Mammary Dysbiosis
An Unwelcome Visitor During Lactation

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Mastitis can be an unwelcome and debilitating visitor to breastfeeding mothers. The mammary gland has its own microbiome that can be affected by reduced polymorphonuclear neutrophil recruitment during the first 3 months postpartum, as well as the receipt of antibiotics during the last trimester of pregnancy. This can leave the breast vulnerable to pathologic bacterial overgrowth. Mammary dysbiosis is a process whereby the population of potential pathogens increases at the expense of the normal mammary microbiota. Multiresistance to antibiotics plus tricky evasion techniques engaged in by bacterial agents can result in microbes that are elusive to antibiotic therapy. Therefore, new strategies are needed for the treatment of this threat to continued breastfeeding. Bacteriotherapy, targeting harmless bacteria to displace pathologic organisms, is an emerging therapeutic intervention that uses probiotics instead of antibiotics. Once more high-quality clinical trials of strain-specific probiotics have been conducted, bacteriotherapy may move into mainstream mastitis treatment.

Keywords: bacteriotherapy; probiotics; mammary dysbiosis; mastitis; subacute mastitis

The human body is host to trillions of bacteria that occupy various locations. The totality of microorganisms (such as bacteria, fungi, and viruses) that inhabit a particular environment, their genetic elements, and their environmental interactions is called the microbiome. Frequently seen in breastfeeding discussions are descriptions of the infant gut microbiome, especially how breast milk and infant formula ingestion result in differences in the microbiota that inhabit the gut (Madan et al., 2016). However, both breast milk and the breast tissue itself also have their own microbiome.

Traditionally, human milk was thought to be sterile, and clinicians often went to great lengths to assure that mothers washed their hands and disinfected their nipples before putting the baby to the breast. In 2003, studies began describing the presence of physiological microbiota or normal bacterial residents in human milk (Heikkilä & Saris, 2003; Martín et al., 2003). Early life is when the adult microbiome is established, with the development of the infant gut microbiome occurring along a well-choreographed path directed in large part by the bacteria present in breast milk. It has also been shown that 27.7% of infant gut bacteria are provided by breast milk, and 10.4% of infant gut bacteria are derived from areolar skin (Pannaraj et al., 2017).

Just as breast milk is not sterile, neither is breast tissue. The breast is primarily composed of glandular and fatty tissue. The microbiome of the breast is distinct from the skin and other body sites, with breast tissue housing a diverse community of bacteria. In a study that collected tissue samples from a variety of locations in the breast, it was shown that numerous taxa were present, both health-conferring and pathogenic bacteria (Urbaniak et al., 2014). Bacteria have been isolated in the ducts and lobules in both the lactating and nonlactating breast, but not yet in the fatty tissue (Urbaniak, Burton, & Reid, 2012). Researchers have proposed that the breast microbiome contributes to maintenance of healthy breast tissue by stimulating resident immune cells (Xuan et al., 2014), allowing the breasts' army of defenders to work together to protect the breast.

Where Do These Bacteria Come From?

There are several potential origins and mechanisms thought to be responsible for bacterial presence in the breast.

- Bacteria from the nipple and areola could enter the breast through the nipple pores.
- Bacteria from the infant's mouth could enter the breast during milk backflow. Milk that is not extracted by the infant has been seen on ultrasound to reverse course and flow back into the milk duct (Ramsay et al., 2006).
- Bacteria can enter the breast through a break in the nipple epithelium when a crack or other damage is present.

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- Bacterial translocation from the maternal gut to the breast. During pregnancy and lactation, it is believed that bacteria-carrying dendritic cells migrate out of the messenteric lymph nodes in the intestines and into the mammary glands (Urbaniak et al., 2012). This is often referred to as the entero-mammary pathway.

All these routes can contribute to the transport of bacteria to various locations within the breast. The complex ductal system in the breast favors the growth of *S. aureus* and *S. epidermidis*, common residents, but also major contributors to mastitis. Mastitis can be a debilitating intruder on lactation and occurs in up to one-third of breastfeeding women (Foxman, D'Arcy, Gillespie, Bobo, & Schwartz, 2002).

Who Are the Culprits in Acute and Subacute Mastitis?

Acute Mastitis

S. aureus is the main etiologic contributor to acute mastitis (Delgado et al., 2011). Under the right conditions, it can proliferate and produce toxins that result in a strong inflammation in the breast tissue. This leads to local intense symptoms of redness, heat, and pain. The toxins are rapidly absorbed into the bloodstream, leading to the systemic flu-like symptoms of fever, muscular pain, and general malaise.

Subacute Mastitis

Coagulase-negative staphylococci (*S. epidermidis*) and viridans streptococci are normal inhabitants in the breast and form thin biofilms that line the ducts, allow normal milk flow, and are swept along during the milk-ejection reflex. Under certain circumstances, overgrowth of these species can occur, leading to subacute or subclinical mastitis. These bacteria do not produce the toxins that are responsible for the debilitating flu-like symptoms of acute mastitis, and the symptoms are generally milder. However, these bacteria can form thick biofilms inside the ducts, inflaming the epithelium, and narrowing the duct so that milk has more difficulty passing through it. The increasing pressure on the inflamed epithelium is felt as a characteristic needle-like pain, breast cramps, and a burning feeling. These bacterial biofilms may even totally fill some ducts, blocking milk flow, and leading to breast engorgement (Fernández et al., 2014).

If left unchecked, milk production may be reduced, and continued discomfort could lead to premature weaning. These symptoms are frequently attributed to candidiasis and treated with antifungals rather than explored further to discover if the symptoms are actually a manifestation of subacute mastitis. Incorrect antifungal treatment may delay appropriate interventions or exacerbate subacute mastitis. Subacute mastitis is characterized by an elevated sodium/potassium ratio in breast milk and an increased concentration of interlukin-8 (an inflammatory marker; Tuaillon et al., 2017). In a study that collected 110 milk samples during the first month postpartum from healthy mothers with no signs or symptoms of mastitis, subacute mastitis (increased inflammatory factors, an elevated sodium/potassium ratio, and markers of an immune response to bacterial exposure) was seen in 23% of the women (Tuaillon et al., 2017). Subacute mastitis is seen more frequently than acute mastitis and may be a precursor to or the initial stage of inflammation that poses an increased risk of progression to acute or clinical mastitis.

Thus, *S. aureus* is suited to develop acute infections, while *S. epidermidis* is typically the villain in subacute and chronic or recurrent mastitis (Angelopoulou et al., 2018). When staph and strep are under stress, they form organized and densely populated collectives on epithelia called biofilms. These develop protective coats that resist antibiotics and the host's immune response, allowing rampant bacterial multiplication. Even though these types of bacteria normally reside in the breast in a state of mutual acceptance or tolerance, disturbance of the balanced state between nonpathologic and pathologic bacteria can tip the balance toward mastitis. The ability to cause an infection depends on the strain of the bacteria, how virulent it is, its resistance to antibiotics, its ability to form biofilms, and the presence of other mechanisms that allow it to evade the body's immune response.

Susceptibility to mastitis may also be related to several other contributors, such as the blood group of the mother and the corresponding types of human milk oligosaccharides in her milk. The balanced state of microbes in the breast can be significantly disrupted by intrapartum antibiotics given to the mother, such as during a cesarean delivery or for group B strep prevention. The dysbiosis caused by the antibiotics may result in the loss of lactobacilli and bifidobacteria with the corresponding overgrowth of mastitis-causing agents. Milk that has been depleted of lactobacilli and bifidobacteria, important players in programming the infant gut microbiome, is then delivered to the baby, resulting in alterations of the baby's gut microflora. Women who received antibiotics in the last trimester of pregnancy and peripartum have 25-fold risk of

TABLE 1. Effects of Mastitis on Milk Quantity and Quality	
Selected Factors Thought to be Associated With or Contribute to Mastitis	
Cracked nipples	Oversupply of breast milk
Plugged ducts	Use of nipple shields
Use of breast pumps	Engorgement
Pacifier and bottle use	Milk stasis
Nipple creams	Nipple bleb
Tight bras and clothing	Poor latch
History of mastitis in previous lactations	Fatigue (holidays)
Use of antibiotics and antifungals	Colonized infant

developing mastitis (Bergmann, Rodríguez, Salminen, & Szajewska, 2014).

Large amounts of lactose and oligosaccharides are present in breast milk. Both Staphylococcus and Streptococcus are efficient lactose/galactose utilizers. Mammary polymorphonuclear neutrophil recruitment is decreased in first 3 months postpartum so not enough of these leukocytes (immune cells) may be available for control of mastitis-causing bacteria during the early weeks of lactation (Bergmann et al., 2014). This is congruent with peak mastitis occurrence seen during the first 6 weeks postpartum (Foxman et al., 2002). While a number of other factors have been associated with mastitis or thought to be the cause (Cullinane et al., 2015; Table 1), dysbiosis caused by microbial factors (overgrowth of pathogenic bacteria, virulence, biofilm formation, antibiotic resistance), host factors (genetic susceptibility, Lewis antigens, human milk oligosaccharides, autoimmune thyroid disease), and medical factors (antibiotic use, cracked nipples) seems to be the underlying entity responsible for both acute and subacute mastitis (Fernández et al., 2014).

Effects of Mastitis on Milk Quantity and Quality

Inflammatory factors in the mastitic breast can change the metabolic activity of milk-producing cells with the resulting reduction in milk synthesis (Say et al., 2016). Edema of the interstitial tissues is facilitated by the opening of paracellular pathways due to protein leakage from blood and milk. This opening increases the sodium and chloride passage into the milk giving it a salty taste and decreases the potassium and lactose content of the milk. Milk stasis may contribute to the observation of white granules in the milk, which are formed from caseins hardened by salts. Fatty or fibrous-looking material can sometimes be seen in the milk (may look like milk clumps). Fat, carbohydrate, and energy levels have been shown to be significantly lower in the milk from a breast with mastitis (Say et al., 2016). These effects on the quantity and quality of milk may mean that mothers with acute mastitis receive a recommendation to breastfeed more frequently during the course of the mastitis. If the baby will not nurse from the affected breast because of the salty taste of the milk or reduced flow and volume, then that breast will need to be pumped to support ongoing milk production.

Therapeutic Interventions

Antibiotics

Antibiotic therapy (typically dicloxacillin, flucloxacillin, cephalexin, clindamycin) has been the traditional treatment for lactation-related mastitis. However, multidrug resistance to antibiotics and the ability to form biofilms as an evasion technique are hallmark abilities of *S. aureus* and *S. epidermidis*. These two skills explain why mastitis can be a recurrent or chronic infection. In a study looking at antibiotic resistance patterns of human mastitis pathogens, it was found that a remarkable percentage of Staphylococcus isolates (>90%) were resistant to at least one antibiotic (Marín, Arroyo, Espinosa-Martos, Fernández, & Rodríguez, 2017). It has been reported that 25% of mothers who abandon breastfeeding due to mastitis have already received antibiotics (cloxacillin, clindamycin, amoxicillin–clavulanic acid, and/or erythromycin) for 2–4 weeks without resolution (Jiménez et al., 2008).

As an alternative to antibiotic use, and in an effort to avoid therapy failure, disruption of the maternal and infant gut microbiome and dysbiosis in the breast microbiome, probiotic therapy has emerged as a new strategy for the treatment of mastitis. Certain bacteria isolated from human milk have been identified as safe and suitable for therapeutic use in the treatment of mastitis as they have the ability to inhibit bacteria such as *S. aureus* (Heikkilä & Saris, 2003).

Probiotics

Due to the escalating problem of antibiotic resistance, bacteriotherapy, using harmless bacteria to displace pathogenic microorganisms, is being increasingly utilized as a means of combatting bacterial infections. Discussed since the beginning of the 20th century, bacteriotherapy is being utilized for infections in numerous areas of the

body, including the breasts. It is important to note that bacteria are commonly referred to by their genus and species names, printed in italics. Strains are a genetic variant or subtype of a microorganism and are designated by strain numbers consisting of several capital letters (the lab where they were constructed) and serial numbering of the strain as a "bookkeeping" method for scientific accuracy and to avoid confusing one mutant with another. In a discussion of probiotics for the treatment of mastitis, only certain strains of a bacterium have been shown to possess specific properties that are effective against the malady.

In 2008, a small pilot trial of 20 women with mastitis, two lactobacilli strains isolated from breast milk were studied as a treatment for Staphylococcal mastitis (Jiménez et al., 2008). Ten mothers received *Lactobacillus salivarius* CECT5713 and *L. gasseri* CECT5714 (10 $\log_{10}$ CFU of each) for a 4-week period, while the other 10 mothers received a placebo. By day 14, clinical signs of mastitis were no longer observed in the probiotic group, while mastitis persisted in the control group. In a larger study of 352 women with mastitis, one group received *Lactobacillus fermentum* CECT5716, one group received *L. salivarius* CECT5713, and a third group received antibiotic therapy (Arroyo et al., 2010). At the end of the 21-day trial, mothers in both probiotic groups had either recovered completely or experienced only slight breast discomfort compared to the antibiotic group where intense pain or breast discomfort persisted for the entire course of antibiotics. The rate of recurrence of mastitis was significantly higher in the antibiotic group. Seeking to discover if varying doses of probiotics would differentially lower the load of *Staphylococcus* in breast milk of women with subacute mastitis and reduce pain levels, a study evaluated the effect of three different escalating doses of *L. fermentum* CECT5716 (Maldonado-Lobón et al., 2015). All three doses lowered *Staphylococcus* levels in breast milk and significantly reduced pain by 30%. A dose–response effect was not seen.

In an effort to prevent mastitis by the prophylactic use of probiotics, two studies explored using probiotics either during pregnancy or for 16 weeks postpartum. In a study of 108 pregnant women who had experienced mastitis after at least one previous pregnancy, one group was given daily oral capsules of *L. salivarius* PS2 from 30 weeks of pregnancy until birth, and the control group received a placebo from 30 weeks of pregnancy until birth (Fernández et al., 2016). Of the 108 women, 41% developed mastitis, 25% in the probiotic group and 57% in the control group, a 56% decrease in mastitis incidence. When mastitis occurred, the milk bacterial counts in the probiotic group were significantly lower than those in the placebo group. In a study of 291 women from birth until 16 weeks, one group received the probiotic *L. fermentum* CECT5716, and one group received a placebo (Hurtado et al., 2017). In the probiotic group, 16 women developed mastitis, while 30 women in the control group did. This represents a 51% reduction in the incidence rate of clinical mastitis in the group taking probiotics. Specific strains of probiotics seem to be demonstrating themselves as an efficient strategy to prevent mastitis in breastfeeding mothers.

Bacteriocins

Bacteriocins are antimicrobial peptides (short amino acid chains) produced by one bacterium that are active against other bacteria, either in the same species (narrow spectrum) or across genera (broad spectrum). They exhibit potent activity against other bacteria, including antibiotic-resistant strains. Many bacteriocins are produced by lactic acid bacteria. Lantibiotics are a group of bacteriocins, one of which is nisin.

Nisin is produced by certain strains of *Lactococcus lactis*, a common species found in breast milk of healthy women. One use of nisin is as an antimicrobial in the food industry to prevent spoilage in food from pathogens and food spoilage microorganisms. It has also been used in the dairy industry to treat bovine mastitis. Nisin was investigated for use in treating infectious mastitis in breastfeeding mothers in a small 2-week study of women who had already received antibiotics for 2 to 4 weeks, but which failed to remedy the infection (Fernández, Delgado, Herrero, Maldonado, & Rodríguez, 2008). One group of mothers applied a topical solution of 0.1 mL nisin to their nipples and areolae after each feeding, while the control group applied a placebo solution. *Staphylococcal* counts in the nisin group were significantly lower after 2 weeks. Clinical symptoms were notably improved in the nisin group by day 7, with the complete disappearance of local inflammation and flu-like symptoms by day 14. In contrast, clinical signs remained persistent in the control group for the entire study period.

Topical Treatment for Inflammation

Markers for mastitis, such as breast pain, breast tension, and erythema (redness) are not uncommon in breastfeeding women and may represent the initial entrance into the mastitis continuum. Seeking alternative treatments to antibiotics, one study was conducted to determine the efficacy of topical curcumin (turmeric) in reducing breast inflammation, as curcumin is known

TABLE 2. Overview of Some Mastitis Treatment Options
Antibiotics (Amir & Academy of Breastfeeding Medicine Protocol Committee, 2014)
If conservative management does not improve symptoms within 24 hours or mother is acutely ill.
• Dicloxacillin (penicillinase-resistant penicillin) • Flucloxacillin (penicillinase-resistant penicillin) • Cephalexin (mother allergic to penicillin) • Clindamycin (mother with severe penicillin allergy) • Vancomycin or trimethoprim/sulfamethoxazole (if mastitis is not improving after 48 hours of treatment and methicillin-resistant *S. aureus* (MRSA) have been confirmed.
Probiotics
As an alternative or complement to antibiotic therapy; as a preventive measure; as first-line therapy for subacute mastitis; as conservative management if mastitis symptoms are mild and have been present for less than 24 hours.
• *L. salivarius PS2*—as a preventive measure, 9 $\log_{10}$ CFU daily from 30 weeks of pregnancy until delivery • *L. fermentum CECT5716*—as a preventive measure, 3 $\log_{10}$ CFU daily from birth to 16 weeks postpartum • *L. fermentum CECT5716*—as a first-line treatment of confirmed acute mastitis, 9 $\log_{10}$ CFU daily for 21 days • *L. salivarius CECT5713*—as a first-line treatment of confirmed acute mastitis, 9 $\log_{10}$ CFU daily for 21 days
Bacteriocins
As an alternative or complement to antibiotics
• *L. lactis ESI515* as the source for nisin—topical nisin solution (0.1 mL) applied to the nipple and areola after each breastfeeding for 14 days for treatment of acute mastitis
Topical Curcumin
For relief of inflammation and breast pain in subacute mastitis
• Curcumin—200 mg cream applied to the breast every 8 hours for 3 days

to have anti-inflammatory properties (Afshariani, Farhadi, Ghaffarpasand, & Roozbeh, 2014). A randomized, double-blind clinical trial was conducted with 70 breastfeeding mothers suffering from breast pain, breast tension, and erythema. The experimental group applied one pump of Curcumin Cream-200 mg (Neurobiologix, TX, USA) to the affected breast every 8 hours for 3 days. At the end of 72 hours of therapy, the curcumin group had significantly decreased markers of lactational mastitis such as pain, breast tension, and erythema compared to the control group.

Where Do We Go From Here?

Bacteriotherapy represents a promising addition to our toolbox of mastitis treatment options (Table 2).

While this is an exciting possibility, bacteriotherapy is still an emerging treatment option. *The best treatment option for mastitis is prevention by prompt and sound lactation management from skilled clinicians.* Mastitis can be thought of as a continuum from plugged ducts and engorgement to cracked nipples, subacute mastitis, and acute mastitis. Interruption of the continuum is a desirable goal to avoid the occurrence of mastitis altogether. Despite the need for more clinical trials (Amir, Griffin, Cullinane, & Garland, 2016), several of the studied probiotics have reached the market and are available for mothers to use. It is important to note that only the specific probiotic strains that have been shown to be effective are possibilities for mastitis treatment. A mother's healthcare team would need to evaluate if and how such therapy would be appropriate as well as if bacteriotherapy were administered exclusively or in conjunction with antibiotics. Several of the studied probiotics are commercially available:

- Lactanza *hereditum* (Angelini, Barcelona, Spain)
- Qiara (Puremedic, Kew, Victoria, Australia)
- Target b^2 (Klaire Labs, Reno, Nevada, USA)

Random probiotic supplements on store shelves that have not been evaluated as therapeutic agents for mastitis are not recommended for such use. Once more high-quality clinical trials of strain-specific probiotics have been conducted, bacteriotherapy may move into mainstream mastitis treatment, a most welcome event.

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Special Section on World Health Assembly Resolution

As reported by *The New York Times*_(https://www.nytimes.com/2018/07/08/health/world-health-breastfeeding-ecuador-trump.html), this spring, the U.S. Delegation to the World Health Assembly undermined a global resolution aimed at supporting breastfeeding_(http://apps.who.int/gb/ebwha/pdf_files/WHA71/A71_ACONF4Rev1-en.pdf), threatening trade sanctions against Ecuador before yielding to a proposal put forth by Russia Breastfeeding organizations and advocates across the country have raised concerns about the role of industry in international policy and the aggressive tactics of the U.S. delegation. See the *Weekly Wire* for a list of member and partner statements: http://www.usbreastfeeding.org/p/bl/et/blogid=1&blogaid=2080

Source: USBC