

# **Understanding how Women with Low Milk Supply Experience Breastfeeding**

BY

ERIN FARAH

BSN, University of Illinois at Chicago, Chicago, IL 2000

MS, University of Illinois at Chicago, Chicago, IL 2005

THESIS

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Defense Committee:

Carrie Klima, Chair and Advisor, Women, Children and Family Health Science

Janet Engstrom, Rush University

Patricia Hershberger, Health Systems Science

Barbara McFarland, Women, Children and Family Health Science

Beverly Rossman, Rush University

This thesis is dedicated to my husband, my children, and my family who have provided the support and time needed to accomplish this endeavor.

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## LIST OF ABBREVIATIONS

DLII	Delayed Lactogenesis II
DM	Diabetes Mellitus
FLII	Failed Lactogenesis II
GDM	Gestation Diabetes Mellitus
HCP	Health Care Provider
IGT	Insufficient Glandular Tissue
IRB	Institutional Review Board
PCOS	Polycystic Ovarian Syndrome
SNS	Supplemental Nursing System
T3	Triiodothyronine
T4	Thyroxine
TSH	Thyroid Stimulating Hormone
UIC	University of Illinois at Chicago

## SUMMARY

Lactation failure affects as many as 1 out of 20 women potentially impacting 365 million mother-infant dyads per year worldwide. The consequences of lactation failure can range from infant hunger and failure to thrive to life-threatening or even fatal dehydration and starvation. The causes of lactation failure are categorized as preglandular, glandular, and postglandular. Unfortunately, despite adequate maternal motivation, knowledge, support, and breastfeeding technique women will still experience lactation failure. Therefore, it is important to understand the experiences of these mother-baby dyads in order to support their breastfeeding experiences.

This hermeneutic phenomenological research study using van Manen's methodology was designed to understand the lived experience women have when breastfeeding with low milk supply. Interviews were conducted with 11 participants who self-reported having breastfed with low milk supply and met the inclusion criteria.

Six themes were identified: Loss of an Expectation; The Emotional Aftermath; Failure of my Body; Searching for Answers; The Hamster Wheel; and It's Not All or Nothing.

## I. LACTATION FAILURE: STATE OF THE SCIENCE

### **Abstract**

It is estimated that as many as 1 in 20 women worldwide are unable to successfully lactate and provide adequate nutrition for their infants through their breast milk alone. This resultant failure of lactation puts the infant at risk for insufficient growth as well as for serious and potentially disabling and life-threatening complications. The purpose of this review is to summarize the known risks associated with lactation failure that can preclude successful lactation despite adequate maternal motivation, knowledge, support, and breastfeeding technique. Although there is no clear way to predict who will experience lactation failure, this knowledge better enables healthcare providers to identify the known primary causes of lactation failure which may help prevent early failure to thrive in the infant.

### **Keywords**

lactation failure, lactogenesis, low milk supply

#### **A. Background**

The number of women in the United States who initiate breastfeeding has been reported at an all-time high of 79%.<sup>1</sup> This can be attributed to improvements in hospital policies and practices brought forth by the Baby-Friendly Hospital Initiative such as rooming-in, skin-to-skin contact immediately after birth, early initiation of breastfeeding, and the training of all health care staff in basic breastfeeding management and support.<sup>2</sup> In general, women are being encouraged to breastfeed primarily due to the health benefits to both mother and child. The health benefits for the child attributable to breastfeeding include improved development of the infant's immune and neurologic systems; decreased incidence or severity of infections including

otitis media, gastroenteritis, bacterial meningitis and upper respiratory infections; protection against sudden infant death syndrome, childhood leukemia and lymphoma, diabetes, obesity and asthma; and promoting socialization and bonding with mother.<sup>3-5</sup> Maternal benefits of breastfeeding include a reduction in risk of breast and ovarian cancers, lifetime development of type 2 diabetes mellitus, cardiovascular disease, postpartum depression, and rheumatoid arthritis.<sup>4,6</sup>

The American Academy of Pediatrics and the World Health Organization have been active in campaigning for increased rates of initiating breastfeeding after birth and encouraging exclusive breastfeeding for the first six months of life.<sup>3,7</sup> Additionally, both organizations recommend that after initiating nutritionally adequate and safe complementary feedings at six months of age, mothers should continue to breastfeed until one year of age and beyond as long as mutually desired by mother and child.<sup>3,7</sup>

Even though breastfeeding has been promoted as a convenient and natural method of infant feeding, there is an entire subset of women for whom this is not true. Women have been misinformed that *all* women can breastfeed<sup>8</sup> when in fact, the literature suggests that despite adequate maternal motivation, knowledge, support, and breastfeeding technique, as many as 1 out of 20 women experience lactation failure.<sup>9,10</sup> The aim of this review is to bring to light the prevalence of lactation failure and its underlying causes in order for health care providers to be able to identify those mother-infant dyads at risk for lactation failure.

## **B. Methods**

Relevant published research was identified by performing a literature search utilizing the databases CINAHL, MEDLINE, and PubMed. A combination of the following search terms was

used: lactogenesis, breast milk, breastfeeding, failure, insufficiency, low milk supply, diabetes mellitus, obesity, polycystic ovarian syndrome, retained placenta, Sheehan's syndrome, theca lutein cysts, thyroid disease, augmentation, reduction, breast surgery, insufficient glandular tissue, hypoplasia, tubular breast, hypoplastic breast, cleft lip, cleft palate, tongue tie, lip tie, preterm birth, and smoking. The databases were searched for articles in the English language and there were no limits placed on publication year. As relevant articles were found, they were read in their entirety and the references were examined for other relevant publications.

### **C. Findings**

#### **1. Development of the Female Breast**

Mammary growth increases exponentially during puberty<sup>11-13</sup> due to the onset of ovulatory cycles and progesterone secretion during the luteal phase which influences progressive mammary development. The newly developed tissue does not regress to its former state after the completion of the cycle.<sup>11</sup> Therefore, a gradual accretion of epithelial tissue occurs with each successive cycle, until about the age of 35 years, forming the characteristic structure of the adult female breast.<sup>13,14</sup>

The breasts attain full development and maturation during pregnancy and lactation.<sup>15</sup> In order for the breast to mature into a milk-secreting organ, two distinct phases known as secretory differentiation and secretory activation must occur.<sup>16</sup> During secretory differentiation, the pregnant mother experiences mammary changes in response to the rising hormone levels of prolactin, serum placental lactogen, estrogen, progesterone, and growth hormones. These changes include nipple growth, expansion of the areolar diameter, increase in the size of the lobular-alveolar system, and proliferation of the ductal system.<sup>11,13,15</sup> Secretory activation is

triggered by the rapid decrease in circulating progesterone that accompanies delivery of the placenta and a corresponding rise in serum prolactin which results in the stimulation of milk synthesis in the breast.<sup>16</sup> This phase also requires the synergistic effects of insulin and cortisol.<sup>15,16</sup>

## **2. Lactogenesis**

Lactogenesis is the process by which the mammary glands secrete milk.<sup>13</sup> The alveoli units within the female breast are comprised of a cluster of secretory cells and surrounded by myoepithelial cells. The secretory cells are composed of a single layer of epithelial cells which are responsible for the secretion of milk into the alveolar lumina where the milk is stored. The milk is later released into the ductal system with the contractions of the myoepithelial cells causing the ejection of milk during the let-down reflex.<sup>13,15,16</sup>

There are two stages of lactogenesis. Lactogenesis I begins during the second trimester of pregnancy when the mammary gland begins to secrete a small amount of milk and lasts until about day two postpartum.<sup>15,16</sup> Lactogenesis II, the onset of copious milk secretion, is triggered in response to the rapid decline in progesterone levels following the delivery of the placenta. Lactogenesis II typically begins on the second or third postpartum day and lasts until day eight.<sup>15,16</sup> Mothers often feel a sense of breast fullness as the milk comes in during lactogenesis II. The maintenance of lactation, once it has been established, is known as galactopoiesis which begins on approximately day nine postpartum and is maintained by the infants demand.<sup>16</sup> Therefore, breast milk synthesis will decline without suckling and milk removal by the infant<sup>16</sup> or a breast pump.

### 3. Hormonal Regulation of Human Lactation

The mechanisms controlling lactation are complex and are regulated by the endocrine system. Successful lactation is dependent upon the endocrine system and its hormones to properly develop the breast during pregnancy, to stimulate the secretion of milk in the immediate postpartum period, to assist in the ejection of milk from the alveolar cells, and to maintain milk production during lactation.<sup>16,17</sup> The key hormones responsible for orchestrating the act of lactation include progesterone, estrogen, insulin, prolactin, thyroid hormones, and multiple growth factors.<sup>18</sup>

After a woman gives birth and the placenta is expelled, there is a withdrawal of progesterone.<sup>17</sup> The anterior pituitary is then triggered to secrete the hormone prolactin which initiates milk secretion. If the woman then breastfeeds her newborn or the breast is stimulated by a breast pump, prolactin continues to be secreted in response to the nipple stimulation and milk secretion is maintained. Milk is ejected from the alveolar cells when the surrounding myoepithelial cells are stimulated by oxytocin which is secreted by the posterior pituitary.<sup>13,17</sup> The elevated levels of prolactin and oxytocin work in conjunction with cortisol, thyroid-stimulating hormone (TSH), and prolactin-inhibiting factor via negative feedback to establish and maintain lactation.<sup>16</sup> Disruption in the interaction of the hormones of the endocrine system may result in delayed or even failed lactogenesis II.<sup>13</sup>

*a. Delayed Lactogenesis II.* Delayed lactogenesis II (DLII) is diagnosed when it takes longer than usual for copious milk production to set in.<sup>19,20</sup> The mother is eventually able to produce a full breast milk supply and exclusively breastfeed her infant; however, the onset of an adequate supply occurs beyond 72 hours postpartum.<sup>19-21</sup>

**b. Failed Lactogenesis II.** Failed lactogenesis II (FLII) is a condition where the mother is unable to achieve an adequate breast milk supply to exclusively breastfeed her infant.<sup>19</sup> It is identified when there is absence of postpartum breast engorgement and milk production even though the mother reports adequate breastfeeding and sufficient stimulation and drainage of the breasts.<sup>8</sup>

This may be due to primary causes, which are intrinsic in nature, whereby the mother is unable to achieve full lactation despite proper technique and sufficient milk removal; or secondary extrinsic causes which impede the ability of a mother with no known physiological risk factors for FLII to fully lactate. Every mother has a different maximal output; some mothers may be able to just make drops of milk and others may only need to supplement a few ounces a day. Regardless of an individual mother's maximal output, if she is unable to exclusively breastfeed, this is considered FLII or low milk supply.<sup>19</sup>

**c. Prevalence of FLII.** While the exact prevalence of FLII is unknown, it has been reported to occur in up to 5% of lactating women,<sup>9,10</sup> potentially impacting 365 million mother-infant dyads per year worldwide.<sup>22</sup> This statistic appears to be first cited by Dr. Spence in 1938 and has been quoted ever since.<sup>10</sup> Interestingly, Neifert et al<sup>23</sup> incidentally found the prevalence of FLII to be 15% in their sample of 319 healthy, primiparous women who were breastfeeding term, healthy newborns despite intensive intervention.<sup>23</sup> There are multiple factors that may contribute to FLII, and the information is limited regarding the ability to predict which factor has a stronger impact on lactation failure than another.

#### 4. Causes of Failed Lactogenesis II

There are both maternal and infant causes of FLII which can be organized into 3 categories as identified by Morton.<sup>24</sup> These categories are known as preglandular, glandular, and postglandular. Preglandular causes involve a disruption in the endocrine system leading to a hormonal imbalance resulting in FLII.<sup>24,25</sup> Glandular causes of FLII involve an anatomical lack of the tissue necessary to create a milk supply sufficient enough to exclusively breast feed.<sup>24,25</sup> This lack of tissue can be the result of inadequate mammary gland development<sup>12</sup> or breast surgery.<sup>26-28</sup> Postglandular causes of FLII involve any infant factors that would lead to ineffective or inadequate emptying of the breast,<sup>24,25</sup> maternal consumption of medications that are known to inhibit milk synthesis,<sup>19</sup> preterm birth,<sup>29-31</sup> and maternal smoking.<sup>19,32</sup> (See Table 1).

##### *a. Preglandular Causes*

**i. Diabetes Mellitus (DM).** DM is a metabolic disease characterized by insulin resistance and impaired insulin production where the affected person has elevated levels of blood glucose circulating in their body.<sup>33</sup> It is known that prior history of DM or newly diagnosed gestational diabetes mellitus (GDM) is a risk factor for FLII or DLII.<sup>9,19,25,34-38</sup> Although the literature suggests that there is an increased risk for both FLII and DLII, there is only evidence for DLII. De Bortoli and Amir<sup>34</sup> conducted a systematic review while Hartmann and Cregan<sup>36</sup> published a case report both highlighting conditions of hyperinsulinemia in which women experienced DLII. Although statistical findings were not reported, both articles concluded that there is an association between diabetes in pregnancy<sup>34</sup> or pre-existing diabetes<sup>36</sup> and DLII. Matias et al<sup>37</sup> performed an observational study on secondary data from the SWIFT project

conducted at Kaiser Permanente from 2008-2011 which enrolled women with GDM who planned to breastfeed (N=1035). Inclusion was limited to those women whose diabetes resolved by 6-9 weeks postpartum and who had information on the desired outcome variable ‘onset of lactation based on maternal perception’ (N=883). They reported that 33% of study participants reported DLII. Additionally, they found that the need for insulin to treat GDM created a greater than three-fold risk for DLII (OR 3.11, 95%CI: 1.37-7.05).<sup>37</sup>

One theory regarding the connection between diabetes and either FLII or DLII is that insulin resistance and the resulting hyperinsulinemia of women with current DM or history of DM promotes hyperandrogenism leading to increased levels of circulating testosterone which inhibits normal lactation.<sup>21,39-41</sup> Elevated levels of testosterone inhibit prolactin and oxytocin secretion from the pituitary gland therefore impairing normal lactogenesis II.<sup>42</sup> Another theory suggests that insulin may act directly on mammary gland cells and affect their ability to secrete milk.<sup>43</sup> Mammary cells need to remain insulin sensitive to develop and work synergistically with the other hormones that control lactation.<sup>43</sup> Therefore, in the presence of any condition that creates insulin resistance, normal lactation may be inhibited.

**ii. Maternal Obesity.** The literature suggests that maternal pre-pregnancy body mass index classified as overweight or obese are risk factors for either FLII or DLII.<sup>19-21,35-37,44-51</sup> Although we do not yet understand the exact mechanism of how obesity may cause FLII or DLII the literature suggests that it could be due to hormonal interference during puberty which inhibits proper mammary gland development affecting future lactation<sup>52</sup>; or decreased prolactin levels in response to a suckling infant in the first 48 hours of life<sup>46</sup>; and/or increased insulin resistance and hyperinsulinemia same effect as in women with DM.

**iii. Polycystic Ovarian Syndrome (PCOS).** PCOS is a hormonal disorder of androgen excess which affects women of reproductive age. Elevated levels of circulating androgens, especially testosterone, may interfere with prolactin receptors within the mammary tissue affecting normal breast tissue growth and milk synthesis.<sup>39</sup> The literature suggests that both FLII and DLII have been observed in women with PCOS who otherwise have good breastfeeding technique, including latch and early frequent feeding.<sup>19,21,39</sup>

**iv. Retained Placenta.** Retained placenta is a condition where some, or all, of the placenta is left within the uterus after delivery. If fragments of the placenta remain within the uterus, the placental lactogenic hormones will continue to block mammary prolactin receptors and the preservation of elevated circulating levels of progesterone will interfere with the onset of lactogenesis II.<sup>53-55</sup> This may cause either a DLII<sup>53,56</sup> or FLII<sup>19,57</sup>.

**v. Sheehan's Syndrome.** Sheehan's syndrome occurs as a result of severe postpartum hemorrhage, hypovolemic shock and damage to the pituitary gland which impedes prolactin secretion.<sup>58</sup> Various publications have noted the negative effects Sheehan's syndrome has on lactogenesis II<sup>9,19,57,59</sup>; however, only 2 case reports<sup>60,61</sup> document the effects of this syndrome on lactation.

**vi. Theca Lutein Cysts.** Theca lutein cysts may develop on the ovary in response to excessive levels of beta-human chorionic gonadotropins in pregnancies compromised by hydatidiform moles, choriocarcinomas, fetal hydrops, diabetes, and multiple gestations or isoimmunized pregnancies.<sup>41</sup> Theca lutein cysts produce high levels of testosterone which may lead to either FLII or DLII.<sup>21</sup> During the postpartum period these cysts tend to resolve

spontaneously which causes the circulating levels of testosterone to return to normal levels.<sup>41</sup> Two case studies<sup>21,41</sup> have been published documenting both FLII and DLII.

**vii. Thyroid Dysfunction.** The thyroid produces the hormones thyroxine (T4) and triiodothyronine (T3) in response to TSH which is secreted by the anterior pituitary. Although the physiological role of T3 and T4 during lactogenesis are not clear,<sup>62</sup> it is known that effective functioning of prolactin and oxytocin are dependent upon normal thyroid function during lactation.<sup>16</sup> Motil et al<sup>63</sup> performed a prospective study with 12 lactating women to understand the role of various hormones in lactation. They found that both T4 ( $p < .05$ ,  $r = 0.57$ ) and T3 ( $p < .05$ ,  $r = 0.61$ ) had significant positive associations with the amount of milk produced. Overall, the literature regarding the relationship between thyroid dysfunction and FLII or DLII is scarce<sup>13,64</sup>; however, it has been suggested that thyroid dysfunction has led to both FLII and DLII.<sup>19,21</sup> Additionally, it is believed that hypothyroidism rather than hyperthyroidism is more frequently associated with FLII or DLII.<sup>64</sup>

#### ***b. Glandular Causes***

**i. Insufficient Glandular Tissue (IGT).** IGT is a condition in which women lack the glandular tissue within the breast for milk production and storage.<sup>65</sup> The majority of mothers diagnosed with IGT and low milk supply will not be able to exclusively breastfeed their infant despite heroic efforts to increase milk supply.<sup>65</sup> IGT can be diagnosed antenatally when a clinician observes abnormal development of at least one breast, breast asymmetry, tubular breast shape, intra-mammary distance greater than 1.5 inches, high mammary fold, disproportionately large or bulbous areolae, or the mother reports absence of typical breast changes that occur during pregnancy.<sup>65,66</sup>

Currently the literature on IGT is limited to documented case reports and one prospective descriptive study. The case reports provide documentation of women who have experienced FLII related to IGT.<sup>8,67-70</sup> A prospective descriptive study by Huggins and Petok<sup>65</sup> identified women with IGT antenatally who planned to breastfeed and followed them until 6 weeks postpartum. Of the 31 participants who completed the study, they found that 1 woman experienced normal lactogenesis II; 11 women experienced DLII and had a full milk supply by week 6 postpartum; and 19 women were found to have chronic low milk supply and were subsequently diagnosed with FLII.<sup>65</sup>

**ii. Breast Surgery.** Breast surgery can cause destruction of breast tissue; interruption of the ducts, nerve supply, or blood supply to the glandular tissue; or damage to the nipple subsequently leading to either FLII or DLII.<sup>58,71-75</sup> Breast surgery includes excisional biopsy, augmentation, reduction, and/or chest surgery involving the breast.<sup>23</sup> Researchers have found that women with previous breast surgery have significantly greater incidence of FLII compared to women with no prior breast surgery.<sup>19,23,26-28</sup> Additionally, it has been noted that women who have had breast reduction surgery may have a higher incidence of FLII due to the disruption of the ductal system compared to women who have breast augmentation; this is due to the fact that the areola and nipple need to be surgically removed and reattached symmetrically.<sup>58</sup>

**c. Postglandular**

**i. Infant Factors.** FLII can occur because of an ineffective or weak suck by the infant caused by functional anomalies such as cleft lip/palate, tongue-tie, or lip-tie.<sup>19,76</sup> Prematurity or anatomical anomalies in the infant such as congenital heart defects can also contribute to the infant having an ineffective suck.<sup>19</sup> Any condition that results in incomplete

emptying of the breast such as improper latch or breastfeeding mismanagement can lead to FLII.<sup>19</sup> Overall, there is very little research regarding the infant causes of FLII; however, the connection between ineffective milk removal and decreased production is well supported.<sup>13,16</sup>

**ii. Maternal Medication.** Use of various medications have been known to cause FLII.<sup>11,19</sup> These medications include pseudoephedrine, androgens, estrogens, dopaminergic agents, anticholinergics, diuretics, antihistamines, and hormonal birth control. Additionally, the herb sage has an antisudorific effect and is said to reduce lactation; and therefore, the entire sage family should be avoided during lactation.<sup>11,19</sup>

**iii. Preterm Birth.** Preterm birth, defined as birth when less than 37 weeks of gestation have been completed, presents challenges to lactation due to the fact that the breast may not be fully developed.<sup>29</sup> The secretory differentiation stage of mammary development may be interrupted due to preterm birth which means the mammary epithelium may not be adequately prepared to produce milk proficiently leading to either FLII or DLII.<sup>29</sup> Literature on management strategies for effectively optimizing milk production in mothers of preterm infants stress the importance of early initiation of milk removal and frequency of milk removal with a breast pump.<sup>29-31</sup>

**iv. Smoking.** Smoking is a known cause of lactation failure.<sup>19,32</sup> Nicotine consumption interferes with the production of prolactin, therefore decreasing the potential volume of milk that can be made in the lactating breast.<sup>32</sup> Additionally, nicotine can interfere with the let-down reflex and not allow milk to flow freely to the suckling infant.<sup>11</sup>

A quantitative descriptive study was published which identified 10 breastfeeding mothers who smoked at least four or more cigarettes per day, and case matched them to 10 breastfeeding

non-smoking mothers.<sup>32</sup> The mothers were matched by maternal age, pre-pregnancy weight and height, absence of morbidity, and no use of medication. Upon enrollment in the study, the babies were within one to three months of age, birth weight of between 3,000-4,000 grams, exclusively breastfed, and free of morbidity. The authors recorded daily milk volumes measured by using the dose-to-mother deuterium-dilution method over 14 days. They found that the difference in maternal milk production between both groups was significant ( $t=5.21$ ,  $p < 0.0001$ ), with a measured average volume of maternal milk of  $961 \pm 120$  g/d for the group of nonsmoking mothers and  $693 \pm 110$  g/d for smoking mothers.<sup>32</sup>

## **5. Consequences of Failed Lactation**

The first week postpartum is critical for the establishment of lactation, and it is important to remember that any woman may encounter breastfeeding complications.<sup>35</sup> The consequences of breastfeeding with low milk supply include hyperbilirubinemia, infant hunger, slow weight gain, infant failure to thrive, hypernatremic dehydration, and life-threatening or even fatal dehydration and starvation.<sup>10,53-58</sup> Additional complications of hypernatremic dehydration include seizures, disseminated intravascular coagulopathy, vascular complications, renal failure, dural thromboses, massive intraventricular hemorrhage, brain damage, and death.<sup>9,77</sup>

Although the consequences of FLII have been well documented for the infant, we do not know what consequences exist for the mother. The inability of a mother to breastfeed her child may lead to significant stress, a longing for an experience she had assumed would happen, and a disconnect between her goals for her new infant and the realities of a difficult and insufficient lactation experience. Frequent healthcare visits for a baby who is not gaining weight, and who may be ill, coupled with the normal stressors of the early postpartum period may impact the

physical and mental health of the new mother and the overall health of the family. One can imagine that there may be psychological ramifications for the mother and research is needed to begin to understand this gap in knowledge.

#### **D. Areas for Future Research**

At this time, we do not have an accurate picture of the extent of the problem of FLII. The prevalence of FLII has been quoted at 5%<sup>9,10</sup> dating back to 1938. However, it is prudent to question the accuracy of this prevalence rate. The prevalence may be much higher based upon the increasing rates of obesity<sup>81</sup> and diabetes<sup>82</sup> both known risk factors for FLII. The existing literature regarding FLII depends on predominately case reports and rare prospective studies. How women and their families experience this phenomenon and its impact upon the mother-infant relationship is unknown. Similarly, the influence of FLII on future infant feeding decisions is not known. We do not know if medical consequences exist for women related to lactation failure. We do not know if there are mental health effects subsequent to lactation failure and if there is an impact on maternal infant attachment and interactions. There are many unanswered questions, and I would argue that until we begin to understand this phenomenon mothers and babies will be put at risk during a vulnerable time and will not receive optimal care to ensure a healthy beginning.

#### **E. Conclusion**

Not all women can breastfeed, and certain conditions place women at risk for lactation failure. Health care providers working with breastfeeding mothers with low milk supply need to be aware of these conditions so mothers at risk for lactation failure can be identified in the early postpartum period and followed closely as they initiate breastfeeding. As more mothers are

encouraged to breastfeed, the incidence of lactation failure will undoubtedly increase. We must recognize that there is a broad range of providers who interact with the mother-infant dyad throughout the perinatal period and early infancy, and ensure that this information is disseminated across all professions. More research is needed to better understand this phenomenon from the perspective of women and their families, and to provide education and support to health care providers to improve recognition of the risks for lactation failure and management options when caring for this cohort of women.

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## F. References

1. Centers for Disease Control and Prevention. *Breastfeeding Report Card-United States, 2014*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 2014.
2. Perrine CG, Galuska DA, Dohack JL, et al. Vital Signs: Improvements in Maternity Care Policies and Practices That Support Breastfeeding - United States, 2007-2013. *MMWR Morb Mortal Wkly Rep*. 2015;64(39):1112-1117. doi:10.15585/mmwr.mm6439a5.
3. Eidelman A, Schanler R. Breastfeeding and the Use of Human Milk. *J Am Acad Pediatr*. 2012;129(3):e827-e841. doi:10.1542/peds.2011-3552.
4. Godfrey JR, Lawrence RA. Toward optimal health: the maternal benefits of breastfeeding. *J Womens Health (Larchmt)*. 2010;19(9):1597-1602. doi:10.1089/jwh.2010.2290.
5. Meyers D. Toward optimal health: maternal benefits of breastfeeding. [An interview with David Meyers by Jodi R. Godfry]. *J Womens Health (Larchmt)*. 2009;18(9):1307-1310. doi:10.1089/jwh.2009.1646.
6. Liu B, Jorm L, Banks E. Parity, breastfeeding, and the subsequent risk of maternal type 2 diabetes. *Diabetes Care*. 2010;33(6):1239-1241. doi:10.2337/dc10-0347.
7. World Health Organization. *Infant and Young Child Feeding*. Geneva, Switzerland: World Health Organization; 2009.  
<http://www.ncbi.nlm.nih.gov.proxy.cc.uic.edu/books/NBK148965/>. Accessed January 4, 2015.
8. Neifert MR, Seacat JM, Jobe WE. Lactation failure due to insufficient glandular development of the breast. *Pediatrics*. 1985;76(5):823-828.  
<http://www.ncbi.nlm.nih.gov/pubmed/4058994>. Accessed February 11, 2016.

9. Neifert M. Prevention of breastfeeding tragedies. *Pediatr Clin North Am.* 2001;48(2):273-297. doi:10.1016/S0031-3955(08)70026-9.
10. Spence JC. The modern decline of breast-feeding. *Br Med J.* 1938;2(4057):729-733. doi:10.1136/bmj.2.4057.729.
11. Lawrence R, Lawrence R. Anatomy of the breast. In: Lawrence R, Lawrence R, eds. *Breastfeeding: A Guide for the Medical Profession.* 8th ed. Philadelphia, PA: Elsevier; 2016:35-55.
12. Rudel RA, Fenton SE, Ackerman JM, Euling SY, Makris SL. Environmental exposures and mammary gland development: state of the science, public health implications, and research recommendations. *Environ Health Perspect.* 2011;119(8):1053-1061. doi:10.1289/ehp.1002864.
13. Wambach K, Watson Genna C. Anatomy and physiology of lactation. In: Wambach K, Riordan J, eds. *Breastfeeding and Human Lactation.* 5th ed. Burlington, MA: Jones & Bartlett Learning; 2016:79-120.
14. Kleinberg DL. Early mammary development: growth hormone and IGF-1. *J Mammary Gland Biol Neoplasia.* 1997;2(1):49-57. doi:10.1023/a:1026373513521.
15. Neville MC. Anatomy and physiology of lactation. *Pediatr Clin North Am.* 2001;48(1):13-34. doi:10.1016/S0031-3955(05)70283-2.
16. Lawrence R, Lawrence R. Physiology of lactation. In: Lawrence R, Lawrence R, eds. *Breastfeeding: A Guide for the Medical Profession.* 8th ed. Philadelphia, PA: Elsevier; 2016:56-90.
17. Tucker HA. Endocrinology of lactation. *Semin Perinatol.* 1979;3(3):199-223. <http://www.ncbi.nlm.nih.gov/pubmed/230600>. Accessed February 24, 2016.

18. Buhimschi CS. Endocrinology of lactation. *Obstet Gynecol Clin North Am.* 2004;31(4):963-979, xii. doi:10.1016/j.ogc.2004.08.002.
19. Hurst NM. Recognizing and treating delayed or failed lactogenesis II. *J Midwifery Womens Health.* 2007;52(6):588-594. doi:10.1016/j.jmwh.2007.05.005.
20. Brownell E, Howard CR, Lawrence RA, Dozier AM. Delayed onset lactogenesis II predicts the cessation of any or exclusive breastfeeding. *J Pediatr.* 2012;161(4):608-614. doi:10.1016/j.jpeds.2012.03.035.
21. Betzold CM, Hoover KL, Snyder CL. Delayed lactogenesis II: a comparison of four cases. *J Midwifery Womens Health.* 2004;49(2):132-137. doi:10.1016/j.jmwh.2003.12.008.
22. Population Reference Bureau. *2015 World Population Data Sheet.* Washington, DC: Population Reference Bureau; 2015.
23. Neifert M, DeMarzo S, Seacat J, Young D, Leff M, Orleans M. The influence of breast surgery, breast appearance, and pregnancy-induced breast changes on lactation sufficiency as measured by infant weight gain. *Birth.* 1990;17(1):31-38. doi:10.1111/j.1523-536X.1990.tb00007.x.
24. Morton JA. The clinical usefulness of breast milk sodium in the assessment of lactogenesis. *Pediatrics.* 1994;93(5):802-806. [http://pediatrics.aappublications.org/content/93/5/802.abstract?ijkey=6a3a7f68fcaa395f1da83b6d7bd0f272133eff8c&keytype2=tf\\_ipsecsha](http://pediatrics.aappublications.org/content/93/5/802.abstract?ijkey=6a3a7f68fcaa395f1da83b6d7bd0f272133eff8c&keytype2=tf_ipsecsha). Accessed February 4, 2016.
25. Neville MC, Morton J. Physiology and endocrine changes underlying human lactogenesis II. *J Nutr.* 2001;131(11):3005S - 3008. <http://jn.nutrition.org/content/131/11/3005S.full>. Accessed January 26, 2016.

26. Hurst N. Breastfeeding after breast augmentation. *J Hum Lact.* 2003;19(1):70-71.  
<http://www.ncbi.nlm.nih.gov/pubmed/12587647>. Accessed February 17, 2016.
27. Michalopoulos K. The effects of breast augmentation surgery on future ability to lactate. *Breast J.* 2007;13(1):62-67. doi:10.1111/j.1524-4741.2006.00364.x.
28. Nommsen-Rivers L. Cosmetic breast surgery--is breastfeeding at risk? *J Hum Lact.* 2003;19(1):7-8. <http://www.ncbi.nlm.nih.gov/pubmed/12587637>. Accessed February 17, 2016.
29. Geddes D, Hartmann P, Jones E. Preterm birth: strategies for establishing adequate milk production and successful lactation. *Semin Fetal Neonatal Med.* April 2013.  
doi:10.1016/j.siny.2013.04.001.
30. Jones E. Initiating and establishing lactation in the mother of a preterm infant. *J Neonatal Nurs.* 2009;15:56-59. doi:10.1016/j.jnn.2008.11.004.
31. Meier PP, Furman LM, Degenhardt M. Increased lactation risk for late preterm infants and mothers: evidence and management strategies to protect breastfeeding. *J Midwifery Womens Health.* 2007;52(6):579-587. doi:10.1016/j.jmwh.2007.08.003.
32. Vio F, Salazar G, Infante C. Smoking during pregnancy and lactation and its effects on breast-milk volume. *Am J Clin Nutr.* 1991;54(6):1011-1016.  
<http://www.ncbi.nlm.nih.gov/pubmed/1957815>. Accessed February 17, 2016.
33. White B, Porterfield S. Energy metabolism. In: White B, Porterfield S, eds. *Endocrine and Reproductive Physiology*. 4th ed. Philadelphia, PA: Elsevier/Mosby; 2013:43-75.
34. De Bortoli J, Amir LH. Is onset of lactation delayed in women with diabetes in pregnancy? A systematic review. *Diabet Med.* 2016;33(1):17-24. doi:10.1111/dme.12846.

35. Dewey KG, Nommsen-Rivers LA, Heinig MJ, Cohen RJ. Risk factors for suboptimal infant breastfeeding behavior, delayed onset of lactation, and excess neonatal weight loss. *Pediatrics*. 2003;112(3 Pt 1):607-619. doi:10.1542/peds.112.3.607.
36. Hartmann P, Cregan M. Lactogenesis and the effects of insulin-dependent diabetes mellitus and prematurity. *J Nutr*. 2001;131(11):3016S - 3020. <http://jn.nutrition.org.proxy.cc.uic.edu/content/131/11/3016S.long>. Accessed February 17, 2016.
37. Matias SL, Dewey KG, Quesenberry CP, Gunderson EP. Maternal prepregnancy obesity and insulin treatment during pregnancy are independently associated with delayed lactogenesis in women with recent gestational diabetes mellitus. *Am J Clin Nutr*. 2014;99(1):115-121. doi:10.3945/ajcn.113.073049.
38. Neville MC, Keller R, Seacat J, et al. Studies in human lactation: milk volumes in lactating women during the onset of lactation and full lactation. *Am J Clin Nutr*. 1988;48(6):1375-1386. <http://www.ncbi.nlm.nih.gov/pubmed/3202087>. Accessed April 16, 2016.
39. Marasco L, Marmet C, Shell E. Polycystic ovary syndrome: a connection to insufficient milk supply? *J Hum Lact*. 2000;16(2):143-148. doi:10.1177/089033440001600211.
40. Nestler JE. Insulin regulation of human ovarian androgens. *Hum Reprod*. 1997;12 Suppl 1:53-62. doi:10.1093/humrep/12.suppl\_1.53.
41. Hoover KL, Barbalinardo LH, Platia MP. Delayed lactogenesis II secondary to gestational ovarian theca lutein cysts in two normal singleton pregnancies. *J Hum Lact*. 2002;18(3):264-268. doi:10.1177/089033440201800309.

42. Kochenour NK. Lactation suppression. *Clin Obstet Gynecol*. 1980;23(4):1045-1059. doi:10.1097/00003081-198012000-00008.
43. Berlato C, Doppler W. Selective response to insulin versus insulin-like growth factor-I and -II and up-regulation of insulin receptor splice variant B in the differentiated mouse mammary epithelium. *Endocrinology*. 2009;150(6):2924-2933. doi:10.1210/en.2008-0668.
44. Chapman DJ, Pérez-Escamilla R. Identification of risk factors for delayed onset of lactation. *J Am Diet Assoc*. 1999;99(4):450-454; quiz 455-456. doi:10.1016/S0002-8223(99)00109-1.
45. Rasmussen KM, Hilson JA, Kjolhede CL. Obesity may impair lactogenesis II. *J Nutr*. 2001;131(11):3009S - 3011. <http://jn.nutrition.org.proxy.cc.uic.edu/content/131/11/3009S.long>. Accessed February 17, 2016.
46. Rasmussen KM, Kjolhede CL. Prepregnant overweight and obesity diminish the prolactin response to suckling in the first week postpartum. *Pediatrics*. 2004;113(5):e465-e471. doi:10.1542/peds.113.5.e465.
47. Lepe M, Bacardí Gascón M, Castañeda-González LM, Pérez Morales ME, Jiménez Cruz A. Effect of maternal obesity on lactation: systematic review. *Nutr Hosp*. 2011;26(6):1266-1269. doi:10.1590/S0212-16112011000600012.
48. Scott JA, Binns CW, Oddy WH. Predictors of delayed onset of lactation. *Matern Child Nutr*. 2007;3(3):186-193. doi:10.1111/j.1740-8709.2007.00096.x.
49. Jevitt C, Hernandez I, Groër M. Lactation complicated by overweight and obesity: supporting the mother and newborn. *J Midwifery Womens Health*. 2007;52(6):606-613. doi:10.1016/j.jmwh.2007.04.006.

50. Chapman DJ. Risk factors for delayed lactogenesis among women with gestational diabetes mellitus. *J Hum Lact*. 2014;30(2):134-135. doi:10.1177/0890334414525566.
51. Hilson JA, Rasmussen KM, Kjolhede CL. High prepregnant body mass index is associated with poor lactation outcomes among white, rural women independent of psychosocial and demographic correlates. *J Hum Lact*. 2004;20(1):18-29. doi:10.1177/0890334403261345.
52. Rasmussen KM. Association of maternal obesity before conception with poor lactation performance. *Annu Rev Nutr*. 2007;27:103-121. doi:10.1146/annurev.nutr.27.061406.093738.
53. Anderson AM. Disruption of lactogenesis by retained placental fragments. *J Hum Lact*. 2001;17(2):142-144. doi:10.1177/089033440101700210.
54. Kent JC. How breastfeeding works. *J Midwifery Womens Health*. 2007;52(6):564-570. doi:10.1016/j.jmwh.2007.04.007.
55. Neville MC, Morton J, Umemura S. Lactogenesis: the transition from pregnancy to lactation. *Pediatr Clin North Am*. 2001;48(1):35-52. doi:10.1016/s0031-3955(05)70284-4.
56. Neifert MR, McDonough SL, Neville MC. Failure of lactogenesis associated with placental retention. *Am J Obstet Gynecol*. 1981;140(4):477-478.  
<http://www.ncbi.nlm.nih.gov/pubmed/7246673>. Accessed February 17, 2016.
57. Livingstone VH, Willis CE, Abdel-Wareth LO, Thiessen P, Lockitch G. Neonatal hypernatremic dehydration associated with breast-feeding malnutrition: a retrospective survey. *CMAJ*. 2000;162(5):647-652.  
<http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1231219&tool=pmcentrez&rendertype=abstract>. Accessed February 22, 2016.

58. Lawrence R, Lawrence R. Medical complications of mothers. In: Lawrence R, Lawrence R, eds. *Breastfeeding: A Guide for the Medical Profession*. 8th ed. Philadelphia, PA: Elsevier; 2016:563-632.
59. Kilicli F, Dokmetas HS, Acibucu F. Sheehan's syndrome. *Gynecol Endocrinol*. 2013;29(4):292-295. doi:10.3109/09513590.2012.752454.
60. Willis CE, Livingstone V. Infant Insufficient Milk Syndrome Associated with Maternal Postpartum Hemorrhage. *J Hum Lact*. 1995;11(2):123-126. doi:10.1177/089033449501100218.
61. Sert M, Tetiker T, Kirim S, Kocak M. Clinical report of 28 patients with sheehan's syndrome. *Endocr J*. 2003;50(3):297-301. doi:10.1507/endocrj.50.297.
62. Anguiano B, Rojas-Huidobro R, Delgado G, Aceves C. Has the mammary gland a protective mechanism against overexposure to triiodothyronine during the peripartum period? The prolactin pulse down-regulates mammary type I deiodinase responsiveness to norepinephrine. *J Endocrinol*. 2004;183(2):267-277. doi:10.1677/joe.1.05711.
63. Motil KJ, Thotathuchery M, Montandon CM, et al. Insulin, cortisol and thyroid hormones modulate maternal protein status and milk production and composition in humans. *J Nutr*. 1994;124(8):1248-1257. <http://www.ncbi.nlm.nih.gov/pubmed/8064373>. Accessed April 20, 2016.
64. Goldstein AL. New-onset graves' disease in the postpartum period. *J Midwifery Womens Health*. 2013;58(2):211-214. doi:10.1111/jmwh.12016.
65. Huggins, KE, Petok ES MO. Markers of lactation insufficiency: a study of 34 mothers. *Issues in Clinical Lactation*. <http://www.sonic.net/~mollyf/igt/>. Published 2000. Accessed February 17, 2016.

66. Cassar-Uhl D. Breastfeeding with hypoplasia: Insufficient glandular tissue. *Breastfeed Today*. 2013;(January-March):16-19.
67. Arbour MW, Kessler JL. Mammary hypoplasia: not every breast can produce sufficient milk. *J Midwifery Womens Health*. 2013;58(4):457-461. doi:10.1111/jmwh.12070.
68. Bodley V, Powers D. Patient with insufficient glandular tissue experiences milk supply increase attributed to progesterone treatment for luteal phase defect. *J Hum Lact*. 1999;15(4):339-343. doi:10.1177/089033449901500415.
69. Michell J. Lactation failure caused by lack of glandular development in the breast. *Breastfeed Rev*. 1997;5(1):27-28.
70. Thorley V. Breast hypoplasia and breastfeeding: a case history. *Breastfeed Rev*. 2005;13(2):13-16. <http://www.ncbi.nlm.nih.gov/pubmed/16127825>. Accessed February 17, 2016.
71. Cruz NI, Korchin L. Breastfeeding after augmentation mammoplasty with saline implants. *Ann Plast Surg*. 2010;64(5):530-533. doi:10.1097/SAP.0b013e3181c925e4.
72. Hill PD, Wilhelm PA, Aldag JC, Chatterton RT. Breast augmentation & lactation outcome: a case report. *MCN Am J Matern Child Nurs*. 2004;29(4):238-242. doi:10.1097/00005721-200407000-00008.
73. Hurst NM. Lactation after augmentation mammoplasty. *Obstet Gynecol*. 1996;87(1):30-34. doi:10.1016/0029-7844(95)00349-5.
74. Strom SS, Baldwin BJ, Sigurdson AJ, Schusterman MA. Cosmetic saline breast implants: a survey of satisfaction, breast-feeding experience, cancer screening, and health. *Plast Reconstr Surg*. 1997;100(6):1553-1557. doi:10.1097/00006534-199711000-00028.

75. Baldwin B, Strom S, Schusterman M. Cosmetic saline breast implants: study of complications in 415 patients. *Plast Surg Forum*. 1995;18:275.
76. Turner L, Jacobsen C, Humenczuk M, Singhal VK, Moore D, Bell H. The effects of lactation education and a prosthetic obturator appliance on feeding efficiency in infants with cleft lip and palate. *Cleft Palate Craniofac J*. 2001;38(5):519-524. doi:10.1597/1545-1569(2001)038<0519:TEOLEA>2.0.CO;2.
77. Alsina-Manrique L, Esteban M, Salvià D, et al. Severe hypernatremic dehydration secondary to undetected lactation failure: usefulness of sodium levels in breast milk. *Clin Pediatr (Phila)*. 2006;45(2):183-186. doi:10.1177/000992280604500211.
78. Boskabadi H, Maamouri G, Ebrahimi M, et al. Neonatal hypernatremia and dehydration in infants receiving inadequate breastfeeding. *Asia Pac J Clin Nutr*. 2010;19(3):301-307. <http://www.ncbi.nlm.nih.gov/pubmed/20805072>. Accessed February 22, 2016.
79. Lavagno C, Camozzi P, Renzi S, et al. Breastfeeding-associated hypernatremia: a systematic review of the literature. *J Hum Lact*. 2016;32(1):67-74. doi:10.1177/0890334415613079.
80. Rand SE, Kolberg A. Neonatal hypernatremic dehydration secondary to lactation failure. *J Am Board Fam Pract*. 2001;14(2):155-158. <http://www.ncbi.nlm.nih.gov/pubmed/11314925>. Accessed February 22, 2016.
81. World Health Organization. *Obesity and Overweight*. Geneva, Switzerland: World Health Organization; 2015.

82. Centers for Disease Control and Prevention. *Incidence of Diagnosed Diabetes per 1,000 Population Aged 18-79 Years, by Age, United States, 1980-2014*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Health Statistics; 2014.

G. Table 1. Causes of Lactation Failure as Categorized by Morton.<sup>24</sup>

Preglandular	Glandular	Postglandular
Diabetes Mellitus	Breast Surgery	Infant Factors
Maternal Obesity	Insufficient Glandular Tissue	-Cleft Lip/Palate
PCOS		-Ineffective Weak Suck
Retained Placenta		- Tongue/Lip Tie
Sheehan's Syndrome		Maternal Medication
Theca Lutein Cysts		Preterm Birth
Thyroid Dysfunction		Smoking

## II. IT'S NOT ALL OR NOTHING: UNDERSTANDING HOW WOMEN WITH LACTATION FAILURE EXPERIENCE BREASTFEEDING

### **Abstract**

**Background.** Although women are encouraged to breastfeed their newborn infants, approximately 5% of women are unable to achieve a sufficient volume of milk to adequately nourish their infants.

**Purpose.** To understand how women with low milk supply experience breastfeeding.

**Methods.** Using a phenomenological approach in this qualitative study, one-time in-depth interviews were conducted with 11 participants who were recruited by purposive sampling via a support social media group, "IGT and Low Milk Supply Support Group". The interviews were analyzed using van Manen's hermeneutic methodology to uncover themes among the interviews.

**Results.** Experiences of breastfeeding with low milk supply revealed six thematic categories: loss of an expectation; the emotional aftermath; failure of my body; searching for answers; the hamster wheel; and making it work.

**Conclusion.** The experiences of these mothers reflect the importance of acknowledging the frustration, disappointment, guilt, and self-blame that mothers may feel when confronted with a diagnosis of low milk supply and the importance of the healthcare provider's role in supporting and caring for the mother.

### **Keywords**

breastfeeding, low milk supply, lactation failure, phenomenology

#### **A. Background**

Breast milk has been widely accepted as the best feeding option for newborns, and its benefits to both mothers and babies are well documented. Ongoing breastfeeding promotion efforts have resulted in steadily rising national breastfeeding rates. In 2011, 79.2% of all women giving birth in the US initiated breastfeeding, representing the highest initiation rate in nearly 7 decades.<sup>1</sup>

These higher rates of breastfeeding are largely due to the implementation of breastfeeding education during the antenatal period.<sup>2</sup> Results from a survey on maternity practices in infant nutrition conducted from 2001-2013 by the Centers for Disease Control and Prevention found

that the majority of US hospitals provide prenatal breastfeeding education (range 91.1%-92.2%) and teach mothers breastfeeding techniques (range 87.8%-92.2%).<sup>3</sup> The Baby-friendly Hospital Initiative launched in 1991 by the World Health Organization and the United Nations International Children's Emergency Fund set out to implement practices that protect, promote and support breastfeeding.<sup>4</sup>

Although there is a progressive movement towards exclusive breastfeeding, it is estimated that between 5% of women<sup>5,6</sup> to 15% of women<sup>7</sup> may experience failed lactogenesis II (FLII). FLII is defined as a condition in which the mother is unable to achieve an adequate breast milk supply to exclusively breastfeed her infant.<sup>8</sup> Causes of FLII may be preglandular, glandular, or postglandular in nature.<sup>9</sup> Preglandular causes are hormonal in nature and are related to a disruption in the endocrine system resulting in FLII.<sup>9,10</sup> Glandular causes are related to an anatomical lack glandular tissue necessary to create a sufficient milk supply<sup>9,10</sup> which can be due to inadequate mammary gland development<sup>11</sup> or breast surgery.<sup>12-14</sup> Postglandular causes include any infant factors that leads to ineffective or inadequate emptying of the breast,<sup>9,10</sup> consumption of medications known to inhibit milk synthesis,<sup>8</sup> preterm birth,<sup>15-17</sup> and maternal smoking.<sup>8,18</sup>

To date no previously published research has focused on women's experiences of breastfeeding with low milk supply. The purpose of this study was to describe the lived experience women have when breastfeeding with low milk supply.

## **B. Methods**

## **1. Design**

A hermeneutic phenomenological research design was selected for this study using van Manen's methodology to guide the analysis.<sup>19</sup> The fundamental goal of phenomenology is to describe the meaning of phenomena from the lived experiences of research participants and to look for meanings embedded within the experience.<sup>19</sup>

## **2. Setting**

This study was conducted in Chicago, Illinois from December 2015 to March 2016. Participants took part in a one-time interview either in-person or using the video conference program GoToMeeting. Women within a 100-mile radius of the University of Illinois at Chicago (UIC) were offered a face-to-face interview in a setting of their choice. Those outside the 100-mile radius completed their interview via the video conference program.

## **3. Sampling Strategy**

Using purposive sampling, 10-15 participants were recruited from a social media platform support group known as "IGT and Low Milk Supply Support Group" after permission was obtained from the groups administrators. At the time of recruitment, there were approximately 5800 members worldwide. Inclusion criteria consisted of females with a biological child; at least 18 years old; able to speak, read and write the English language; residing within the United States; self-reported having breastfed with low milk supply after a full term (delivery after 37 weeks' gestation) singleton pregnancy. Only those who indicated that they could remember and articulate their breastfeeding experience(s) were included. The institutional review board (IRB) of UIC granted approval of this study, and all procedures were followed in accordance with the standards of the IRB.

#### **4. Data Collection**

Informed consent was obtained from each participant. Participants who lived outside the 100-mile radius of UIC were mailed 2 copies of the consent form and were asked to return one signed copy in the self-addressed stamped envelope provided. One-time in-depth interviews were conducted in an environment that facilitated confidentiality and privacy. The interviews consisted of open-ended questions that encouraged the participant to discuss their experience of breastfeeding with low milk supply. Interviews ranged in duration from 27-75 minutes. At the conclusion of each interview, demographic information was collected. The interviews were digitally recorded and transcribed verbatim. Transcripts were then uploaded to the program Dedoose for analysis.

#### **5. Data Analysis**

For this study, van Manen's<sup>19</sup> highlighting approach to data analysis was applied. Beginning with hermeneutic reflection, each interview was read and re-read in-depth and essential statements or phrases were highlighted and given a unique code using the qualitative data analysis tool Dedoose. A code book containing definitions of the codes was developed simultaneously. Coding of the data was a continuous and ongoing process which allowed for reflection of the experience of breastfeeding with low milk supply. Once all the data was complete, the codes were grouped based on shared meanings and the essential themes emerged. Collaborative analysis was then performed by the research team.

Rigor in this study was achieved through several means. A reflective journal and audit trail were kept to perceived biases that might affect data analysis and interpretation.<sup>20</sup>

Trustworthiness or credibility of the data was ensured through peer debriefing with members of

the research team and member checking. A brief descriptive summary of the themes was sent to all of the participants. No new data or clarifications emerged from this process.

## **C. Findings**

### **1. Description of Participants**

Sixteen women responded to the recruitment post on the social media platform where the support group “IGT and Low Milk Supply Support Group” is located. All 16 women were deemed eligible for inclusion based on the eligibility assessment form that was completed during our initial phone conversation. Fifteen of the women lived outside the 100-mile radius of UIC and were mailed 2 hard copies of the consent form; one for their personal records and one to sign and return in the self-addressed, stamped envelope. Thirteen women returned their signed consent form, however only 10 set up and completed the interview process via video conferencing; one participant completed an in-person interview. Participants were primarily married, college-educated, and had planned to breastfeed.

### **2. Themes**

Six themes were identified: Loss of an Expectation; The Emotional Aftermath; Failure of my Body; Searching for Answers; The Hamster Wheel; and It’s Not All or Nothing.

#### *a. Theme 1: Loss of an Expectation*

Participants described their decisional experiences during pregnancy in relation to feeding choices. They all expressed that they had made the decision to exclusively breastfeed their newborn prior to delivery and were looking forward to this experience. Participants indicated that the decision to breastfeed was made with a sense of pride, and multiple participants expressed that they wanted to do what was best for their baby:

“I wanted what was best for my baby and what was best for me. And I just did a lot of research; talked to other mothers and friends; and you know, read things and just decided that was the best thing for everyone involved. So, yeah, and I was really excited to do that, you know, for all reasons, to bond with my baby and to give him the best nutrients.”

When asked, the majority of the participants had no idea that it was even possible to have low milk supply; especially if they were continuously putting their baby to the breast and not introducing any supplements. One participant said,

“I just thought, you know, how cool is it that we as women can grow a person and then nourish them with our body. And I just thought that that was like the most powerful thing that we can do as women. And I was always really an advocate for us, and I, I believed in the process.”

Only two participants understood having low milk supply may be possible but felt it occurred only in extreme cases.

Each participant expressed shock and devastation upon learning that their baby had lost more than an acceptable amount of weight in the newborn stage or that their baby was not gaining weight but just maintaining their weight. One participant explained her experience:

“Oh, I was devastated. I cried. I probably, not kidding, I probably cried for a week straight, maybe even two. Anybody that mentioned breastfeeding, anybody that said anything about breastfeeding, if I saw somebody breastfeeding, I cried. It was like this flood of emotion that I wanted to do it so bad and I can't.”

One participant had successfully breastfed her first two children beyond their first year and was unable to make a full supply with her third child. While reflecting on her experience, she reported feeling paralyzed and being unable to comprehend exactly what was happening when on day 4 postpartum she learned that her baby had lost too much weight and needed to start supplementation at that very moment with formula.

Participants reported that they had prepared for the experience in different ways which included: reading literature or books about breastfeeding; speaking with their healthcare providers (HCPs), family, and friends about breastfeeding; or taking breastfeeding classes prior to delivery. Except for the two participants who acknowledged that it may be possible for a woman to not be able to exclusively breastfeed, all the other participants felt that breastfeeding was something that they just had to choose to do and had trust in the process. Upon learning that they were experiencing low milk supply and would have to initiate supplementation, they each experienced this loss of expectation for exclusive breastfeeding.

*b. Theme 2: The Emotional Aftermath*

The participants in the study expressed a multitude of emotions when describing how they felt after realizing they were going to be unable to fulfill their desire to exclusively breastfeed. Participants described their emotions using terms such as anger, denial, grief, self-blame, depression, resentment, disappointment, guilt, devastation, shame, and frustration during the immediate period following the realization that they were not producing a full milk supply and would need to start supplementation. However, each participant's experience was individual and unique in terms of the emotions that she reported feeling as well as the trajectory of her

personal emotional journey. Although the emotions that each participant described feeling varied, some commonalities emerged.

One participant very succinctly described her strong emotional reaction in the immediate period after discovering her low milk supply.

“I felt a lot of grief, and I felt like I was really mad at my body for not doing what it was supposed to do. I really was questioning like, did I do the right things or is this my fault for doing something wrong in the beginning? I was really like an emotional wreck for like a little while.”

In contrast, other participants articulated that there are many different emotions one might feel and acknowledged there is no right way to go through the process.

"It's okay to cry; it's okay to feel guilty; it's okay to internalize it. You know, it's -- you have a flood of emotions that are going to run through you, and you need to process all of those.”

*c. Theme 3: Failure of my Body*

The participants often referred to their body as being broken when sharing their experience with having low milk supply. There are certain functions that women expect their bodies to be able to perform, one being breastfeeding. When their breasts were not functioning the way they had anticipated, they each explained in their own way how they felt that their bodies had failed them. One participant articulated her experience of her broken body as:

“And it's also frustrating to feel like you are defective, and you don't know why. You don't know what happened. Was it something I did? Was there something

else I could have done? You are always questioning yourself and second guessing yourself.”

Participants struggled with the idea that their babies were doing what they needed to do but their bodies didn't respond the way they expected. They discussed the failure they felt at not being able to nurture their babies: “I felt like I was a bad cow”; and acknowledged the stigma of feeling like a failure: “I felt like I got an ‘F’ on a test, and I didn't want to tell anybody.”

*d. Theme 4: Searching for Answers*

Participants all experienced this phase where they were searching for the cause of their low milk supply. There was a tendency to question everything that transpired from birth to the moment when they realized that they had low milk supply. Every decision or action related to breastfeeding was put under the microscope. Related to this, participants expressed an intense need to understand ‘why’ to be able to process their experiences. Sometimes participants got caught in a loop of searching for answers, receiving false information that led to hope, and then more frustration, and ultimately more searching:

“And meanwhile, I have everyone I know said just do this. It will be fine. And I'm like, it's not working. And I don't know anyone who ever had this type of experience before. I mean, it always just worked for everybody. I wanted answers. Like why -- why is this happening to me? Is there something that had to do with the birth? Is it something to do with me?”

During the quest for answers, participants acknowledged that the online support group, from where they were recruited, provided reliable resources and information about low milk supply. Through the support group, they knew there were laboratory tests available to help

diagnose whether or not they had any of the contributing factors associated with low milk supply. Some participants reported that a barrier in their search for answers was their health care provider's unwillingness to explore possible causes, while others expressed relief if they were lucky enough to discover the cause of their low milk supply.

*e. Theme 5: The Hamster Wheel*

Participants expressed a sense of determination after discovering their low milk supply and a need to maximize their output. They discussed the many different modalities that they tried in order to attain the most breast milk possible. These modalities included: use of prescription medications, herbs, supplements, homeopathics, and tinctures; round-the-clock pumping; massage of their breasts and hand expressing milk; use of a supplemental nursing system (SNS) in order to keep the baby at the breast and optimize stimulation; and calling upon professional help from IBCLCs, pediatric providers, and the health care providers from their pregnancy.

There was a rhythm that was expressed by some of the participants that started with breastfeeding, supplemental feeding with either a bottle or SNS, and then pumping; this regimen would continue all day and night round the clock.

“Setting the alarm to wake up and pump, and taking fenugreek and drinking tons of water, and doing physical therapy and expressing into a bottle and eating cookies. And I was like I'm doing all of these things and nothing is working, and this is just pissing in the wind.”

Participants that were following these strict regimens felt that their every moment was consumed by feeding. There seemed to be no time for anything else which included enjoying their new

child and bonding. Ultimately some participants expressed remorse over the loss of time while working at trying to increase their breast milk supply, “I wish that I had all that time back that I could have just been with my baby.”

*f. Theme 6: It's Not All or Nothing*

Participants discussed accepting not being able to exclusively breastfeed their baby and focusing instead on maintaining personal balance while caring for their child. One said:

“It is mentally, physically, emotionally just the most hardest thing I have ever done. But at the end of the day, I kind of pat myself on the back because I have pushed through. So, with it being the hardest thing, it's probably one of the most rewarding things as well.”

Similarly, there was an agreement among the women that it is not all or nothing; that providing any amount of breast milk that they could was beneficial and that needing to supplement wasn't a reason to stop providing their own milk. There was also a sense of pride that quitting was not an option and they were persevering even with a low milk supply. For some mothers, just seeing any amount of their own milk was enough reason for them to keep going as it meant that their baby would be getting some of their nutrients.

“And it wasn't all or nothing, or less than a full supply isn't good enough or worth messing with.”

This participant seemed to sum up perfectly this final theme of “*It's Not All or Nothing*”:

“Every day that I nurse her, I feel thankful. Every time I look at her nursing, I feel thankful. I feel anger still that I failed. I feel annoyed that I have to go through all of these processes for an ounce or two of milk. So I still feel them all,

and they just aren't as strong as they were in the beginning. I don't cry about it anymore, and I found acceptance in where we are at now.”

#### **D. Discussion**

Mothers in this study experienced a profound sense of loss, failure, and desperation for answers after learning they had an insufficient volume of milk to exclusively breastfeed their child. This is the first study to clearly describe mothers' reflections upon the timeframe immediately following the moments when they learned of their low milk supply and their journey while breastfeeding.

The participants in the study were able to clearly articulate their experiences of breastfeeding with low milk supply. Each of them recounted their overwhelming feeling of a *loss of an expectation*. The women expressed that they had made a decision to breastfeed based on their understanding that breast milk was the healthiest option for their baby. The sense of loss occurred when they felt that the decision to exclusively breastfeed their baby was taken from them. Findings from this research show that reactions to the loss of an expectation are processed with individual coping styles and how women cope and feel from one day to the next may change due to the multitude of emotions experienced in the early days after learning of their low milk supply.

Despite making the conscious decision to exclusively breastfeed, women in this study found that their body could not perform this function, and some women expressed feeling broken and embarrassed. Breastfeeding is something only a woman can do, and is oftentimes considered synonymous with being a good mother.<sup>21</sup> Therefore, the inability to exclusively provide their own milk impacted how women in this study defined their role as a woman and

even as a mother. Equating breastfeeding with being a good mother puts an already marginalized group of women at further risk of peripartum complications such as postpartum depression.

Another key finding of this research was the participants' experiences navigating the health care system as they sought to get to the "why" of their low milk supply. The knowledge base of the HCPs whom the participants encountered seemed to vary widely in terms of diagnosing and treating low milk supply. In a study examining HCPs' attitudes and beliefs about breastfeeding, Radzyminski and Callister<sup>22</sup> found that HCPs' reported feeling ill-equipped to handle any issues that may come up during a breastfeeding session. Shakespeare et al<sup>23</sup> reported a similar finding stating that the participants described their interactions with HCPs as "unhelpful and highly charged". Although the participants in this study also discussed that some HCPs were dismissive and resistant when women asked about laboratory testing to search for the cause of low milk supply, others were willing to explore possible causes and provide accurate information about medicinal and herbal therapies.

This study provided the first in-depth exploration of the experiences of women who are breastfeeding with low milk supply. Health care professionals can use the results of this study to guide their care for women during this time. This study also provides insight into future research needs including: how and what information regarding low milk supply should be included in prenatal breastfeeding education; the discussion and implementation of support systems for women and their families who are dealing with low milk supply; additional education needed for health care providers regarding the diagnosis and treatment of low milk supply; and what resources are available.

### **E. Limitations of the Study**

The study participants were all recruited from an on-line support forum for women with low milk supply therefore, it is possible that these findings only represent the unique experiences of women who seek this specific form of support. Women who seek support and are participants on this forum represent a subset of women within the greater cohort of women with low milk supply. As with all qualitative research and this methodological approach, the findings are not generalizable to the larger population of women experiencing low milk supply.

### **F. Conclusion**

Ultimately, all participants in this study had to make compromises during their breastfeeding journey. Women should be reminded during this time that they can still provide their breast milk if they desire and supplement the needs of their baby with another safe option. It's also important for women to hear and learn about breast supplementation systems that can be used to keep their baby exclusively at their breast. Women may need to hear that every drop of their milk is also important. Even though they are not producing a full supply, they can still breastfeed.

“I just really hope that, you know, moving into the future that the medical community is more kind of cognizant of low milk supply issues. That they're more open and willing to help moms with low milk supply. That people are more educated about it. I didn't realize -- and realize how important it is and that it is a thing. And early intervention, like with anything, is the most important. We exist. I'm not a unicorn; I'm sitting here. And I'm a breathing person. I do exist.”

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## G. References

1. Centers for Disease Control and Prevention. *Breastfeeding Report Card-United States, 2014*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 2014.
2. Neifert M, Bunik M. Overcoming clinical barriers to exclusive breastfeeding. *Pediatr Clin North Amer*. 2013;60(1):115-145. doi:10.1016/j.pcl.2012.10.001.
3. Perrine CG, Galuska DA, Dohack JL, et al. Vital Signs: Improvements in Maternity Care Policies and Practices That Support Breastfeeding - United States, 2007-2013. *MMWR Morb Mortal Wkly Rep*. 2015;64(39):1112-1117. doi:10.15585/mmwr.mm6439a5.
4. World Health Organization and UNICEF. *Baby-Friendly Hospital Initiative*. Geneva, Switzerland: World Health Organization; 2009.  
[http://apps.who.int/iris/bitstream/10665/43593/1/9789241594967\\_eng.pdf](http://apps.who.int/iris/bitstream/10665/43593/1/9789241594967_eng.pdf).
5. Neifert M. Prevention of breastfeeding tragedies. *Pediatr Clin North Am*. 2001;48(2):273-297. doi:10.1016/S0031-3955(08)70026-9.
6. Spence JC. The modern decline of breast-feeding. *Br Med J*. 1938;2(4057):729-733. doi:10.1136/bmj.2.4057.729.
7. Neifert M, DeMarzo S, Seacat J, Young D, Leff M, Orleans M. The influence of breast surgery, breast appearance, and pregnancy-induced breast changes on lactation sufficiency as measured by infant weight gain. *Birth*. 1990;17(1):31-38. doi:10.1111/j.1523-536X.1990.tb00007.x.
8. Hurst NM. Recognizing and treating delayed or failed lactogenesis II. *J Midwifery Womens Health*. 2007;52(6):588-594. doi:10.1016/j.jmwh.2007.05.005.

9. Morton JA. The clinical usefulness of breast milk sodium in the assessment of lactogenesis. *Pediatrics*. 1994;93(5):802-806.  
[http://pediatrics.aappublications.org/content/93/5/802.abstract?ijkey=6a3a7f68fcaa395f1da83b6d7bd0f272133eff8c&keytype=tf\\_ipsecsha](http://pediatrics.aappublications.org/content/93/5/802.abstract?ijkey=6a3a7f68fcaa395f1da83b6d7bd0f272133eff8c&keytype=tf_ipsecsha). Accessed February 4, 2016.
10. Neville MC, Morton J. Physiology and endocrine changes underlying human lactogenesis II. *J Nutr*. 2001;131(11):3005S - 3008. <http://jn.nutrition.org/content/131/11/3005S.full>. Accessed January 26, 2016.
11. Rudel RA, Fenton SE, Ackerman JM, Euling SY, Makris SL. Environmental exposures and mammary gland development: state of the science, public health implications, and research recommendations. *Environ Health Perspect*. 2011;119(8):1053-1061.  
doi:10.1289/ehp.1002864.
12. Hurst N. Breastfeeding after breast augmentation. *J Hum Lact*. 2003;19(1):70-71.  
<http://www.ncbi.nlm.nih.gov/pubmed/12587647>. Accessed February 17, 2016.
13. Michalopoulos K. The effects of breast augmentation surgery on future ability to lactate. *Breast J*. 2007;13(1):62-67. doi:10.1111/j.1524-4741.2006.00364.x.
14. Nommsen-Rivers L. Cosmetic breast surgery--is breastfeeding at risk? *J Hum Lact*. 2003;19(1):7-8. <http://www.ncbi.nlm.nih.gov/pubmed/12587637>. Accessed February 17, 2016.
15. Geddes D, Hartmann P, Jones E. Preterm birth: strategies for establishing adequate milk production and successful lactation. *Semin Fetal Neonatal Med*. April 2013.  
doi:10.1016/j.siny.2013.04.001.
16. Jones E. Initiating and establishing lactation in the mother of a preterm infant. *J Neonatal*

- Nurs.* 2009;15:56-59. doi:10.1016/j.jnn.2008.11.004.
17. Meier PP, Furman LM, Degenhardt M. Increased lactation risk for late preterm infants and mothers: evidence and management strategies to protect breastfeeding. *J Midwifery Womens Health.* 2007;52(6):579-587. doi:10.1016/j.jmwh.2007.08.003.
  18. Vio F, Salazar G, Infante C. Smoking during pregnancy and lactation and its effects on breast-milk volume. *Am J Clin Nutr.* 1991;54(6):1011-1016.  
<http://www.ncbi.nlm.nih.gov/pubmed/1957815>. Accessed February 17, 2016.
  19. van Manen M. *Researching the Lived Experience: Human Science for an Action Sensitive Pedagogy.* 2nd ed. London, Ont: Althouse Press; 1997.
  20. Ortlipp M. Keeping and using reflective journals in the qualitative research process. *Qual Rep.* 2008;13(4):695-705. <http://www.nova.edu/ssss/QR/QR13-4/ortlipp.pdf>.
  21. Marshall JL, Godfrey M, Renfrew MJ. Being a “good mother”: managing breastfeeding and merging identities. *Soc Sci Med.* 2007;65(10):2147-2159.  
doi:10.1016/j.socscimed.2007.06.015.
  22. Radzyninski S, Callister LC. Health professionals’ attitudes and beliefs about breastfeeding. *J Perinat Educ.* 2015;24(2):102-109. doi:10.1891/1058-1243.24.2.102.
  23. Shakespeare J, Blake F, Garcia J. Breast-feeding difficulties experienced by women taking part in a qualitative interview study of postnatal depression. *Midwifery.* 2004;20(3):251-260. doi:10.1016/j.midw.2003.12.011.

## **APPENDICES**

## Appendix A

### Research Proposal

#### I. SPECIFIC AIMS

The purpose of this research project is to better understand the breastfeeding experiences of women who have low breast milk production. An understanding of women's lived experiences may lead to the development of interventions, geared towards clinicians, to improve the care of these women after diagnosis.

Breastfeeding is promoted as a convenient and natural method of infant feeding, and mothers are encouraged to breastfeed primarily due to the health benefits to both mother and child (Godfrey, 2009; Godfrey & Lawrence, 2010; Shaikh & Chantry, 2006). The health benefits for the child attributable to breastfeeding include improved development of the infant's immune and neurologic systems; decreased incidence or severity of infections including otitis media, gastroenteritis, bacterial meningitis and upper respiratory infections; protection against sudden infant death syndrome (SIDS), childhood leukemia and lymphoma, diabetes, obesity and asthma; and promoting socialization and bonding with mother (Eidelman & Schanler, 2012; Godfrey, 2009; Godfrey & Lawrence, 2010). Maternal benefits of breastfeeding include a reduction in risk of breast and ovarian cancers and lifetime development of type 2 diabetes mellitus (Godfrey & Lawrence, 2010; Liu, Jorm, & Banks, 2010). Infant formula, on the other hand, may increase a child's risk of cardiovascular disease, childhood obesity, gastrointestinal infections, mortality, ear infections, and side effects from environmental contaminants (INFACT Canada, 2002).

The American Academy of Pediatrics (AAP) and the World Health Organization (WHO) have been active in campaigning for increased rates of initiating breastfeeding after birth and encouraging exclusive breastfeeding for the first six months of life (Eidelman & Schanler, 2012; World Health Organization, 2009). Additionally, both organizations recommend that after initiating nutritionally adequate and safe complementary feedings at six months of age, mothers should continue to breastfeed until one year of age or beyond as long as mutually desired by mother and child (Eidelman & Schanler, 2012; World Health Organization, 2009).

Women, however, have been misinformed that *all* women can breastfeed (Neifert, Seacat, & Jobe, 1985). There are multiple conditions of both the mother and infant that may interfere with a woman's ability to produce an adequate milk supply. Maternal conditions may include: thyroid dysfunction, polycystic ovarian syndrome (PCOS), gestational ovarian theca lutein cysts, retained placenta after delivery, postpartum hemorrhage with Sheehan's syndrome, insufficient glandular tissue (IGT), diabetes mellitus (DM), obesity, preterm birth, smoking, history of breast surgery, and maternal medications (Anderson, 2001; Chapman, 2014; Hopkinson, Schanler, & Garza, 1988; Hurst, 1996; Hurst, 2007; Jevitt, Hernandez, & Groer, 2007; Marasco, Marmet, & Shell, 2000; Souto, Giugliani, Giugliani, & Schneider, 2003). Infant conditions may include: structural deformities (e.g., tongue ties, lip ties, cleft lip/palate), prematurity, and an ineffective or disorganized sucking reflex (Hopkinson et al., 1988; Lindberg & Berglund, 2014).

Low milk production, also known as failed lactogenesis II, is identified when there is absence of postpartum breast engorgement and milk production even though the mother reports adequate breastfeeding and sufficient stimulation and drainage of the breasts (Neifert et al.,

1985). Mothers, who unknowingly have low milk production and who are determined to exclusively breastfeed their infant, place their newborn at risk for medical complications (Neifert, 2001). Medical complications include infant failure to thrive, slow weight gain, hyperbilirubinemia, infant hunger, hypernatremic dehydration, and fatal dehydration and starvation. Additional complications of hypernatremic dehydration include seizures, disseminated intravascular coagulopathy, vascular complications, renal failure, dural thromboses, massive intraventricular hemorrhage, brain damage, and death (Alsina-Manrique et al., 2006; Neifert, 2001). Despite adequate maternal motivation, knowledge, support, and breastfeeding technique, it is speculated that the prevalence of women with low milk production affects as many as 1 out of 20 women (Neifert et al., 1990; Neifert, 2001).

To date there are no studies published regarding the lived experiences of breastfeeding women who have low milk supply. This lack of research represents a missed opportunity to hear the stories and insights of those whose interests are at stake. In this qualitative study, using hermeneutic phenomenology, 10-15 participants will be recruited by purposive sampling. Data collection will stop when theoretical saturation has been met. Participants will be at least 18 years of age and have experienced low milk supply after a full-term (delivery after 37 weeks gestation), singleton pregnancy. This phenomenological study will give a voice to the women who experience low milk supply when breastfeeding and will contribute to this gap in knowledge.

#### Specific Aims:

1. Describe the lived experiences of breastfeeding women who have low milk supply

## II. RESEARCH STRATEGY

### A. Significance

**Psychological impact of failed lactogenesis II.** The current literature does not provide a perspective on the experiences of women who breastfeed with low milk supply. Labbok (2008) looked at the physician's role in the prevention, diagnosis, and treatment of guilt in a mother when she does not breastfeed either by choice or imposed by lactation failure. This article suggests that feelings of guilt, loss, and anger may arise when a mother's expectations are not met (Labbok, 2008). Other than Labbok's 2008 article, there has been no other suggestion as to the psychological impact failed lactogenesis may have on a mother. Given that there is very little known about this topic, it is the short-term goal of this project to describe the lived experiences of women who breastfeed with low milk supply and to reveal, if any, the psychological impact of lactation failure.

**Development of the female breast.** The anatomical structures of the female mammary glands are developed during a process known as mammogenesis. The first stage of mammogenesis occurs during the embryogenic period starting at the fourth week of gestation when two primitive milk streaks grow from the axilla to the groin of the embryo. These milk streaks become the mammary ridge by the fifth week gestation and are comprised of a thickening of epithelial cells which continue to grow through weeks seven and eight (Lawrence & Lawrence, 2011). Growth then continues inward toward the chest wall and between 12 and 16 weeks gestation the epithelial cells begin to differentiate into the smooth muscle of the nipple and areola (Riordan & Wambach, 2010). During this same timeframe, the epithelial cells develop into mammary buds which proliferate to form epithelial branches and are later known as

alveoli (Riordan & Wambach, 2010). The ducts of the breast are also forming during this stage and continue to elongate and form a mammary sprout, which invades the fat pad, forming the rudimentary mammary ductal system present at birth. By the time the fetus is 18-19 weeks gestation, a bulb-shaped mammary bud can be seen on *ultrasonography*. *It is within this bud that the rudimentary mammary ductal system is formed. The placental sex hormones continue to stimulate the formation of the ductal system until the fetus is 32 weeks gestation* (Riordan & Wambach, 2010). *Then from 32 to 40 weeks gestation, the alveolar structures will create and store colostrum, and the fetal mammary gland mass increases four fold. The nipple and areola will also become pigmented, and at birth there are some neonates who will secrete their colostrum* (Neville, 2001; Riordan & Wambach, 2010).

The second stage of mammogenesis occurs during pubertal development. During this stage, estrogen and a pituitary factor, likely growth hormone, influence ductal and lobular growth which results in growth of the breast parenchyma with its surrounding fat pad. This process is also known as organogenesis and occurs in a girl between 10 and 12 years of age (Riordan & Wambach, 2010). With the onset of menses and ovulatory cycles, progesterone is secreted by the ovary during the luteal phase which causes further mammary development that does not regress to its former state after the completion of the cycle (Lawrence & Lawrence, 2011). Therefore, a gradual accretion of epithelial tissue occurs with each successive cycle until about the age of 35 years (Riordan & Wambach, 2010). It is these effects of estrogen and progesterone that facilitate the formation of the characteristic structure of the adult female breast (Kleinberg, 1997). However, full alveolar development and maturation of the epithelium requires the hormones of pregnancy (Neville, 2001).

This leads to the third stage of mammogenesis which occurs during pregnancy and is influenced by multiple hormones. During this stage, there are two distinct phases that occur. The first phase is called secretory differentiation which represents the mammary changes experienced by the pregnant mother (Lawrence & Lawrence, 2011). The nipple grows in response to rising serum prolactin levels and the areola increases in diameter in response to serum placental lactogen (Lawrence & Lawrence, 2011; Riordan & Wambach, 2010). The rising levels of estrogen cause the ductal system to proliferate, and progesterone promotes an increase in size of the lobular-alveolar system (Neville, 2001). Lastly, adrenocorticotrophic hormone and growth hormone combine with prolactin and progesterone to further promote mammary growth. The second phase, secretory activation, is triggered when the placenta is expelled and there is a withdrawal of progesterone causing the initiation of milk secretion (Lawrence & Lawrence, 2011). This phase also requires the synergistic effects of prolactin, insulin, and cortisol (Lawrence & Lawrence, 2011; Neville, 2001).

The change that occurs in the breast from pregnancy to lactation is called lactogenesis and is the process by which the mammary glands secrete milk (Riordan & Wambach, 2010). The structures of the female breast that are involved in lactogenesis are the alveoli units and the ductules (Neville, 2001). Each alveoli unit is comprised of a cluster of secretory cells and surrounded by myoepithelial cells. The secretory cells are composed of a single layer of epithelial cells which are responsible for the secretion of milk into the alveolar lumina where the milk is then stored. The milk is later released into the ductal system with the contractions of the myoepithelial cells causing the ejection of milk during the let-down reflex (Lawrence & Lawrence, 2011; Neville, 2001; Riordan & Wambach, 2010).

Lactogenesis occurs in two stages. Lactogenesis I begins during the second trimester of pregnancy when the mammary gland begins to secrete a small amount of milk, and lactose is present in the maternal blood and urine, which lasts until about day two postpartum (Black, Jarman, & Simpson, 1998; Lawrence & Lawrence, 2011; Neville, 2001). Lactogenesis II is initiated in the postpartum period by the rapid decline in progesterone levels that occur following delivery of the placenta. This process with begins on approximately day three postpartum and lasts until day eight postpartum is not dependent on suckling by the infant (Black et al., 1998; Lawrence & Lawrence, 2011; Neville, 2001). The onset of copious milk secretion associated with lactogenesis II is regulated by the endocrine system and should occur by the third or fourth postpartum day for a woman delivering a full-term infant (Lawrence & Lawrence, 2011). Galactopoiesis, or the maintenance of lactation once it has been established, is under the control of the autocrine system. This phase begins about day nine postpartum and is the maintenance of lactation once it has been established and will only be maintained by the infants demand (Lawrence & Lawrence, 2011). Therefore, breast milk synthesis will decline without suckling and milk removal by the infant (Black et al., 1998; Lawrence & Lawrence, 2011)

**Hormonal regulation of human lactation.** The mechanisms controlling lactation are complex and are regulated by the endocrine system. The endocrine system refers to the glands in the human body which secrete hormones. These glands include the pineal gland, pituitary gland, pancreas, ovaries (female), testes (male), thyroid gland, parathyroid gland, hypothalamus, and adrenal glands (White & Porterfield, 2013). The components necessary for successful lactation which are dependent upon the endocrine system include proper development of the breast during pregnancy, stimulation of secretion of milk in the immediate postpartum period, ejection of milk

from the alveolar cells, and maintenance of milk production during lactation (Lawrence & Lawrence, 2011; Tucker, 1979).

As the breast is prepared during pregnancy for lactation the local effects of estrogen and progesterone prevent milk from being secreted. After a woman gives birth and the placenta is expelled there is a withdrawal of the hormone progesterone (Tucker, 1979). The anterior pituitary is then triggered to secrete the hormone prolactin which initiates milk secretion. If the woman then breastfeeds her newborn, prolactin continues to be secreted in response to the nipple stimulation and milk secretion is maintained. Milk is ejected from the alveolar cells when the surrounding myoepithelial cells are stimulated by the hormone oxytocin which is secreted by the posterior pituitary (Riordan & Wambach, 2010; Tucker, 2000; Tucker, 1979). The elevated levels of prolactin and oxytocin work in conjunction with the hormones cortisol, thyroid-stimulating hormone (TSH), and prolactin-inhibiting factor via negative feedback to establish and maintain lactation (Hiller-Sturmhofel & Bartke, 1998; Riordan & Wambach, 2010).

In general, bodily functions such as lactation, are regulated by several hormones that regulate each other known as the negative feedback mechanism. This mechanism starts with a target gland which sends signals to the hypothalamus and/or pituitary glands depending on the hormone involved (Hiller-Sturmhofel & Bartke, 1998). The signals that are sent back to the hypothalamus and/or pituitary glands can either trigger the release of additional hormones needed or turn off the release of the hormone (Hiller-Sturmhofel & Bartke, 1998).

The hormones cortisol, thyroid-stimulating hormone (TSH), and prolactin-inhibiting factor are the other essential hormones in the maintenance of lactation (Riordan & Wambach, 2010). Cortisol is a glucocorticoid hormone secreted by the adrenal glands. The role of cortisol

in lactation is seen during the final differentiation of the alveolar epithelial cells when they become secretory cells. It is prolactin that initiates the cell differentiation within the alveoli cells only after the cells have come into contact with both cortisol and insulin (Riordan & Wambach, 2010). Thyroid-stimulating hormone (TSH), which is secreted by the anterior pituitary gland, has been shown to aid in the regulation of both prolactin and oxytocin. An imbalance in the thyroid hormones may impact the ability of prolactin and oxytocin to function effectively for normal lactation to occur (Lawrence & Lawrence, 2011). Prolactin-inhibiting factor controls the amount of prolactin that is secreted via the hypothalamus (Riordan & Wambach, 2010). Any disruption to how the hormones of the endocrine system interact with one another may cause delayed lactogenesis or even failed lactogenesis (Riordan & Wambach, 2010).

**Definition of failed lactogenesis II.** Failed lactogenesis II is a condition where the mother is unable to achieve an adequate breast milk supply in order to exclusively breastfeed her infant (Hurst, 2007). This can be due to primary causes, which are intrinsic in nature, whereby the mother is unable to achieve full lactation despite proper technique and sufficient milk removal; or secondary extrinsic causes which impede the ability of a mother with no known physiological risk factors for failed lactogenesis to fully lactate. It is important to note that when a woman experiences failed lactogenesis II, or low milk supply, she may decide to continue to breastfeed her baby in addition to using a supplement. Every mother has a different maximal output; some mothers may be able to just make drops of milk for their babies and others may only need to supplement a few ounces a day. Regardless of an individual mother's maximal output, if she is not able to exclusively breastfeed, this is considered failed lactogenesis II or low milk supply (Hurst, 2007).

**Definition of delayed lactogenesis II.** Delayed lactogenesis II is different from failed lactogenesis II in that the mother eventually is able to produce an adequate breast milk supply to exclusively breastfeed her baby (Hurst, 2007). The problem is that the onset of adequate supply occurs beyond the fifth day postpartum (Betzold, Hoover, & Snyder, 2004; Hurst, 2007).

**Prevalence of failed lactogenesis II.** While the exact prevalence of failed lactogenesis II is unknown, it has been reported to occur in up to 5% of lactating women (Neifert et al., 1990; Neifert, 2001). Estimates for 2014 state the world's population is approximately 7.2 billion with 144 million births occurring per year (Population Reference Bureau, 2014). This would mean that lactation failure could potentially impact 7.2 million mother/baby dyads per year worldwide. There are multiple factors that may contribute to failed lactogenesis, and the information is limited regarding the ability to predict which factor has a stronger impact on lactation failure than another (Berens, 2004).

**Maternal causes of failed lactogenesis II.** Known causes of primary lactation failure associated with the mother include: thyroid dysfunction, PCOS, gestational ovarian theca lutein cysts, retained placenta after delivery, postpartum hemorrhage with Sheehan's syndrome, IGT, DM, obesity, and preterm birth (Anderson, 2001; Chapman, 2014; Hopkinson et al., 1988; Hurst, 1996; Hurst, 2007; Jevitt et al., 2007; Marasco et al., 2000; Souto et al., 2003). The associated secondary causes of lactation failure in the mother include smoking (Hurst, 2007), history of breast surgery (Hurst, 2007; Neifert et al., 1990), and maternal medications (e.g. pseudoephedrine, progestin only and/or estrogen containing birth control) (Hurst, 2007).

**Thyroid Dysfunction.** Thyroid dysfunction, either acute or chronic, during the postpartum period is a maternal health problem that may have a negative effect on lactation. In

normal thyroid function there is a negative feedback loop between the hypothalamus, pituitary gland, and the thyroid itself. The hormones produced by the thyroid include thyroxine ( $T_4$ ), triiodothyronine ( $T_3$ ), and calcitonin (Riordan & Wambach, 2010). Hormonal output from the thyroid is regulated by thyroid-stimulating hormone (TSH) produced by the anterior pituitary, which itself is regulated by thyrotropin-releasing hormone (TRH) secreted from the hypothalamus (Goldstein, 2013). In general, the thyroid's function is to control the body's metabolism and promote normal development of the central nervous system (Riordan & Wambach, 2010). During lactation, healthy thyroid function is necessary so the hormones prolactin and oxytocin can function effectively and allow for normal lactation to occur (Lawrence & Lawrence, 2011). Within the literature it is stated that hypothyroidism rather than hyperthyroidism is more frequently associated with failed lactogenesis II or delayed lactogenesis (Goldstein 2013). In general, the literature regarding the relationship between thyroid dysfunction and lactation failure is scarce (Goldstein, 2013; Riordan & Wambach, 2010).

***Polycystic Ovarian Syndrome (PCOS).*** PCOS is a hormonal disorder of androgen excess which affects women of reproductive age. The prevalence of PCOS is thought to be anywhere from 3% to as high as 20% of the female population (Marasco et al., 2000; Riordan & Wambach, 2010). PCOS is characterized by the presence of multiple cysts on the ovaries which influences the ovaries to overproduce androgens, most commonly testosterone. When a woman has elevated levels of circulating androgens in her body, she may experience irregular menses, erratic ovulation or anovulation, infertility, hirsutism, oligo- or amenorrhea, acne, unusual breast development, and be more likely to be obese (Lawrence & Lawrence, 2011; Marasco et al., 2000; Riordan & Wambach, 2010). Elevated levels of circulating androgens may interfere with

prolactin receptors within the mammary tissue affecting normal breast tissue growth and milk synthesis (Marasco et al., 2000). Riordan and Wambach (2010) maintain that low milk production has been observed in women with PCOS who otherwise do everything else right, including early frequent feeding and breastfeeding technique. Marasco et al., (2000) have also described three cases of breastfeeding women with PCOS who all experienced failed lactogenesis II.

***Gestational Ovarian Theca Lutein Cysts.*** An ovarian theca lutein cyst may develop on the ovary in response to excessive levels of beta-human chorionic gonadotropins (beta-HCG). The types of pregnancies that are more likely to produce excessive levels of beta-HCG include hydatidiform moles, choriocarcinomas, fetal hydrops, diabetes, and multiple or isoimmunized pregnancies (Hoover, Barbalinardo, & Platia, 2002). However, there are case studies of women with otherwise normal pregnancies who have been diagnosed with theca lutein cysts (Hoover et al., 2002). These cysts produce high levels of testosterone, 10 to 150 times the normal level, which may lead to either failed or delayed lactogenesis II (Riordan & Wambach, 2010). During the postpartum period these cysts tend to resolve spontaneously which causes the circulating levels of testosterone to return to normal levels (Hoover et al., 2002). Betzold et al. (2004) described four cases of women with gestational ovarian theca lutein cysts in which 3 of the 4 experienced delayed lactogenesis II and one experienced failed lactogenesis II. The physiology regarding how testosterone interferes with normal lactation is the same as for PCOS.

***Retained Placenta.*** Retained placenta is a condition where some, or all, of the placenta is left within the uterus after the birth of the baby (King et al., 2013). If fragments of the placenta remain within the uterus unknowingly, the placental lactogenic hormones will continue to block

mammary prolactin receptors and the preservation of elevated circulating levels of progesterone will interfere with the onset of lactogenesis II (Kent, 2007; Taylor, 1995). This may cause either a delay in lactogenesis II or failed lactogenesis II. Neifert, McDonough, and Neville (1981) describe three cases of placental retention and interference of the normal onset of lactogenesis II. In two of the three cases, once the diagnosis of retained placenta was given and treated by curettage, the women experienced an increase in milk production but never attained adequate lactation. In the third case, after diagnosis and subsequent curettage, the mother went on to produce an adequate milk supply and continued to breastfeed her infant for 16 months (Neifert et al., 1981). Anderson (2001) also discusses a case of delayed lactogenesis II where a woman was found to have retained placental products secondary to placenta increta after seeking help for low milk supply. After treatment with methotrexate, and passage of the remaining placenta, the mother was able to produce an adequate milk supply on day 36 postpartum (Anderson, 2001).

***Postpartum Hemorrhage with Sheehan's Syndrome.*** Sheehan syndrome occurs due to severe postpartum hemorrhage which leads to hypovolemic shock and damage to the pituitary gland. The pituitary gland is a highly vascular organ that increases in size during pregnancy and its function is sensitive to hypoperfusion. The result of hypoperfusion can lead to pituitary thrombotic infarction and necrosis or other vascular injury to the pituitary gland (Lawrence & Lawrence, 2011). If the pituitary gland is not functioning properly postpartum, there will be a lack of secreted prolactin which is necessary for normal lactation to occur. Willis and Livingstone (1995) reported 10 cases of women who experienced postpartum hemorrhage and subsequently had either failed or delayed lactogenesis II. Upon exam and history taking of the 10 mothers, two had small hypoplastic appearing breasts which may have accounted for their

low milk supply; four had minor difficulties with breastfeeding techniques, however, the authors did not indicate that they felt this was the cause of their lactation insufficiency; and the rest had normal appearing breasts and effective breastfeeding techniques were observed (Willis & Livingstone, 1995). After providing breastfeeding support and correcting techniques as needed, two mothers went on to fully lactate; five continued to breastfeed with the assistance of an at breast supplement system; one mother chose to stop breastfeeding; and the remaining two were lost to follow up. The authors did note that laboratory exams exploring the correlation between pituitary function and prolactin levels were not measured (Willis & Livingstone, 1995).

***Insufficient Glandular Tissue (IGT).*** IGT is a primary cause of lactation failure or delay and is a condition in which women lack the glandular tissue in the anatomy of the breast for milk production and storage (Huggins, Petok, & Mireles, 2000). The majority of mothers diagnosed with IGT and low milk supply will not be able to exclusively breastfeed their baby despite heroic efforts to increase milk supply (Huggins et al., 2000). Given that IGT is rare, it may not be diagnosed antenatally or in the early stages of breastfeeding. Mothers may be given other rationale as to why they are experiencing low milk supply, and some mothers may be given false hope that with additional efforts and changes they will be able to produce an adequate milk supply and exclusively breastfeed their infant (Cassar-Uhl, 2013).

IGT can be diagnosed antenatally when a clinician observes abnormal development of at least one breast, breast asymmetry, tubular breast shape, intra-mammary distance greater than 1.5 inches, high mammary fold, disproportionately large or bulbous areolae, or the mother reports absence of typical breast changes that occur during pregnancy (Cassar-Uhl, 2013; Huggins et al., 2000). Currently the literature on IGT is limited to documented case reports and one prospective

descriptive study. The case reports describe the clinical presentation of women who have experienced failed lactogenesis II related to IGT (Arbour & Kessler, 2013; Bodley & Powers, 1999; Michell, 1997; Neifert et al., 1985; Thorley, 2005); and the prospective descriptive study (Huggins et al., 2000) identified women with IGT antenatally who planned to breastfeed and followed them until one month postpartum to quantify their milk production. Huggins et al. (2000) found that some of the women experienced delayed lactogenesis and by six weeks postpartum they had achieved an adequate milk supply. However, this was not the case for all of the subjects who were otherwise found to have chronic low milk supply and subsequently diagnosed with failed lactogenesis II.

***Diabetes Mellitus (DM).*** DM is a metabolic disease where the affected person has elevated levels of blood glucose circulating in their body. This is either due to the pancreas not producing enough insulin or because the cells of the body do not respond properly to the insulin that is produced. There are three types of diabetes: Type 1 DM, Type 2 DM and gestational diabetes mellitus (GDM). Type I DM results from the body's failure to produce insulin and typically is detected during childhood and requires lifetime insulin therapy. Type 2 DM results from insulin resistance, a condition in which the body's cells fail to use insulin properly and may sometimes be accompanied by an absolute insulin deficiency. Type 2 DM can be managed with diet, oral medication, or insulin. Lastly, GDM is a condition that develops only during pregnancy and can be managed with diet, oral medication, or insulin depending on the severity of the disease, and typically resolves after pregnancy (White & Porterfield, 2013).

It is known that prior history of DM or newly diagnosed GDM is a risk factor for failed or delayed lactogenesis II in the postpartum period (Chapman, 2014; Hartman & Cregan, 2001;

Hurst, 2007; Matias, Dewey, Quesenberry, & Gunderson, 2014). Matias et al. (2014) performed a secondary data analysis from a study being conducted between Kaiser Permanente of Northern California and the University of California, Davis. This was the Study of Women, Infant Feeding and Type 2 Diabetes after GDM Pregnancy (SWIFT). Matias and colleagues required that the participants had initiated breastfeeding and were able to provide data on the timing of lactogenesis stage II. They ultimately enrolled 883 participants and found that 33% of the women reported a delay in lactogenesis II. They also reported that a key risk factor for delayed lactogenesis was the need of insulin for the treatment of GDM (Matias et al., 2014). The authors did not provide statistics on those women who continued to have chronic low milk supply indicating failed lactogenesis II. Lastly, it is believed that the hyperinsulinaemia of women with DM promotes hyperandrogenism allowing for increased levels of circulating testosterone which may lead to failed lactogenesis II (Marasco et al., 2000; Nestler, 1997).

***Maternal Obesity.*** Obesity refers to body weight that is greater than what is considered healthy for a certain height. This is measured by body mass index (BMI) which estimates body fat based on a height to weight ratio (USHHS, 2012). Someone with a BMI greater than or equal to 30 is considered obese, and morbidly obese if their BMI is greater than or equal to 40 (USHHS, 2012).

The literature suggests that maternal obesity is a risk factor for failed or delayed lactogenesis II (Chapman & Perez-Escamilla, 1999; Hurst, 2007; Rasmussen & Kjolhede, 2004; Rasmussen, Hilson, & Kjolhede, 2001). Rasmussen & Kjolhede (2004) identified 40 mothers antenatally who were either of normal pre-pregnant BMI defined as  $<26$  ( $n=23$ ) or overweight/obese with BMI  $\geq 26$  ( $n=17$ ). They obtained blood samples from the participants

before and after a breastfeeding session on day two postpartum and again on day seven. The blood samples were later analyzed for concentrations of prolactin, insulin, estradiol, progesterone, leptin, and glucose. They also timed the breastfeeding session on these days and recorded the times. They concluded that women with  $\geq 26$  BMI had significantly lower levels of prolactin than normal weight women on day two postpartum; but by seven days postpartum, there were no significant differences between groups. They also found that infants of the women in the overweight/obese category suckled on average for 23 minutes compared to the infants of women with normal BMI who suckled on average for 15 minutes. These findings suggested to the authors that the infants of overweight/obese women suckled longer due to early low milk production in order to help increase milk supply (Rasmussen & Kjolhede, 2004). Additionally, the authors theorized that delayed onset of lactogenesis II experienced by the mothers with higher BMI may lead to early supplementation and eventually less milk production.

***Preterm Birth.*** Preterm birth, defined as birth when less than 37 weeks of gestation have been completed, presents challenges to lactation (King et al., 2013). Mothers of preterm infants are encouraged to provide breast milk for their babies because of the benefits which include reduction in the incidence of infections and necrotizing enterocolitis, improved feeding tolerance, and enhanced neurological development (Jones, 2009). Women who deliver preterm may experience either failed or delayed lactogenesis II because their breasts may not be fully developed (Geddes, Hartmann, & Jones, 2013). During the third stage of mammogenesis, which occurs during pregnancy, there are two important phases that the breast tissue needs to complete in order to achieve lactation. These are the secretory differentiation and secretory activation phases. During secretory differentiation the mother experiences nipple growth, increase in size

of the areola, proliferation of the ductal system, and increase in size and growth of the lobular-alveolar system. The secretory activation, or milk making phase, is then triggered when the placenta is expelled and there is a subsequent drop in circulating progesterone (Lawrence & Lawrence, 2011). However, when the secretory differentiation phase is interrupted due to preterm birth, the mammary epithelium may not be adequately prepared to produce milk proficiently leading to either failed or delayed lactogenesis II (Geddes et al., 2013).

**Smoking.** Smoking is a secondary cause of low milk supply (Hurst, 2007). In a mother who otherwise has no physiological problems that would interfere with lactation, nicotine use will interfere with the let-down reflex and not allow milk to flow freely to the suckling infant (Lawrence & Lawrence, 2011). Vio, Salazar, and Infante (1991) add that nicotine consumption also interferes with the production of prolactin, therefore decreasing the potential volume of milk that can be made in the lactating breast. Vio et al., (1991) published a quantitative descriptive study where they indentified 10 breastfeeding mothers who smoked at least four or more cigarettes per day, and case matched them to 10 breastfeeding non-smoking mothers. The mothers were matched by maternal age, pre-pregnancy weight and height, absence of morbidity, and no use of medication. Upon enrollment in the study, the babies were within one to three months of age, birth weight of between 3,000-4,000 grams, exclusively breastfed, and free of morbidity. The authors recorded daily milk volumes measured by using the dose-to-mother deuterium-dilution method over 14 days. They found that the difference in maternal milk production between both groups was significant ( $t=5.21$ ,  $p < 0.0001$ ), with a measured average volume of maternal milk of  $961 \pm 120$  g/d for the group of nonsmoking mothers and  $693 \pm 110$  g/d for smoking mothers (Vio et al., 1991).

**Breast Surgery.** Breast surgery is a secondary cause of either failed or delayed lactogenesis II. The surgery itself can cause destruction of breast tissue; interruption of the ducts, nerve supply, or blood supply to the glandular tissue; or damage to the nipple leading to either failed or delayed lactogenesis later in life (Lawrence & Lawrence, 2011). Breast surgery includes excisional biopsy, augmentation, reduction, and/or chest surgery involving the breast (Neifert et al., 1990). Neifert et al. (1990) performed a prospective study investigating the effects of breast surgery on lactation outcomes. Their sample consisted of 319 healthy, motivated, primiparous women, who were breastfeeding term, healthy newborns that were appropriate or large for gestational age. Of the 319 participants, 22 women (6.9%) reported previous breast surgery. The participants were visited twice in the first 2 weeks postpartum and their babies were weighed to determine if their mother had an adequate milk supply. Adequacy was defined as the infant being exclusively breastfed and gaining 28.5 grams or more per day between the two consecutive visits. Overall, the participants with previous breast surgery had greater than a threefold risk of low milk supply compared to the women with no prior breast surgery (RR=3.11, (95% CI 1.65-5.85;  $p < 0.01$ ) (Neifert et al., 1990). Hurst (1996) also replicated the significance of the results found by Neifert et al. (1990) in her retrospective study of breast-feeding mothers. She identified 42 mothers who had experienced breast surgery and 42 mothers who had not. She identified these women through the records kept by the Lactation Program at Texas Children's Hospital in Houston, Texas between May 1990 through February 1995. This lactation program is a hospital-based service that also offers outpatient community-based care. Upon review of the 84 records, the author found a significantly greater incidence of low milk supply in the women who had prior breast surgery compared to those who had not

( $p < .001$ ). Ultimately she reported that of the 42 women with prior breast surgery, 27 (64%) had low milk supply; this is compared to the 3 (<7%) mothers out of 42 with no prior breast surgery who experienced low milk supply (Hurst, 1996).

**Maternal Medication.** Use of various medications have been known to cause a secondary effect of lactation insufficiency in the lactating women (Hurst, 2007; Lawrence & Lawrence, 2011). These medications include pseudoephedrine, androgens, estrogens, dopaminergic agents, anticholinergics, diuretics, and antihistamines. Additionally, the herb sage has an antisudorific effect and is said to reduce lactation; and therefore, the entire sage family should be avoided during lactation (Hurst, 2007; Lawrence & Lawrence, 2011).

**Infant causes of failed lactogenesis II.** Hurst (2007) states that secondary lactation failure can occur because of an ineffective or weak suck by the infant. Conditions that can cause an ineffective or weak suck include functional anomalies such as cleft lip/palate, tongue-tie, or lip-tie (Hurst, 2007). Prematurity or anatomical anomalies in the infant such as congenital heart defects can also contribute to the infant having an ineffective suck (Hurst, 2007). Lastly, any condition that results in incomplete emptying of the breast such as improper latch or breastfeeding mismanagement can lead to lactation insufficiency (Hurst, 2007). Turner et al. (2001) also discuss that slow weight gain has been reported in breastfed infants with palatal anomalies and they contribute this finding to decreased ability of the infant to effectively remove milk from the maternal breast. Overall, there is very little research regarding the infant causes of decreased milk supply; however, the connection between ineffective milk removal and decreased production is well supported (Black et al., 1998; Lawrence & Lawrence, 2011; Riordan & Wambach, 2010).

**Failed lactogenesis II and infant risks.** The consequences of breastfeeding with unknown low milk supply include hyperbilirubinemia, infant hunger, slow weight gain, infant failure to thrive, and life-threatening or even fatal dehydration and starvation (Alsina-Manrique et al., 2006; Boskabadi et al., 2010; Dewey, Nommsen-Rivers, Heinig, & Cohen, 2003; Livingstone, Willis, Abdel-Wareth, Thiessen, & Lockitch, 2000; Neifert, 2001; Rand & Kolberg, 2001).

Alsina-Manrique et al. (2006) described a case in which a first-time mother delivered a healthy, full-term baby and was exclusively breastfeeding her baby. By day 10 postpartum, the baby was readmitted due to 30% body weight loss, dehydration, and lethargy. The laboratory findings revealed that the baby had elevated serum sodium levels and was diagnosed with hypernatremic dehydration. The authors stated that this infant was fortunate to have received treatment prior to further complications including seizures and permanent neurological and vascular damage (Alsina-Manrique et al., 2006).

Livingstone et al. (2000) asserted that the number of parents reporting that their baby had been re-admitted for hypernatremic dehydration has been increasing. These authors performed a retrospective survey in which they identified 21 cases of hypernatremic dehydration from the medical charts at British Columbia's Children's Hospital. The presenting complaints from the parents when they brought their infants in included: weight loss, failure to gain weight, lethargy, poor feeding, infrequent or absent bowel movements, or seizures (Livingstone et al., 2000). They concluded that as breastfeeding rates rise, we may continue to see more cases of breastfeeding failure and the consequences to the baby.

Neifert (2001) described two stories of babies whose consequences were fatal. The first narrative was of a teenage mother whose eight week old son died of starvation due to failed lactogenesis II; the second narrative was of a mother, a registered nurse, whose 10 day old son was diagnosed with severe hypernatremic dehydration that left him severely brain damaged to which he succumbed to related complications by nine months of age (Neifert, 2001).

The first week postpartum is a critical period for the establishment of breastfeeding and it is important to remember that any woman may encounter breastfeeding complications (Dewey et al., 2003). Health care providers working with breastfeeding mothers and their babies need to be aware of the conditions that may cause either failed or delayed lactogenesis. Early follow-up after birth allows healthcare providers to assess for adequate milk production. If failed lactogenesis II is suspected, frequent follow-up should be recommended to ensure the baby receives adequate nutrition (Boskabadi et al., 2010; Dewey et al., 2003).

## **B. Innovation**

The number of women who initiate breastfeeding is at an all-time high at 77% (CDC, 2013), primarily due to rooming-in, skin-to-skin contact and the efforts of the baby friendly movement. As more mothers breastfeed, the incidence of failed lactogenesis II will undoubtedly increase, and women will look to their healthcare providers for guidance. Therefore, clinicians need to be aware of the potential causes of low milk supply as well as what can be done to preserve the breastfeeding relationship and how to properly care for the mother-baby dyad from both clinical and emotional perspectives. This study is an important first step in understanding the breastfeeding experiences of women diagnosed with low breast milk production from the woman's perspective. The proposed study is innovative because it is the first known study that

proposes to better understand how women feel when they learn they have low milk supply, how they cope with the diagnosis, and how they manage their breastfeeding after the diagnosis. The stories of women and the emotional and/or psychological impact that failed lactogenesis II has had on their lives is the critical factor that has been missing in the research of women with low milk supply.

### III. Approach

#### **Research Design**

**Qualitative research methodologies.** Qualitative research is a method of inquiry which may be employed to gain a deeper understanding of human experience. There are five approaches that a qualitative researcher may choose from to answer their research question. These approaches include: grounded theory, ethnography, narrative analysis, qualitative descriptive, and phenomenology (Weinberg, 2002).

Grounded theory is a method that enables the researcher to produce a theoretical understanding of a concept or explanation of a phenomenon, from the perspective of the persons who have experienced that phenomenon. With this approach the theoretical understanding of a concept is grounded in data rather than the testing of hypotheses from existing theories. In the end, the researcher will have developed conceptual categories and a theory regarding the phenomenon under study (Sandelowski, 2000; Weinberg, 2002).

Ethnography is the study of people and cultures in their physical location. With this approach the researcher collects data via field work, observation, and the taking of field notes. The product of ethnographic research is a description of a group of people grounded in their culture (Sandelowski, 2000; Weinberg, 2002).

Narrative analysis, also known as case study, is a research method commonly used to understand the operation or function of a person, event, or group. The unit of study can vary widely in size and can range from a single individual, to an organization or community. The outcome of narrative analysis, is a description of the unit of study which includes an understanding of how it operates (Sandelowski, 2000; Weinberg, 2002).

Qualitative descriptive approach, which originated in journalism and with World War II propaganda, provides a thematic summary of the phenomenon. In qualitative descriptive analysis, codes are generated from the data under study, which tends to answer the who, what, and where questions of an event or experience (Sandelowski, 2000; Weinberg, 2002).

Phenomenology originated in German philosophy in the first half of the 20<sup>th</sup> Century. The philosophical underpinnings of phenomenology are based on the works of Edmund Husserl and Martin Heidegger. The purpose of this approach is to describe the lived experience of an individual in the first-person point of view. Experiences can range from perceptions, thoughts, memories, embodied action, and social activity (Stanford Encyclopedia of Philosophy, 2013). Data collection occurs primarily through in-depth interviews, but can also include literature, biographies, poetry, and art. The products from this approach are patterns or themes regarding the meaning of the phenomenon under study (Sandelowski, 2000; Weinberg, 2002).

Husserl was a positivist who believed in descriptive phenomenology in which one seeks to purely describe the phenomena from the perspective of those who have experienced it (Dowling, 2007). In order to avoid the researcher's subjective bias, Husserl developed phenomenologic reduction which is also known as bracketing. This is the process by which the researcher acknowledges their personal experiences with the phenomenon that they are studying, and sets aside any assumptions that they may have so not to affect the study (Connelly, 2010; Dowling, 2007; van Manen, 1990). Husserlian phenomenology is descriptive, which means the observer only describes what he or she sees, hears, or feels without any input from the self (Lavery, 2003). Lastly, when applying the philosophical underpinnings of Husserlian phenomenology, the researcher must believe in the idea of universal essences, or eidetic

structures, which are common to all persons who have a lived experience; meaning there is one correct interpretation of the experience (Flood, 2010).

In contrast, Martin Heidegger, a student of Husserl, challenged the idea of bracketing by saying that interpretation cannot be free of judgment or influence of the researcher. He believed that one's ideas could not be completely put aside because they are a part of the person. Instead, he challenges the researcher to be aware of their presuppositions and any effects that they have on the research (Connelly, 2010; Snow, 2009). Heideggerians believe that the researcher is as much a part of the research as the participant; that it is impossible to rid the mind of background knowledge and understandings; and that the researcher's ability to interpret the data is reliant on previous knowledge. Heidegger called this prior understanding fore-structure and felt that it was an essential aspect for analysis (McConnell-Henry, Chapman, & Francis, 2009). *Heideggerian phenomenology is interpretive, whereby the researcher seeks to interpret and analyze the experiences of the study participants and ultimately identify themes of the phenomenon. Another term for this style of phenomenology is hermeneutic (Cerbone, 2008; Dowling, 2007).*

Although Husserl is credited as the founding father of phenomenology, there have been other phenomenologists who have come after him (Dowling, 2007). Each phenomenologist has their own interpretation of phenomenology based in their discipline of study including disciplines such as psychology, sociology, philosophy, and nursing. This study will be analyzed using the human science approach described by Max van Manen (1990), a sociologist and a proponent of Heideggerian phenomenology.

*Max van Manen is a phenomenologist with a background in sociology.* Like Heidegger, van Manen does not embrace Husserl's view of bracketing and asks: "If we simply try to forget

or ignore what we already “know”, we might find that the presuppositions persistently creep back into our reflections” (van Manen, 1990, p. 47). Instead, he advises that the researcher make clear their understandings, beliefs, biases, assumptions, presuppositions, and theories regarding the phenomena in which they are studying (van Manen, 1990).

Van Manen’s (1990) method is interpretive and it encourages the investigator to really understand the information they receive and to reflect on it. He describes interpretive research as a systematic search for a deeper understanding of the experiences of an individual. He developed a systematic methodological structure with six research activities:

- 1) turning to a phenomenon which seriously interests us and commits us to the world;
- 2) investigating experience as we live it rather than as we conceptualize it;
- 3) reflecting on the essential themes which characterize the phenomenon;
- 4) describe the phenomenon through the art of writing and rewriting;
- 5) maintaining a strong and oriented pedagogical relation to the phenomenon;
- 6) balancing the research context by considering parts and wholes (van Manen, 1990, p. 30).

Even though van Manen developed this systematic methodological structure it is not meant to be used as a sequence of procedural steps, and instead is meant to guide the investigator in their methodology (van Manen, 1990).

According to van Manen (1990), the lived experience is the starting and ending point of phenomenological research. Although an individual continuously experiences daily life, their lived experiences gather hermeneutic significance by “giving memory to them” (van Manen, 1990, p. 37). Lived experiences have temporal characteristics in that they can only be reflected

on as past experiences. The ultimate goal then of phenomenology is to transform one's lived experience into written expression whereby meaning is then assigned to the experience (van Manen, 1990).

This qualitative study will be conducted using van Manen's hermeneutic phenomenological approach. Phenomenology is an appropriate methodology because the research topic has not yet been explored and to date there are no published studies that have looked at the breastfeeding experiences of women diagnosed with low milk supply.

Phenomenology is the best method to employ when a researcher wants to come to a greater understanding of an individual's unique experience. Lastly, van Manen's (1990) hermeneutic phenomenological method was chosen because it is interpretive and ultimately helps the researcher achieve a deeper understanding of the experience under investigation.

### **Setting and Sample**

The aim of this study is to understand the experience of women who have breastfed after having been diagnosed with low milk supply. In order to understand this phenomenon, women willing to share their stories will need to be recruited and interviewed.

**Inclusion and exclusion criteria.** The inclusion criteria required for participation is as follows: (a) participant is a women with a biological child and at least 18 years old (b) participant self-reports having breastfed with a diagnosis of low milk supply (c) the breastfeeding encounter(s) occurred after a full term (delivery after 37 weeks gestation) singleton pregnancy (d) participant indicates that they remember and can articulate their breastfeeding experience (e) participant is able to speak and read in the English language. Participants who do not meet these basic inclusion criteria will not be permitted to participate in this study.

**Sampling Strategy.** Using purposive sampling, 10-15 women will be recruited who self-report having experienced low milk supply during a breastfeeding encounter(s). At this time there is no guidance in the literature regarding the recommended time approximation to the experience a researcher is interested in. For that reason, I will ask each potential participant if they are able to remember the details of their breastfeeding experience after their diagnosis with low milk supply during their assessment for eligibility (see Appendix A).

Recruitment will occur via a social media platform support group which I have been personally involved in for the last two years. This is a support group for women breastfeeding with low milk supply and is named “IGT and Low Milk Supply Support Group”. At this time there are approximately 4,700 members worldwide. I have already received approval from the support groups moderators to post my research study to the board in order to recruit participants; which I will do after IRB approval. I believe that this will be an appropriate and successful way to recruit the participants needed to conduct this study.

**Sample Size.** The sample size for interpretive phenomenological research is small and typically the research can be accomplished with 10 or less participants (Roberts, 2013).

**Setting.** The interview will consist of a one-time encounter. How we accomplish the individual interviews will be based on the proximity of the participant to Chicago. For those women who are close in proximity to Chicago I will travel to a setting of their choice for a face-to-face interview. For those women who are outside of my travel boundaries I will complete the interviews using an internet based modality such as Skype.

## **Measures**

Interview Guide, See Appendix B

## **Procedure**

**Data Collection.** IRB approval will be obtained prior to data collection. Data will be collected through the means of a one-time interview (either in person or via an on-line modality). The interview will be audio-taped and later transcribed verbatim. Notes during the interview will also be collected as needed and added to the transcripts. Data collection will end when theoretical saturation has been met.

**Data Analysis.** Interviews will be transcribed verbatim, read in their entirety and coded by the primary investigator. Analysis will be conducted using Max van Manen's approaches to uncovering themes among the interviews. Van Manen (1990) describes three approaches to isolating themes within data. These approaches include: the wholistic or sententious approach; the selective or highlighting approach; and the detailed or line-by-line approach. For the purpose of this study the second approach will be used which is the selective or highlighting approach. In this approach the researcher is interested in those statements or phrases that seem essential to the experience being described. These statements or phrases are then highlighted. Once all of the essential statements or phrases have been highlighted from the interviews, codes and themes will be developed.

**Data Interpretation.** Data will be interpreted using the method described by Van Manen (1990) as guidance. I will begin by reading my transcribed interviews and selecting or highlighting applicable quotes. A code book containing definitions of the codes will be developed from in-depth reading of the transcribed interview and highlighting of applicable quotes. Coding the data is a continuous and on-going process. Theme development will occur once coding of the data is complete.

## **Trustworthiness of the Research**

The aim of trustworthiness in a qualitative inquiry is to support the argument that the inquiry's findings are "worth paying attention to" (Lincoln & Guba, 1985, p.290). In qualitative research four criteria are needed to establish trustworthiness: credibility, dependability, confirmability, and transferability (Lincoln & Guba, 1985).

**Credibility.** Credibility is an evaluation of whether or not the research findings represent a "credible" conceptual interpretation of the data drawn from the participants' original interview (Lincoln & Guba, 1985, p.296). According to Miles and Huberman (1994) the purpose of credibility ensures that the findings of the study make sense. Credibility for this study will be ensured through peer debriefing and member checking. Peer debriefing is a process by which the researcher works together with one or several colleagues who hold an impartial view of the study. The peer(s) may examine the recorded interviews, handwritten notes, transcripts, final reports and methodology of the study. After examination feedback is provided to the researcher to enhance credibility and ensure validity. The peer debriefing also helps the researcher become more aware of his or her own views regarding the data (Lincoln & Guba, 1985; Miles & Huberman, 1994). Member checking will also be employed. This is when data, analytic categories, interpretations and conclusions are shared with the participants of the study and asked if they feel like this is a true interpretation of their experience (Lincoln & Guba, 1985).

**Dependability.** Dependability is an assessment of the quality of the processes of data collection, data analysis, and theory generation. It also refers to the stability of data over time (Mile & Huberman, 1994). In this study dependability will be achieved by establishing an audit

trail thereby ensuring the data collection and analysis process is free from error (Lincoln & Guba, 1985).

**Confirmability.** Confirmability is a measure of how well the inquiry's findings are supported by the data collected (Lincoln & Guba, 1985). This requires an audit trail and reflexivity by the researcher (Lincoln & Guba, 1985). In this study confirmability will be achieved by keeping an accurate audit trail so to ensure that the findings, conclusions, and recommendation are supported by the data. Also, for reflexivity I will continue to make my beliefs and biases explicit to myself and others by keeping a notebook with my thoughts as they come up.

**Transferability.** Transferability is the degree to which the findings of this inquiry can apply or transfer beyond the bounds of the project (Lincoln & Guba, 1985). In order for this to be considered, the researcher needs to provide thick descriptive data and descriptions of the topic. This is ultimately the goal in this proposal.

### **Risks to Human Subjects**

**Human subjects involvement, characteristics, and design.** In this qualitative study, using a phenomenological approach, 10-15 women will be recruited who self-report having been previously diagnosed with low milk supply and have had a breastfeeding encounter(s). The purposive sample will consist of women who are at least 18 years of age who meet inclusion criteria. Recruitment will occur via a social media platform support group which I have been personally involved in for the last two years. This support group is for women breastfeeding with low milk supply and is named "IGT and Low Milk Supply Support Group". At this time there are approximately 3,400 members worldwide. The participants will be involved in a one-

time interview either face-to-face or via an internet based modality such as Skype. The interview will last between one to two hours.

**Inclusion Criteria.** The inclusion criteria required for participation is as follows: (a) participant is a women with a biological child and at least 18 years old (b) participant self-reports having breastfed with a diagnosis of low milk supply (c) the breastfeeding encounter(s) occurred after a full term (delivery after 37 weeks gestation) singleton pregnancy (d) participant indicates that they remember and can articulate their breastfeeding experience (e) participant is able to speak and read in the English language. Participants who do not meet these basic inclusion criteria will be excluded.

**Exclusion Criteria.** Women who do not meet the basic inclusion criteria, as above, will not be permitted to participate in this study.

**Sources of Materials.** Participants will receive an identification number once enrolled in the study. The identification number will be the only information on the written and electronic copies of the transcribed interview. Any personal information collected will be stored in a secured cabinet in a locked research office when not in use. Interviews will be recorded, transcribed verbatim, and then checked for accuracy. Once transcription is complete the recorded interviews will be properly destroyed after data analysis is complete. Any electronically collected personal information will be secured in a password protected file on an encrypted computer in a locked research office when not in use. Only the principle investigator and designated members of the research team will have access to any personal information or data collected to use solely for the purpose of the study. Prior to data analysis, all personal

information will be removed. Contact information, such as phone numbers or email addresses, may be used for follow-up only with the consent of participants.

**Potential Risks.** Potential risks may include minor psychological stress to the women due to having to relive an experience and tell their story. However, it is unlikely that the interview questions will cause an emotional reaction that is more than they would experience in their everyday life. Prior to meeting with each woman, I will have prepared a list of mental health providers in their area for them to contact if they need to speak to someone.

### **Adequacy of Protection Against Risks**

**Recruitment and informed consent.** All materials will be approved by the IRB at the University of Illinois at Chicago and will be HIPPA compliant prior to the start of recruitment. Recruitment will occur via a social media platform support group for women breastfeeding with low milk supply entitled “IGT and Low Milk Supply Support Group”. At this time there are approximately 3,400 members worldwide. After women express interest in the study, I will discuss with them the purpose, protocol, and rights of study participants. Written consent forms explaining the purpose, protocol, and rights of study participants will also be provided to potential subjects. Women will then provide written documentation of their consent to participate in the study. The consent form will include consent to be contacted after completion of the analysis for member checking purposes.

**Protections against risk.** Members of the research team will be trained on the ethical treatment of human research subjects and will sign confidentiality agreements prior to their involvement in the study. The impact of potential risks will be minimized by continual support and debriefing as necessary. Resources for mental health services will be prepared by me prior

to the interview and provided to a woman if she desires them. Participant confidentiality will be assured by de-identifying data through the assignment of identification numbers specific to each woman. All collected personal and de-identified data will be used solely for the purpose of the study. Women reserve the right to withdraw from participation in the study at any time.

**Potential benefits of the proposed research to human subjects and others.** Women participating in this study may or may not directly benefit from the study. Some women may find the act of telling their story and being heard to be therapeutic.

**Importance of the knowledge to be gained.** Study findings may benefit other women by describing the lived experience of women with low milk supply and their breastfeeding encounter(s).

**Data safety and monitoring plan.** To ensure confidentiality, each woman will be assigned a code number. Recorded interviews will be transcribed verbatim and then recordings will be properly destroyed after data analysis is complete. Electronically stored data will be kept in a locked research room on an encrypted computer secured by a password. Access to the data will be restricted to me, the principle investigator (PI), and research staff. Continuous, close monitoring conducted by the PI will be an adequate and appropriate format for monitoring, with prompt reporting of adverse effects to the IRB. Known potential adverse effects will be monitored, such as psychological stress.

**Inclusion of women and minorities.** The purpose of the research study is to look at the lived experience of women who have been diagnosed with low milk supply and their breastfeeding experience. All women regardless of race, ethnicity, religion, educational level, and socioeconomic status may participate in this study as long as they meet the inclusion criteria.

**Inclusion of children.** No children will be included in this study.

**Inclusion of vulnerable populations.** Pregnant women may be included as long as they have had a previous child and meet the inclusion criteria.

## References

1. Alsina-Manrique, L., Esteban, M., Salvià, D., Miracle, X., Rodríguez-Miguélez, J., Figueras, J., & Carbonell, X. (2006). Severe hypernatremic dehydration secondary to undetected lactation failure: usefulness of sodium levels in breast milk. *Clinical Pediatrics*, *45*(2), 183-186. doi: 10.1177/000992280604500211
2. Anderson, A. (2001). Disruption of lactogenesis by retained placental fragments. *Journal Of Human Lactation*, *17*(2), 142-144. doi: 10.1177/089033440101700210
3. Arbour, M. W., & Kessler, J. (2013). Mammary Hypoplasia: Not Every Breast Can Produce Sufficient Milk. *Journal Of Midwifery & Women's Health*, *58*(4), 457-461. doi:10.1111/jmwh.12070
4. Berens, P. (2004). Applied physiology in the peripartum management of lactation. *Clinical Obstetrics & Gynecology*, *47*(3), 643-655. doi: 10.1097/01.grf.0000135342.38287.58
5. Betzold, C., Hoover, K., & Snyder, C. (2004). Delayed lactogenesis II: a comparison of four cases. *Journal Of Midwifery & Women's Health*, *49*(2), 132-137. doi: 10.1016/S1526-9523(03)00537-3
6. Black, R. F., Jarman, L., & Simpson, J. B. (1998). Lactation specialist self-study series. *The Science of Breastfeeding*. Sudbury, MA: Jones and Bartlett Publishers.
7. Bodley, V., & Powers, D. (1999). Patient with insufficient glandular tissue experiences milk supply increase attributed to progesterone treatment for luteal phase defect. *Journal of Human Lactation*, *15*(4), 339-343. doi:10.1177/089033449901500415
8. Boskabadi, H., Maamouri, G., Ebrahimi, M., Ghayour-Mobarhan, M., Esmaeily, H., Sahebkar, A., & Ferns, G. (2010). Neonatal hypernatremia and dehydration in infants

- receiving inadequate breastfeeding. *Asia Pacific Journal Of Clinical Nutrition*, 19(3), 301-307. Retrieved from <http://apjcn.nhri.org.tw/server/APJCN/19/3/301.pdf>
9. Cassar-Uhl, D. (2013). Breastfeeding with hypoplasia: Insufficient glandular tissue. *Breastfeeding Today*, January-March 2013(17). Retrieved from <http://viewer.zmags.com/publication/7aece22c#/7aece22c/1>
  10. Centers for Disease Control and Prevention (CDC). (July 2013). Breastfeeding report card: United States/2013. Retrieved from <http://www.cdc.gov/breastfeeding/pdf/2013breastfeedingreportcard.pdf>
  11. Cerbone, D. R. (2008). *Heidegger: A guide for the perplexed*. London: Continuum International Publishing Group.
  12. Chapman, D. J. (2014). Risk Factors for Delayed Lactogenesis among Women with Gestational Diabetes Mellitus. *Journal Of Human Lactation*, 30(2), 134-135. doi:10.1177/0890334414525566
  13. Chapman, D. J., & Perez-Escamilla, R. (1999). Identification of risk factors for delayed onset of lactation. *Journal of the American Dietetic Association*, 99(4), 450-454. doi 10.1016/S0002-8223(99)00109-1
  14. Connelly, L. M. (2010). What is phenomenology?. *MEDSURG Nursing*, 19(2), 127-128. Retrieved from <http://web.ebscohost.com.proxy.cc.uic.edu/ehost/pdfviewer/pdfviewer>
  15. Dewey, K., Nommsen-Rivers, L., Heinig, M., & Cohen, R. (2003). Risk factors for suboptimal infant breastfeeding behavior, delayed onset of lactation, and excess neonatal weight loss. *Pediatrics*, 112(3), 607-619. doi 10.1542/peds.112.3.607
  16. Dowling, M. (2007). From Husserl to van Manen. A review of different phenomenological

- approaches. *International Journal Of Nursing Studies*, 44(1), 131-142. doi:  
10.1016/j.ijnurstu.2005.11.026
17. Eidelman, A. K., & Schanler, R. J. (2012). Breastfeeding and the Use of Human Milk. *Pediatrics*, 129(3), 598-601. doi:10.1542/peds.2011-3552
18. Flood, A. (2010). Understanding phenomenology. *Nurse Researcher*, 17(2), 7-15. doi:  
10.7748/nr2010.01.17.2.7.c7457
19. Geddes, D., Hartmann, P., & Jones, E. (2013). Preterm birth: Strategies for establishing adequate milk production and successful lactation. *Seminars in Fetal & Neonatal Medicine*, 18, 155-159. doi: 10.1016/j.siny.2013.04.001
20. Godfrey, J. (2009). Toward optimal health: Maternal benefits of breastfeeding... David Meyers. *Journal Of Women's Health*, 18(9), 1307-1310. doi:10.1089/jwh.2009.1646
21. Godfrey, J., & Lawrence, R. (2010). Toward optimal health: the maternal benefits of breastfeeding.... Ruth A. Lawrence, M.D. *Journal Of Women's Health*, 19(9), 1597-1602. doi:10.1089/jwh.2010.2290
22. Goldstein, A. L. (2013). New-Onset Graves' Disease in the Postpartum Period. *Journal Of Midwifery & Women's Health*, 58(2), 211-214. doi:10.1111/jmwh.12016
23. Hartmann, P., & Cregan, M. (2001). Lactogenesis and the effects of insulin-dependent diabetes mellitus and prematurity. *The Journal of Nutrition*, 131, 3016S-3020S. Retrieved from
24. Hiller-Sturmhofel, S., & Bartke, A. (1998). The endocrine system: an overview. *Alcohol Health & Research World*, 22(3), 153-164. doi: none

25. Hoover, K., Barbalinardo, L., & Platia, M. (2002). Delayed lactogenesis II secondary to gestational ovarian theca lutein cysts in two normal singleton pregnancies. *Journal Of Human Lactation*, 18(3), 264-268. doi: 10.1177/089033440201800309
26. Hopkinson, J., Schanler, R., & Garza, C. (1988). Milk production by mothers of premature infants. *Pediatrics*, 81(6), 815-820. Retrieved from
27. Huggins, K. E., Petok, E. S., & Mireles O. Markers of lactation insufficiency: A study of 34 mothers. In Auerbach KG, ed. *Current issues in clinical lactation*. Boston, MA: Jones and Bartlett; 2000:25–35. Retrieved from <http://www.sonic.net/~mollyf/igt/>
28. Hurst, N. M. (1996). Lactation after augmentation mammoplasty. *Obstetrics & Gynecology*, 87(1), 30-34. doi: 10.1016/0029-7844(95)00349-5
29. Hurst, N. M. (2007). Recognizing and treating delayed or failed lactogenesis II. *Journal of Midwifery and Women's Health*, 52(6), 588-594. doi: 10.1016/j.jmwh.2007.05.005
30. INFACt Canada. (2002). A brief annotated bibliography: Fourteen risks of formula feeding. [Pamphlet]. Retrieved from <http://www.infactcanada.ca/pdf/14-Risks-Small.pdf>
31. Jevitt, C., Hernandez, I., & Groër, M. (2007). Lactation complicated by overweight and obesity: supporting the mother and newborn. *Journal Of Midwifery & Women's Health*, 52(6), 606-613. doi: 10.1016/j.jmwh.2007.04.006
32. Jones, E. (2009). Initiating and establishing lactation in the mother of a preterm infant. *Journal Of Neonatal Nursing*, 15(2), 56-59. Retrieved from
33. Kent, J. C. (2007). How breastfeeding works. *Journal of Midwifery and Women's Health*, 52(6), 564-570. doi: 10.1016/j.jmwh.2007.04.007

34. Kleinberg, D. L. (1997). Early mammary development: Growth hormone and IGF-1. *Journal of Mammary Gland Biology and Neoplasia*, 2(1), 49-57. doi:10.1023/A:1026373513521
35. King, T. L., Brucker, M. C., Kreibs, J.M., Fahey, J. O., Geger, C.L., & Varney, H. (2013) *Varney's Midwifery* (5<sup>th</sup> ed.). Burlington, MA: Jones and Bartlett Learning.
36. Labbok, M. (2008). Exploration of guilt among mothers who do not breastfeed: the physician's role. *Journal Of Human Lactation*, 24(1), 80-84. doi:10.1177/0890334407312002
37. Lavery, S. M. (2003). Hermeneutic phenomenology and phenomenology: A comparison of historical and methodological considerations. *International Journal of Qualitative Methods*, 2(3), 21-49. Retrieved from <http://wigan-ojs.library.ualberta.ca/index.php/IJQM/article/viewFile/4510/3647>
38. Lawrence, R. A., & Lawrence, R. M. (2011). *Breastfeeding: A guide for the medical profession* (7<sup>th</sup> ed.). Maryland Heights, MO: Elsevier Mosby
39. Lincoln, Y. S., & Guba, E. G. (1985). *Naturalistic inquiry*. Newbury, CA: Sage Publications Inc
40. Lindberg, N., & Berglund, A. (2014). Mothers' experiences of feeding babies born with cleft lip and palate. *Scandinavian Journal Of Caring Sciences*, 28(1), 66-73. doi:10.1111/scs.12048
41. Liu, B., Jorm, L., & Banks, E. (2010). Parity, breastfeeding, and the subsequent risk of maternal type 2 diabetes. *Diabetes Care*, 33(6), 1239-1241. doi:10.2337/dc10-0347
42. Livingstone, V. H., Willis, C. E., Abdel-Wareth, L. O., Thiessen, P., & Lockitch, G. (2000). Neonatal hypernatremic dehydration associated with breast-feeding malnutrition: a

retrospective survey. *Canadian Medical Association Journal*, 162(5), 647-652. Retrieved from

43. Marasco, L., Marmet, C., & Shell, E. (2000). Insights in practice. Polycystic ovary syndrome: a connection to insufficient milk supply?. *Journal Of Human Lactation*, 16(2), 143-148. doi: 10.1177/089033440001600211
44. Matias, S. L., Dewey, K. G., Quesenberry Jr., C. P., & Gunderson, E. P. (2014). Maternal prepregnancy obesity and insulin treatment during pregnancy are independently associated with delayed lactogenesis in women with recent gestational diabetes mellitus. *American Journal Of Clinical Nutrition*, 99(1), 115-121. doi:10.3945/ajcn.113.073049
45. McConnell-Henry, T., Chapman, Y., & Francis, K. (2009). Unpacking Heideggerian phenomenology. *Southern Online Journal Of Nursing Research*, 9(1), 6p. Retrieved from <http://web.ebscohost.com.proxy.cc.uic.edu/ehost/detail?sid=5c34f660-988f-48f4-a66f-867bd1c87c4b%40sessionmgr111&vid=5&hid=104&bdata=JnNpdGU9ZWZWhvc3QtbGl2ZQ%3d%3d#db=rzh&AN=2010235015>
46. Michell, Jane. (1997). Lactation failure caused by lack of glandular development in the breast. *Breastfeeding Review*, 5(1), 27-28. Retrieved from
47. Miles, M.B., & Huberman, A.M. (1994). *Qualitative Data Analysis* (2<sup>nd</sup> ed.). Thousand Oaks, CA: Sage Publications
48. Neifert, M.R., McDonough, S.L., & Neville, M.C. (1981). Failure of lactogenesis associated with placental retention. *American Journal of Obstetrics and Gynecology*, 140(4), 477-478. Retrieved from

49. Neifert, M. R., Seacat, J. M., & Jobe, W. E. (1985). Lactation failure due to insufficient glandular development of the breast. *Pediatrics*, 76, 823. Retrieved from
50. Neifert, M., DeMarzo, S., Seacat, J., Young, D., Leff, M., & Orleans, M. (1990). The influence of breast surgery, breast appearance, and pregnancy-induced breast changes on lactation sufficiency as measured by infant weight gain. *Birth*, 17(1), 31-38. doi: 10.1111/j.1523-536X.1990.tb00007.x
51. Neifert, M. R. (2001). Prevention of breastfeeding tragedies. *Pediatric Clinics of North America*, 48(2), 273-297. doi: 10.1016/S0031-3955(08)70026-9
52. Nestler, J. E. (1997). Insulin regulation of human ovarian androgens. *Human Reproduction*, 12(1), 53-62. doi: 10.1093/humrep/12.suppl\_1.53
53. Neville, M. (2001). Anatomy and physiology of lactation. *Pediatric Clinics of North America*, 48(1), 13-34. doi: 10.1016/S0031-3955(05)70283-2
54. Phenomenology. (December 16, 2013). In *Stanford Encyclopedia of Philosophy online*. Retrieved from <http://plato.stanford.edu/entries/phenomenology/>
55. Population Reference Bureau. (2014). *2014 World population data sheet*. Retrieved from [http://www.prb.org/pdf14/2014-world-population-data-sheet\\_eng.pdf](http://www.prb.org/pdf14/2014-world-population-data-sheet_eng.pdf)
56. Rand, S. E., & Kolberg, A. (2001). Neonatal hypernatremic dehydration secondary to lactation failure. *Journal of the American Board of Family Medicine*, 14(2), 155-158. Retrieved from
57. Rasmussen, K.M, Hilson, J.A., & Kjolhede, C.L. (2001). Obesity may impair lactogenesis II. *The Journal of Nutrition*, 131(11), 3009s-3011s. Retrieved from

<http://jn.nutrition.org.proxy.cc.uic.edu/content/131/11/3009S.full.pdf+html?sid=20b5d8ac-df60-4a17-8409-a41f8a5b8721>

58. Rasmussen, K., & Kjolhede, C. (2004). Prepregnant overweight and obesity diminish the prolactin response to suckling in the first week postpartum. *Pediatrics*, *113*(5), e465-71. doi: 10.1542/peds.113.5.e465
59. Riordan, J., & Wambach, K. (2010). *Breastfeeding and human lactation* (4<sup>th</sup> ed.). Sudbury, MA: Jones and Bartlett Publishers.
60. Roberts, T. (2013). Understanding the research methodology of interpretative phenomenological analysis. *British Journal Of Midwifery*, *21*(3), 215-218. doi: 10.12968/bjom.2013.21.3.215
61. Sandelowski, M. (2000). Focus on research methods. Whatever happened to qualitative description?. *Research In Nursing & Health*, *23*(4), 334-340. doi: 10.1002/1098-240X(200008)23:4%3C334::AID-NUR9%3E3.0.CO;2-G
62. Shaikh, U., & Chantry, C. (2006). Reflections on the american academy of pediatrics' 2005 policy statement on "breastfeeding and the use of human milk". *Journal of Human Lactation*, *22*(1), 108-110. doi: 10.1177/0890334405279359
63. Snow, S. (2009). Nothing ventured, nothing gained: a journey into phenomenology (part 1). *British Journal Of Midwifery*, *17*(5), 288-290. doi: 10.12968/bjom.2009.17.5.42216
64. Souto, G., Giugliani, E., Giugliani, C., & Schneider, M. (2003). The impact of breast reduction surgery on breastfeeding performance. *Journal Of Human Lactation*, *19*(1), 43. doi: 10.1177/0890334402239733

65. Taylor, B. (1995). Retained placenta and suppressed lactogenesis? *Journal of Human Lactation*, 11(4), 261. doi: 10.1177/089033449501100403
66. Thorley, V. (2005). Breast hypoplasia and breastfeeding: A case history. *Breastfeeding Review*, 13(2), 13-16. Retrieved from
67. Tucker, H. (1979). Endocrinology of lactation. *Seminars in Perinatology*, 3(3), 199-223. Doi: none found
68. Tucker, H. (2000). Symposium: hormonal regulation of milk synthesis. Hormones, mammary growth, and lactation: a 41-year perspective. *Journal of Dairy Science*, 83(4), 874-884. doi: 10.3168/jds.S0022-0302(00)74951-4
69. Turner, L., Jacobsen, C., Humenczuk, M., Singhal, V., Moore, D., & Bell, H. (2001). The effects of lactation education and a prosthetic obturator appliance on feeding efficiency in infants with cleft lip and palate. *Cleft Palate-Craniofacial Journal*, 38(5), 519-524. doi: 10.1597/1545-1569(2001)
70. U.S. Department of Health & Human Service, National Institutes of Health. (2012, July 13). How are overweight and obesity diagnosed? Retrieved from <http://www.nhlbi.nih.gov/health/health-topics/topics/obe/diagnosis.html>
71. van Manen, M. (1990) Researching lived experience. *Human Science for an Action Sensitive Pedagogy*. The Althouse Press, Toronto, Canada.
72. Vio, F., Salazar, G., & Infante, C. (1991). Smoking during pregnancy and lactation and its effects on breast-milk volume. *American Journal of Clinical Nutrition*, 54(6), 1011-1016. Retrieved from [ajcn.nutrition.org.proxy.cc.uic.edu/content/54/6/1011.long](http://ajcn.nutrition.org.proxy.cc.uic.edu/content/54/6/1011.long)

73. White, B.A., & Porterfield, S.P. (2013). *Endocrine and Reproductive Physiology* (4<sup>th</sup> ed.). Philadelphia, PA: Elsevier Mosby
74. Weinberg, D. (Ed.). (2002). *Qualitative research methods*. Malden, MA: Blackwell Publishers Inc.
75. Willis, C., & Livingstone, V. (1995). Infant insufficient milk syndrome associated with maternal postpartum hemorrhage. *Journal Of Human Lactation*, 11(2), 123-126.  
doi: 10.1177/089033449501100218
76. World Health Organization. (2009). *Infant and young child feeding*. France: WHO Press

## APPENDICES

## APPENDIX A

### Assessment for Eligibility

- 1) Are you at least 18 years of age? Yes No
- 2) Are you able to speak and read in the English language? Yes No
- 3) Have you had a biological child that you breastfed? Yes No
- 4) Was your pregnancy full-term with one baby? Yes No
- 5) During your breastfeeding experience were you diagnosed with low milk supply by either a lactation consultant, pediatric provider, or your midwife/OB provider? Yes NO
- 6) After diagnosis, did you continue to breastfeed and if so for what duration of time?
- 7) Are you able to recall the details of your experience of breastfeeding with low milk supply? Yes No

## APPENDIX B

### Interview Guide

Hello \_\_\_\_\_, first I would like to thank you for agreeing to participate in this interview. I would also like to take a moment to explain what we will be doing today and answer any questions if you have them. I am currently a PhD student in the College of Nursing at UIC and I am completing my dissertation. I am interested in the breastfeeding experiences of women with low milk supply. I became interested in this after my own breast feeding difficulties and milk supply issues. The interview that we are completing today will later become part of my dissertation. I will be tape recording this interview and taking notes. I also want you to know that this interview is confidential and your name will not be used in the write-up; instead each interview will be given a number and contain no identifiable information. Do you have any questions before we begin? Lastly, before we get started I would like you to know that you can take as much time in answering these questions as you need to give as much detail as you can remember.

- I would like you to begin with your first pregnancy and tell me your memories about how you came to your decision to breastfeed (Depending on if the mother has had more than one child and more than one breastfeeding experience with low milk supply)
- Please tell me about your earliest breastfeeding memories after the birth of your child
- Please share with me the details about when you first learned that you had low milk supply and the details of what happened after that

#### Probes

- ❖ Where were you when you learned about your low milk supply
- ❖ Were you diagnosed with a reason for your low milk supply
- ❖ Did you receive support after finding out about your low milk supply
- ❖ How did you choose to supplement your infant; what factors played a role in your decision
- ❖ How long did you choose to give your own milk; what factors played a role in your decision
- ❖ How did this experience impact you
- ❖ How did this experience influence your subsequent pregnancies if applicable
- If there are additional pregnancies we will discuss the same questions for each additional pregnancy

During the process of the interview I plan to use silence as needed and also observe the non-verbal cues of my participant.

At the end I will thank my participant for sharing her story with me and for her time.

## Appendix B IRB Approval Letter

### UNIVERSITY OF ILLINOIS AT CHICAGO

Office for the Protection of Research Subjects (OPRS)  
Office of the Vice Chancellor for Research (MC 672)  
203 Administrative Office Building  
1737 West Polk Street  
Chicago, Illinois 60612-7227

#### Approval Notice Initial Review (Response To Modifications)

August 6, 2015

Erin Farah, MSN  
Women, Child, & Family Health Science  
845 S Damen Avenue  
M/C 802  
Chicago, IL 60612

**RE: Protocol # 2015-0597**  
**“Understanding how Women with Low Milk Supply Experience Breastfeeding”**

Dear Ms. Farah:

**Please note that the training credits for faculty sponsor, Carrie Klima will expire on August 20, 2015.** All UIC investigators and key research personnel involved in human subject research must complete a minimum of two hours of continuing education in human subject protection every two years. For further information, please see the OPRS website:  
<http://tigger.uic.edu/depts/ovcr/research/protocolreview/irb/education/index.shtml>

Your Initial Review (Response To Modifications) was reviewed and approved by the Expedited review process on July 28, 2015. You may now begin your research

Please note the following information about your approved research protocol:

**Protocol Approval Period:** July 28, 2015 - July 27, 2016  
**Approved Subject Enrollment #:** 20  
**Additional Determinations for Research Involving Minors:** These determinations have not been made for this study since it has not been approved for enrollment of minors.  
**Performance Sites:** UIC  
**Sponsor:** None  
**PAF#:** Not applicable  
**Research Protocol(s):**  
a) Understanding how Women with Low Milk Supply Experience Breastfeeding; Version 1, 06/03/2015

**Recruitment Material(s):**  
a) Script for Eligibility; Version 2, 08/04/2015  
b) Assessment Eligibility Form; Version 2, 08/04/2015

Phone: 312-996-1711

<http://www.uic.edu/depts/ovcr/oprs/>

FAX: 312-413-2929

c) "Research Participants Needed" no footer

**Informed Consent(s):**

- a) Low Milk Production; Version 2, 07/22/2015
- b) A waiver of documentation of informed consent has been granted under 45 CFR 46.117 and an alteration of consent has been granted under 45 CFR 46.116(d) for recruitment purposes only; minimal risk; verbal consent to screening/eligibility questions will be obtained; written consent/ will be obtained at enrollment.

Your research meets the criteria for expedited review as defined in 45 CFR 46.110(b)(1) under the following specific category(ies):

- (6) Collection of data from voice, video, digital, or image recordings made for research purposes.,
- (7) Research on individual or group characteristics or behavior (including but not limited to research on perception, cognition, motivation, identity, language, communication, cultural beliefs or practices and social behavior) or research employing survey, interview, oral history, focus group, program evaluation, human factors evaluation, or quality assurance methodologies.

**Please note the Review History of this submission:**

Receipt Date	Submission Type	Review Process	Review Date	Review Action
06/03/2015	Initial Review	Expedited	06/09/2015	Modifications Required
07/22/2015	Response To Modifications	Expedited	07/28/2015	Approved

Please remember to:

→ Use your **research protocol number** (2015-0597) on any documents or correspondence with the IRB concerning your research protocol.

→ Review and comply with all requirements on the enclosure,

**"UIC Investigator Responsibilities, Protection of Human Research Subjects"**

<http://tiger.uic.edu/depts/ovcr/research/protocolreview/irb/policies/0924.pdf>

**Please note that the UIC IRB has the prerogative and authority to ask further questions, seek additional information, require further modifications, or monitor the conduct of your research and the consent process.**

**Please be aware that if the scope of work in the grant/project changes, the protocol must be amended and approved by the UIC IRB before the initiation of the change.**

We wish you the best as you conduct your research. If you have any questions or need further help, please contact OPRS at (312) 996-1711 or me at (312) 355-0816. Please send any correspondence about this protocol to OPRS at 203 AOB, M/C 672.

Sincerely,



Alison Santiago, MSW, MJ  
IRB Coordinator, IRB # 2  
Office for the Protection of Research Subjects

Enclosure(s):

- 1. UIC Investigator Responsibilities, Protection of Human Research Subjects**
- 2. Informed Consent Document(s):**
  - a) Low Milk Production; Version 2, 07/22/2015
- 3. Recruiting Material(s):**
  - a) Script for Eligibility; Version 2, 08/04/2015
  - b) Assessment Eligibility Form; Version 2, 08/04/2015
  - c) "Research Participants Needed" no footer

cc: Barbara McFarlin, Women, Child, & Family Health Science, M/C 802  
Carrie Klima (faculty sponsor), Women, Child, & Family Health Science, M/C 802

Curriculum Vitae

**Erin F. Farah, PhD, APN, CNM**  
**1836 W. 17<sup>th</sup> St.**  
**Chicago, IL 60608**

**Home: (312) 698-8893**

**Cell: (312) 330-3138**

**E-mail: [efarah1@uic.edu](mailto:efarah1@uic.edu)**

**Education:** University of Illinois at Chicago, College of Nursing  
Doctorate of Philosophy in Nursing, 2016

University of Illinois at Chicago, College of Nursing  
Master of Science, 2005  
Certified Nurse-Midwife  
Women's Health Care Nurse Practitioner

University of Illinois at Chicago, College of Nursing  
Bachelor of Science in Nursing, 2000

**Professional Experience:**

**October 2005-present University of Illinois at Chicago**

Certified Nurse Midwife

- ❖ Health care provider for well women care, antenatal care, intrapartum care and postpartum care
- ❖ Coordinator for Midwifery practice from July 2012-present

**August 2002-December 2005 Loyola University Medical Center**

Clinical Nurse III in Labor and Delivery

- ❖ Work as a bedside nurse in Labor and Delivery
- ❖ Perinatal HIV liaison
- ❖ Developed the units educational in-service meetings held once a month regarding various topics encountered in labor and delivery and women's health issues
- ❖ Serve as a resource to other nurses
- ❖ Preceptor of new nurses to the unit

**January 2005-May 2005 University of Illinois at Chicago**

Teaching Assistant

- ❖ Clinical Instructor for the undergraduate nursing course NUSC 345: Clinical Concepts and Processes in Women's and Family Health
- ❖ Instructor at the University of Illinois at Chicago Hospital holding a 50% position

**August 2004-December 2004 Office of the Inspector General**

Research Assistant

- ❖ Nurse consultant on various health care issues surrounding children and families within the DCFS system
- ❖ Assisted in the City of Chicago outreach project for perinatal HIV infection and participated in developing new protocol for minors entering into DCFS system

**January 15, 2001- June 2002 Rush Presbyterian St. Lukes Medical Center**

Clinical Nurse II in the Maternal-Child Float Pool

- ❖ Work as a bedside nurse in Postpartum, General Care Nursery, Special Care Nursery, Pediatrics, and the Pediatric Intensive Care Unit

**May 1999-May 2004 University of Illinois at Chicago**

Research Assistant to Dr. J. Holden

- ❖ Collaborating research assistant in a Neuroscience Laboratory
- ❖ In charge of running personal experiments and recording data
- ❖ In charge of inputting data and running statistical measures
- ❖ Collaborate with the coordinator to properly maintain laboratory. Including keeping track of materials, making solution, processing tissues, and using a Neurolucida program to locate markers in brain tissue

**Professional Service:**

2005-present      Active Member, American College of Nurse-Midwives

**Licensure and Certification:**

ACNM Certification Council, Licensed 2005, exp 12/2018  
 RN, Illinois, Licensed 2001, Reactivated 2012, exp 5/2016  
 APN, Illinois, Licensed 2005, Reactivated 2012, exp 5/2016  
 Cardiopulmonary Resuscitation Provider (CPR), current status  
 Neonatal Resuscitation Provider (NRP), current status