# 〈月經的重要性與演化〉

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**關鍵詞**

演化、自然發生之蛻膜化、表型可塑性、遺傳固着、幹細胞、月經抑制

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**摘要（翻譯）**

在歷史上，有關月經演化起源的學說主要有二：其一認為月經可排除與精子一併帶入子宮之病原；其二則主張相較於完全不行經，行經可節約能量。近年新理論指出，自發性蛻膜化（spontaneous decidualization）才是月經物種的關鍵適應機制；蛻膜化乃母體對胚胎具侵襲性的防禦措施。就生理而言，月經是子宮內膜藉由發炎反應與血管調控所驅動的複雜過程，得以穩定內膜並使組織與血液適度脫落。多項人類疾病（如反覆性流產、前置胎盤植入、子宮外孕、子宮內膜異位症、子宮腺肌症、痛經與慢性骨盆腔疼痛）可被視為此一演化機制之「易感性」所衍生。透過演化觀點，我們不僅得以理解上述疾病何以發生，亦能為治療、預防及未來研究奠定基礎。

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## 緒論

自古以來，月經功能即備受關注——從古典文獻、早期文化習俗與禁忌，至當代之性別研究 [1]。近年來，特別是在人類慢性疾病的演化醫學視角中，月經的機制與意義日益成為研究焦點。月經可定義為：於未受孕之月經週期末，因黃體素下降而引發的子宮內膜功能層剝落及出血。本篇旨在探討月經的演化起源、生理機制及其在臨床疾病中的意義，相關論述可參閱多篇綜述 [1–4]。

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## 月經的演化

### 歷史觀點

演化透過適應使個體在生存與繁殖上獲益。因此，月經的演化必須能夠提升個體適存度。那麼，月經的適應利益為何？

- **亞里斯多德**認為月經血為「無生命物質」，與精液結合後促成胚胎發育 [5]。
- **蓋倫**則視月經為排出「過多血液」的方式 [6]。
- 月經亦曾被拿來類比犬隻動情前的陰道出血，惟後者並非子宮來源且週期時相不符 [7]。
- 近代亦提出月經與性反應相關 [8]，或用以排除未著床胚胎 [9]。

較具影響力的兩大理論分別為：

1. **抗感染理論**——月經可清除性交時精子攜帶入宮腔的病原體 [10]；此說已被 Finn 反駁 [11]。

2. **能量節約理論——**與維持持續增厚之內膜相比，行經較為省能 [12]；但考量子宮須執行複雜而精細的妊娠功能，此說亦遭質疑 [2]。

上述理論皆未能充分說明月經如何經由自然選擇而演化。

**現代理論——自發性蛻膜化**

月經發生之前，子宮內膜已經歷一連串複雜變化。最新證據顯示，「**自發性蛻膜化**」是月經物種的核心適應 [13–15]。

- **非月經動物**：蛻膜化發生於受精後胚胎進入子宮，或實驗性外傷刺激。
- **月經動物**（人類、大猩猩、黑猩猩、象鼩、部分蝙蝠 [16]）：僅因排卵後黃體素上升即行蛻膜化，即使無受精卵亦然。

自發性蛻膜化被視為母體對具高度侵襲性胚胎的一種「先發制人」保護機制，具體表現為：

- **血管新生**與大量 **子宮自然殺手細胞 (uNK)** 浸潤，藉以限制胚胎滋養層過度侵入 [17,18]。
- 蛻膜巨大細胞形成緊密結連，構築物理屏障 [20]。

兩大推論：

1. **母體防禦說——**蛻膜化降低胚胎過度侵襲所造成之母體風險 [16]；
2. **胚胎篩選說——**蛻膜化使子宮得以「評估」胚胎品質並淘汰劣質胚 [21]。

分子層面上，**cAMP** 途徑為蛻膜化共同訊號，無論是荷爾蒙驅動或胚胎誘導 [22]。

**表型可塑性與遺傳固着**

表型可塑性指生物體在不同環境下可產生不同表現型 [23,24]；當環境壓力持續，該表現型可能經「**遺傳固着 (genetic assimilation)**」成為遺傳性狀 [26,27]。

就月經而言，子宮對「高度侵襲胚胎」的環境刺激，可能經過表型可塑性的中介，逐漸演化出自發性蛻膜化並最終固着——具備更高的母體適存度 [30]。

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**月經的生理學**

月經的探討橫跨社會、性別乃至生物醫學層面 [1]。下文聚焦於**生物學機制**及其與疾病的關聯。月經為「孕失敗」後，黃體素急降所觸發：功能層崩解並出血 [31]。機制可從**發炎反應**與**血管調控**兩方面說明。

**發炎機制**

一種「二步驟假說」[1]：

1. **黃體素撤退** → 蛻膜細胞釋放細胞激素、趨化因子與黏附分子 [34]；
2. **白血球（尤以中性球）** 滲入 → 分泌 **基質金屬蛋白酶 (MMPs)**，分解細胞外基質，引發組織脫落 [35–38]。

   抑制 COX-2 或 NF-κB 可減少月經血量，證實前列腺素與發炎途徑對白血球遷移及組織破壞之調控作用 [39]。

若此序列失衡，將導致**功能性子宮出血** [40,41]。

**血管調控**

- **動脈解剖**：子宮／卵巢動脈 → 弓狀動脈 → 放射動脈 → 基底動脈與螺旋動脈。
- **螺旋動脈** 對荷爾蒙極敏感。月經來潮時，其短暫痙攣可造成內膜局部缺血，隨後血流恢復並出血 [42,43]。
- **前列腺素平衡**：$PGF_2\alpha$、ET-1（血管收縮）與 $PGE_2$（血管舒張）比例改變影響血流 [44]；螺旋動脈平滑肌增生亦影響管徑 [43,45,46]。
- **止血機制**：血小板、膠原、組織因子及 von Willebrand 因子 (vWF) 形成血栓；纖溶系統參與月經量調控。抗纖溶藥（如 **tranexamic acid**）可治療過多月經 [47]。vWF 缺陷（von Willebrand 病）亦是**經血過多**之常見原因 [48,49]。

研究月經的演化與生理機制，可為多種重要疾病帶來洞見。有論者指出，**分娩過程本質上可被視為一種月經現象**：其仍依賴「自發性蛻膜化」，而懷孕與分娩併發症的異常與缺陷，早在未孕狀態的自發性蛻膜化或月經伊始即已可偵測。亦即，這些限制可能在懷孕前就已存在 [50]。

**· 反覆性流產（Recurrent Pregnancy Loss, RPL）**

- 與子宮內膜腔幹細胞功能異常有關。
- 幹細胞不足、間質老化加速及蛻膜化缺陷，皆會削弱內膜分化能力，導致妊娠失敗 [51]。

**· 子宮內膜異位症與子宮腺肌症**

- 兩者皆表現出具「高度侵襲性」的內膜幹細胞 [53]。
- 內膜異位症常被解釋為逆行性月經將內膜細胞帶入腹膜所致，然而逆行性月經在無病女性中也相當普遍。
- 內膜細胞並非同質；其中部分為具再生能力的幹細胞 [52]。逆行性月經中幹細胞的比例，可能影響內膜異位症的發生率 [1]。
- 亦可解釋為何部分內膜異位病灶對黃體素治療具有抗藥性 [53]。

**· 植入性胎盤（Placenta Accreta）**

- 指胎盤過度侵入甚至穿透子宮壁，嚴重者可侵犯膀胱等鄰近器官。
- 常見於曾接受剖宮產的孕婦，且帶來高病死率與併發症 [54–57]。
- 可能因**母體與胎兒力量失衡**：胎兒需侵入以獲取資源，母體則以蛻膜化（含 uNK 細胞誘發滋養層凋亡）自我保護。蛻膜化不足 → 胎盤侵襲過度 → 分娩後胎盤不易剝離 → 大量出血。
- 類似機制亦見於輸卵管異位妊娠：缺乏/蛻膜不足 → 胚胎侵入→ 破裂大出血。

· **慢性骨盆腔疼痛（Chronic Pelvic Pain, CPP）**

- 多來自反覆的盆腔器官疼痛。當周邊疼痛訊號頻繁且強烈，脊髓可發生可塑性變化，導致痛覺敏化 → 慢性痛。
- 臨床表徵：腹壁與會陰部**皮膚痛覺異常（allodynia）** [58,59]。
- 具 cutaneous allodynia 之 CPP 女性，過去多有**嚴重痛經** [60]。
- 百年間人類生理最大變化之一即為「終生月經次數」大增：因初經提前、壽命延長、懷孕與泌乳期相對縮短。現代女性若活至 52 歲、僅生兩胎並哺乳，終身月經次數 >400 次（見 Fig. 1）[61]。
- 對部分女性而言，**重複性劇痛**超出疼痛系統之調節能力 → 「高痛覺致敏前置 (hyperalgesic priming)」[62,63] → 慢性疼痛。
- 臨床意涵：及早辨識/治療嚴重痛經（NSAIDs、月經抑制等）或可預防 CPP [64–66]。

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**總結**

醫學文獻多聚焦於「疾病是什麼、何時發生、如何發生」，而演化觀點則解釋「**為何**罹病」，特別有助於慢性病之理解。洞察月經之演化與意義，可為多種人類疾病之治療與研究帶來新契機。隨著我們更加了解蛻膜在妊娠穩定中的要角，以及幹細胞研究的進展，生殖健康問題有望獲得重大改善。

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**實務重點（Practice points）**

- 🔍 **詳細詢問月經影響**：需主動提問，以免忽略患者「嚴重痛經」的真實程度。
- 💊 **前列腺素抑制 & 月經抑制**：痛經治療兩大要點。
- ⏳ **月經抑制可望預防 CPP**：亦可逆轉已出現的痛覺敏化（allodynia），但需時間。

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**研究議程（Research agenda）**

1. **內膜幹細胞異常**—內膜異位症、子宮腺肌症、RPL 可能皆與幹細胞功能失調有關，亟需深入探討其異質性與治療策略 [67,68]。
2. **剖宮產教育**—過度施行初次/再次剖宮產 → 植入性胎盤、前置胎盤甚至羊水栓塞風險升高，應加強孕婦衛教 [69]。
3. **預防慢性骨盆痛**—青少女痛經應積極治療與適度月經抑制，以避免 30% 女性從痛經進展為慢性痛 [70,71]。
4. **再生醫學**—應用於子宮，改善蛻膜化、增進著床，治療 RPL 具潛力 [62,72]。

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J. Jarrell / Best Practice & Research Clinical Obstetrics and Gynaecology 50 (2018) 18e26 25

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