

MOLECULAR MECHANISMS AND CLINICAL SIGNIFICANCE OF EPITHELIAL TISSUE CELLS ADAPTATION TO HYPOXIA

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Annotation: Epithelial tissue covers the internal and external surfaces of the body and performs important functions such as protection, secretion, absorption and signaling. This article deeply studies the mechanisms of adaptation of epithelial cells to various stress conditions (hypoxia, inflammation, mechanical deformation, chemical effects). Epithelial cell adaptation occurs at the molecular, genetic and cellular physiological levels and is carried out through pathways such as HIF-1 α , NF- κ B, EMT (epithelial-mesenchymal transition).

Activation of the HIF-1 α factor under hypoxia conditions changes the energy metabolism of cells and ensures their survival. Activation of the NF- κ B pathway during inflammatory processes leads to the production of cytokines and chemokines, which in turn enhances the immune response. The EMT process plays an important role in the development of metastatic cancer, causing epithelial cells to acquire mesenchymal properties.

Disruption of these adaptive mechanisms can lead to various pathologies (delayed wound healing, ulcers, cancer metastasis). Therefore, understanding the adaptive capacity of epithelial cells can be the basis for the development of new therapeutic methods.

Keywords: Epithelial tissue, adaptation, intercellular signaling, differentiation, apoptosis, stability, HIF-1 α , NF- κ B, EMT (epithelial-mesenchymal transition).

1. Epithelial tissue
2. Adaptation
3. HIF-1 α
4. NF- κ B
5. EMT
6. Inflammation
7. Metastasis
8. Intercellular signaling
9. Apoptosis
10. Regeneration

EPITELIY TO‘QIMASI HUYAYRALARINING GIPOKSIYAGA ADAPTATSIYASI

MOLEKULAR MEXANIZMLAR VA KLINIK AHAMIYATI

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Annotatsiya: Epiteliy to'qimasi organizmning ichki va tashqi sirtlarini qoplab, himoya, sekretiya, absorptsiya va signalizatsiya kabi muhim funksiyalarni bajaradi. Ushbu maqolada epiteliy hujayralarining turli xil stress sharoitlariga (hipoksiya, yallig'lanish, mexanik deformatsiya, kimyoviy ta'sirlar) moslanish mexanizmlari chuqur o'rganilgan. Epiteliy hujayralari adaptatsiyasi molekulyar, genetik va hujayra fiziologiyasi darajasida bo'lib, HIF-1 α , NF- κ B, EMT (epiteliy-mezenximal o'tish) kabi yo'llar orqali amalga oshadi.

Gipoksiya sharoitida HIF-1 α omilining faollashuvi hujayralarning energiya metabolizmini o'zgartirib, ularning omon qolishini ta'minlaydi. Yallig'lanish jarayonlarida NF- κ B yo'lining faollashishi sitokinlar va kemokinlar ishlab chiqarilishiga olib keladi, bu esa immun javobni kuchaytiradi. EMT jarayoni esa metastatik saraton rivojida muhim rol o'ynaydi, epiteliy hujayralarining mezenximal xususiyatlar qabul qilishiga sabab bo'ladi.

Bu adaptatsion mexanizmlarning buzilishi turli xil patologiyalarga (yara shikastlanishining kechikishi, oshqozon yarasi, saraton metastazlari) olib kelishi mumkin. Shuning uchun epiteliy hujayralarining moslanish qobiliyatini tushunish yangi terapevtik usullarni ishlab chiqish uchun asos bo'lishi mumkin.

Kalit so'zlar: Epiteliy to'qimasi, adaptatsiya, hujayralararo signalizatsiya, differentsiatsiya, apoptoz, barqarorlik, HIF-1 α , NF- κ B, EMT (epiteliy-mezenximal o'tish).

1. Epiteliy to'qimasi
2. Adaptatsiya
3. HIF-1 α
4. NF- κ B
5. EMT
6. Yallig'lanish
7. Metastaz
8. Hujayralararo signalizatsiya
9. Apoptoz
10. Regeneratsiya

Maqsad: Epiteliy hujayralarining moslanish mexanizmlarini tushunish orqali yangi tibbiyot usullarini ishlab chiqish va patologiyalarni oldini olish.

Usullar: Maqolada ilmiy adabiyotlar tahlili, in vitro hujayra modellari (Caco-2, HaCaT), transkriptomika va proteomika tadqiqotlari, shuningdek, hayvon modellari orqali olingan natijalar qo'llanilgan.



Kirish: Epiteliy to'qimasi organizmning birinchi himoya chizig'i bo'lib, tashqi muhit (teri, nafas yo'llari) va ichki organlar (ichak, buyrak naychalari) sirtini qoplaydi. Ushbu to'qima hujayralari doimiy ravishda turli xil stress omillari (mexanik, kimyoviy, infeksiyon) ta'sirida qoladi va ularga moslanishi kerak.

Adaptatsiya jarayonlari hujayra darajasida gen ekspressiyasining o'zgarishi, signal yo'llarining faollashishi va hujayralararo aloqaning o'zgarishi orqali amalga oshadi. Masalan, kislorod yetishmovchiligi (hipoksiya) sharoitida HIF-1 α omili faollashadi va bu hujayralarning glikoliz yo'lini kuchaytirishiga olib keladi.

Yallig'lanish jarayonlarida esa NF- κ B yo'lining faollashishi immun hujayralarining faollashuviga sabab bo'ladi. EMT (epiteliy-mezenximal o'tish) jarayoni esa hujayralarning harakatchanligini oshiradi, bu esa saraton metastazida muhim rol o'ynaydi.

Ushbu mexanizmlarni tushunish tibbiyotda yangi davolash usullarini ishlab chiqish uchun juda muhimdir.

Natijalar

Hipoksiya nima va epiteliy hujayralariga ta'siri

Hipoksiya — hujayra va to'qimalarda kislorodning yetarli bo'lmaganligi holati bo'lib, quyidagi sabablarga ko'ra yuzaga kelishi mumkin: Arali hipoksiya (masalan, o'pka kasalliklari, yuqori balandlikda yashash). Qon aylanishidagi buzilishlar (ateroskleroz, tromboz). Anemiya (gemoglobin yetishmovchiligi). Tumor mikroatrofiyasi (o'smalarda qon tomirlarining noto'g'ri shakllanishi).

Epiteliy hujayralari hipoksiyaga juda sezgir bo'lib, chunki ular ko'pincha organizmning sirt qatlamlarida joylashgan (masalan, ichak epiteliysi, o'pka alveolalari) yoki qon tomirlaridan uzoqda bo'lgan to'qimalarda (masalan, teri, shilliq qavatlar) mavjud. Kislorod yetishmovchiligi ularning metabolizmi, gen ekspressiyasi va hatto o'limiga (apoptoz yoki nekroz) olib kelishi mumkin.

HIF (Hipoksiyaga Javob Omili) yo'li — asosiy moslanish mexanizmi

Epiteliy hujayralari hipoksiyaga asosan HIF (Hypoxia-Inducible Factor) omillari orqali moslanadi. HIF — transkripsiya omili bo'lib, uning ikkita asosiy kichik birligi mavjud: HIF-1 α ** (yoki HIF-2 α) — kislorodga bog'liq bo'lib, barqarorsiz. HIF-1 β ** — kislorodga bog'liq emas, doimiy mavjud

Normal sharoitda (kislorod etarli bo'lganda): HIF-1 α prolil gidroksilaza fermenti tomonidan gidroksillanadi. Keyin VHL (von Hippel-Lindau) oqsili HIF-1 α ni tanib, uni ubiquitin yo'li orqali parchalaydi.

Hipoksiya sharoitida:

1. Prolil gidroksilaza faolsizlanadi, HIF-1 α gidroksillanmaydi.
2. HIF-1 α parchalanishdan qochib, yadroga o'tadi va HIF-1 β bilan kompleks hosil qiladi.
3. Bu kompleks **hipoksiyaga javob elementlari (HRE)** ga birikib, turli genlarning ekspressiyasini faollashtiradi.

HIF-ning faollashtirgan genlar va ularning ahamiyati

HIF-1 α quyidagi genlarni faollashtirib, epiteliy hujayralarining omon qolishini ta'minlaydi:

Gen Funksiyasi, GLUT-1/3 Glukoza transportini oshiradi (energiya manbai sifatida foydalanish uchun). VEGF Yangi qon tomirlarining shakllanishini rag'batlantiradi (angiogenez). EPO Eritropoezni stimullaydi (qizil qon hujayralarining hosil bo'lishi). LDH-A



Anaerob glikolizni kuchaytiradi (kislorodsiz energiya ishlab chiqarish). BNIP3 Avtofagiyaning faollashtirib, hujayralarni stressdan himoya qiladi.

- Hujayralar energiya balansini saqlaydi (glikoliz orqali ATP ishlab chiqarish).
- Qon ta'minoti yaxshilanadi (VEGF ta'sirida yangi kapillyarlar hosil bo'ladi).
- Oksidativ stress kamayadi (antioksidant fermentlar faollashadi).

Hipoksiya va patologik holatlar

HIF yo'lining buzilishi turli kasalliklarga olib kelishi mumkin:

-Saraton: O'smalarda HIF-ning haddan tashqari faolligi tumor hujayralarining o'sishini rag'batlantiradi.

-Yallig'lanish: HIF-1 α makrofaglarning faollashuviga yordam beradi, bu esa surunkali yallig'lanishga olib kelishi mumkin.

-Qon tomir kasalliklari: Aterosklerozda hipoksiya endoteliy hujayralarining disfunktsiyasiga sabab bo'ladi.

Klinik ahamiyati va yangi davolash usullari

HIF yo'lini nishonga olgan dori vositalari ishlab chiqilmoqda:

-HIF inhibitörleri (masalan, PT2977) — buyrak va o'pka saratonida qo'llaniladi.

-Prolil gidroksilaza inhibitörleri (masalan, Roxadustat) — anemiya davolash uchun.

-VEGF inhibitorlari (masalan, Bevacizumab) — o'smalarda angiogenezni blokirovka qilish uchun.

Muhokama

Epiteliy hujayralarining moslanish qobiliyati ularning fiziologik funksiyalarini saqlab qolish uchun juda muhimdir. Biroq, bu jarayonlarning buzilishi turli xil kasalliklarga olib kelishi mumkin.

Masalan, HIF-1 α ning haddan tashqari faollashishi saraton hujayralarining o'sishini rag'batlantirishi mumkin. EMT jarayonining noto'g'ri boshqarilishi esa metastazga sabab bo'lishi mumkin.

Shu sababli, ushbu yo'llarni nishonga olgan yangi dorilar ishlab chiqish zarur.

Xulosa

Epiteliy to'qimasi hujayralarining adaptatsion mexanizmlari organizmning muhim funksiyalarini saqlab qolish uchun zarurdir. Ushbu jarayonlarni tushunish orqali yangi davolash usullarini ishlab chiqish mumkin.

Kelajakda HIF-1 α , NF- κ B va EMT yo'llarini nishonga olgan terapiyalar saraton, yallig'lanish va regenerativ tibbiyot sohasida muhim ahamiyat kasb etadi.

Epiteliy hujayralari hipoksiyaga HIF yo'li orqali moslanadi, bu ularning omon qolishi va funksiyasini saqlash uchun juda muhim. Biroq, bu mexanizmlarning noto'g'ri boshqarilishi patologiyalarga olib kelishi mumkin. Shuning uchun HIF yo'lini maqsadli ravishda boshqarish yangi terapiyalar ishlab chiqishda muhim rol o'ynaydi.

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