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Review Article

Lactation and cardiovascular health: A comprehensive review

Xuban Palau Villarreal^a, Viviana De Assis^b, Daniela R. Crousillat^c, Amanda L. Nielson^d, Alex M. Parker^e, Maureen W. Groër^f, Adetola F. Louis-Jacques^{g,*}

^a College of Medicine, University of Florida, Gainesville, FL, USA

^b Department of Obstetrics and Gynecology, Brooke Army Medical Center, San Antonio, TX, USA

^c Division of Cardiovascular Sciences, Department of Medicine and Obstetrics and Gynecology, University of South Florida Morsani College of Medicine, Tampa, FL, USA

^d College of Medicine, Florida State University, Tallahassee, FL, USA

^e Section of Cardiovascular Medicine, Department of Medicine, University of Florida, Gainesville, FL, USA

^f College of Nursing, University of Tennessee, Knoxville, TN, USA

^g Department of Obstetrics and Gynecology, University of Florida, Gainesville, FL, USA

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ABSTRACT

Lactation is increasingly recognized as a modifiable factor influencing long-term maternal cardiovascular health. This review synthesizes current evidence on the association between lactation and cardiometabolic outcomes such as hypertension, type 2 diabetes, coronary artery disease, dyslipidemia, metabolic syndrome, and cardiovascular events. This narrative review also addresses clinical, systemic, and policy barriers that impede lactation success.

1. Introduction

Evidence on the positive association between lactation and maternal cardiometabolic health has been developing over the past three decades [1–9]. Although women are traditionally diagnosed with cardiovascular disease (CVD) at an older age as compared with men, CVD risk factors, which are associated with lifetime CVD risk, are rising among younger women [10,11]. Given the rising burden of CVD risk among younger women, lactation may represent an effective strategy to reduce CVD incidence [12].

Over the last decade, reproductive factors such as early and late menarche, polycystic ovarian syndrome, premature menopause, adverse pregnancy outcomes (APOs), and *suboptimal lactation* have been recognized as CVD risk factors [13–22]. Additionally, adverse social determinants of health are barriers to optimal lactation and CV health in women. In the United States, American Indian or Alaskan Native and non-Hispanic Black women have the lowest rates of breastfeeding and the highest rates of CV-related mortality [23,24]. Persisting structural inequities continue to widen the gaps in health care quality and access that contribute to non-biological differences in CV risk [25].

The objective of this review is to synthesize current evidence on the association between lactation and cardiometabolic outcomes. This review also addresses clinical and systemic barriers that impede lactation success. Our goal is to increase knowledge of maternal cardiometabolic benefits of lactation, and the recognition of suboptimal lactation as a cardiovascular risk factor.

2. Pregnancy, lactation, and the cardiovascular system

2.1. Cardiovascular adaptations during pregnancy

During pregnancy, the maternal CV system undergoes adaptations to support both maternal and fetal needs. In the first trimester, one of the earliest changes observed is an increase in maternal heart rate (HR) mediated by estrogen-regulated increase in myocardial alpha-receptors, which progressively rises to 15–25 % above baseline by the third trimester. This change supports a significant increase in cardiac output (CO) as the systemic vascular resistance drops to maximize uteroplacental circulation [26,27].

In the second trimester, progesterone primarily induces vasodilation,

* Corresponding author at: Department of Obstetrics and Gynecology, University of Florida, PO Box 100294, 1600 SW Archer Road, Gainesville, FL 32610, USA.
E-mail address: louisjacquesa@ufl.edu (A.F. Louis-Jacques).

which lowers the resistance in the placental bed and contributes to a gradual decrease in blood pressure (5–15 mmHg in systolic and diastolic blood pressure about 24 weeks gestation) [28]. Concurrently, plasma volume continues to increase, reaching an approximate 40 % increase by the third trimester. This increase in plasma volume drives a 45 % increase in CO. [29] These CV changes are vital for optimal perfusion of both maternal and fetal tissues [30].

By the third trimester, systemic blood pressure often returns to pre-pregnancy levels [28,31]. Physical manifestations of CV adaptations can include mild tachycardia, peripheral edema, systolic flow murmur, S3 gallop, and lateral displacement of the left ventricular apex [28,31]. Immediately following delivery, there is a rapid increase in systemic vascular resistance, autotransfusion of blood from the contracting uterus, and a gradual decrease in the maternal HR to baseline by approximately 10 days postpartum [26,30]. Women who consistently lactated for 5 months had lower systolic blood pressure and heart rate compared to their counterparts who formula-fed their newborns (Fig. 1) [32].

2.2. Hormonal regulation and cardiovascular changes during lactation

The neuropeptide hormone oxytocin, synthesized in the hypothalamus and released into the circulation by the posterior pituitary (Fig. 1) surges during lactation and causes vasodilatory effects [33]. These vasodilatory effects promote relaxation of the vascular smooth muscle which leads to decreased peripheral resistance, HR, and blood pressure, as well as increased CO. [33] Moreover, oxytocin has been shown to

have anti-atherosclerotic properties and anti-obesogenic effects [34]. Oxytocin moderates factors directly linked to atherosclerotic CVD such as inflammation, weight gain, food intake, and insulin resistance [34]. Additionally, oxytocin surge with lactation initiation can promote uterine involution protecting against postpartum blood loss which can indirectly benefit CV health [35].

Oxytocin is an important trigger for prolactin secretion [36]. Prolactin is a hormone secreted by the maternal pituitary gland which acts on the mammary glands to promote breast milk synthesis (Fig. 1) [37]. Prolactin secretion increases during late gestation and in the postpartum period. Prolactin can be cleaved into a vasculo-toxic product that in mice has been associated with endothelial dysfunction, microvascular disease, and myocyte death [37]. Dysregulation of the prolactin axis in pregnancy and postpartum has been linked to impaired vascular adaptation that has been implicated in both preeclampsia and peripartum cardiomyopathy [37,38]. In obese or overweight women, lower levels of oxytocin may reduce prolactin secretion, which could lead to lactation difficulties and earlier weaning [39,40].

2.3. Metabolic adaptations during pregnancy and lactation

Weight gain and an increase in visceral adiposity during pregnancy are theorized to support the increase in caloric expenditure postpartum which is evolutionarily designed to support a growing infant with lactation [41,42]. Insulin resistance and glucose intolerance increase during pregnancy, resulting in greater maternal fat reserves [43]. Significant elevations of low-density lipoprotein and triglycerides during

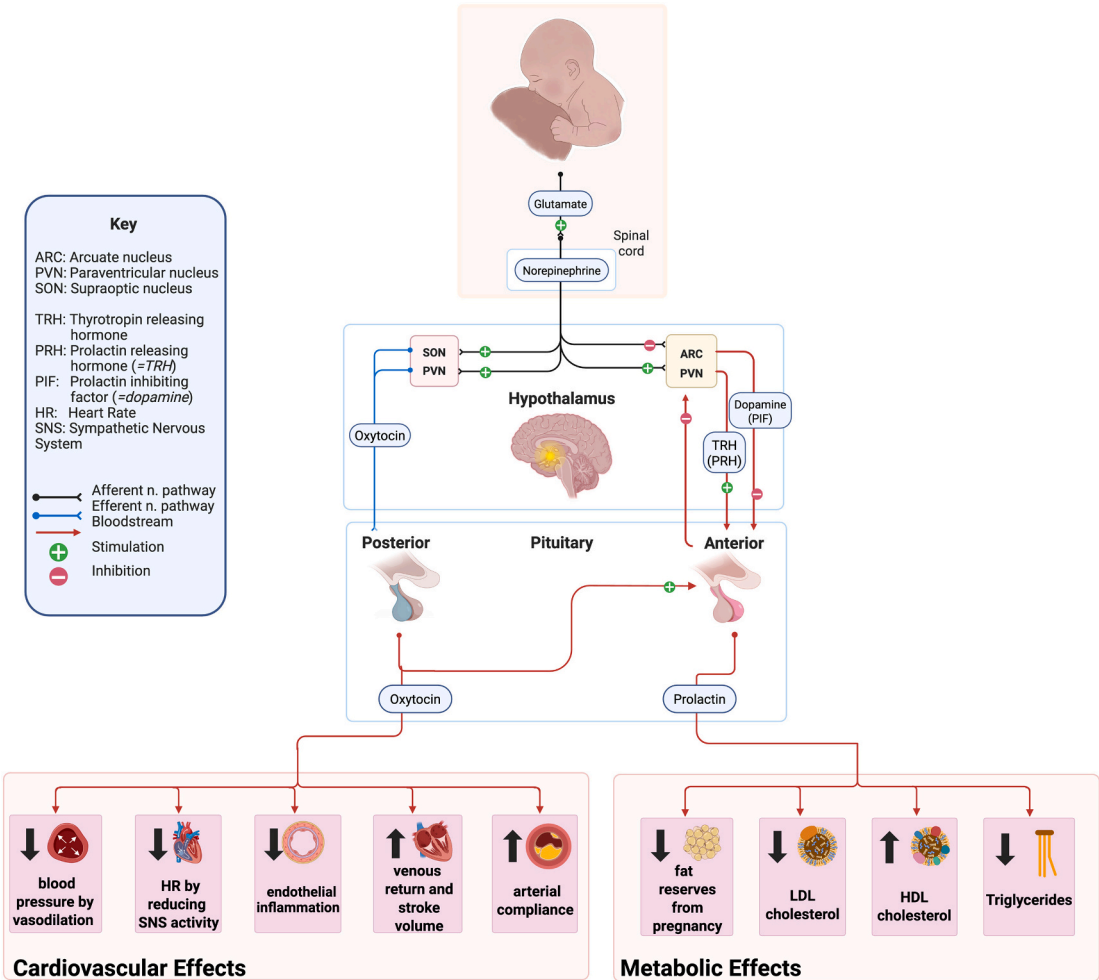


Fig. 1. Breastfeeding and its effect on the cardiovascular and metabolic systems [61].

pregnancy allow for greater transfer of fatty acids to the growing fetus while creating a more atherogenic profile in the mother that may lead to endothelial damage [5,44].

During lactation, large quantities of carbohydrates, protein, and lipids are exported through the mammary gland into the offspring via breast milk. This process is energetically expensive for the mother, and to meet the nutrient demand, increased food intake or mobilization of storage depots is required [45]. This lipid mobilization and increased energy expenditure associated with lactation is thought to lead to a faster return to maternal pre-pregnancy homeostasis [46].

Dietary intake and energy expenditure during lactation can be associated with weight loss [47]. Butte et al. found a mean mobilization of 156 kcal/day from fat stores in women who exclusively lactated [48]. Women who consumed more calories, had less weight loss [49]. Within the first 2–3 months postpartum, multiple studies have shown that formula-feeding mothers consumed 600–800 fewer calories than lactating mothers [50,51]. Another study found that women consumed an additional 300 kcal/day during lactation and expended 200 kcal/day less in physical activity to support the estimated 480 kcal/day needed for lactation [12,52]. Women may compensate for the increased energy demand of lactation by increasing their dietary intake and reducing their energy expenditure, rather than mobilizing fat stores [50–52].

Women who lactated beyond 3 months postpartum experienced greater weight loss compared to those who did not lactate or only lactated for 3 months [53]. However, the association between postpartum weight loss and lactation is mixed [54–57]. A systemic review of 43 observational studies found insufficient evidence to suggest a relationship between lactation and postpartum weight change [58]. Of these 43 studies, five obtained objective measurements for postpartum weight change and adjusted for key covariates such as gestational weight gain and parity [58]. Four of the 5 studies reported a direct positive association between duration of lactation and postpartum weight loss [58]. Moreover, there may be an association between the duration of lactation and a reduction in visceral adipose tissue [59,60].

3. Implications of lactational history on maternal cardiovascular health

Lactation has been associated with reduction in major CV risk factors such as metabolic syndrome, type II diabetes mellitus (T2DM) and hypertension (Fig. 2) [62,63]. Each added month of lactation was associated with a 1 % decreased lifetime risk of developing T2DM [62,64]. Individuals affected by GDM who lactate for >6 months had the lowest risk of developing postpartum diabetes with the reduced risk extending up to 2 years post-delivery [44,65]. This T2DM risk reduction associated with lactation can reduce the risk of CVD [7].

Lactation is also associated with hypertension reduction (Fig. 2) [4,32,40,66]. Lee et al. reported that women who had never lactated showed an increased risk of premenopausal hypertension [5]. Women with cumulative lactation of one year or more can experience a 12 % lower risk of hypertension [4,32,40,66]. This risk reduction appears to be dose-dependent with women who lactated for 0–6 months, 6–12 months, or > 12 months showing an 8 %, 11 %, and 12 % lower risk, respectively [63,67–69]. Compared to women who never lactated, those who lactated had a 7 % lower risk of developing hypertension, after adjusting for age, body mass index, and smoking [63,69]. Additionally, lactation intensity may influence hypertension risk [70]. A study comparing exclusive, almost exclusive, predominant, and partial lactation showed that exclusive and almost exclusive lactation was associated with lower diastolic blood pressure [70].

Lactation has been recognized for its long-lasting metabolic benefits, mothers who lactate were on average 8 kg lighter 6 years after cessation of lactation [64]. Besides, lactation can improve insulin requirements, glucose tolerance, and lipid metabolism in the postpartum period [52,71]. These benefits may be associated with the 500 daily caloric deficit with full lactation [47].

In the U.S., dyslipidemia is highest in Hispanic/Latina women (37 % with total cholesterol levels of ≥ 200 mg/dL) [72]. While atherogenic lipid profiles have been shown to increase the long-term risk of CVD, lactation has been associated with improved lipid homeostasis (Fig. 2) [2,3,73,74]. Duration of lifetime lactation has been inversely associated

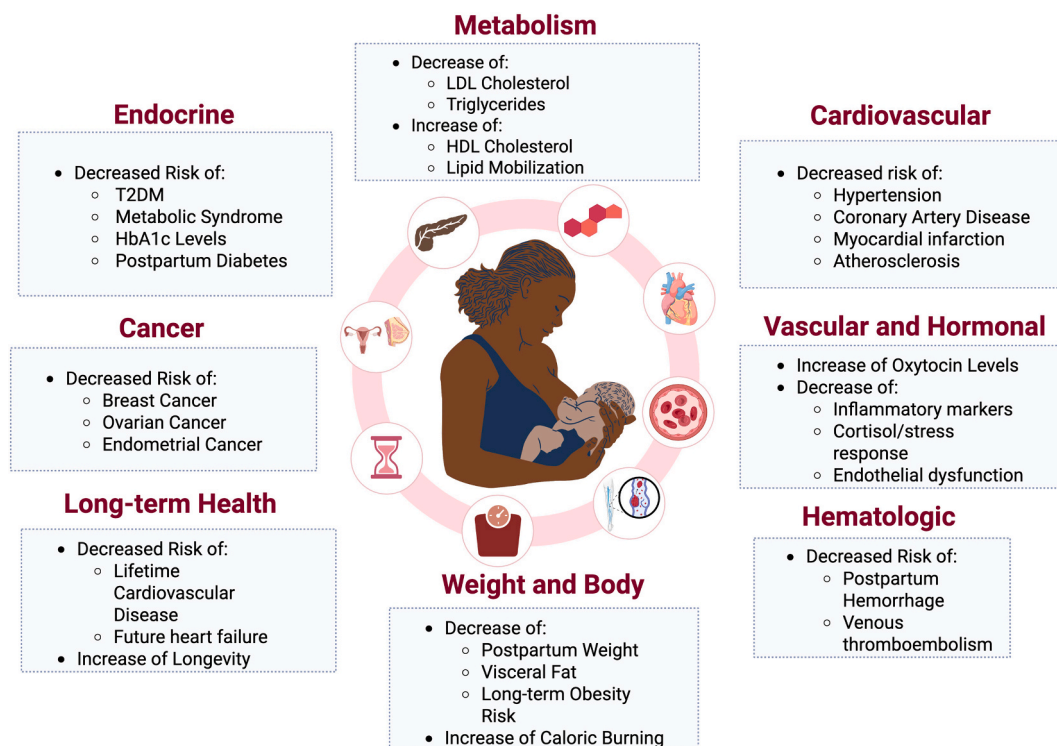


Fig. 2. Maternal benefits associated with lactation [76].

with serum triglycerides, total cholesterol, and low-density lipoprotein cholesterol [46]. A positive association between lactation and HDL metabolism has been reported at 6 weeks and 3 months postpartum, with higher HDL levels until weaning for those who lactate for 1 year [2,3,73,74]. In the Keiser SWIFT cohort, a greater lactation frequency was associated with higher HDL and lower LDL at 6 to 9 weeks postpartum [41]. Prior evaluation of triglyceride levels, has shown a return to baseline 13 weeks earlier in lactating than non-lactating women [42]. Lactation may decrease the risk of CVD by improving lipid homeostasis [7,75].

Metabolic syndrome refers to a clustering of metabolic abnormalities including insulin resistance, dyslipidemia, hypertension, and obesity [43]. Metabolic syndrome can increase the risk of diabetes mellitus, CVD, and major CV events [8,43]. Nguyen et al. observed that lactation can decrease the risk of developing metabolic syndrome [46]. Several studies have reported an inverse association between the duration of lactation and the incidence of metabolic syndrome in midlife parous women, indicating that longer lactation duration showed a lower incidence of metabolic syndrome [9,74]. Moreover, Magnus et al. highlighted that lactation could improve the cardiometabolic profile by reducing lower body mass index, waist circumference, and levels of C-reactive protein, triglycerides, insulin, and proinsulin [45]. This improved cardiometabolic profile was observed decades after cessation of lactation, further demonstrating the potential long-term metabolic benefits of lactation [45].

3.1. Implications of lactation history on cardiovascular health in animal studies

Current animal models for lactation and CV health, focus on the effects of early weaning, exclusive feeding, overfeeding, or on the on the maternal dietary composition and its impact on long-term CVD risk and possible changes in metabolism through epigenetic mechanisms on the offspring [77–79]. Investigators, measured regional changes in lipoprotein lipase activity as a proxy for fat deposition in the rat [80]. Fat deposition was noted to increase during pregnancy, but with lactation, storage of lipids in adipose tissue ceased, and uptake into mammary tissue increased as lipids were transferred into milk [80]. At 20 days postpartum, lactating animals had smaller adipose cells and lower peripheral lipoprotein lipase activity than nonlactating controls [81]. These animal studies of longer-term lactation show similar results to human studies after longer durations of lactation [53].

Some limitations of animal studies include the controlled nutrition in laboratory settings and sometimes separation of pups. The diverse diets and caloric expenditures of humans, mixed feeding—combining formula and lactation in differing proportions—makes it more challenging to draw direct comparisons to studies in animals.

4. Lactation's influence in mitigating sequelae of hypertensive disorders of pregnancy (HDP)

Hypertensive disorders of pregnancy (HDP) can present as a risk factor for developing CVD later in life [82]. Efforts are being made to understand the cardioprotective effect of lactation in HDP [82]. Countouris et al. compared lactating women with preeclampsia, lactating women with gestational hypertension, and normotensive lactating women and found both lower systolic and diastolic blood pressures in women with gestational hypertension [49]. Another study assessed the impact of lactation on preeclampsia, and showed women who had lactated in previous pregnancies had a lower risk of preeclampsia than women who had never lactated [83].

5. Lactation's influence on future cardiovascular events

5.1. Myocardial infarction (MI)

In a cohort study, an inverse association was found between lactation and MI, independent of risk factors of CVD [84]. Women with a lifetime lactation of >23 months presented a higher risk reduction for MI compared to those with shorter lactation duration [84]. Furthermore, a meta-analysis demonstrated that parous women who had ever lactated had a reduced risk of MI (HR 0.86 [95 % CI 0.78, 0.95]) compared to parous women who had never lactated [63].

5.2. Coronary artery disease

There is also a relationship between lactation and coronary artery disease (CAD). Schwarz et al. reported that mothers who had never lactated were more likely to develop aortic and coronary artery calcification compared to mothers who had lactated for at least 3 months after adjusting for CVD risk factors [85]. However, there were no differences between exclusive and non-exclusive lactation groups [85]. Tschiderer et al. reported that 12-month lactation duration was negatively associated with CAD risk, and longer than 12 months of lactation had a progressive decreased risk for CAD [63]. However, another study noted a plateau of reduced CAD risk between 12 and 48 months, suggesting the unknown additional CAD risk reduction past 12 months of lactation [86].

Even though no studies have assessed the effect of lactation on carotid flow, many have studied the relationship between lactation and carotid intima-media thickness. In a study by Augoulea et al., postmenopausal women who lactated for at least 6 months had lower carotid intima-media thickness and reduced prevalence of subclinical atherosclerosis [87]. Perez-Roncero et al. found that women, who had lactated for ³6 months, had a lower carotid intima-media thickness and higher HDL cholesterol levels than women who lactated for <6 months [88].

5.3. Stroke and cerebrovascular accidents

Lactation is associated with a reduced risk of stroke and cerebrovascular accidents in parous postmenopausal women [86,89,90]. A study showed that women with a lifetime lactation duration of 7 months had a lower risk of stroke (HR 0.52 [95 % CI, 0.50–0.55] to 0.64 [95 % CI, 0.59–0.69]) and intracerebral hemorrhage (HR 0.56 [95 % CI, 0.49–0.63] to 0.78 [95 % CI, 0.64–0.96]) compared to those who never lactated [89]. However, reduced risk of subarachnoid hemorrhage was observed only with a lifetime duration of lactation >24 months (HR 0.61 [95 % CI, 0.57–0.66]) [89]. Additionally, Jacobson et al. reported that women with a lifetime lactation of 1 month or longer had a 23 % decreased risk of stroke (HR 0.77 [95 % CI, 0.70–0.84]) compared to those who had never lactated [90].

6. The interplay between cardiovascular health and lactation

We discussed the potential benefits of lactation on CV health, however, it is known that CV health can also influence lactation [91]. Burgess et al. suggests that women with a history of HDP are less likely to initiate and continue lactation when compared to normotensive women [91]. In addition, data from the All Our Families Cohort demonstrated that women with HDP had lower rates of non-exclusive lactation at 4 months and a shorter lactation duration at 12 months compared to women without HDP [92].

The lower rates of lactation duration and exclusivity were observed in groups with an increased need for medical interventions and postpartum complications, which may present as lactation barriers [91]. Among these, women with HDP reported that the most common reason to stop or not initiate lactation was feeling sick or that they had to stop for medical reasons [91]. Moreover, the study notes that late-onset

preeclampsia (≥ 34 weeks' gestation) displayed higher lactation intent than those women with early-onset preeclampsia (< 34 weeks' gestation) [91]. Women with early onset preeclampsia were more likely to have infants in the NICU highlighting increased maternal/infant separation and hindered lactation initiation and establishment of the milk supply [91]. The maternal-infant separation from preterm birth, maternal complications postpartum, and psychosocial stressors associated with HDP diagnosis may discourage lactation initiation [91]. Implementation of Spatz et al.'s ten steps for promoting and protecting breastfeeding for vulnerable infants during hospitalization may improve lactation support [93]. Women with cardiac disease managed at a comprehensive cardio obstetrical program for their pregnancy demonstrated high lactation initiation rates (85 %) but low continuation rates in the acute postpartum period (40 %) [94] highlighting the need for interventions both at the time of delivery admission in addition to continuation of resources and support postpartum.

While few medications are contraindicated during lactation, there is a gap in clinician knowledge regarding medication compatibility with lactation [95,96]. Tigka et al. reported that 39 out of 57 lactating women were inaccurately counseled to discontinue lactation by a clinician despite medication compatibility [97]. For instance, Colaceci et al. found medication package leaflets reported higher risk profiles than evidence-based scientific sources for the use of antihypertensive medications during lactation [98]. Inconsistent or inaccurate information on the use of medications during lactation may lead to cautious and harmful recommendations [98].

7. Potential negative impact of lactation on cardiovascular health

Peripartum cardiomyopathy (PPCM) is defined as a decline in maternal cardiac function that occurs in the third trimester or first few months postpartum in the absence of other identifiable causes [99]. PPCM is more prevalent among women of advanced maternal age, multi-gestational pregnancies, and HDP, however, the role of prolactin and lactation have also been linked in both the pathogenesis as well as in the recovery of left ventricular function [100,101]. During pregnancy and postpartum, an increase in oxidative stress upregulates cathepsin D which cleaves prolactin into 16-kDa prolactin fragment which in mice models has been shown to lead to endothelial damage and increase cardiomyocyte apoptosis. Therefore, bromocriptine, a D2 receptor agonist that inhibits prolactin secretion, has been shown in several studies to improve the likelihood of recovery of LV systolic function. A systematic review of 8 studies of 593 patients with PPCM demonstrated increased survival (91.6 % versus 83.9 %) and showed a higher LVEF (53.3 % versus 41.8 %) with bromocriptine treatment [37,102].

The current ACC/AHA guidelines recommend consideration of use of bromocriptine for lactation suppression in severe LV systolic dysfunction (LVEF < 25 %) or cardiogenic shock in the context of PPCM but highlight the marked benefits of lactation and recommend shared decision-making until more data is available. Among the Investigations of the Pregnancy-Associated Cardiomyopathy (IPAC) study, Koczo et al. followed 100 women diagnosed with PPCM throughout their first year postpartum and assessed the lactation impact on PPCM in lactating and non-lactating women. The study found higher circulating levels of prolactin among the lactation cohort and no differences were observed in LVEF recovery between non-lactating women vs 6 and 12-month lactating women [103]. The U.S.-based REBIRTH (Randomized Evaluation of Bromocriptine in Myocardial Recovery Therapy for Peripartum Cardiomyopathy, NCT05180773) comparing bromocriptine for lactation suppression versus lactation is currently ongoing and aims to determine if bromocriptine is associated with better outcomes than standard medical therapy for heart failure [100].

8. Optimizing lactation in socially vulnerable populations

8.1. Barriers to lactation

In the U.S. the prevalence of lactation initiation is high (84.1 %) with disparities based on social determinants of health [104]. Black women have the lowest rate of lactation initiation (75.4 %) compared to the highest rate observed in Asian mothers (92.7 %) [104]. Differences in lactation rates are also influenced by income, language, age, parity, education, employment benefits, access to health care, and lactation support [105–109]. Among 142,643 new mothers in the 2016–2019 Pregnancy Risk Assessment Monitoring System (PRAMS), 60.4 % reported lactating up to 3 months and 54.7 % up to 6 months. High school education or lower, Medicaid insurance coverage, and absence of lactation support from the health care team was associated with lower breastfeeding rates [105]. Mothers who reported food insecurity, including participation in WIC (Women, Infants, and Children), were less likely to continue lactation. Participants reported that the financial burden of increased food intake to meet caloric demands of lactation, financial pressures to return to work sooner, and the cost of lactation equipment were reasons why they ceased lactation [106]. Lower breastfeeding education and support, and lower household income was associated with lower lactation initiation among Black women [107].

Hispanic women have slightly lower rates of lactation initiation compared to White women. However, duration and exclusivity disparities are present [104]. Cultural factors including nativity and acculturation can present as additional elements impacting lactation rates among Hispanic women. Foreign-born Hispanic women and Spanish-speaking women had higher lactation rates compared to U.S.-born Hispanic women [108]. Lastly, Singh et al. found that immigrant women had higher rates of lactation initiation and longer lactation duration than U.S.-born women [109]. Thus, lactation promotion programs should target ethnic-immigrant and social groups with lower lactation rates (Fig. 3) [109].

8.2. Strategies for improving lactation support

Multi-level interventions including national, state legislative, and medical provider support are needed to enable breastfeeding, and to maximize the health benefit for all lactating individuals. Interventions include paid parental leave, workplace accommodations, and expansion of insurance coverage for lactation support (Fig. 3). The expansion of postpartum Medicaid for up to 12 months postpartum in up to 48 U.S. states has demonstrated improved access to lactation supplies and increased favorable lactation outcomes [110]. Community-based peer support strategies include the engagement of community health workers, doulas, and community outreach to help reinforce lactation, particularly among historically marginalized communities with low lactation rates (Fig. 3) [111]. Importantly, educating healthcare professionals on the effect of systemic racism and implicit bias on lactation is essential (Fig. 3). Educational campaigns surrounding the benefits of lactation and the facilitation of community peer counseling and support groups in the community are vital to addressing cultural norms, stigma, and challenges.

9. Future research directions

Despite the growing evidence of the benefits of lactation in maternal CV health, there are remaining gaps in evidence [68]. Longitudinal studies with detailed lactation history such as duration, frequency, and exclusivity, biomarkers, and maternal CV health outcomes are needed. Such longitudinal studies could enhance clinical recommendations in lactation practice to maximize maternal CV health benefits. Mechanistic investigations are crucial to understanding the biological pathways through which lactation benefits maternal CV health. Racial and ethnic differences (socially based) in metabolic and inflammatory changes may

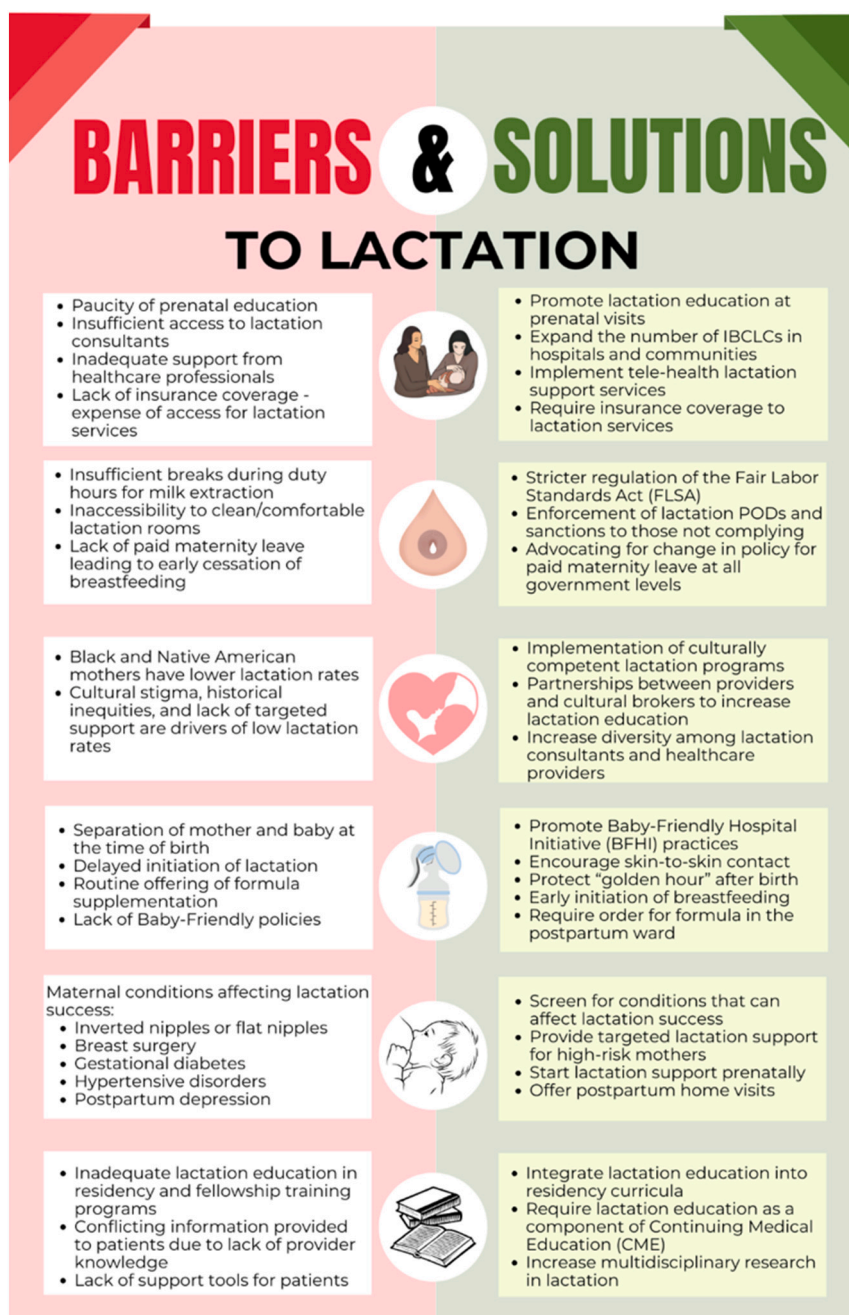


Fig. 3. Barriers and solutions to lactation.

show variations in CV benefits that need to be explored [112,113]. For instance, social determinants can contribute to the observed higher rates in diabetes and hypertension in Hispanic and Black women respectively which underscores the need for tailored preventative efforts [114]. Future studies investigating structural barriers such as inadequate maternity leave, limited access to lactation support, and healthcare inequities can aid the design of lactation interventions in diverse populations [115].

10. Conclusion

Lactation offers a unique period, with significant implications for prevention and intervention strategies for maternal CV health. By deepening our understanding of these connections, healthcare providers can better advocate for lactation as a cornerstone of maternal health.

Additionally, ensuring equitable access to lactation education and support through public health strategies can enhance maternal CV health across all populations [115].

CRediT authorship contribution statement

Xuban Palau Villarreal: Conceptualization, Investigation, Project administration, Visualization, Writing – original draft, Writing – review & editing. **Viviana De Assis:** Conceptualization, Investigation, Project administration, Visualization, Writing – original draft, Writing – review & editing. **Daniela R. Crousillat:** Conceptualization, Investigation, Writing – original draft, Writing – review & editing. **Amanda L. Nielson:** Investigation, Writing – original draft, Writing – review & editing. **Alex M. Parker:** Writing – review & editing. **Maureen W. Groër:** Writing – review & editing. **Adetola F. Louis-Jacques:** Writing – review & editing.

Conceptualization, Investigation, Supervision, Writing – original draft, Writing – review & editing.

Ethical statement

All authors affirm that this manuscript is an original work and complies with ethical standards for research and publication.

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Declaration of competing interest

All authors declare that they have no conflicts of interest.

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