.aureus* which showed resistance to Cefuroxime and Augmentin, one showed resistance to Ceftriaxone with the zones of inhibition measured below the criteria as shown in Table 4.10

Furthermore, samples collected from La polyclinic (LP) had three samples which were contaminated with *S. aureus* showed resistance to Cefuroxime and two to Ceftriaxone. Four samples contaminated with *Salmonella spp*, showed resistance to Cefuroxime and one to Co-trimoxazole with the zones of inhibition measured below MIC. The *E. coli* isolated from this health centre however showed no resistance to all the antibiotics,

From a total of 90 samples collected from the six health centres in the Greater Accra region 25.6% were positive for *S. aureus*, 14.4% were positive for *Salmonell*a and 2.2% of the samples were positive for *E. coli* as shown below in Table 4.13

A total of 13 samples representing 14.4% did not meet criteria for safe infant‘s formula due to the detection of the presence of *Salmonella* in them. However, there are no published

studies from Ghana. On the other hand, work done by On *et al*. (2010) on risk profile on *Salmonell*a in cereal grains reported similar trends in prevalence.

Also, 23 samples representing 25.6% collected from the health centres were contaminated with *S. aureus* which were above the specification of 100cfu/g for infant foods. (ICSMF8, 2011)

Githaiga (2012), posited that the presence of *Staphylococcus aureus* suggests a contamination which emanates from food handling which might have occurred in the foods during handling, processing or vending.. *Staphylococcus aureus* being part of the micro flora present in several parts of the human body is a good indicator of contamination due to poor personnel hygiene practices

E. coli was detected in *2* samples representing 2.2% of the total samples collected. Edberg *et al. (*2000) suggested that presence of *E. coli*, which is an enteric bacterium, has been widely accepted as an indicator of faecal contamination in milk and dairy products and other foods. Figure1 below show the percentile contamination of each health centre.

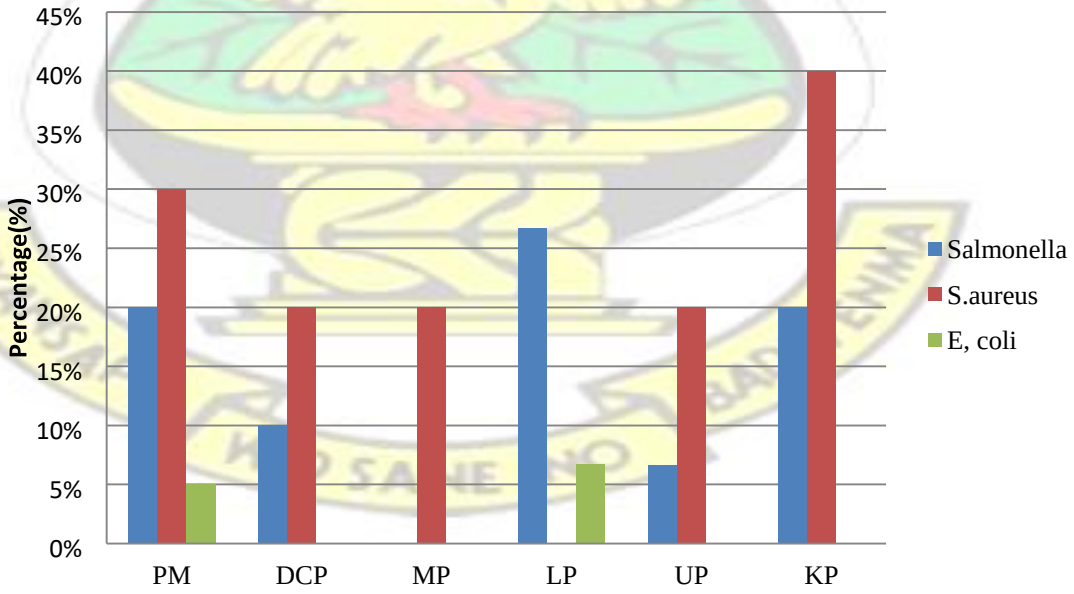

Figure1: Summary of the pathogens presents in the food samples.

4.2 Antibiotics Susceptibility

This current study reveals that, the 13(100%) samples which showed positive for *Salmonella spp.* were susceptible to Ciprofloxacin, 69.2% to Ceftriaxone, 23.1% to Augmentin, 61.5% to Co-trimoxazole and 46.2% to Cefuroxime (Table 4.14). This result contradicts the observation in China by Dong *et al.* (2016), in cereal based foods. However, it is consistent with studies in Romania by Colobatiu *et al.* (2015). This observation is also lower than the 90% resistance observed by Mebrat *et al.* (2016) and Ahmed *et al.* (2016) in their studies on *salmonella* isolates in maize based food products to major antibiotics. This analysis has become necessary due to infection that infants get through food borne organism and how it would be treated.

Likewise, 23 samples representing 100% which tested positive for *S. aureus* were susceptible to Ciprofloxacin, 34.8% to Ceftriaxone, 73.9% to Augmentin, 95.7% to Cotrimoxazole and 21.7% to Cefuroxime. *E. coli* showed 100% susceptibility to all the antibiotics except Cefuroxime which was 50%. This pattern observed is consistent with what was observed by Adzitey and Huda *(*2010) in Northern Ghana. *S. aureus* is one of the microbes that can easily develop resistance and hence high resistance to the most used antibiotics is therefore worrying. According to the report of Bechtold, and Mussak, (2009) the sources of microbial contamination of cereals are many, but all are traceable to the environment in which grains are grown, handled and processing methods. Microorganisms that contaminate cereal grains may come from air, dust, soil, water, insects, rodents, birds, animals, humans, storage, means of transports, and handling and processing equipment.

Table 4.14: Summary of the Antibiotics Susceptibility on isolated organisms

Antibiotics	Salmonella	S.aureus	E.coli
Ciprofloxacin	13(100%)	23(100%)	2(100%)
Ceftriaxone	9(69.2%)	8(34.8%)	2(100%)
Augmentin	3(23.1%)	17(73.9%)	2(100%)
Co-trimoxazole	8(61.5%)	22(95.7%)	2(100%)
Cefuroxime	6(46.2%)	5(21.7%)	1(50%)

Ruiz *et al*. (2012) propounded that significant number of *Salmonella* isolates proved to be resistant to quinolones (Qs) (for example nalidixic acid) and fluoroquinolones (FQs) (for example Ciprofloxacin). This he said is worrying because Qs and FQs are potent broadspectrum antibiotics used in the treatment of infections in both humans and animals and the significant number of isolates proving resistance to the various antibiotics could be due to the indiscriminate use of antibiotics in the study area and across the country. This may result in selection pressure that increases the advantage of maintaining resistant genes in bacteria according to Ruiz *et al.* (2012)

From Table 4.15 the results reveals that all the organisms has a zero resistant with Ciprofloxacin , *E.coli* has zero resistant with four of the antibiotics except Cefuroxime which was resistant of 50% of the *E.coli* spp, however 30.8% of the *salmonella* species and 65.2% of *S.aureus* species were resistant to ceftriaxone, 76.9% and 26.1% of the sample that tested positive for *Salmonella* and *S.aureus* respectively were resistant to Augmentin

likewise Co-trimoxazole was not effective on 38.5% and 4.3% of *Salmonella* and *S.aureus* spp. 53.8% and 78.3% of *Salmonell*a and *S.aureus spp* were resistant to Cefuroxime. . From this Ciprofloxacin proved to be more effective on all the pathogens isolated as shown below.

Table 4.15: Summary of the Antibiotics resistance on isolated organisms

Antibiotics	**Salmonella**	***S.aureus***	***E.coli***
Ciprofloxacin	0(100%)	0(100%)	0(100%)
Ceftriaxone	4(30.8%)	15(65.2%)	0(100%)
Augmentin	10(76.9%)	6(26.1%)	0(100%)
Co-trimoxazole	5(38.5%)	1(4.3%)	0(100%)
Cefuroxime	7(53.8%)	18(78.3%)	1(50%)

CHAPTER FIVE

5.0. CONCLUSIONS AND RECOMMENDATIONS

5.1. Conclusions

From the study, complementary foods or weaning food (―weanimix‖), an important food for feeding infants was found to be highly contaminated with *Salmonella, S. aureus* and *E. coli.* It was observed from the variation in the individual sample results that, the products sold to mothers at the health centres come from different grain combination and different producers. Also, the weanimix were widely different from each other and with most of them not labelled. The fundamental target of this examination is to assess the microbiological safety of weanimix and its antimicrobial susceptibility.

The study revealed that 13 samples representing 14.4% did not meet the criteria for safe food for infants as *salmonella* species were detected in them; data on the prevalence of *salmonella* in cereal are not readily available in Ghana.

However, similar prevalence as reported in this current work has been reported in other parts of the continent. Adding to the preceding points, 23 samples representing 25.6% collected from the health centres were contaminated with *S. aureus* which were above the specification of 100cfu/g (ICSMF8, 2011) for infant foods. *E. coli* which is supposed to be absent was detected in *2* samples representing 2.2% of the total samples collected. The presences of enteric bacteria example *E. coli* have been widely accepted as indicators of faecal contamination. *E. coli* has been classically used as indicator of the possible presence of enteric pathogens in milk and dairy products and other foods (Reij *et al.* 2004).,

This study also revealed that 69.2% out of the 13 samples that tested positive for *Salmonella* were susceptible to Ciprofloxacin, 84.6% were susceptible to Ceftriaxone,

55.8% was susceptible to Augmentin, and 30.8% was susceptible to Co-trimoxazole and 23.1% to Cefuroxime. This result contradicts the observation in China by Dong et al (2016), in cereal based foods however, it is consistent with studies in Romania by Colobatiu *et al.* (2015). Likewise, it was revealed that some isolates from weanimix were susceptible to some antibiotics while other isolates were resistant as shown in Table 4.14 and 4.15

5.2. Recommendations

The findings demonstrate that weaning food or ―weanimix‖ were contaminated with *Salmonella, S. aureus and E. coli.* This has negative implication on the health of infants. In view of the discoveries above the following recommendations have been made;

- Regulatory agencies(FDA, Ghana Health Services, Local District Assemblies) should take up the challenge in helping local food manufactures to produce safe foods especially _weanimix‘ as most of these products are unregulated (unregistered). .
- Region by region research should be conducted to ascertain the safety of weanimix sold in the health centres across the entire country.
- Therefore, the Ministry of Health (MoH) needs to educate mothers to develop an economically feasible strategy to eliminate exposure of children fed homemade weanimix to *Salmonella, S. aureus* and *E. coli.*

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7.0. APPENDICES

APPENDIX 1

Results for Total Plate Counts (TVC)

DANSOMAN POLYCLINIC

sample	Serial dilutions								Cfu/g
	10^{-1}		10^{-2}		10^{-3}		10^{-4}		
	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	
DCP1	TNTC	TNTC	>300	>300	36	38	3	2	**3.4×10^4**
DCP2	206	211	23	25	3	2	0	0	**1.9×10^3**
DCP3	>300	>300	41	37	5	4	0	0	**3.5×10^3**
DCP4	1	1	0	0	0	0	0	0	**<10**
DCP5	0	0	0	0	0	0	0	0	**<10**
DCP6	25	28	3	2	0	0	0	0	**2.6×10^2**
DCP7	38	43	11	12	1	1	0	0	**3.7×10^3**
DCP8	0	0	0	0	0	0	0	0	**<10**
DCP9	1	0	0	0	0	0	0	0	**<10**
DCP10	1	1	0	0	0	0	0	0	**<10**
DCP11	35	39	4	4	1	1	0	0	**3.4×10^2**
DCP12	>300	>300	56	53	2	1	0	0	**5.0×10^3**
DCP13	26	29	2	3	0	0	0	0	**2.8×10^2**
DCP14	>300	>300	90	86	8	6	0	0	**8.0×10^3**
DCP15	>300	>300	68	74	6	6	0	0	**6.5×10^3**
DCP16	>300	>300	36	37	2	1	0	0	**3.3×10^3**
DCP17	280	286	31	29	2	2	0	0	**2.8×10^3**
DCP18	22	23	4	4	0	0	0	0	**2.3×10^2**
DCP19	1	0	0	0	0	0	0	0	**<10**

DCP20	43	46	3	4	0	0	0	0	**4.0×10^2**

USSHER POLYCLINIC

Sample	**Serial dilutions**								**Cfu/g**
	10^{-1}		**10^{-2}**		**$.0^{-3}$**		**10^{-4}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
UP21	86	88	7	6	2	3	0	0	**7.9×10^2**
UP22	2	2	1	0	0	0	0	0	**<10**
UP23	0	0	0	0	0	0	0	0	**<10**
UP24	4	2	0	0	0	0	0	0	**<10**
UP25	>300	>300	118	126	15	15	2	2	**1.1×10^4**
UP26	TNTC	TNTC	>300	>300	147	151	11	8	**1.3×10^5**
UP27	>300	>300	172	184	20	21	3	3	**1.6×10^4**
UP28	TNTC	TNTC	>300	>300	182	190	20	18	**1.7×10^5**
UP29	0	0	0	0	0	0	0	0	**<10**
UP30	2	3	1	1	0	0	0	0	**<10**
UP31	>300	>300	152	156	17	15	4	5	**1.4×10^4**
UP32	TNTC	TNTC	>300	>300	78	73	2	3	**6.9×10^4**
UP33	82	80	9	10	3	4	0	0	**7.4×10^2**
UP34	68	64	7	5	1	1	0	0	**6.0×10^2**
UP35	122	131	12	12	3	3	1	1	**1.2×10^3**

PRINCESS MARIE CHILDREN'S HOSPITAL

sample	Serial dilutions								Cfu/g
	10^{-1}		10^{-2}		10^{-3}		10^{-4}		
	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	
PM36	>300	>300	68	72	7	8	0	0	6.3×10^3
PM37	48	50	7	9	1	1	0	0	4.5×10^2
PM38	>300	>300	149	144	4	3	1	0	1.3×10^4
PM39	159	163	18	20	2	2	0	0	1.5×10^3
PM40	TNTC	TNTC	>300	>300	118	124	12	13	1.1×10^5
PM41	TNTC	TNTC	>300	>300	195	193	20	23	1.8×10^5
PM42	>300	>300	98	104	10	8	2	2	9.2×10^3
PM43	>300	>300	108	115	2	4	1	2	1.0×10^4
PM44	158	155	13	13	4	2	0	0	1.4×10^3
PM45	179	182	17	20	2	1	0	0	1.2×10^3
PM46	TNTC	TNTC	>300	>300	126	129	13	15	1.2×10^5
PM47	50	52	6	5	1	1	0	0	4.6×10^2
PM48	>300	>300	185	198	10	12	0	0	1.7×10^4
PM49	>300	>300	158	164	21	18	2	5	1.5×10^4
PM50	>300	>300	195	198	20	18	1	3	1.8×10^4
PM51	159	164	16	18	1	1	0	0	1.5×10^3
PM52	TNTC	TNTC	>300	>300	73	75	0	0	6.7×10^4

PM53	TNTC	TNTC	>300	>300	188	192	18	19	1.7×10^5
PM54	>300	>300	108	110	16	17	0	0	9.9×10^3
PM55	TNTC	TNTC	>300	>300	121	124	15	16	1.1×10^5

KANESHIE POLYCLINIC

sample	Serial dilutions								Cfu/g
	10^{-1}		10^{-2}		10^{-3}		10^{-4}		
	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	
KP 56	TNTC	TNTC	TNTC	TNTC	>300	>300	40	44	3.8×10^5
KP 57	>300	>300	139	141	5	3	0	0	1.3×10^4
KP 58	>300	>300	221	223	23	22	2	2	2.0×10^4
KP 59	>300	>300	230	225	25	24	3	2	2.1×10^4
KP 60	48	46	5	6	1	1	0	0	4.3×10^2
KP 61	>300	>300	78	80	7	9	2	4	7.2×10^3
KP 62	10	12	1	1	0	0	0	0	1.1×10^2
KP 63	>300	>300	126	137	14	12	5	4	1.2×10^4
KP 64	16	11	0	0	0	0	0	0	1.3×10^2
KP 65	TNTC	TNTC	>300	>300	168	179	10	6	1.6×10^5

MAMPROBI POLYCLINIC

sample	Serial dilutions								Cfu/g
	10^{-1}		10^{-2}		10^{-3}		10^{-4}		
	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	
MP 66	>300	>300	55	57	4	5	2	1	5.1×10^3
MP 67	>300	>300	140	141	4	3	1	1	1.3×10^4
MP 68	11	10	2	3	0	0	0	0	1.1×10^2

MP 69	TNTC	TNTC	>300	>300	135	137	4	4	**1.2×10^5**
MP 70	>300	>300	165	169	7	8	0	0	**1.5×10^4**
MP 71	83	84	10	9	1	1	0	0	**7.6×10^2**
MP 72	1	1	0	0	0	0	0	0	**<10**
MP 73	98	92	12	10	0	1	0	0	**8.6×10^2**
MP 74	74	76	7	6	0	0	0	0	**6.8×10^2**
MP 75	>300	>300	48	45	2	2	0	0	**4.2×10^3**

LA POLYCLINIC

sample	**Serial dilutions**								**Cfu/g**
	10^{-1}		**10^{-2}**		**10^{-3}**		**10^{-4}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
LP 76	TNTC	TNTC	>300	>300	143	139	3	4	**1.3×10^5**
LP 77	TNTC	TNTC	>300	>300	210	212	21	25	**1.9×10^5**
LP 78	>300	>300	136	132	9	13	2	1	**1.2×10^4**
LP 79	179	171	28	29	3	2	0	0	**1.6×10^3**
LP 80	35	39	3	2	0	0	0	0	**3.4×10^2**
LP 81	4	3	0	0	0	0	0	0	**<10**
LP 82	24	29	3	2	0	1	0	0	**2.7×10^2**
LP 83	>300	>300	98	96	10	11	1	0	**8.8×10^3**
LP 84	26	28	2	3	1	1	0	0	**2.7×10^2**
LP 85	TNTC	TNTC	>300	>300	128	123	11	15	**1.1×10^5**
LP 86	>300	>300	142	145	15	20	2	2	**1.3×10^4**
LP 87	TNTC	TNTC	>300	>300	101	106	6	8	**9.4×10^4**
LP 88	TNTC	TNTC	>300	>300	158	162	4	5	**1.5×10^5**
LP 89	>300	>300	49	56	5	7	0	0	**4.8×10^3**
LP 90	109	118	15	14	2	4	0	0	**1.0×10^3**

RESULTS FOR *STAPHLOCOCCUS AUREUS*

DANSOMAN POLYCLINIC

sample	Serial dilutions								Cfu/g
	10^{-1}		10^{-2}		10^{-3}		10^{-4}		
	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	
DCP1	0	0	0	0	0	0	0	0	**<10**
DCP2	35	36	2	3	0	0	0	0	**3.5×10^2**
DCP3	0	0	0	0	0	0	0	0	**<10**
DCP4	23	21	2	1	0	0	0	0	**2.1×10^2**
DCP5	0	0	0	0	0	0	0	0	**<10**
DCP6	0	0	0	0	0	0	0	0	**<10**
DCP7	0	0	0	0	0	0	0	0	**<10**
DCP8	0	0	0	0	0	0	0	0	**<10**
DCP9	0	0	0	0	0	0	0	0	**<10**
DCP10	0	0	0	0	0	0	0	0	**<10**
DCP11	0	0	0	0	0	0	0	0	**<10**
DCP12	143	151	14	15	2	2	0	0	**1.5×10^3**

DCP13	0	0	0	0	0	0	0	0	**<10**
DCP14	0	0	0	0	0	0	0	0	**<10**
DCP15	>300	>300	31	35	3	4	0	0	**3.3×10^3**
DCP16	0	0	0	0	0	0	0	0	**<10**
DCP17	0	0	0	0	0	0	0	0	**<10**
DCP18	0	0	0	0	0	0	0	0	**<10**
DCP19	0	0	0	0	0	0	0	0	**<10**
DCP20	0	0	0	0	0	0	0	0	**<10**

USSHER POLYCLINIC

sample	**Serial dilutions**								**Cfu/g**
	10^{-1}		**10^{-2}**		**10^{-3}**		**10^{-4}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
UP21	0	0	0	0	0	0	0	0	**<10**
UP22	0	0	0	0	0	0	0	0	**<10**
UP23	0	0	0	0	0	0	0	0	**<10**
UP24	0	0	0	0	0	0	0	0	**<10**
UP25	121	119	12	11	0	0	0	0	**1.2×10^3**
UP26	0	0	0	0	0	0	0	0	**<10**
UP27	0	0	0	0	0	0	0	0	**<10**
UP28	33	38	4	4	0	0	0	0	**3.6×10^2**
UP29	0	0	0	0	0	0	0	0	**<10**
UP30	100	112	11	10	1	0	0	0	**1.1×10^3**

UP31	0	0	0	0	0	0	0	0	**<10**
UP32	18	15	2	2	0	0	0	0	**1.7×10^2**
UP33	0	0	0	0	0	0	0	0	**<10**
UP34	0	0	0	0	0	0	0	0	**<10**
UP35	0	0	0	0	0	0	0	0	**<10**

PRINCESS MARIE CHILDREN'S HOSPITAL

sample	**Serial dilutions**								**Cfu/g**
	10^{-1}		**10^{-2}**		**10^{-3}**		**10^{-4}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
PM36	6	8	2	4	0	0	0	0	**1.0×10^2**
PM37	>300	>300	201	209	20	21	2	0	**2.1×10^4**
PM38	0	0	0	0	0	0	0	0	**<10**
PM39	0	0	0	0	0	0	0	0	**<10**
PM40	10	13	2	1	0	0	0	0	**1.2×10^2**
PM41	0	0	0	0	0	0	0	0	**<10**
PM42	150	149	34	45	14	17	1	1	**1.9×10^3**
PM43	0	0	0	0	0	0	0	0	**<10**

PM44	0	0	0	0	0	0	0	0	**<10**
PM45	0	0	0	0	0	0	0	0	**<10**
PM46	0	0	0	0	0	0	0	0	**<10**
PM47	0	0	0	0	0	0	0	0	**<10**
PM48	>300	>300	151	148	14	16	2	3	**1.5×10^3**
PM49	0	0	0	0	0	0	0	0	**<10**
PM50	0	0	0	0	0	0	0	0	**<10**
PM51	132	150	14	13	2	2	0	0	**1.4×10^3**
PM52	0	0	0	0	0	0	0	0	**<10**
PM53	0	0	0	0	0	0	0	0	**<10**
PM54	0	0	0	0	0	0	0	0	**<10**
PM55	0	0	0	0	0	0	0	0	**<10**

KANESHIE POLYCLINIC

sample	**Serial dilutions**								**Cfu/g**
	10^{-1}		**10^{-2}**		**10^{-3}**		**10^{-4}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
KP 56	0	0	0	0	0	0	0	0	**<10**
KP 57	36	44	19	14	5	2	0	0	**5.5×10^2**
KP 58	0	0	0	0	0	0	0	0	**<10**
KP 59	>300	>300	189	177	27	14	2	8	**1.7×10^4**
KP 60	12	19	5	5	2	1	0	0	**2.0×10^2**
KP 61	0	0	0	0	0	0	0	0	**<10**
KP 62	0	0	0	0	0	0	0	0	**<10**

KP 63	73	57	5	7	2	0	0	0	**6.5×10^2**
KP 64	0	0	0	0	0	0	0	0	**<10**
KP 65	0	0	0	0	0	0	0	0	**<10**

MAMPROBI POLYCLINIC

sample	**Serial dilutions**								**Cfu/g**
	10^{-1}		**10^{-2}**		**10^{-3}**		**10^{-4}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
MP 66	0	0	0	0	0	0	0	0	**<10**
MP 67	0	0	0	0	0	0	0	0	**<10**
MP 68	0	0	0	0	0	0	0	0	**<10**
MP 69	0	0	0	0	0	0	0	0	**<10**
MP 70	0	0	0	0	0	0	0	0	**<10**

MP 71	>300	>300	110	112	10	11	1	2	**1.1×10^4**
MP 72	0	0	0	0	0	0	0	0	**<10**
MP 73	138	130	15	14	2	3	0	0	**1.4×10^3**
MP 74	0	0	0	0	0	0	0	0	**<10**
MP 75	0	0	0	0	0	0	0	0	**<10**

LA POLYCLINIC

sample	**Serial dilutions**								**Cfu/g**
	10^{-1}		**10^{-2}**		**10^{-3}**		**10^{-4}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
LP 76	0	0	0	0	0	0	0	0	**<10**
LP 77	>300	>300	58	65	6	7	1	1	**6.2×10^3**
LP 78	0	0	0	0	0	0	0	0	**<10**
LP 79	0	0	0	0	0	0	0	0	**<10**
LP 80	0	0	0	0	0	0	0	0	**<10**
LP 81	158	149	15	18	2	4	1	0	**1.6×10^3**
LP 82	0	0	0	0	0	0	0	0	**<10**
LP 83	0	0	0	0	0	0	0	0	**<10**
LP 84	28	33	4	4	1	0	0	0	**3.2×10^2**
LP 85	0	0	0	0	0	0	0	0	**<10**
LP 86	0	0	0	0	0	0	0	0	**<10**
LP 87	0	0	0	0	0	0	0	0	**<10**
LP 88	0	0	0	0	0	0	0	0	**<10**
LP 89	0	0	0	0	0	0	0	0	**<10**

LP 90	0	0	0	0	0	0	0	0	**<10**

RESULTS FOR *E.COLI* DANSOMAN POLYCLINIC

sample	**Serial dilutions**						
	10^{-1}		**10^{-2}**		**10^{-3}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	
DCP1	0	0	0	0	0	0	
DCP2	0	0	0	0	0	0	
DCP3	0	0	0	0	0	0	
DCP4	0	0	0	0	0	0	
DCP5	0	0	0	0	0	0	
DCP6	0	0	0	0	0	0	
DCP7	0	0	0	0	0	0	
DCP8	0	0	0	0	0	0	
DCP9	0	0	0	0	0	0	
DCP10	0	0	0	0	0	0	
DCP11	0	0	0	0	0	0	
DCP12	0	0	0	0	0	0	
DCP13	0	0	0	0	0	0	
DCP14	0	0	0	0	0	0	
DCP15	0	0	0	0	0	0	
DCP16	0	0	0	0	0	0	
DCP17	0	0	0	0	0	0	
DCP18	0	0	0	0	0	0	
DCP19	0	0	0	0	0	0	

DCP20	0	0	0	0	0	0	

USSHER POLYCLINIC

sample	Serial dilutions						
	10^{-1}		10^{-2}		10^{-3}		
	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	
UP21	0	0	0	0	0	0	
UP22	0	0	0	0	0	0	
UP23	0	0	0	0	0	0	
UP24	0	0	0	0	0	0	
UP25	0	0	0	0	0	0	
UP26	0	0	0	0	0	0	
UP27	0	0	0	0	0	0	
UP28	0	0	0	0	0	0	
UP29	0	0	0	0	0	0	
UP30	0	0	0	0	0	0	
UP31	0	0	0	0	0	0	
UP32	0	0	0	0	0	0	
UP33	0	0	0	0	0	0	
UP34	0	0	0	0	0	0	
UP35	0	0	0	0	0	0	

PRINCESS MARIE CHILDREN'S HOSPITAL

sample	Serial dilutions						
	10^{-1}		10^{-2}		10^{-3}		CFU/g
	Plate1	Plate2	Plate1	Plate2	Plate1	Plate2	
PM36	0	0	0	0	0	0	

PM37	0	0	0	0	0	0	
PM38	0	0	0	0	0	0	
PM39	0	0	0	0	0	0	
PM40	0	0	0	0	0	0	
PM41	0	0	0	0	0	0	
PM42	0	0	0	0	0	0	
PM43	0	0	0	0	0	0	
PM44	0	0	0	0	0	0	
PM45	0	0	0	0	0	0	
PM46	0	0	0	0	0	0	
PM47	0	0	0	0	0	0	
PM48	16	15	1	0	0	0	
PM49	0	0	0	0	0	0	
PM50	0	0	0	0	0	0	
PM51	132	150	14	13	2	2	
PM52	0	0	0	0	0	0	
PM53	0	0	0	0	0	0	
PM54	0	0	0	0	0	0	
PM55	0	0	0	0	0	0	

KANESHIE POLYCLINIC

sample	**Serial dilutions**						
	10^{-1}		**10^{-2}**		**10^{-3}**		
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**	

KP 56	0	0	0	0	0	0	
KP 57	0	0	0	0	0	0	
KP 58	0	0	0	0	0	0	
KP 59	10	12	2	2	0	0	
KP 60	0	0	0	0	0	0	
KP 61	0	0	0	0	0	0	
KP 62	0	0	0	0	0	0	
KP 63	0	0	0	0	0	0	
KP 64	0	0	0	0	0	0	
KP 65	0	0	0	0	0	0	

MAMPROBI POLYCLINIC

sample	**Serial dilutions**					
	10^{-1}		**10^{-2}**		**10^{-3}**	
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**
MP 66	0	0	0	0	0	0
MP 67	0	0	0	0	0	0
MP 68	0	0	0	0	0	0
MP 69	0	0	0	0	0	0
MP 70	0	0	0	0	0	0
MP 71	0	0	0	0	0	0
MP 72	0	0	0	0	0	0
MP 73	0	0	0	0	0	0
MP 74	0	0	0	0	0	0
MP 75	0	0	0	0	0	0

LA POLYCLINIC

sample	**Serial dilutions**

	10^{-1}		10^{-2}		10^{-3}	
	Plate1	**Plate2**	**Plate1**	**Plate2**	**Plate1**	**Plate2**
LP 76	0	0	0	0	0	0
LP 77	0	0	0	0	0	0
LP 78	0	0	0	0	0	0
LP 79	0	0	0	0	0	0
LP 80	0	0	0	0	0	0
LP 81	0	0	0	0	0	0
LP 82	0	0	0	0	0	0
LP 83	0	0	0	0	0	0
LP 84	0	0	0	0	0	0
LP 85	0	0	0	0	0	0
LP 86	0	0	0	0	0	0
LP 87	0	0	0	0	0	0
LP 88	0	0	0	0	0	0
LP 89	0	0	0	0	0	0
LP 90	0	0	0	0	0	0

RESULTS FOR SALMONELLA DETECTION

DANSOMAN POLYCLINIC

sample	**Growth on selected plates**	
	XLD	**BSA**
DCP1	**Red colonies with black centered**	**Brown metallic sheen colonies**
DCP2	No growth	No growth
DCP3	No growth	No growth
DCP4	No growth	No growth
DCP5	No growth	No growth
DCP6	No growth	No growth
DCP7	No growth	No growth
DCP8	No growth	No growth
DCP9	No growth	No growth
DCP10	No growth	No growth
DCP11	No growth	No growth
DCP12	No growth	No growth

DCP13	No growth	No growth
DCP14	No growth	No growth
DCP15	**Red colonies with black centered observed**	**Black metallic sheen colonies observed**
DCP16	No growth	No growth
DCP17	No growth	No growth
DCP18	No growth	No growth
DCP19	No growth	No growth
DCP20	No growth	No growth

USSHER POLYCLINIC

sample	**Growth on selected plates**	
	XLD	**BSA**
UP 21	No growth	No growth
UP 22	No growth	No growth
UP 23	No growth	No growth
UP 24	No growth	No growth
UP 25	**Red colonies with black centered**	**Black metallic sheen colonies**
UP 26	No growth	No growth
UP 27	No growth	No growth
UP 28	No growth	No growth
UP 29	No growth	No growth
UP 30	No growth	No growth
UP 31	No growth	No growth
UP 32	**Red colonies with black centered**	**Brown metallic sheen colonies**
UP 33	No growth	No growth
UP 34	No growth	No growth
UP 35	No growth	No growth

PRINCESS MARIE CHILDRENS HOSPITAL

sample	**Growth on selected plates**	
	XLD	**BSA**
PM36	No growth	No growth
PM37	**Red colonies with black centered**	**Black metallic sheen colonies**

PM38	No growth	No growth
PM39	**Red colonies with black centered**	**Black metallic sheen colonies**
PM40	No growth	No growth
PM41	No growth	No growth
PM42	**Red colonies with black centered**	**Black metallic sheen colonies**
PM43	No growth	No growth
PM44	No growth	No growth
PM45	No growth	No growth
PM46	No growth	No growth
PM47	No growth	No growth
PM48	**Red colonies with black centered**	**Black metallic sheen colonies**
PM49	No growth	No growth
PM50	No growth	No growth
PM51	**Red colonies with black centered**	**Black metallic sheen colonies**
PM52	No growth	No growth
PM53	No growth	No growth
PM54	**Red colonies with black centered**	**Black metallic sheen colonies**
PM55	No growth	No growth

KANESHIE POLYCLINIC

sample	**Growth on selected plates**	
	XLD	**BSA**
KP 56	No growth	No growth
KP 57	**Red colonies with black centered**	**Black metallic sheen colonies**
KP 58	No growth	No growth
KP 59	**Red colonies with black centered**	**Black metallic sheen colonies**
KP 60	**Red colonies with black centered**	**Black metallic sheen colonies**

KP 61	No growth	No growth
KP 62	No growth	No growth
KP 63	**Red colonies with black centered**	**Black metallic sheen colonies**
KP 64	No growth	No growth
KP 65	No growth	No growth

MAMPROBI POLYCLINIC

sample	**Growth on selected plates**	
	XLD	**BSA**
MP 66	No growth	No growth
MP 67	No growth	No growth
MP 68	No growth	No growth
MP 69	No growth	No growth
MP 70	No growth	No growth
MP 71	No growth	No growth
MP 72	No growth	No growth
MP 73	No growth	No growth
MP 74	No growth	No growth
MP 75	No growth	No growth

LA POLYCLINIC

sample	**Growth on selected plates**	
	XLD	**BSA**
LP76	No growth	No growth
LP77	No growth	No growth
LP78	No growth	No growth
LP79	No growth	No growth
LP80	No growth	No growth

LP81	Red colonies with black centered	Black metallic sheen colonies
LP82	No growth	No growth
LP83	No growth	No growth
LP84	No growth	No growth
LP85	No growth	No growth
LP86	No growth	No growth
LP87	No growth	No growth
LP88	No growth	No growth
LP89	No growth	No growth
LP90	No growth	No growth

calculations for S. aureus = $\frac{\sum C}{V\,(n_1 + 0.1n_2)\,d}$

Where d is the first dilution at which there was a count

C is the colonies counted on each plate.

n_1 is the number of plate used in the 1st dilution

. n_2 is the number of plate used in the 2nd dilution

V is the amount of volume used

Calculations for APC = $\frac{\sum C}{n \times 1.1 \times d}$

Where d is the first dilution at which there was a counts C is the average colonies counted.

n is the amount of sample inoculated.

NB; TNTC_ TOO NUMERIOUS TO COUNT

ANTIBIOTICS SUSCEPTIBILITY

For S.aureus

Sample no.	MINIMUM INHIBITORY CONCENTRATIONS OF ANTIBIOTICS USED (MM)				
	Cefuroxime 30ug/ml	Ceftriaxone 30ug/ml	Ciprofloxacin 5ug/ml	Augmentin 20ug/10ug/ml	C0-trimoxazole 23.75ug/1.25ug/ml
DCP2	0	0	20	16	18
DCP4	14	19	19	18	18
DCP12	0	0	19	17	15
DCP15	10	12	21	16	16
UP25	0	0	23	17	18
UP28	0	0	21	18	19
UP30	9	8	20	17	19
UP32	0	12	16	15	18
PM36	0	0	22	14	18
PM37	0	0	20	16	18
PM40	0	0	21	15	16
PM42	0	0	19	0	15
PM48	0	0	24	18	18
PM51	12	11	22	19	19
KP 57	9	13	21	10	15
KP 59	14	19	19	18	18
KP 60	12	14	20	17	16
KP 63	8	11	21`	17	19
MP 71	10	15	23	10	17
MP 73	0	0	19	17	15
LP 77	14	19	18	13	18
LP 81	0	0	21	17	18
LP 84	0	0	15	15	12

FOR SALMONELLA

Sample no.	MINIMUM INHIBITORY CONCENTRATIONS OF ANTIBIOTICS USED (MM)				

	Cefuroxime 30ug/ml	Ceftriaxone 30ug/ml	Ciprofloxacin 5ug/ml	Augmentin 20ug/10ug/ml	C0-trimoxazole 23.75ug/1.25ug/ml
DCP1	16	26	29	13	12
DCP15	13	27	26	14	12
UP25	11	24	26	11	10
UP32	0	25	30	12	14
PM37	0	28	32	12	13
PM39	7	21	24	0	8
PM48	6	30	32	11	8
PM54	0	25	29	11	11
KP 57	16	28	28	13	9
KP 59	0	25	31	13	14
KP 60	0	16	25	0	11
KP 63	0	17	26`	11	12
LP 81	7	22	30	13	9

FOR *E. COLI*

Sample no.	MINIMUM INHIBITORY CONCENTRATIONS OF ANTIBIOTICS USED (MM)				
	Cefuroxime 30ug/ml	Ceftriaxone 30ug/ml	Ciprofloxacin 5ug/ml	Augmentin 20ug/10ug/ml	C0-trimoxazole 23.75ug/1.25ug/ml
KP 59	13	19	32	16	30
PM51	12	18	34	15	35

Summary of results

Sample location	Total sample collected			
		Salmonella	*S.aureus*	*E, coli*
PM	20	4	6	1
DCP	20	2	4	0
MP	10	0	2	0
LP	15	4	4	1
UP	15	1	3	0
KP	10	2	4	0

TOTAL	**90**	**13**	**23**	**2**

ANTIBIOTICS SUSCEPTIBILITY

Antibiotics	salmonella	s.aureus	e.coli
Ciprofloxacin	9(69.2%)	21(91.3%)	2(100%)
Ceftriaxone	11(84.6%)	19(82.6%)	2(100%)
Augmentin	7(55.8%)	11(47.8%)	1(50%)
Co-trimoxazole	4(30.8%)	7(30.4%)	2(100%)
Cefuroxime	3(23.1%)	12(52.2%)	2(100%)

Table 4.1: Log CFU/g of microbes present in the food samples from Dansoman Polyclinic (DCP)

Code of Health Centre	Total viable count (TVC) Log cfu/g	*S. aureus* Log cfu/g	*Salmonella* detection 0/25g	*E. coli* detection 0 cfu/g	*B. cereus* Log cfu/g
DCP1	3.53	1	**detected**	Non detected	0
DCP2	3.28	2.54	Non detected	Non detected	0
DCP3	3.54	1	Non detected	Non detected	0
DCP4	1	2.32	Non detected	Non detected	0
DCP5	1	1	Non detected	Non detected	0
DCP6	2.41	1	Non detected	Non detected	0
DCP7	3.57	1	Non detected	Non detected	0

DCP8	1	1	Non detected	Non detected	0
DCP9	1	1	Non detected	Non detected	0
DCP10	1	1	Non detected	Non detected	0
DCP11	2.53	1	Non detected	Non detected	0
DCP12	3.70	3.18	Non detected	Non detected	0
DCP13	2.45	1	Non detected	Non detected	0
DCP14	3.90	1	Non detected	Non detected	0
DCP15	3.81	3.52	**detected**	Non detected	0
DCP16	3.52	1	Non detected	Non detected	0
DCP17	3.45	1	Non detected	Non detected	0
DCP18	2.36	1	Non detected	Non detected	0
DCP19	1	1	Non detected	Non detected	0
DCP 20	2.60	1	Non detected	Non detected	0

Table 4.2 Zone of Inhibition for pathogens Isolated DCP

Code for health centre	**NCENTRATION (MM)**									
	Cefuroxime 30ug/ml		Ceftriaxone 30ug/ml		Ciprofloxacin 5ug/ml		Augmentin 20ug/10ug/ml		Co-trimoxazole 23.75ug/1.25ug /ml	
	SA	*Sal*	*SA*	*Sal*	*SA*	*Sal*	*SA*	*Sal*	*SA*	*Sal*
DCP1	-	16	-	26	-	29	-	13	-	12
DCP2	0	-	0	-	20	-	16	-	18	-
DCP4	14	-	19	-	19	-	18	-	18	-
DCP12	0	-	0	-	19	-	17	-	15	-
DCP15	**10**	13	**12**	27	21	26	16	14	16	12

Table 4.3: Log CFU/g of microbes present in the food samples from Ussher Polyclinic (UP)

Code of Health Centre	Total viable count (TVC) Log cfu/g	*S. aureus* Log cfu/g	*Salmonella* detection 0/25g	*E. coli* detection 0 cfu/g	*B. cereus* Log cfu/g
UP21	2.90	1	Non detected	Non detected	0
UP22	1	1	Non detected	Non detected	0
UP23	1	1	Non detected	Non detected	0
UP24	1	1	Non detected	Non detected	0
UP25	4.04	3.08	**detected**	Non detected	0
UP26	5.11	1	Non detected	Non detected	0
UP27	5.20	1	Non detected	Non detected	0
UP28	5.23	2.56	Non detected	Non detected	0
UP29	1	1	Non detected	Non detected	0
UP30	1	3.04	Non detected	Non detected	0
UP31	4.15	1	Non detected	Non detected	0
UP32	4.84	1	Non detected	Non detected	0
UP33	2.87	1	Non detected	Non detected	0
UP34	2.78	1	Non detected	Non detected	0
UP35	3.08	1	Non detected	Non detected	0

Table 4.4: Zone of Inhibition for pathogens Isolated from Ussher polyclinic (UP)

Code for health centre	**NCENTRATION (MM)** Cefuroxime 30ug/ml		Ceftriaxone 30ug/ml		Ciprofloxacin 5ug/ml		Augmentin 20ug/10ug/ml		Co-trimoxazole 23.75ug/1.25ug /ml	
	SA	*Sal*	*SA*	*Sal*	*SA*	*Sal*	*SA*	*Sal*	*SA*	*Sal*
UP25	0	11	0	24	23	26	17	11	18	10
UP28	0	-	0	-	21	-	18	-	19	-
UP30	9	-	8	-	20	-	17	-	19	-

Table 4.5: Log CFU/g of microbes present in the food samples from Princess Marie Children's Hospital (PMCH)

Code of Health Centre	Total viable count (TVC) Log cfu/g	*S. aureus* Log cfu/g	*Salmonella* detection 0/25g	*E. coli* detection 0 cfu/g	*B. cereus* Log cfu/g
PM36	3.80	1	Non detected	Non detected	0
PM37	2.65	4.32	detected	Non detected	0
PM38	4.11	1	Non detected	Non detected	0
PM39	3.18	1	detected	Non detected	0
PM40	5.04	2.08	Non detected	Non detected	0
PM41	5.26	1	Non detected	Non detected	0
PM42	3.96	3.28	Non detected	Non detected	0
PM43	4	1	Non detected	Non detected	0
PM44	3.15	1	Non detected	Non detected	0
PM45	3.08	1	Non detected	Non detected	0
PM46	5.08	1	Non detected	Non detected	0
PM47	2.66	1	Non detected	Non detected	0
PM48	4.23	1	detected	Non detected	0
PM49	4.18	3.18	Non detected	Non detected	0
PM50	4.26	1	Non detected	Non detected	0
PM51	3.18	3.15	Non detected	detected	0
PM52	4.83	1	Non detected	Non detected	0
PM53	5.23	1	Non detected	Non detected	0
PM54	3.99	1	detected	Non detected	0
PM55	5.04	1	Non detected	Non detected	0

Table 4.6 Zone of Inhibition for pathogens Isolated from PMCH

Code for health center	**(MM)**						**CENTRATIONS Ol**								
		Cefuroxime 30ug/ml		Ceftriaxone 30ug/ml			Ciprofloxacin 5ug/ml			Augmentin 20ug/10ug/ml			C0-trimoxazole 23.75ug/1.25ug/ml		
	SA	*Sal*	*E.C*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*
PM36	0	-	-	0	-	-	22	-	-	14	-	-	18	-	-
PM37	0	0	-	0	28	-	20	32	-	16	12	-	18	13	-
PM 39	-	7	-	-	21	-	-	24	-	-	0	-	-	8	-
PM40	0	-	-	0	-	-	21	-	-	15	-	-	16	-	-
PM42	0	-	-	0	-	-	19	-	-	0	-	-	15	-	-
PM48	0	6	-	0	30	-	24	32	-	18	11	-	18	8	-
PM51	12	-	12	11	-	18	22	-	34	19	-	15	19	-	35
PM 54	-	0	-	-	25	-	-	29	-	-	11	-	-	11	-

Table 4.7: Log CFU/g of microbes present in the food samples from Kaneshie polyclinic (KP)

Code of Health Centre	Total viable count (TVC) Log cfu/g	*S. aureus* Log cfu/g	*Salmonella* detection 0/25g	*E. coli* detection 0 cfu/g	*B. cereus* Log cfu/g
KP 56	5.58	1	Non detected	Non detected	0
KP 57	4.11	2.74	detected	Non detected	0

KP 58	4.30	1	Non detected	Non detected	0
KP 59	4.32	4.23	detected	Non detected	0
KP 60	2.63	2.30	Non detected	Non detected	0
KP 61	3.86	1	Non detected	Non detected	0
KP 62	2.04	1	Non detected	Non detected	0
KP 63	4.08	2.81	Non detected	Non detected	0
KP 64	2.11	1	Non detected	Non detected	0
KP 65	5.20	1	Non detected	Non detected	0

Table 4.8 Zone of Inhibition for pathogens Isolated from Kaneshie Polyclinic

Code for health center	**(MM)** Cefuroxime 30ug/ml		Ceftriaxone 30ug/ml		**CENTRATION USED** Ciprofloxacin 5ug/ml		Augmentin 20ug/10ug/ml		C0-trimoxazole 23.75ug/1.25ug/ml	
	S.A	*Sal*	*S.A*	*Sal*	*S.A*	*Sal*	*S.A*	*Sal*	*S.A*	*Sal*
KP 57	9	16	13	28	21	28	10	13	15	9
KP 59	14	0	19	25	19	31	18	13	18	14
KP 60	12	-	14	-	20	-	17	-	16	-
KP 63	8	-	11	-	21	-	17	-	19	-

Table 4.9: Log CFU/g of microbes present in the food samples from Mamprobi Polyclinic (MP)

Code of Health Centre	Total viable count (TVC) Log cfu/g	*S. aureus* Log cfu/g	*Salmonella* detection 0/25g	*E. coli* detection 0 cfu/g	*B. cereus* Log cfu/g
MP 66	3.71	1	Non detected	Non detected	0
MP 67	4.11	1	Non detected	Non detected	0

MP 68	2.04	1	Non detected	Non detected	0
MP 69	5.08	1	Non detected	Non detected	0
MP 70	4.18	1	Non detected	Non detected	0
MP 71	2.88	4.04	Non detected	Non detected	0
MP 72	1	1	Non detected	Non detected	0
MP 73	2.93	3.15	Non detected	Non detected	0
MP 74	2.83	1	Non detected	Non detected	0
MP 75	3.62	1	Non detected	Non detected	0

Table 4.10 Zone of Inhibition for pathogens Isolated from Mamprobi Polyclinic

Code for MINIMUM INHIBITORY CONCENTRATIONS OF ANTIBIOTICS USED health (MM) center	**Cefuroxime 30ug/ml**	**Ceftriaxone 30ug/ml**	**Ciprofloxacin 5ug/ml**	**Augmentin 20ug/10ug/ml**	**C0-trimoxazole 23.75ug/1.25ug/ml**
	S.aureus	***S.aureus***	***S.aureus***	***S.aureus***	***S.aureus***
MP 71	10	15	23	10	17
MP 73	0	0	19	17	15

Table 4.11: Log CFU/g of microbes present in the food samples from La Polyclinic (LP)

Code of Health Centre	Total viable count (TVC) Log cfu/g	*S. aureus* Log cfu/g	*Salmonella* detection 0/25g	*E. coli* detection 0 cfu/g	*B. cereus* Log cfu/g
LP 76	2.90	1	Non detected	Non detected	0
LP 77	1	3.79	detected	Non detected	0
LP 78	1	1	Non detected	Non detected	0
LP 79	1	2.23	detected	detected	0
LP 80	4.04	1	Non detected	Non detected	0
LP 81	5.11	3.20	detected	Non detected	0
LP 82	5.20	1	Non detected	Non detected	0
LP 83	5.23	1	Non detected	Non detected	0
LP 84	1	2.50	detected	Non detected	0
LP 85	1	1	Non detected	Non detected	0
LP 86	4.15	1	Non detected	Non detected	0
LP 87	4.84	1	Non detected	Non detected	0
LP 88	2.87	1	Non detected	Non detected	0
LP 89	2.78	1	Non detected	Non detected	0
LP 90	3.08	1	Non detected	Non detected	0

Table 4.12 Zone of Inhibition for pathogens Isolated from LA Polyclinic

MINIMUM INHIBITORY CONCENTRATIONS OF ANTIBIOTICS USED (MM)

Code for health center	Cefuroxime 20ug/10ug/ml			Ceftriaxone 30ug/ml			Ciprofloxacin 30ug/ml			Augmentin 5ug/ml			C0-trimoxazole 23.75ug/1.25ug/ml		
	SA	*Sal*	*EC*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*	*SA*	*Sal*	*EC*
LP 77	14	0	-	19	16	-	18	25	-	13	0	-	18	11	-
LP 81	0	7	-	0	22	-	21	30	-	17	13	-	18	9	-
LP 84	0	10	-	0	20	-	15	22	-	15	19	-	12	12	-
LP 79	15	0	13	20	17	19	24	26	32	18	11	16	19	12	30

Table 4.13 Summary of the pathogens present in the food samples collect

Sample location	Total sample collected	Microorganisms		
		Salmonella	*S.aureus*	*E, coli*
PM	20	4(20%)	6(30%)	1(5%)
DCP	20	2(10%)	4(20%)	0(0%)
MP	10	0(0%)	2(20%)	0(0%)
LP	15	4(26.7%)	4(26.7%)	1(6.7%)
UP	15	1(6.6%)	3(20%)	0(0%)
KP	10	2(20%)	4(40%)	0(0%)
TOTAL	**90**	**13(14.4%)**	**23(25.6%)**	**2(2.2%)**

REGULATORY LIMITS FOR MICROBIAL COUNTS ON INFANTS FOODS

Parameter	Regulatory Limit(cfu/g)	Log cfu/g
S. aureus	10_2	2
TVC	$5x10^3$	3.7
Salmonella	0/25	0
E. coli	0	0
B. cereus	10^2	2

Source: ICMSF, 2011