

Endometrial Receptivity and the Window of Implantation: A Narrative Review

Dr. Umme Aman Saba¹, Dr. Amreen Begum², and Dr. Syeda Abidunnisa³

^{1,2,3} *Government Nizamia Tibbi College and General Hospital, Charminar, Hyderabad, India*

Abstract—Endometrial receptivity is a transient, hormonally regulated state in which the uterine endometrium becomes capable of supporting blastocyst attachment and the earliest stages of implantation. The classical window of implantation is generally placed in the mid-secretory phase, although contemporary evidence indicates that receptivity is better understood as a dynamic, multicellular and molecular process rather than as a single fixed calendar interval. This narrative review summarizes the physiology of endometrial preparation, epithelial and stromal remodeling, decidualization, adhesion molecules, cytokines and growth factors, immune regulation, endometrial transcriptomics, extracellular vesicles, microRNAs, long non-coding RNAs, the endometrial microbiome and clinically used markers of receptivity. It also examines endometrial thickness, chronic endometritis, endometriosis and adenomyosis as clinical contexts in which receptivity may be altered. Particular attention is given to endometrial receptivity analysis and other personalized embryo-transfer approaches, including the emerging evidence that routine receptivity-guided transfer has not demonstrated a consistent live-birth advantage over standard timing in unselected patients. A cautious Unani perspective is also presented, emphasizing that traditional concepts concerning uterine milieu and fertility may provide a complementary conceptual framework but should not be equated directly with modern molecular pathways without evidence. Future work is likely to integrate multi-omics, functional models, extracellular vesicles and patient-specific endometrial phenotyping to improve the biological and clinical definition of receptivity.

Index Terms—endometrial receptivity, implantation, decidualization, embryo transfer, endometrium, window of implantation.

I. INTRODUCTION

Successful implantation requires coordinated interaction between a developmentally competent

blastocyst and an appropriately prepared endometrium. In humans, implantation occurs within a restricted period after ovulation, with successful implantation most often occurring approximately 8–10 days after ovulation.[1] The concept of a receptive endometrium therefore refers not simply to endometrial thickness, but to a coordinated functional state involving epithelial transformation, stromal decidualization, vascular remodeling, immune adaptation, extracellular matrix changes and regulated communication with the embryo.[2-5]

The endometrium undergoes cyclical proliferation under estrogen followed by secretory transformation under progesterone. During the mid-secretory phase, epithelial and stromal cells acquire characteristics associated with implantation competence, while progesterone-dependent decidualization prepares the tissue for trophoblast interaction and early pregnancy.[3,6,7] Early histological dating provided a useful framework for describing these changes, but subsequent molecular studies have shown substantial biological variability between individuals and between cycles.[2,10,40]

The clinical importance of receptivity has increased with the widespread use of assisted reproductive technology (ART), particularly frozen embryo transfer. A central question is whether the timing of embryo transfer can be personalized by molecular testing of the endometrium. Although transcriptomic tools such as the endometrial receptivity analysis (ERA) were developed for this purpose, randomized and systematic-review evidence has not established a routine benefit in unselected patients.[11-13,16,35] Current approaches consequently emphasize careful assessment of established uterine factors and individualized decision-making rather than assuming that a single molecular test defines receptivity in every patient.[15,36]

II. PHYSIOLOGY OF THE WINDOW OF IMPLANTATION

The window of implantation (WOI) describes the limited period during which the endometrium is optimally prepared for embryo attachment. Classical clinical observations place this period in the mid-secretory phase, but the biological state is better viewed as a continuum of progesterone-driven transformation rather than a universally identical 24–48-hour interval.[3,5,6] The timing is influenced by ovarian steroid exposure, tissue response and the developmental stage of the embryo.

Progesterone converts the estrogen-primed proliferative endometrium into a secretory and decidualizing tissue. Stromal cells enlarge and undergo functional differentiation, glandular secretion changes, extracellular matrix components are remodeled, and local immune and vascular environments are altered.[6,7] The luminal epithelium also undergoes structural changes that may facilitate embryo attachment. Pinopodes, for example, have been investigated as potential morphological markers of receptivity, although their precise clinical value remains uncertain.[28]

The historical histological dating system of Noyes and colleagues helped establish the relationship between endometrial morphology and the menstrual cycle.[2] However, later molecular work demonstrated that the endometrium cannot be characterized reliably by morphology alone. Global gene-expression studies identified cycle-dependent transcriptional changes during the receptive phase, supporting a more complex molecular definition of receptivity.[10,34,40]

The physiological sequence is illustrated in Fig. 1.

Figure 1. Physiological window of implantation

Cyclical endometrial preparation and the mid-secretory receptive period

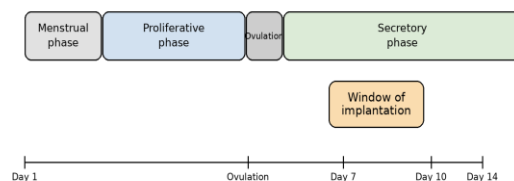


Fig. 1. Schematic representation of the menstrual cycle and the physiological window of implantation.

III. MOLECULAR AND CELLULAR BASIS OF ENDOMETRIAL RECEPTIVITY

Endometrial receptivity is produced by coordinated signaling among epithelial cells, stromal fibroblasts, endothelial cells, immune cells and extracellular matrix components. Adhesion molecules such as integrins have been studied extensively, with $\alpha\beta3$ integrin expression showing cycle-dependent associations with the receptive phase.[8] Leukemia inhibitory factor (LIF), cytokines, chemokines and growth factors participate in the local signaling environment required for implantation.[4,9] These pathways do not operate independently; rather, they form an interconnected network that integrates steroid signaling, tissue remodeling and embryo–endometrium communication.

Decidualization is a central component of receptivity. Human endometrial stromal cells differentiate under progesterone and cyclic AMP-related signaling, acquiring secretory and immunomodulatory functions that support embryo implantation and regulate trophoblast invasion.[7] This process is also increasingly understood as a biosensor of embryo quality: decidualized stromal cells can respond to signals associated with embryonic competence, suggesting that receptivity is not simply passive preparation of the uterus.[6,7]

At the epithelial surface, the balance between adhesion and anti-adhesion molecules is important for controlling embryo attachment. Glycoproteins, integrins, selectins and extracellular matrix components contribute to this interface.[4,8,29] Cytokines and growth factors, including LIF and members of the interleukin and transforming growth factor families, further regulate epithelial–stromal communication and trophoblast interaction.[4,9]

The integrated molecular relationships are summarized in Fig. 2.

Figure 2. Molecular regulation of endometrial receptivity
Steroid signalling coordinates epithelial, stromal, immune and extracellular-matrix responses

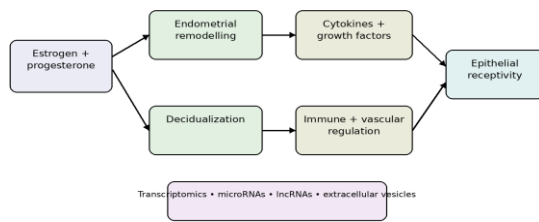


Fig. 2. Integrated molecular regulation of endometrial receptivity.

IV. IMMUNE REGULATION AND VASCULAR REMODELING

Implantation requires an immune environment that is neither simply inflammatory nor immunosuppressive. Uterine natural killer (uNK) cells, macrophages and other immune populations are abundant in the secretory endometrium and contribute to vascular remodeling, decidualization and regulation of trophoblast invasion.[30,31] The available evidence does not support the use of peripheral or uterine immune-cell measurements as simple stand-alone diagnostic tests for implantation failure, and contemporary guidance recommends avoiding unvalidated immune testing in routine practice.[31,36] Angiogenesis and vascular adaptation are also essential. The receptive endometrium must support tissue oxygenation and subsequent placentation while maintaining controlled trophoblast invasion. Cytokines, chemokines, growth factors and matrix-remodeling pathways participate in these processes.[4,7] Consequently, an apparently normal endometrial surface does not necessarily indicate normal functional receptivity.

The principal immune and vascular components are summarized in Fig. 3.

Figure 3. Immune and vascular remodelling

Local immune adaptation and vascular change support decidualization and implantation

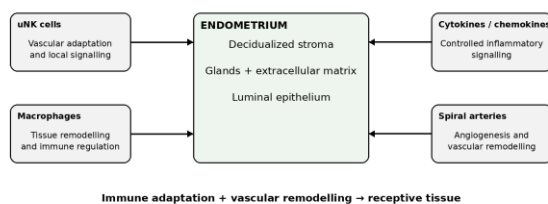


Fig. 3. Immune and vascular remodelling during implantation.

V. TRANSCRIPTOMICS, OMICS AND MOLECULAR TESTING

Transcriptomic studies have demonstrated reproducible cycle-related changes in endometrial gene expression. Kao et al. described global gene-expression patterns associated with the human window of implantation, providing an important foundation for molecular characterization of receptivity.[10] The development of the ERA subsequently translated a transcriptomic signature into a clinical assay intended to identify the timing of receptivity.[11]

However, molecular receptivity is not synonymous with clinical implantation potential. Systematic reviews have identified substantial heterogeneity among transcriptomic studies, including inconsistent reporting of differentially expressed genes and methodological differences.[34,40] More recent omics research emphasizes integration of transcriptomics with proteomics, metabolomics, epigenetic regulation and extracellular vesicle biology rather than reliance on a single gene-expression signature.[14,39]

MicroRNAs and long non-coding RNAs are emerging regulators of genes and signaling pathways involved in endometrial receptivity.[37,38] These molecules may influence steroid responsiveness, cell proliferation, decidualization, immune signaling and embryo–endometrium communication. At present, however, most such findings remain mechanistic or exploratory and require prospective clinical validation before routine diagnostic application.[14,17,37,38]

VI. EXTRACELLULAR VESICLES AND EMBRYO–ENDOMETRIUM COMMUNICATION

Extracellular vesicles provide a biologically plausible mechanism for communication between endometrial cells and the developing embryo. They can carry proteins, lipids, messenger RNAs and non-coding RNAs, potentially modifying recipient-cell behavior.[39] Human studies have shown that endometrial extracellular vesicles can influence processes related to embryo development and implantation in blastocysts, supporting the concept that receptivity includes active biochemical communication rather than simple physical attachment.[19]

These observations may eventually lead to functional biomarkers that measure endometrial–embryo interaction directly. Nevertheless, extracellular-vesicle signatures remain investigational, and differences in isolation methods, cargo characterization and study populations currently limit clinical translation.[14,19,39]

The sequence of embryo–endometrium interaction and implantation is summarized in Fig. 4.

Figure 4. Sequential events in embryo implantation

from diastocyst contact to adhesion, invasion and decidual response

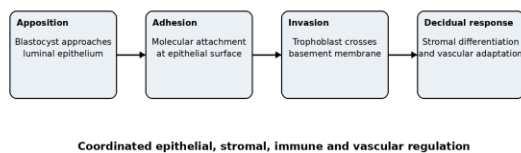


Fig. 4. Sequential events in embryo implantation: apposition, adhesion, invasion and decidual response.

VII. ENDOMETRIAL THICKNESS AND STRUCTURAL ASSESSMENT

Transvaginal ultrasonography remains a practical component of endometrial assessment in ART. Endometrial thickness is associated with reproductive outcomes at a population level, but it is an imperfect surrogate for receptivity. A 2025 systematic review and meta-analysis found that endometrial thickness behaves as a gradient prognostic factor in fresh and frozen-thawed embryo transfer, while also noting limitations in evidence quality and substantial heterogeneity.[24]

Thus, a thin endometrium should prompt clinical evaluation of underlying causes, but thickness alone should not be interpreted as a direct molecular measurement of receptivity. Conversely, a satisfactory thickness does not guarantee successful implantation because epithelial, stromal, immune and molecular functions may remain abnormal.[5,24,40]

VIII. RECEPTIVITY IN ENDOMETRIOSIS, ADENOMYOSIS AND CHRONIC ENDOMETRITIS

Endometriosis is associated with altered eutopic endometrial biology, including changes in steroid

responsiveness, inflammation and molecular pathways that may affect implantation.[20,32] The precise contribution of receptivity defects to infertility in endometriosis remains complex because pelvic inflammation, altered anatomy, ovarian factors and embryo-related variables may coexist.

Adenomyosis has likewise been associated with altered endometrial and uterine biology, including inflammatory and immune changes that may contribute to impaired fertility and ART outcomes.[23] Current evidence supports a multifactorial mechanism rather than a single receptivity defect.

Chronic endometritis has received attention in recurrent reproductive failure. A 2024 systematic review and meta-analysis found associations between chronic endometritis and infertility and recurrent pregnancy loss, but did not demonstrate a significant association with recurrent implantation failure specifically.[22] This distinction is important because treatment decisions should be based on the clinical context and quality of evidence rather than assuming that every inflammatory finding represents an implantation defect.

IX. PERSONALIZED EMBRYO TRANSFER AND THE ERA DEBATE

The ERA and related endometrial receptivity tests aim to identify an individual patient's receptive timing and adjust progesterone exposure or embryo-transfer timing accordingly.[11,16] The biological premise is attractive, particularly in patients with repeated implantation failure, but clinical efficacy must be separated from biological plausibility.

A randomized clinical trial published in 2022 found no significant improvement in live birth with receptivity-test-guided frozen embryo transfer compared with standard timing in the studied population; live birth occurred in 58.5% of the receptivity-guided group and 61.9% of the standard-timing group.[12] A 2023 systematic review and meta-analysis likewise found insufficient evidence for routine personalized embryo transfer in the general IVF population.[35] A 2026 systematic review and meta-analysis of endometrial receptivity-guided embryo transfer concluded that current evidence does not support routine use in patients without recurrent implantation failure, while evidence in recurrent implantation failure remains

uncertain and particularly limited in euploid embryo transfer.[13]

These findings illustrate a broader principle: a biomarker can be biologically associated with receptivity without improving patient outcomes when used to alter treatment. Current evidence therefore favors established clinical assessment and careful selection of patients for investigational or individualized receptivity testing rather than routine testing for all IVF cycles.[13,15,16,36]

The clinical assessment pathway and transfer strategies are summarized in Fig. 5.

Figure 5. Assessment and personalized embryo transfer
Established clinical measures remain central while molecular approaches are evaluated

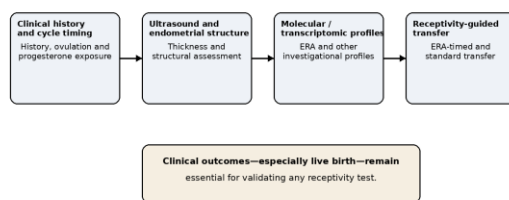


Fig. 5. Assessment of endometrial receptivity and personalized embryo transfer.

X. EMERGING ROLE OF THE ENDOMETRIAL MICROBIOME

The endometrial microbiome has been proposed as another component of the uterine implantation environment. Research has examined whether microbial composition and host–microbe interactions influence inflammation, epithelial function and embryo implantation.[21] However, there is still no universally accepted definition of a normal endometrial microbiome, and studies have been limited by small cohorts, differences in sampling and laboratory methods, and potential contamination issues.[21]

Accordingly, microbiome-based tests should presently be regarded as investigational. Future studies need standardized sampling, rigorous controls and prospective outcome-based validation before microbial profiles can be considered reliable measures of endometrial receptivity.[21]

XI. FUNCTIONAL MODELS AND ENDOMETRIUM-ON-CHIP TECHNOLOGY

An important recent development is the use of functional experimental systems to study endometrial receptivity. A 2026 human endometrium-on-chip platform was used to assess luminal epithelial receptivity and blastoid attachment under hormonally controlled conditions.[18] Such models can help separate the effects of progesterone exposure, epithelial maturation and embryo attachment from the confounding factors present in clinical observational studies.

Functional models may therefore complement transcriptomic biomarkers by asking whether a tissue actually performs the biological function of receptivity. Their current role is research-based, but they represent a promising direction for mechanistic validation and future precision reproductive medicine.[18]

The major emerging research directions are summarized in Fig. 6.

Figure 6. Emerging research in endometrial receptivity
Integrated, functional and patient-specific approaches to defining receptivity

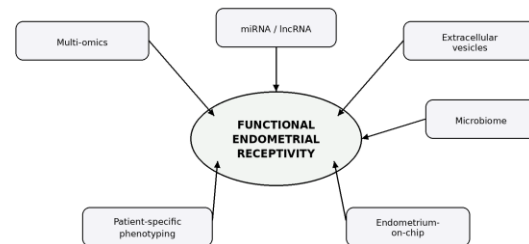


Fig. 6. Emerging research directions in endometrial receptivity.

XII. UNANI PERSPECTIVE

Classical Unani medicine approaches fertility through the condition and function of the reproductive organs, the quality of humoral balance and the broader physiological state of the individual. Historical Unani medical literature, including Ibn Sina's *Al-Qanun fi al-Tibb*, contains discussions relevant to infertility and reproductive health.[25] Modern reviews of Unani approaches to female infertility describe traditional concepts and therapeutic practices directed toward reproductive function.[26]

From a contemporary scientific perspective, it is reasonable to consider the uterine environment described in traditional frameworks as a broad conceptual parallel to the modern idea that implantation depends on a suitable local environment. However, direct equivalence between classical Unani concepts and specific molecular markers such as LIF, integrins, microRNAs or transcriptomic signatures has not been established. Therefore, Unani concepts should be presented as a complementary conceptual framework rather than as a claimed molecular explanation of endometrial receptivity.

Some Unani or herbal interventions have been investigated for aspects of female reproductive function. For example, a randomized controlled trial evaluated *Asparagus racemosus* (Satavar) in anovulatory infertility and reported effects related to follicular growth and ovulation.[27] Such evidence addresses ovarian function rather than demonstrating improved endometrial receptivity or implantation. Similarly, isolated case reports cannot establish efficacy for implantation-related outcomes.[38] Rigorous clinical trials with validated reproductive endpoints are required before traditional interventions can be incorporated into evidence-based receptivity management.

XIII. CLINICAL IMPLICATIONS

Endometrial receptivity should be assessed as a multidimensional biological phenomenon. In clinical practice, evaluation begins with menstrual and reproductive history, uterine imaging, endometrial development during the treatment cycle and identification of structural or inflammatory disorders when clinically indicated.[15,36] Endometrial thickness may provide prognostic information but should not be treated as a definitive receptivity test.[24]

For recurrent implantation failure, contemporary recommendations emphasize systematic assessment of embryo-related, uterine, endometrial and other relevant factors rather than indiscriminate use of add-on tests.[15,36] Receptivity assays may be considered selectively in research-oriented or carefully selected clinical settings, but patients should be informed that evidence for improved live birth remains uncertain, particularly outside well-defined subgroups.[13,16,35]

XIV. LIMITATIONS AND FUTURE DIRECTIONS

Several limitations complicate the field. First, the definition of a receptive endometrium varies among studies. Second, molecular signatures are influenced by cycle timing, hormone exposure and tissue heterogeneity. Third, implantation is an interaction between embryo competence and maternal endometrial function, making it difficult to attribute failure to the endometrium alone.[5,14]

Future research should prioritize prospective, adequately powered trials with live birth as the primary outcome; standardized sampling and laboratory methods; integration of transcriptomic, proteomic, epigenetic, microbiome and extracellular-vesicle data; and functional validation using advanced human endometrial models.[13,14,18,21,39] The most clinically useful biomarker may ultimately be a composite phenotype rather than a single molecular test.

XV. CONCLUSION

Endometrial receptivity is a dynamic state generated by coordinated hormonal, cellular, molecular, vascular and immunological changes in the endometrium. The traditional window of implantation remains a useful clinical concept, but modern evidence indicates that receptivity is more complex than a fixed time interval or a single biomarker. Transcriptomics, non-coding RNAs, extracellular vesicles, microbiome research and functional endometrial models are expanding understanding of the implantation environment. At the same time, randomized and systematic-review evidence cautions against routine use of receptivity-guided embryo transfer in unselected IVF patients. A clinically meaningful definition of receptivity should ultimately be based on validated prediction of reproductive outcomes. In a multidisciplinary framework, Unani concepts may be explored as complementary perspectives, while claims of molecular equivalence should await direct evidence.

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