

Control of Growth and Differentiation of the Endometrium: The Role of Tissue Interactions

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ABSTRACT: Early work with neonatal mice showed that estrogen receptor-negative uterine epithelium responded to estrogen treatment. Since the underlying mesenchymal cells were estrogen receptor-positive, it was suggested that these cells mediated the hormonal response through elaboration of a paracrine factor. Cell culture work showed that mesenchymal cells produced soluble factors that stimulate uterine epithelium, but hormonal regulation was absent or minimal. The paracrine hypothesis of estrogen action has been proved by the use of tissue recombination studies in which epithelium from estrogen receptor-alpha knockout mice was combined with wild-type mesenchyme; estrogen stimulated the ER α -negative epithelium if the underlying stromal cells were receptor-positive. Also, it is hypothesized that there is a reciprocal paracrine interaction during stimulation with progesterone and estrogen. Accordingly, under progesterone dominance, the epithelium elaborates factors that direct the underlying stroma to proliferate when estrogen is administered. Although this hypothesis needs further testing, it has been shown that the uterine epithelium is required for stromal responsiveness to hormones. The question arises: What are the factors that mediate the effects of the steroid hormones in the uterus? Several peptide growth factors are regulated by estrogen and/or progesterone. Use of knockout animals will allow a determination of the role that these factors play in the uterus. However, ablation of many of these growth factor genes has proved lethal to the newborn animals, making it impossible to study hormonal effects using standard techniques. Tissue xenograft and tissue recombination studies offer a means of defining the role of specific growth factors in uterine physiology.

KEYWORDS: tissue interactions; paracrine factors; cell proliferation; estrogen; progesterone; tissue recombinations; xenograft; gene knockout

One of the hallmarks of endometriosis is the inclusion of endometrial stroma in the ectopic tissue implant. Is this merely a diagnostic tool for pathologists or does it point to a biologically important aspect of the disease process? Endometriomas are responsive to hormonal changes, but the hormonal effects are not always identical to those occurring in the normal endometrium. Do the hormonal responses of endometrioma tissues derive from an interaction of the stromal and epithelial tissues and

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Table of Contents Control Of Growth Differentiation In

1. Understanding the eBook Control Of Growth Differentiation In
 - The Rise of Digital Reading Control Of Growth Differentiation In
 - Advantages of eBooks Over Traditional Books
2. Identifying Control Of Growth Differentiation In
 - Exploring Different Genres
 - Considering Fiction vs. Non-Fiction
 - Determining Your Reading Goals
3. Choosing the Right eBook Platform
 - Popular eBook Platforms
 - Features to Look for in an Control Of Growth Differentiation In
 - User-Friendly Interface
4. Exploring eBook Recommendations from Control Of Growth Differentiation In
 - Personalized Recommendations
 - Control Of Growth Differentiation In User Reviews and Ratings
 - Control Of Growth Differentiation In and Bestseller Lists
5. Accessing Control Of Growth Differentiation In Free and Paid eBooks
 - Control Of Growth Differentiation In Public Domain eBooks
 - Control Of Growth Differentiation In eBook Subscription Services
 - Control Of Growth Differentiation In Budget-Friendly Options
6. Navigating Control Of Growth Differentiation In eBook Formats
 - ePub, PDF, MOBI, and More
 - Control Of Growth Differentiation In Compatibility with Devices
 - Control Of Growth Differentiation In Enhanced eBook Features
7. Enhancing Your Reading Experience
 - Adjustable Fonts and Text Sizes of Control Of Growth Differentiation In
 - Highlighting and Note-Taking Control Of Growth Differentiation In
 - Interactive Elements Control Of Growth Differentiation In
8. Staying Engaged with Control Of Growth Differentiation In

- Joining Online Reading Communities
 - Participating in Virtual Book Clubs
 - Following Authors and Publishers Control Of Growth Differentiation In
9. Balancing eBooks and Physical Books Control Of Growth Differentiation In
- Benefits of a Digital Library
 - Creating a Diverse Reading Collection Control Of Growth Differentiation In
10. Overcoming Reading Challenges
- Dealing with Digital Eye Strain
 - Minimizing Distractions
 - Managing Screen Time
11. Cultivating a Reading Routine Control Of Growth Differentiation In
- Setting Reading Goals Control Of Growth Differentiation In
 - Carving Out Dedicated Reading Time
12. Sourcing Reliable Information of Control Of Growth Differentiation In
- Fact-Checking eBook Content of Control Of Growth Differentiation In
 - Distinguishing Credible Sources
13. Promoting Lifelong Learning
- Utilizing eBooks for Skill Development
 - Exploring Educational eBooks
14. Embracing eBook Trends
- Integration of Multimedia Elements
 - Interactive and Gamified eBooks

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