Hormonal Physiology of Childbearing: Evidence and Implications for Women, Babies, and Maternity Care

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Executive Summary

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Introduction

This report examines current understandings of the hormonal physiology of childbearing, first in relation to the physiologic onset of labor at term and scheduled birth, and then through chapters addressing four impactful hormonal systems: oxytocin; beta-endorphins; epinephrine-norepinephrine (adrenaline-noradrenaline) and related stress hormone systems; and prolactin. Each chapter addresses physiologic hormonal processes followed by the possible impacts of common maternity care practices and interventions. The final chapter presents conclusions, a summary table, and recommendations.

The "hormonal physiology of childbearing" here refers to reproduction-related biologic processes from pregnancy through the postpartum and newborn periods in relation to innate, endogenous hormone systems. "Physiologic childbearing" refers to childbearing conforming to healthy biologic processes. Consistent and coherent evidence finds that physiologic childbearing facilitates beneficial (salutogenic) outcomes in women and babies by promoting fetal readiness for birth and safety during labor, enhancing labor effectiveness, providing physiologic help with labor stress and pain, promoting maternal and newborn transitions and maternal adaptations, and optimizing breastfeeding and maternal-infant attachment, among many processes.

The perinatal period is highly sensitive for mother and baby in relation to hormonal and other biologic processes. Practices that promote (through favorable policies and system capacities), support (with direct facilitating practices), and protect (from disturbance) physiologic childbearing may have amplified, ongoing benefits—for example, through supporting breastfeeding.

Contemporary childbearing has benefitted from many medical advances, and from highly skilled and committed maternity care providers, especially for mothers and babies who require special care. However, current high rates of maternity care interventions may be disadvantageous for the healthy majority. Common maternity care practices and interventions can impact the hormonal physiology of mother and baby, according to physiologic understandings and human and animal studies. Impacts on hormonal physiology and consequences for mother and/or baby may occur in the perinatal period or beyond. For example, prelabor cesareans are associated with reduced fetal/newborn epinephrine-norepinephrine due to loss of the "catecholamine surge," which may contribute to increased respiratory and other morbidities. Longer-term impacts from perinatal hormonal disruptions are possible in women and babies, according to provisional human findings and solid animal research.

Core hormonal physiology themes and principles recur throughout results synthesized in this report, revealing profound interconnections at many levels and over time, as follows:

Evolutionary origins. The hormonal physiology of childbearing has evolved over millions of years to optimize reproductive success. Maternal and infant survival at birth is obviously critical for reproductive success, but equally important for long-term survival are successful lactation and maternal-infant attachment immediately following birth. These hormonally-mediated processes are intertwined and continuous with the biologic processes of parturition. Disruption of perinatal hormonal physiology may thus impact not only labor and birth, but also breastfeeding and maternal-infant attachment. As humans share many reproductive processes with other mammals, animal research helps illuminate human hormonal physiology, especially where human research is currently limited.

Mother-baby dyad. Hormonal physiology is interrelated, coordinated, and mutually regulated between mother and baby to optimize outcomes for both. For example, maternal and fetal readiness for labor is precisely aligned at the physiologic onset of term labor to optimize labor efficiency and maternal and newborn transitions. Similarly, skin-to-skin contact after birth mutually regulates maternal and newborn oxytocin systems. As a general principle, effects on maternal hormonal physiology impact fetal/newborn hormonal physiology, and vice versa.

Beneficial hormonal physiology pathway. From pregnancy through labor and birth, breastfeeding, and maternal-infant attachment, hormonal processes of physiologic childbearing anticipate and prepare for upcoming processes and biological needs. For example, prelabor upregulation of maternal uterine oxytocin receptors promotes labor efficiency, and prelabor epinephrine-norepinephrine receptor upregulation optimizes fetal adaptations to labor hypoxia and newborn transitions via the fetal catecholamine surge.

Interorchestration among hormone systems. The hormone systems described here have complex interactions in the perinatal period, including promoting or inhibiting one another's activity. This can amplify hormonal effects, leading to the peaks that characterize physiologic birth. For example, late-labor oxytocin peaks, promoted by high levels of prolactin and oxytocin itself, assist with the pushing stage. Similarly, excessive stress and stress hormones may disrupt labor progress via hormonal interorchestration.

Cascade of intervention. Hormonal disruptions can be amplified when one intervention necessitates and leads to another that is used to monitor, prevent, or treat its side effects. This escalation of technology can further disrupt hormonal physiology and introduce extra risks for mother and baby. For example, the reduction in maternal oxytocin that generally follows administration of epidural analgesia may lead to use of synthetic oxytocin to compensate. Prolonged use of synthetic oxytocin may desensitize the oxytocin receptor system and increase the risk of postpartum hemorrhage.

Concern about long-term impacts. Non-physiologic exposures during the sensitive perinatal period may disrupt offspring hormone systems, with amplified and/or enduring biological, developmental, and/or behavioral impacts, as found in animal offspring, likely via epigenetic programming effects. High-quality, long-term human studies following fetal/newborn exposure to perinatal drugs and interventions are very limited. Thus, the current evidence-based approach to identifying safe and effective care, based on short-term follow-up and limited examination of hormonally-mediated outcomes such as breastfeeding, may not provide adequate safeguards for mothers and babies. Similarly, conventional shorter-term pharmacologic considerations of fetal/newborn drug exposure (e.g., dose, duration, metabolism) may not adequately safeguard the baby. Current levels of uncertainty about long-term impacts suggest research priorities and support avoiding unneeded interventions.

Physiologic Onset of Labor at Term

The physiologic (spontaneous) onset of term labor is a complex and incompletely understood process. Critical for survival, its timing is thought to be essentially determined by the baby's maturity, via fetal cortisol production, coordinated with the mother's readiness for parturition, via estrogen production and other processes. Timing of the physiologic onset of term labor is difficult to predict due to normal variation in the length of human gestation.

With the physiologic onset of labor at term, maternal and fetal systems are fully primed and precisely aligned for safe, effective, labor and birth, and for optimal postpartum physiologic transitions, including breastfeeding initiation and maternal-newborn attachment, according to physiologic understandings,

and human and animal studies. Physiologic prelabor preparations occur in the weeks, days, and (in animal studies) hours before the onset of labor. Maternal preparations include:

- rising estrogen levels, activating the uterus for an efficient labor
- cervical ripening due to increases in oxytocin and prostaglandin activity (receptors, levels)
- increasing inflammation, which also activates the cervix and uterus
- increasing uterine oxytocin receptors, giving effective contractions during labor, and after birth to reduce bleeding
- increasing brain-based (central) receptors for beta-endorphins (animal studies), contributing to endogenous analgesia in labor
- elevations in mammary and central oxytocin and prolactin receptors (animal studies), which promote breastfeeding and maternal-infant attachment after birth

Similarly, processes before and during labor foster the baby's adaptations for labor and peak readiness for the critical transition to life outside the womb. These include:

- prelabor maturing of the lungs and other organ systems, and of the processes that clear lung fluid in labor
- prelabor development of oxytocin neuroprotective processes (animal studies)
- prelabor increase in epinephrine-norepinephrine receptors, giving protection from labor hypoxia via the late-labor epinephrine-norepinephrine (catecholamine) surge
- in-labor preservation of blood supply to heart and brain, via the catecholamine surge, with neuroprotective effects
- in-labor catecholamine-mediated preparations that will promote newborn breathing, energy and glucose production, and heat regulation

Possible Impacts of Scheduled Birth

Scheduled birth—whether by labor induction or prelabor cesarean section—benefits mother and/or baby in selected circumstances. However, it may also significantly disrupt the processes discussed above.

Possible maternal impacts of scheduled birth include:

- reduced contraction efficiency leading to risks of failed induction, instrumental birth (induction), and postpartum hemorrhage (induction, prelabor cesarean)
- reduction in prelabor oxytocin and prolactin receptor peaks in the breasts and brain (animal studies) with potential impacts on breastfeeding, maternal adaptations, and maternal-infant attachment (induction, prelabor cesarean)

Possible impacts of scheduled birth on the baby include:

- immature protective processes, including the catecholamine surge, with increased vulnerability to labor hypoxia and "fetal distress" (induction)
- increased risks of postpartum breathing difficulties, hypoglycemia, and hypothermia due to lack of exposure to catecholamine surge (prelabor cesarean)
- reduced maturity of brain, brain-hormone, and other organ systems (induction, prelabor cesarean)
- long-term offspring impacts (animal studies), likely via epigenetic programming effects (cesarean section, plausibly relevant to induction)

These are crucial knowledge gaps given the high incidence of scheduled birth.

Oxytocin: Normal Physiology

Oxytocin is a powerful reproductive hormone with widespread effects on the brain and body of all mammals, for example, by mediating sperm ejection, labor contractions, and milk ejection. Oxytocin also reduces stress by centrally activating the parasympathetic nervous system, which promotes calm, connection, healing, and growth; and by reducing activity in the sympathetic nervous system, which reduces fear, stress, and stress hormones, and increases sociability. Oxytocin has a short half-life, but its effects can be prolonged because it modulates other brain-hormone systems (neuromodulation).

In the perinatal period, oxytocin optimizes labor, birth, and postpartum transitions of mother and baby through:

- central oxytocin release into the maternal bloodstream, causing rhythmic uterine contractions, including the late-labor oxytocin surge that benefits pushing (Ferguson reflex)
- central calming and analgesic effects in mothers and babies in labor through the postpartum period
- positive feedback of central oxytocin on itself, especially in multiparous mothers, augmenting and accelerating in-labor effects (animal studies)
- postpartum maternal adaptations that reduce stress, increase sociability, and prime reward centers, imprinting pleasure with infant contact and care, therefore promoting longer-term infant survival

Prelabor increases in uterine oxytocin receptors (human studies) and oxytocin receptors in brain and mammary glands (animal studies) maximize these effects.

The hour or so after physiologic birth is a sensitive period, when skin-to-skin maternal-newborn interactions foster peak oxytocin activity. Benefits may include:

- stronger contractions, likely reducing postpartum hemorrhage risk
- natural warming for the newborn through vasodilation of mothers' chest
- activation of hormonally-mediated maternal-infant biologic bonding
- facilitation of breastfeeding initiation, including by reducing maternal and newborn stress

Common Maternity Care Practices That May Impact Oxytocin Physiology

Common maternity care practices may disrupt these and other beneficial oxytocin effects, with short- and longer-term impacts in mothers and babies. High-quality research is lacking.

While the administration of synthetic oxytocin for induction or augmentation is beneficial in selected circumstances, adverse impacts have been found in women and babies. Synthetic oxytocin administered in labor is not thought to cross into the maternal brain in biologically significant amounts, and so may lack calming and analgesic effects. However, when synthetic oxytocin stimulates contractions, positive feedback cycles may lead to central oxytocin release, promoting further contractions, labor progress, and continued central release.

Synthetic oxytocin may impact maternal oxytocin and physiology. Possible effects include:

- uterine hyperstimulation with potential fetal hypoxia, requiring monitoring
- stronger contractions and increased pain without central oxytocin analgesia
- synthetic oxytocin overexposure causing desensitization of oxytocin receptors, contributing to reduced contractility, prolonged pushing, instrumental birth, and/or postpartum hemorrhage
- disruption of newborn breastfeeding behaviors, reduced maternal oxytocin release with breastfeeding, and possible reduced breastfeeding duration

Physiologic principles, animal studies, and evolving human evidence suggest that perinatal synthetic oxytocin exposure may have longer-term impacts on offspring. While high-quality research is lacking, potential mechanisms include:

- direct fetal brain-hormone effects from synthetic oxytocin transfer through placenta
- indirect signaling of maternal oxytocin to fetal brain
- indirect effects from subclinical hypoxia
- interference with fetal neuroprotective mechanisms (animal studies)
- fetal/newborn impacts from synthetic oxytocin co-interventions such as epidural
- long-term programming of offspring hormonal systems, likely via epigenetic effects (animal studies)
- indirect effects via disruptions to maternal oxytocin systems that impact attachment, reward, breastfeeding, and/or mutual regulation

Epidural analgesia reduces maternal oxytocin in labor, likely due to numbing of the sensory feedback that promotes central oxytocin release. Possible impacts include:

- slowed labor with increased need for synthetic oxytocin
- prolonged pushing stage with increased use of assisted vaginal birth
- disruption of maternal adaptations and attachment

These can also adversely affect the newborn. High-quality research is lacking.

With prelabor cesarean section, mothers and babies miss their complete prelabor physiologic oxytocin preparations; and with any cesarean section, the full oxytocin processes, including the maternal late-labor oxytocin surge and postpartum oxytocin peaks, may be reduced or absent. Impacts on breastfeeding, maternal adaptations, and postpartum hemorrhage have been found. Scheduled cesarean carried out after the physiologic onset of labor may have fewer adverse oxytocin impacts than prelabor cesarean section.

Postpartum separation of healthy mothers and newborns may have detrimental short-and longer-term impacts on the oxytocin system, including:

- reduced oxytocin due to lack of skin-to-skin contact, with increased newborn stress and stress hormones, hypoglycemia, and hypothermia
- disruptions to breastfeeding initiation and long-term success
- deficits in maternal hormones and adaptations, with longer-term impacts on maternal-infant attachment

In animal studies, variations in maternal caregiving in the newborn period lead to epigenetic programming of offspring oxytocin systems, with enduring effects on offspring stress reactivity, and on the maternal care given by female offspring.

Beta-Endorphins: Normal Physiology

Beta-endorphins are endogenous opioids that give analgesic and adaptive responses to stress and pain. Beta-endorphins also activate brain reward and pleasure centers, motivating and rewarding reproductive and social behaviors, and support immune function, physical activity, and psychological well-being.

From labor through the postpartum period, beta-endorphins promote**:**

- endogenous analgesia though prelabor increase in central receptors (animal studies) and increases in beta-endorphins as labor progresses
- an altered state of consciousness that may help with labor stress and pain
- fetal neuroprotection from hypoxia (animal studies)
- postpartum peaks of beta-endorphins (along with oxytocin) that may facilitate maternal euphoria and prime reward centers, imprinting pleasure with infant contact and care
- reward and reinforcement of breastfeeding in both mother and baby
- newborn support with the stress of postpartum transition, including via beta-endorphins in colostrum

Excessive maternal stress in labor may lead to excessive (supraphysiologic) beta-endorphins, which may inhibit oxytocin and slow labor (animal studies). Alternatively, too-low levels of beta-endorphins (infraphysiologic) may not give adequate stress and pain reduction, or activate postpartum pleasure and reward. Optimal levels of beta-endorphins to reduce stress and pain and promote labor progress likely vary among women.

Common Maternity Care Practices That May Impact Beta-Endorphins Physiology

Laboring women may experience excessive stress in relation to their maternity care providers and birth environments (e.g., if not familiar, calm, and private), which may increase BEs to supraphysiologic levels and slow labor. (Stress mechanisms in women are not clear but may also involve oxytocin and/or epinephrine-norepinephrine.)

Labor analgesia that effectively reduces pain will reduce maternal beta-endorphins to some degree. This may be beneficial if excessive stress is inhibiting labor. However, reduced beta-endorphins, as found with epidurals, may also reduce postpartum reward center activation and priming, potentially impacting hormonally-mediated maternal adaptations and attachment, also involving oxytocin.

Women experiencing a cesarean section may miss prelabor opioid receptor increases (animal studies), in-labor peaks of beta-endorphins, and/or postpartum reward center activation. Cesarean newborns have lower levels of beta-endorphins at birth than vaginally born babies, but levels may rise after birth with separation stress.

Separation of mother and newborn in the early sensitive period following physiologic birth, when levels of beta-endorphins are elevated, may interfere with reward center activation of both. In animal studies, repeated brief separations in the newborn period leads to detrimental impacts on offspring opioid systems, likely via epigenetic programing, with enduring effects on pain sensitivity and addiction.

Epinephrine-Norepinephrine and Related Stress Hormones: Normal Physiology

Epinephrine (adrenaline) and norepinephrine (noradrenaline) mediate "fight or flight" stress responses. Epinephrine-norepinephrine release with perceived danger has promoted safety for laboring females in the wild through human evolution by:

- slowing or stopping labor, giving time for fight or flight
- redistributing blood to heart, lungs, and major muscle groups, and away from uterus and baby, to maximize fight-or-flight actions

This epinephrine-norepinephrine response, which acts at an instinctive, subcortical level in all laboring mammals, may inhibit labor when women do not feel private, calm, safe, and undisturbed in labor. However, if the laboring female perceives stress or danger in late labor, epinephrine-norepinephrine elevations may paradoxically stimulate contractions via differential receptor effects. This "fetus ejection reflex" may also occur physiologically when labor has been largely undisturbed, creating powerful, effective, and involuntary pushing. High-quality research in relation to this reflex and its implications for birth is lacking.

In addition to maternal epinephrine-norepinephrine elevations with perceived stress or danger, a physiologic rise in epinephrine with advancing labor has been found in women. This may benefit laboring women by promoting alertness and may promote labor progress by increasing prostaglandin production. The healthy stress (eustress) of labor also elevates the medium-term stress hormone cortisol as much as tenfold. Cortisol may promote contractions, increase central oxytocin effects on maternal adaptations and attachment, and enhance postpartum mood.

For the baby, late-labor epinephrine-norepinephrine elevations (catecholamine surge) provide critical adaptations to labor hypoxia and facilitate newborn transitions, e.g., by:

- preserving blood flow to heart and brain
- promoting respiratory transitions, including clearing of lung fluid
- mobilizing metabolic fuels for the newborn period
- promoting newborn thermoregulation by burning brown fat
- promoting newborn alertness and energy for breastfeeding initiation

After birth, epinephrine-norepinephrine levels drop steeply in mother and baby. These decreases promote uterine contractions, which may limit maternal bleeding, and, for the newborn, reduce energy consumption. Warmth and undisturbed skin-to-skin contact may be important in facilitating maternal and newborn epinephrine-norepinephrine reductions.

Common Maternity Care Practices That May Impact Epinephrine-Norepinephrine and Related Stress Hormones

Aspects of contemporary pregnancy care may have unintended negative (nocebo) effects by increasing maternal stress and anxiety. Stress and anxiety in pregnancy can elevate maternal stress hormones, including epinephrine-norepinephrine and cortisol, with detrimental long-term effects on offspring, including impacts on brain development and stress responsiveness, as established in human and animal studies. Studies suggest that maternal relaxation techniques may reduce pregnancy stress and its detrimental effects, but high-quality research is lacking in this important area.

In labor, anxiety or situations in which the woman does not feel private, safe, and undisturbed may provoke epinephrine-norepinephrine elevations, which may slow or stall labor and reduce fetal blood supply via epinephrine-norepinephrine effects. Stress may also slow labor by reducing pulsatile oxytocin and/or by increasing beta-endorphins.

Attention to emotional well-being may promote labor progress. The reduced need for labor interventions associated with doula and midwifery care may reflect this beneficial focus. Conversely, many common maternity care practices may be stressful for laboring women. High-quality research is lacking in relation to physiologic aspects of labor stress, and methods for ameliorating this.

Epidural analgesia can beneficially reduce maternal pain and epinephrine levels, which may have been inhibiting labor. However, the rapid drop in epinephrine may contribute to hypotension and uterine hyperstimulation. More commonly, contractions reduce over time because oxytocin also decreases. Reductions in both epinephrine-norepinephrine and oxytocin with epidural analgesia may contribute to a prolonged pushing stage and assisted vaginal birth. Epidurals do not assist with, and may increase, fetal hypoxia, stress, and stress hormones in labor, and the risk of cesarean for fetal distress.

With cesarean section, both mothers and babies may miss late-labor epinephrine-norepinephrine elevations, and be less alert after birth for breastfeeding initiation. Lack of the fetal catecholamine surge may significantly contribute to newborn morbidities following cesarean section, including breathing difficulties, hypoglycemia, hypothermia, and drowsiness that may impact interactions and breastfeeding. Cesarean birth may impair newborn and infant stress responses.

Separation of healthy mothers and newborns is more likely following cesarean section, leading to newborn stress and stress hormone elevations. Early separation may also be stressful to the mother, depriving her of the opportunity to reduce epinephrine-norepinephrine for herself and her baby through oxytocin elevations with skin-to-skin contact and mutual interactions. In animal studies, repeated brief separations in the newborn period can lead to detrimental impacts on offspring stress hormone systems, likely via epigenetic programming, with enduring effects including depression-like behaviors in adult offspring and also in separated new mothers.

Prolactin: Normal Physiology

Prolactin is a major hormone of reproduction as well as breast-milk synthesis. Prolactin adapts maternal physiology for pregnancy and breastfeeding, promotes maternal adaptations, and is a caregiving hormone in mammalian mothers and fathers. Outside of reproduction, it is a stress and growth hormone.

Maternal prolactin elevations from early pregnancy may have stress-reducing effects that also benefit the fetus. Late-pregnancy prolactin elevations promote the formation of prolactin receptors in the brain and mammary gland (animal studies). Near term, prolactin production also increases in the uterine lining (decidua), and may be involved in labor processes. Prolactin in amniotic fluid, which fills the fetal lungs, may assist with respiratory preparations. Fetal prolactic production increases close to the physiologic onset of labor, and may promote newborn transitions.

Maternal prolactin paradoxically declines as labor advances (outside of labor, stress triggers prolactin release). Prolactin increases steeply as birth nears, likely due to peaks of beta-endorphins and oxytocin, both of which stimulate prolactin release. In addition, prolactin stimulates oxytocin release, contributing to oxytocin peaks in late labor and birth.

Postpartum prolactin elevations, persisting for several hours after birth, may promote breast-milk production and maternal adaptations. Peaks in prolactin and cortisol, together with early and frequent breastfeeding, may promote prolactin receptor formation, with benefits to ongoing milk production ("prolactin receptor theory"). Prolactin levels released during early breastfeeding have been correlated with maternal adaptations, including: reduced anxiety, aggression, and muscular tension; and increased social desirability (conformity), which may help mothers to prioritize infant care.

Common Maternity Care Practices That May Impact Prolactin Physiology

High-quality research is lacking in relation to possible impacts of maternity care practices on prolactin physiology. Stress in labor may paradoxically reduce prolactin secretion, giving infraphysiologic levels in labor and birth, possibly contributing to the negative impacts of labor stress on breastfeeding. Epidurals may cause in-labor prolactin elevations and postpartum prolactin reductions, with unknown impacts. Induction with synthetic oxytocin may also impact physiologic prolactin release. Prostaglandins may inhibit prolactin with possible impacts on breastfeeding success.

With cesarean section, the expectant mother may miss her pre-labor prolactin elevation, late-labor peak and/or postpartum elevations, which may all impact milk production and maternal adaptations. Following cesarean section, prolactin release with early breastfeeding may be reduced or absent. These and other factors may contribute to reduced breastfeeding success following prelabor cesarean section. Following cesarean section, newborns may have lower prolactin levels, possibly contributing to breathing difficulties and low temperature. Lack of the catecholamine surge may also contribute.

Separation of mothers and their healthy newborns, which typically follows cesarean section, may also impact postpartum maternal prolactin levels. If separation interferes with early breastfeeding initiation and frequency, disruption to prolactin receptor formation may impact ongoing milk production and breastfeeding success.

Conclusions and Recommendations

Overall, consistent and coherent evidence from physiologic understandings and human and animal studies finds that that the innate, hormonal physiology of mothers and babies—when promoted, supported, and protected—has significant benefits for both in childbearing, and likely into the future, by optimizing labor and birth, newborn transitions, breastfeeding, maternal adaptations, and maternal-infant attachment.

There are likely additional benefits from avoiding potential harms of unnecessary interventions, including possible adverse epigenetic programming effects.

From the perspective of hormonal physiology, these are not all-or-nothing benefits, but rather accrue along a continuum. Every mother and baby is likely to benefit from additional support for physiologic childbearing, as far as safely possible, including when interventions are used. The hormonal physiology perspective provides additional considerations for weighing possible benefits and harms of maternity care interventions, and suggests new agendas for research. Research priorities include better understanding of many aspects of hormonal physiology and of impacts of maternity interventions on breastfeeding, maternal adaptations, maternal mood, and other short-, medium-, and longer-term hormonally-mediated and developmental outcomes.

Given the uncertainty and potential for significant harms to women and babies in relation to maternity care interventions, application of the Precautionary Principle would be wise in maternity care. Such a standard would involve**:**

- rigorously verifying the benefits of proposed interventions in individual circumstances before undertaking them
- limiting routine practices to those of proven benefit to healthy mothers and babies
- avoiding the use of interventions for the convenience of women or maternity care providers and systems
- initially using less invasive measures to address challenges, and stepping up to more consequential interventions only as needed

A table in the report summarizes the established and potential effects of the maternity care practices addressed in the report on the four hormone systems.

The following recommendations for education, policy, practice, and research arise from the synthesis presented here. Care practice recommendations below are intended to apply whenever safely possible. To optimize hormonal physiology in childbearing**:**

- Educate all maternity care providers in the hormonal physiology of childbearing.
- Use effective policies and quality improvement strategies to foster consistent access to physiologic childbearing.
- Strengthen and increase access to care models that promote physiologic childbearing and safely limit use of maternity care interventions.
- Use effective consumer engagement strategies to inform women about physiologic childbearing and involve them in related aspects of their care.
- Provide prenatal care that reduces stress and anxiety in pregnant women.
- Foster the physiologic onset of labor at term.
- With hospital birth, encourage admission in active labor.
- Foster privacy and reduce anxiety and stress in labor.
- Make nonpharmacologic comfort measures for pain relief routinely available, and use analgesic medications sparingly.
- Make nonpharmacologic methods of fostering labor progress routinely available, and use pharmacologic methods sparingly.

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- Promote continuous support during labor.
- Foster spontaneous vaginal birth and avoid unneeded cesareans.
- Support early and unrestricted skin-to-skin contact after birth between mother and newborn.
- Support early, frequent, and ongoing breastfeeding after birth.
- Identify and carry out priority research into hormonal physiology of childbearing, and routinely incorporate this perspective in maternity care research.

The Appendix identifies resources for learning more and improving maternity care, including a booklet that presents essential findings from this report to childbearing women.

About the National Partnership for Women & Families

At the National Partnership for Women & Families, we believe that actions speak louder than words, and for four decades we have fought for every major policy advance that has helped women and families.

Today, we promote reproductive and maternal-newborn health and rights, access to quality, affordable health care, fairness in the workplace, and policies that help women and men meet the dual demands of work and family. Our goal is to create a society that is free, fair and just, where nobody has to experience discrimination, all workplaces are family friendly and no family is without quality, affordable health care and real economic security.

Founded in 1971 as the Women's Legal Defense Fund, the National Partnership for Women & Families is a nonprofit, nonpartisan 501(c)3 organization located in Washington, D.C.

About Childbirth Connection Programs

Founded in 1918 as Maternity Center Association, Childbirth Connection became a core program of the National Partnership for Women & Families in 2014. Throughout its history, Childbirth Connection pioneered strategies to promote safe, effective evidence-based maternity care, improve maternity care policy and quality, and help women navigate the complex health care system and make informed decisions about their care. Childbirth Connection Programs serve as a voice for the needs and interests of childbearing women and families, and work to improve the quality and value of maternity care through consumer engagement and health system transformation.