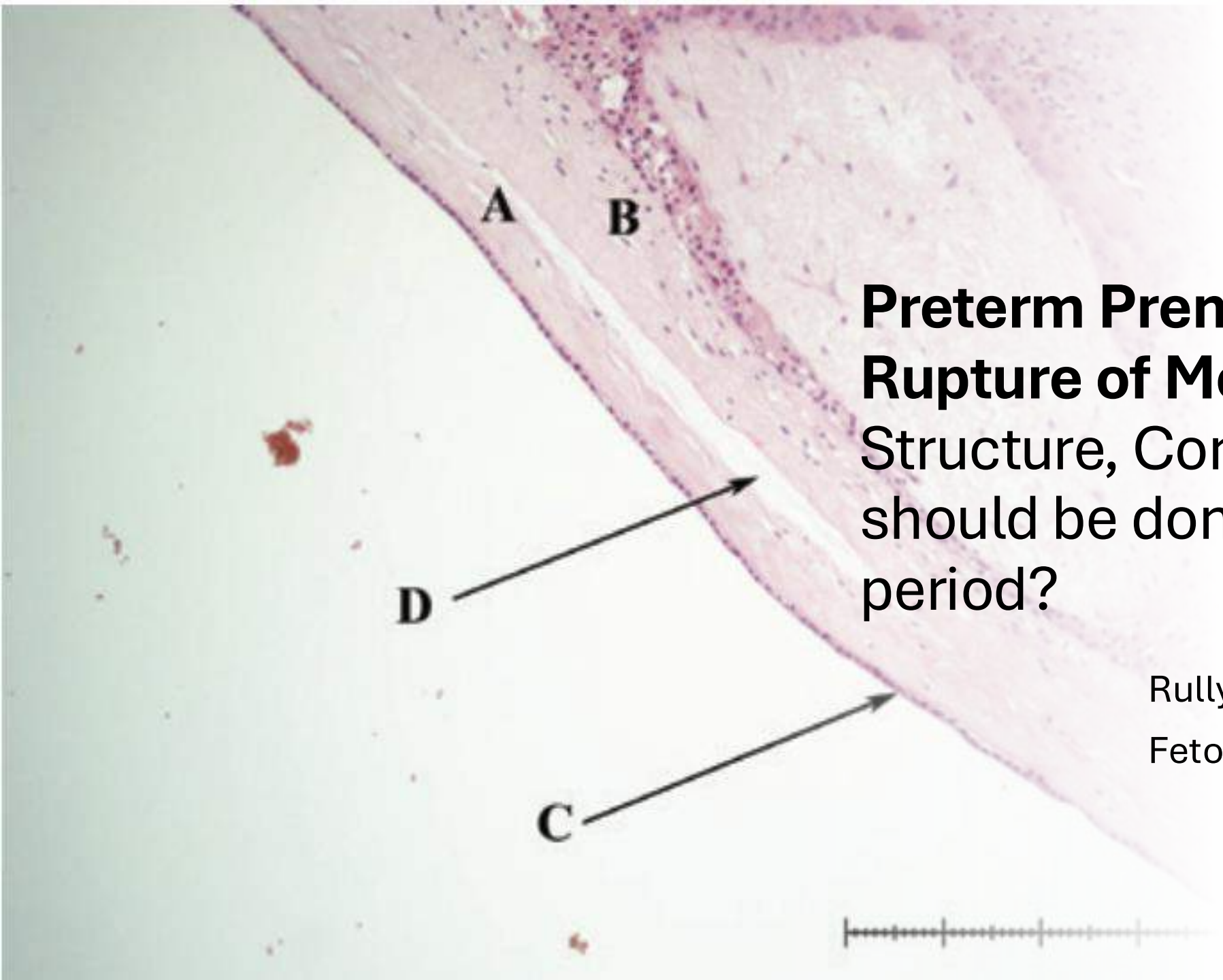


Preterm Premature Rupture of Membrane

Structure, Concept & What should be done in latency period?

Rully Ayu Nirmalasari H.P.

Feto Meeting, July 7th, 2025

Preterm prelabor rupture of membranes (PPROM) is defined as membrane rupture before labor that occurs before 37 0/7 weeks of gestation

PPROM occurs in < 1% of all pregnancies associated with substantial maternal and neonatal infectious morbidity and mortality

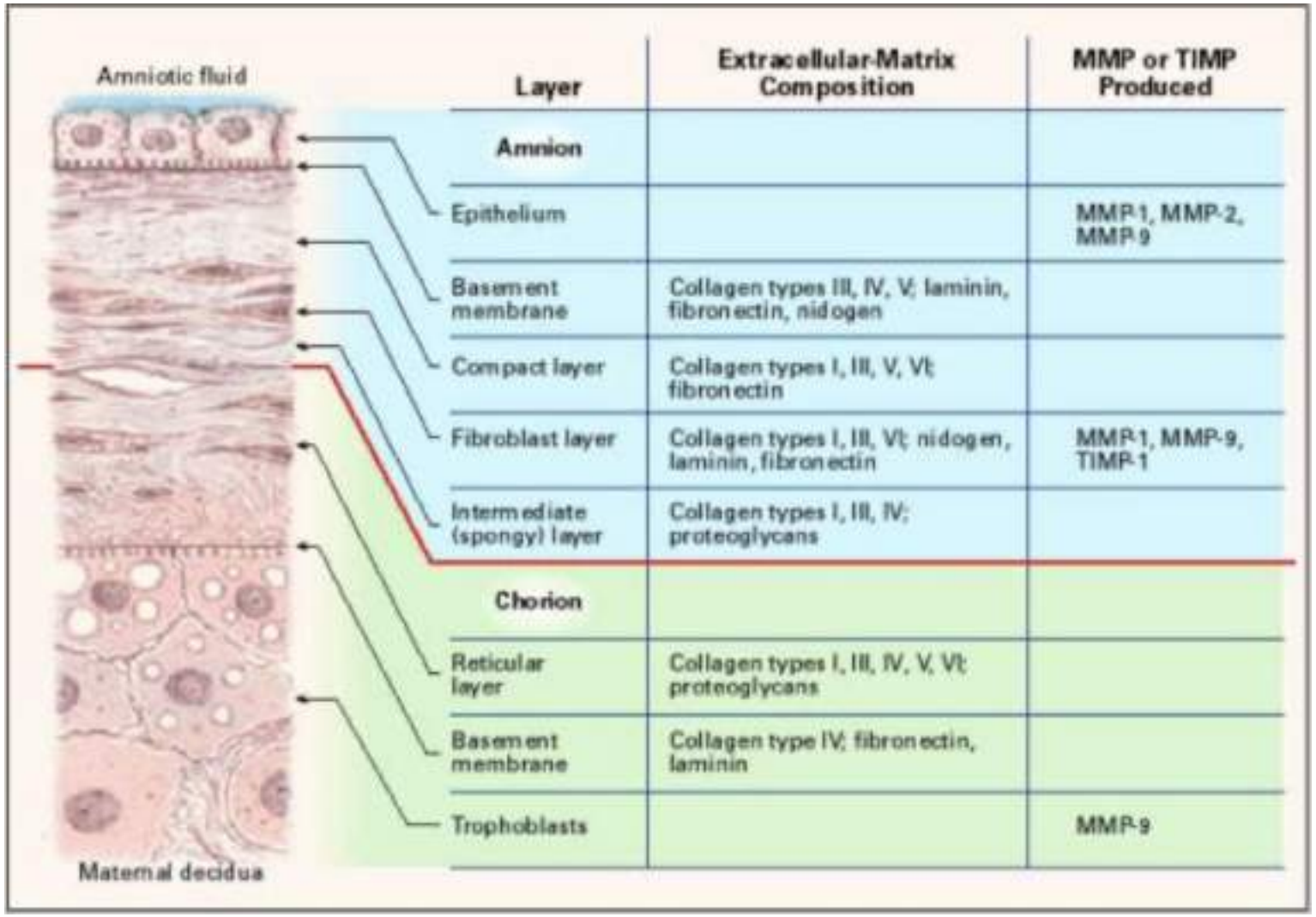
Outline

01	Structure of human fetal membrane
02	Novel concept of PPROM
03	Management during the Latency Period of Preterm Prelabour Rupture of Membranes (pPROM)

Structure of human fetal membrane

Fetal Membrane Structure

- Composed of two distinct layers: amnion and chorion
- Connected by extracellular matrix, rich in collagen
- Amnion, highly elastic, consist of five distinct histological layer, human amnion epithelial cells (hAECs) and human amnion mesenchymal stem cells (hAMSCs)



Layer	Extracellular-Matrix Composition	MMP or TIMP Produced
Amnion		
Epithelium		MMP-1, MMP-2, MMP-9
Basement membrane	Collagen types III, IV, V; laminin, fibronectin, nidogen	
Compact layer	Collagen types I, III, V, VI; fibronectin	
Fibroblast layer	Collagen types I, III, VI; nidogen, laminin, fibronectin	MMP-1, MMP-9, TIMP-1
Intermediate (spongy) layer	Collagen types I, III, IV; proteoglycans	
Chorion		
Reticular layer	Collagen types I, III, IV, V, VI; proteoglycans	
Basement membrane	Collagen type IV; fibronectin, laminin	
Trophoblasts		MMP-9

clinical subtypes of PPROM:

(1) intra-amniotic infection (defined by the presence of both MIAC and intra-amniotic inflammation);

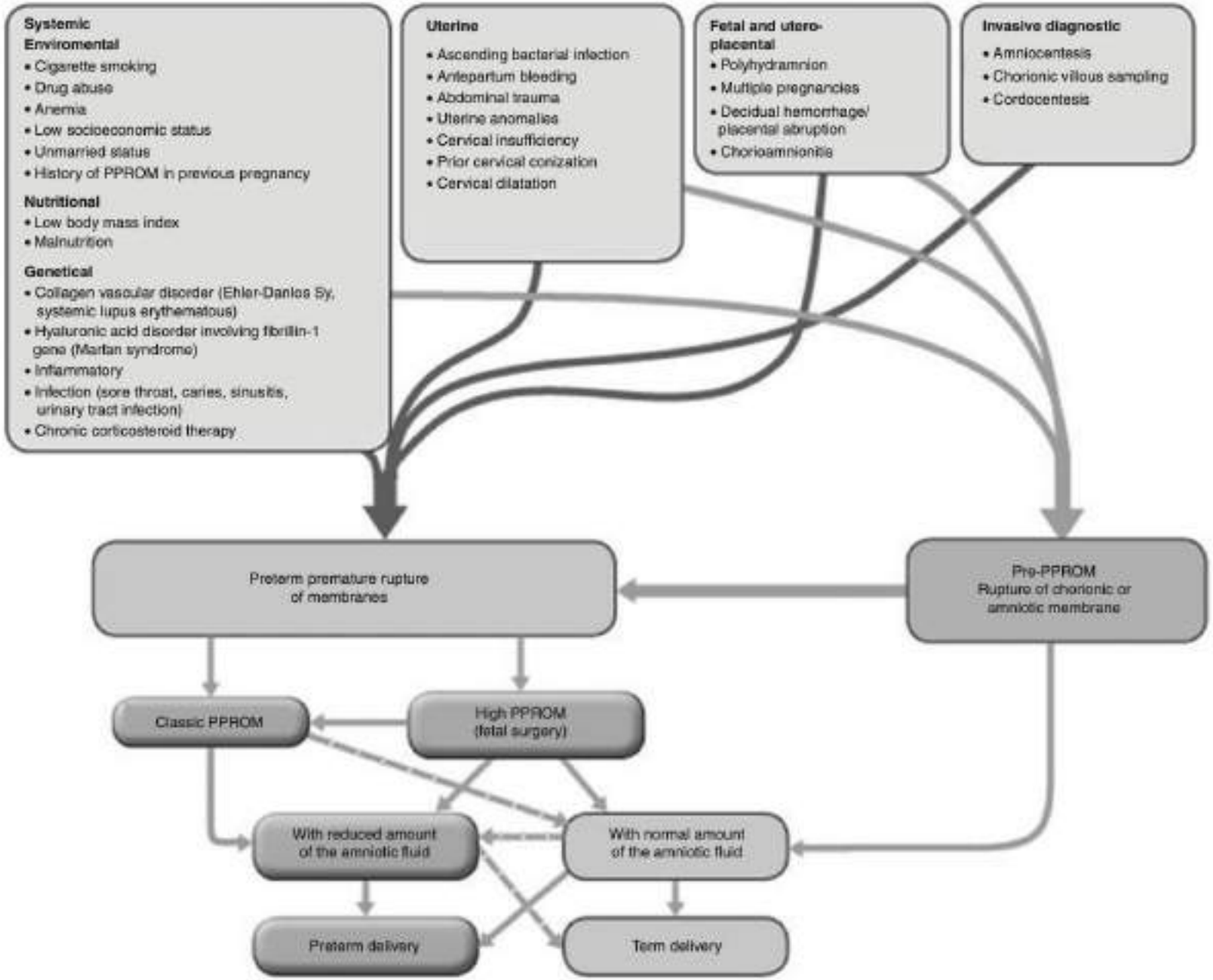
(2) sterile intra-amniotic inflammation;

(3) MIAC without intra-amniotic inflammation;

(4) Without either MIAC or intra-amniotic inflammation.

Novel Concepts in PPROM

PPROM risk factor & classification in second trimester



Tchirikov M, Schlabritz-Loutsevitch N, Maher J, Buchmann J, Naberezhnev Y, Winarno AS, Seliger G. Mid-trimester preterm premature rupture of membranes (PPROM): etiology, diagnosis, classification, international recommendations of treatment options and outcome. Journal of perinatal medicine. 2018 Jul 26;46(5):465-88.

Pathophysiology of Membrane Rupture

Microbial induced infection	membrane stretching	oxidative stress	infammatory responses
premature senescence	external infuences	individual circumstances	medical procedures

Physiologic weakening at Term

- Amniochorion loses its structural and mechanical integrity
- Near-term myometrial smooth muscle cells (MSMC) release chemokines like CCL-2, attracting leukocytes into the myometrium → produce various cytokines → activating infammatory pathways → disrupt fetal membrane function
- Pro-inflammatory biochemical signals from the fetus, such as platelet-activating factor, fetal lung surfactant protein, epidermal growth factor, and endothelins, present in amniotic fluid →exceed immune homeostasis thresholds and induce inflammation in fetal membrane cells
- increase in **Matrix Metalloproteinases (MMPs)** and an imbalance with their inhibitors, Tissue Inhibitors of Metalloproteinases (TIMPs). MMPs, such as MMP-1, MMP-8, and MMP-9, degrade extracellular proteins including collagen

Microbial-Induced Infections

- **70% of PPROM cases are caused by microbial infections**
- *Ureaplasma species* (*Ureaplasma urealyticum*, *Ureaplasma parvum*), *Gardnerella vaginalis*, *Mycoplasma hominis*, and *Streptococcus agalactiae*
- Invasion of microorganisms into the amniotic cavity → intra-amniotic inflammation & fetal inflammatory response
- viral infections can alter cervical mucosal immunity, compromising the ability of the female reproductive tract to resist bacterial infection of the uterus

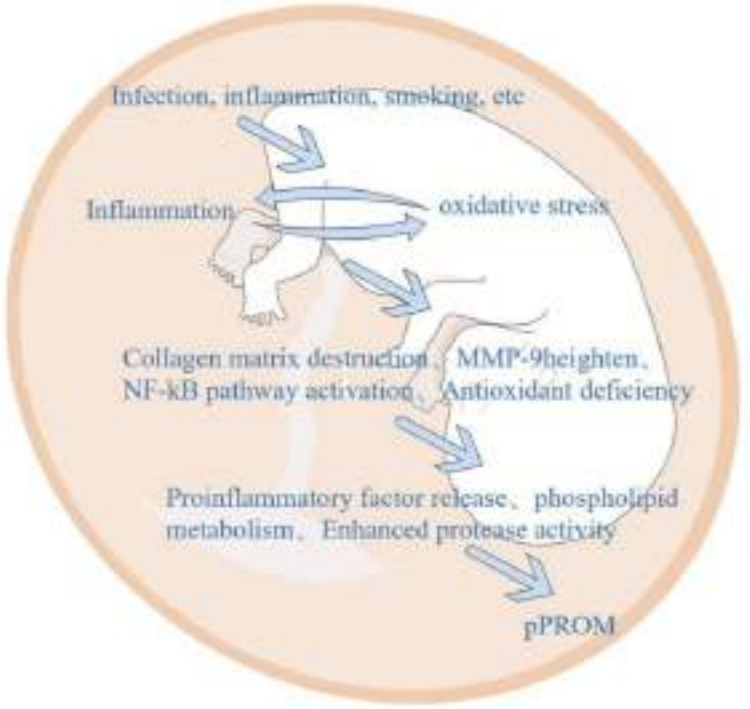
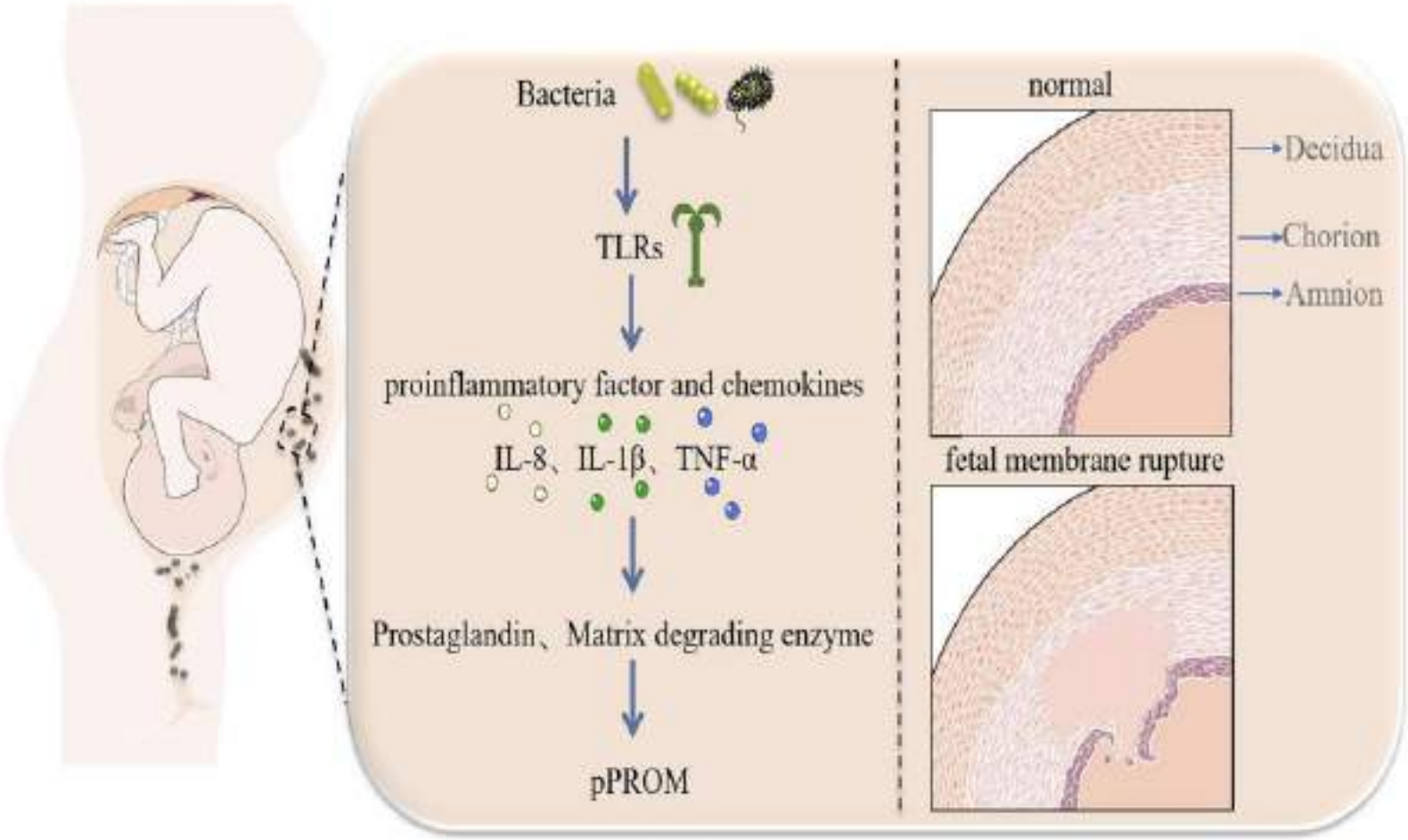


Fig. 3 Mechanisms by which maternal risk leads to pPROM through induction of inflammatory oxidative stress in utero. Abbreviations: MMP-9, Matrix metalloproteinase-9; NF-κB, nuclear factor kappa-B

Xu L, Yang T, Wen M, Wen D, Jin C, An M, Wang L, Liu Y, Fan J. Frontiers in the etiology and treatment of preterm premature rupture of membrane: from molecular mechanisms to innovative therapeutic strategies. Reproductive Sciences. 2024 Apr;31(4):917-31.

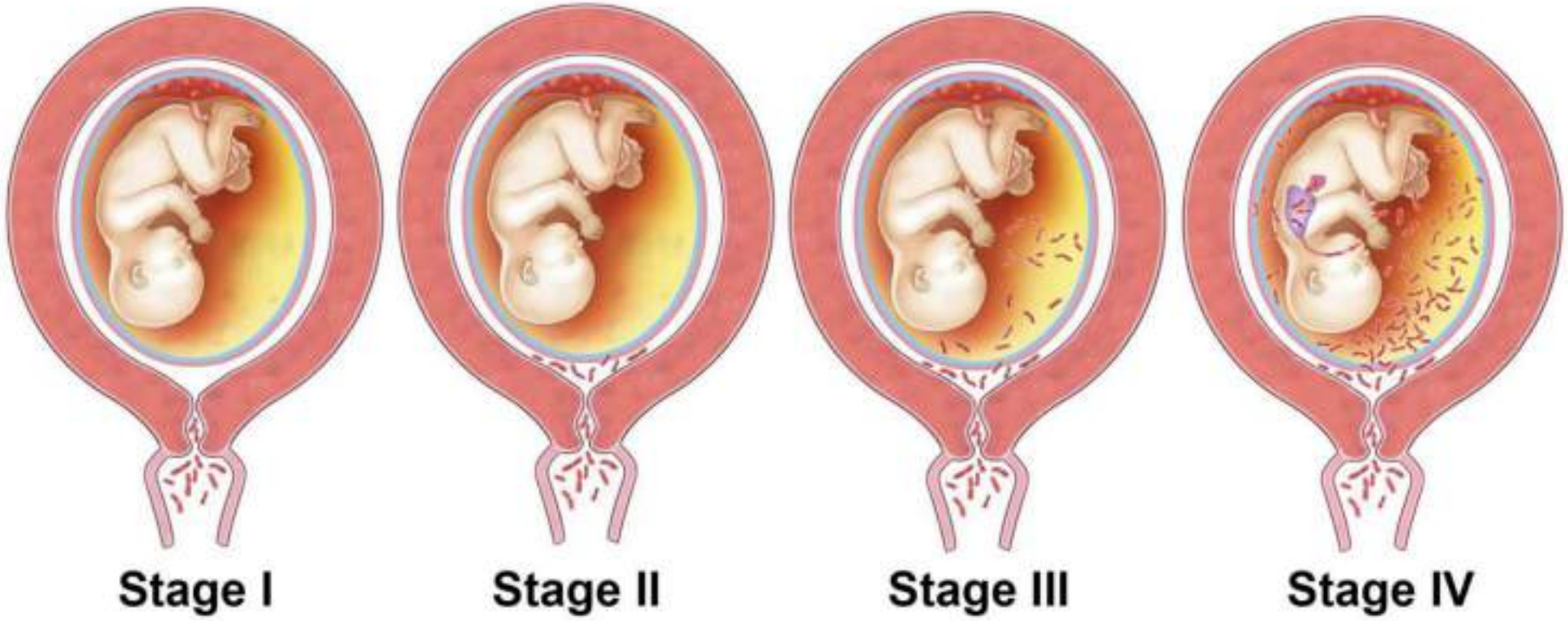
The amniotic cavity is sterile for microorganisms using cultivation and molecular microbiologic techniques, based on the detection of the 16S rRNA gene (present in all bacteria, but not in mammalian cells).

- ❑ upstream infection through the vagina
- ❑ bloodstream transmission through the placenta
- ❑ inadvertent initiation through invasive procedures
- ❑ fallopian tube retrograde transmission

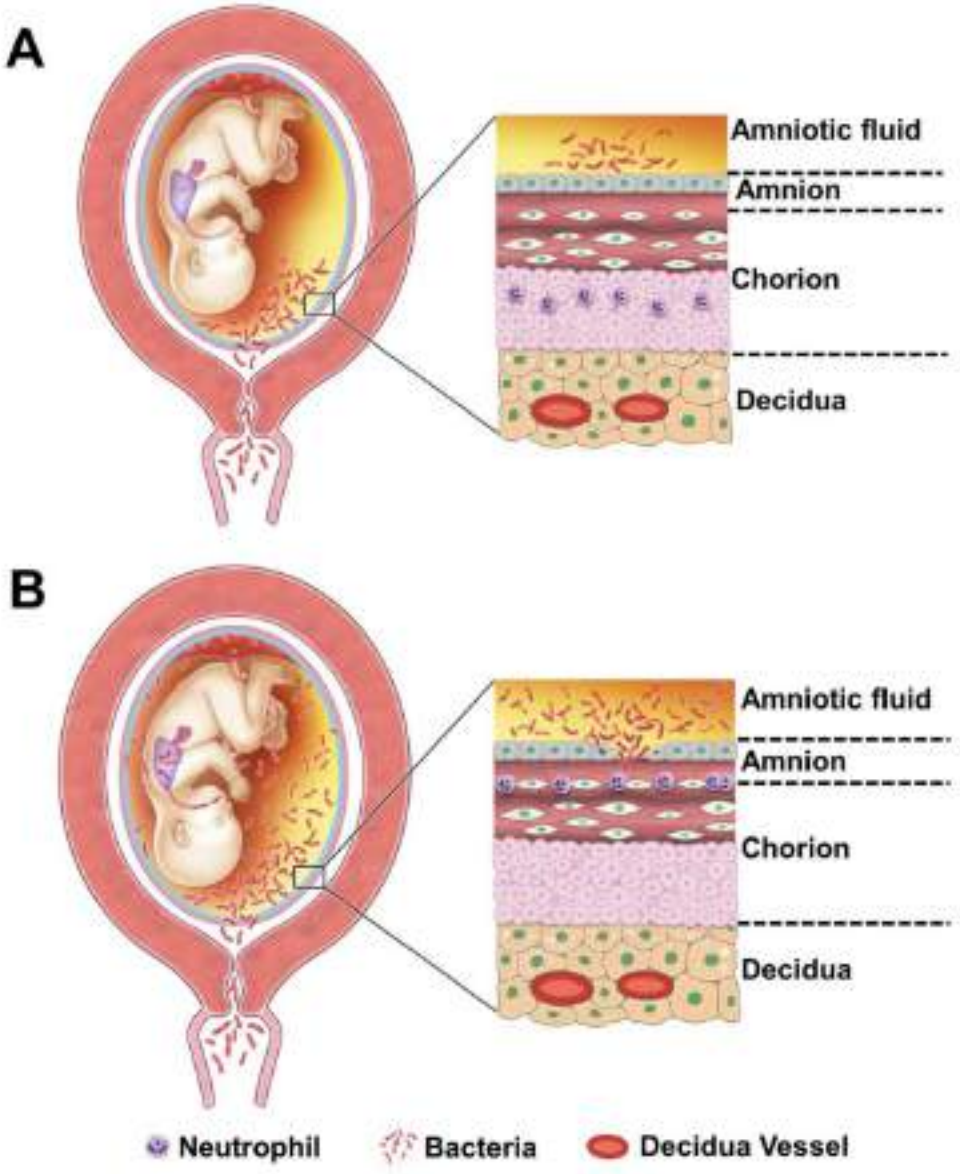


Rupture of membranes is not necessary for bacteria to reach the amniotic cavity – indeed, there is experimental evidence that bacteria can cross intact membranes

The stages of ascending infection in preterm labor



Stage I in the process of ascending infection is corresponding to a change in the vaginal/cervical microbial flora or the presence of pathologic organisms in the cervix. Once microorganisms gain access to the amniotic cavity, they reside in the lower pole of the uterus between the membranes and the chorion (Stage II). The microorganisms may invade the fetal vessels (choriovasculitis) or proceed through the amnion (amnionitis) into the amniotic cavity leading to an intra-amniotic infection (Stage III). The microorganisms may invade the fetus by different ports of entry (Stage IV).

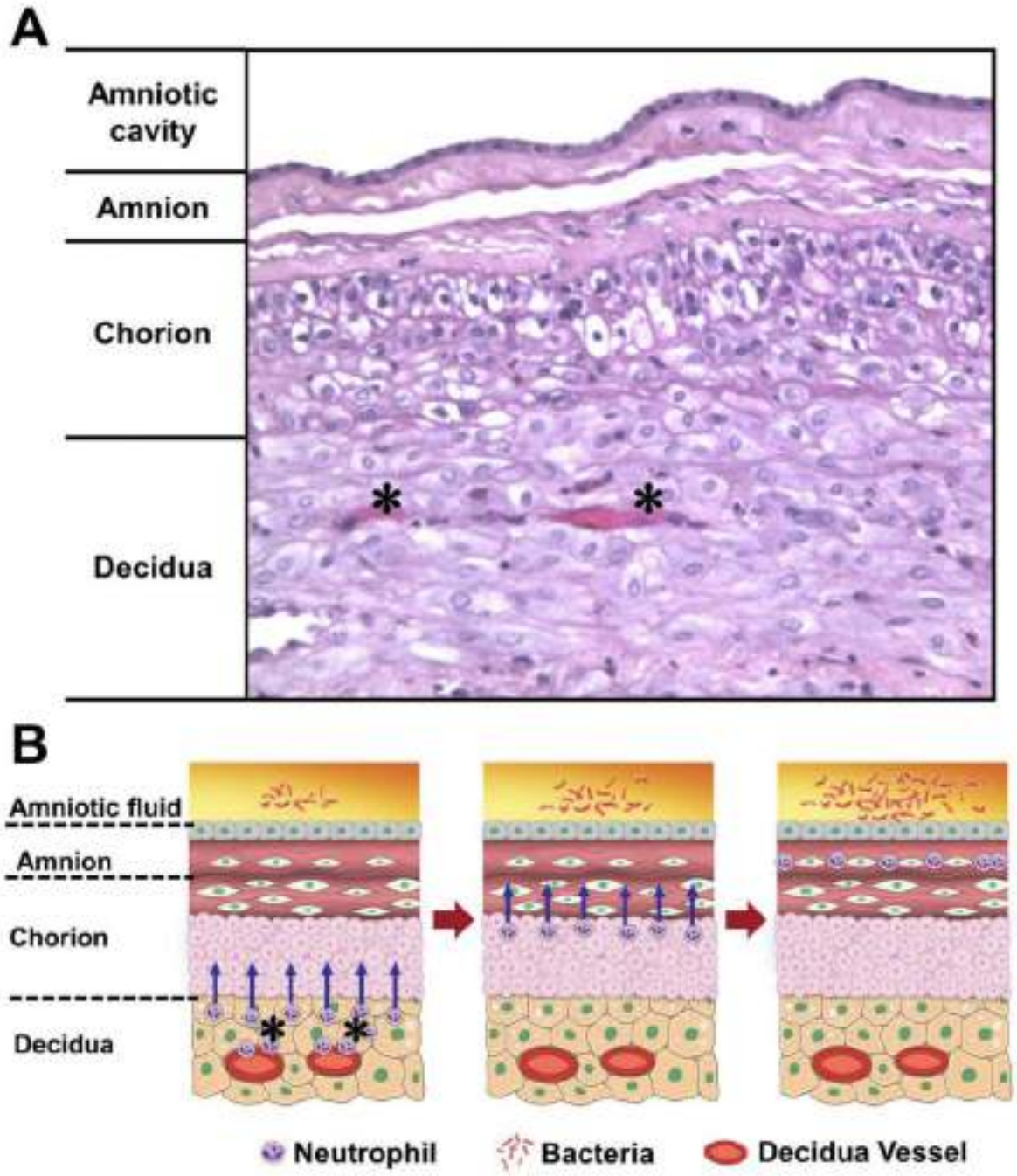


Pathways of intra-amniotic infection

(A) Most cases of microbial invasion of the amniotic cavity are the result of ascending infection from the vagina and cervix.

(B) Extensive microbial invasion of the amniotic cavity can result in fetal infection (bacteria are located in the fetal lung) and damaged chorioamniotic membranes (i.e. necrotizing chorioamnionitis). The destruction of the amnion epithelium is a cardinal feature of necrotizing chorioamnionitis

Kim CJ, Romero R, Chaemsaithong P, Chaiyasit N, Yoon BH, Kim YM. Acute chorioamnionitis and funisitis: definition, pathologic features, and clinical significance. American journal of obstetrics and gynecology. 2015 Oct 1;213(4):S29-52.

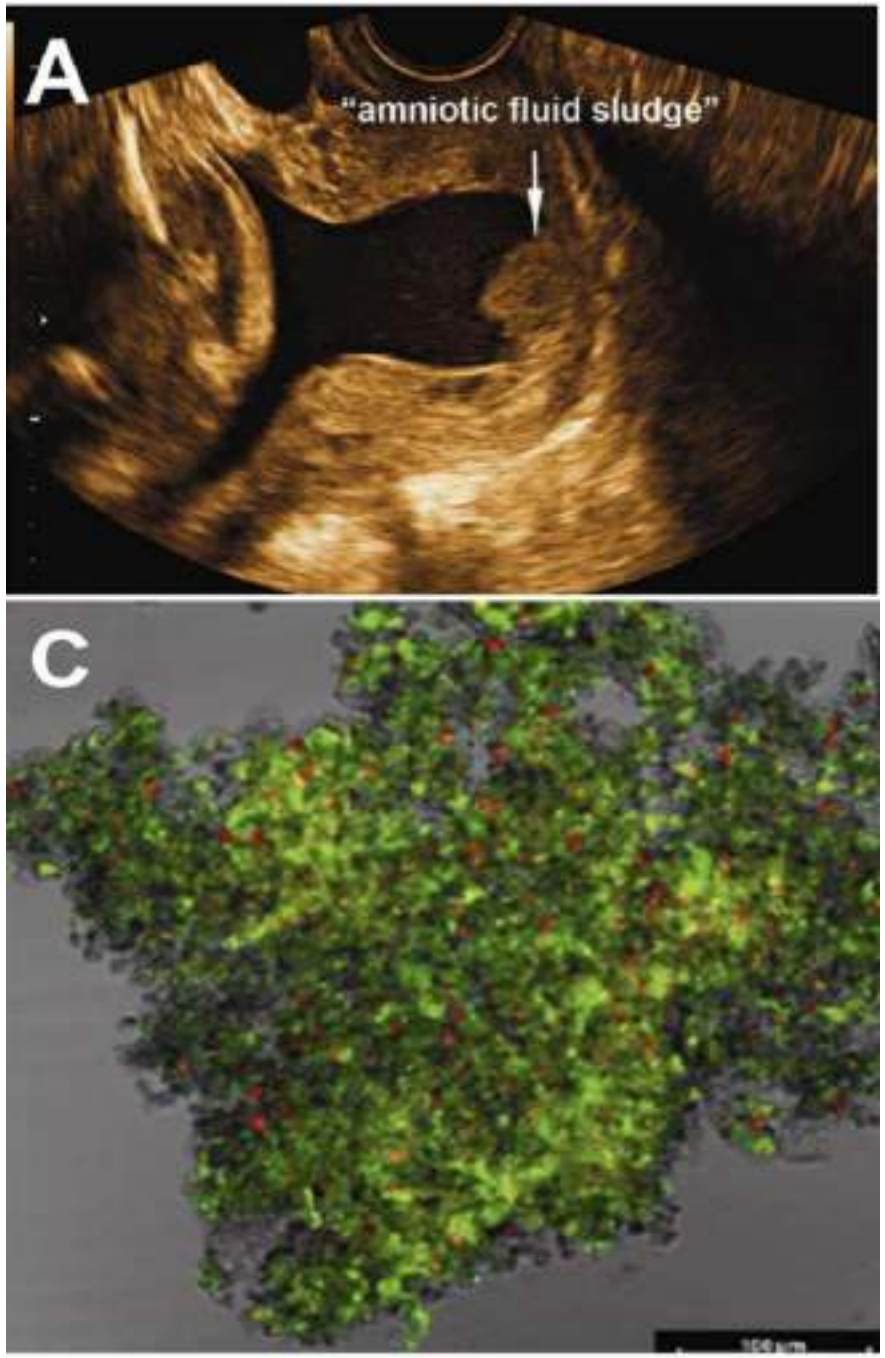


Migration of the neutrophils from decidual vessels into the chorioamniotic membranes. (A) Normal histology of the chorioamniotic membranes, which are composed of amnion and chorion laeve. The decidua is adjacent to the chorion and contains maternal capillaries (black asterisk). Neutrophils migrate from the maternal circulation in the presence of chemotactic gradient (increased amniotic fluid neutrophil chemokine concentrations). (B) Progression of neutrophils from the decidual vessels (in red) towards the amnion.

Kim CJ, Romero R, Chaemsaithong P, Chaiyasit N, Yoon BH, Kim YM. Acute chorioamnionitis and funisitis: definition, pathologic features, and clinical significance. American journal of obstetrics and gynecology. 2015 Oct 1;213(4):S29-52.

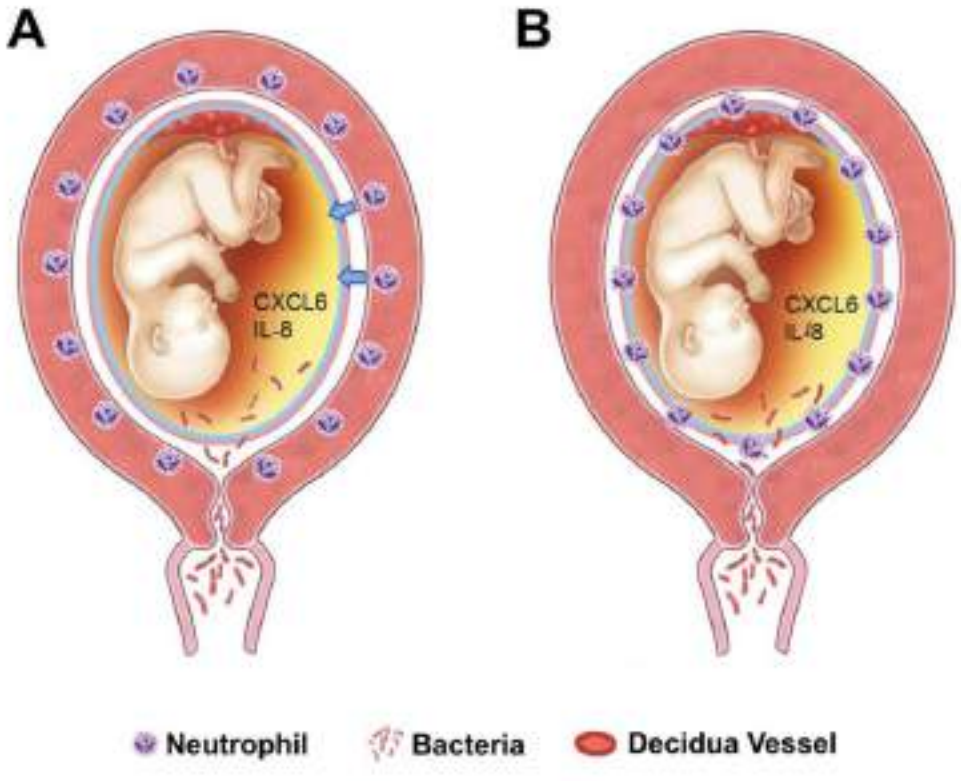
“amniotic fluid sludge”.

Recent evidence suggests that amniotic fluid bacteria can form biofilms. Biofilms are defined as communities of sessile organisms that attach to a substratum or to each other. The presence of biofilms can be clinically suspected when sludge is detected as particulate matter in the amniotic fluid using ultrasound



Kim CJ, Romero R, Chaemsaithong P, Chaiyasit N, Yoon BH, Kim YM. Acute chorioamnionitis and funisitis: definition, pathologic features, and clinical significance. American journal of obstetrics and gynecology. 2015 Oct 1;213(4):S29-52.

Chemotactic signals in the amniotic cavity are responsible for chorioamnionitis and funisitis

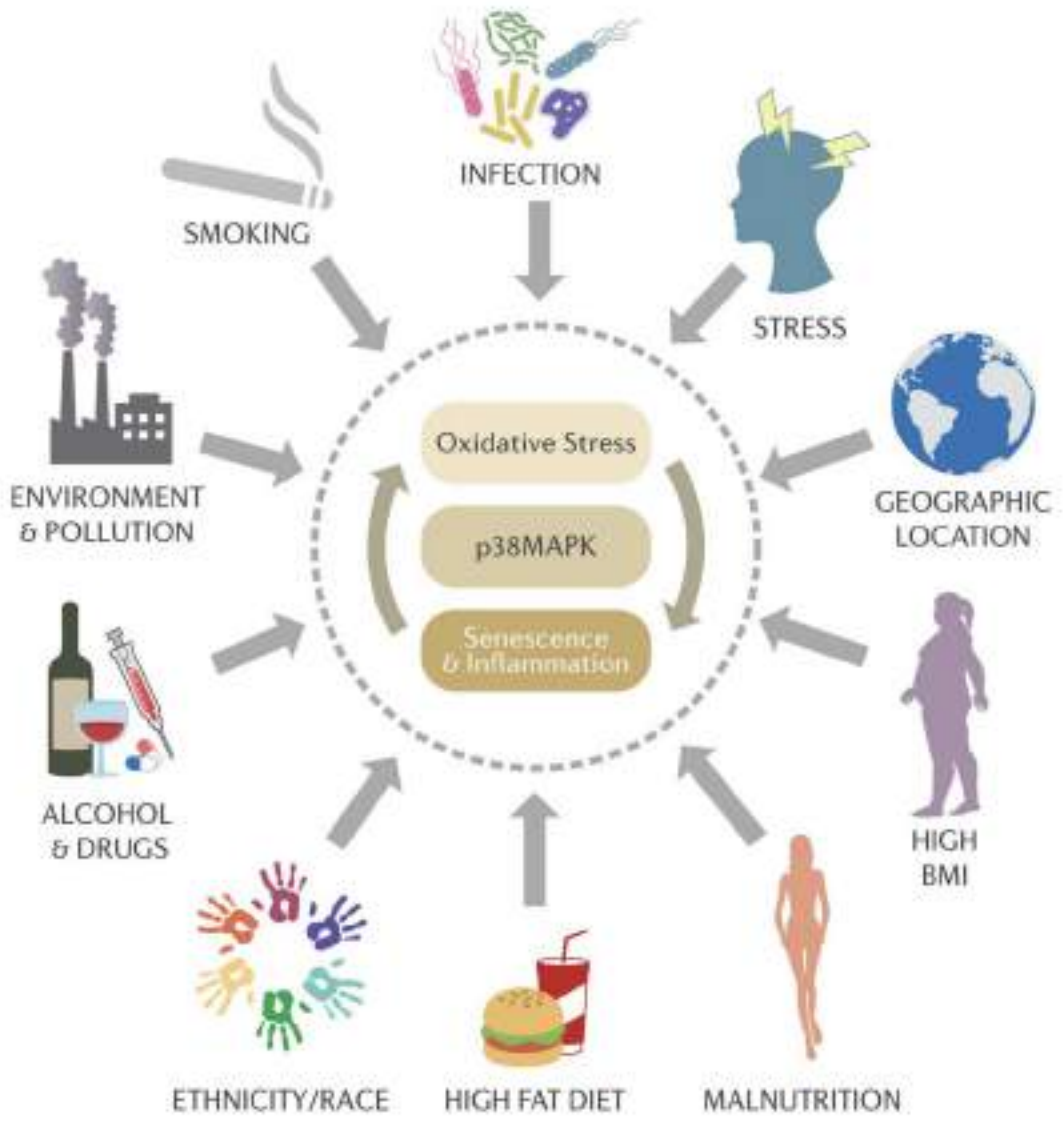


In the absence of microorganisms, danger signals released by cells under stress conditions or cell death can induce intra-amniotic inflammation (“sterile inflammation”)

Microorganism → Chemotactic stimuli provided by neutrophil chemokines (e.g. IL-8, also known as neutrophil activating peptide, and CXCL6 – granulocyte chemotactic protein→ amniotic fluid chemokine concentrations are elevated → “danger signals” → establish a chemotactic gradient favoring migration of neutrophils. In the absence of microorganisms, danger signals released by cells under stress conditions or cell death can induce intra-amniotic inflammation (“sterile inflammation”)3

Sterile intra-amniotic inflammation

- More frequent than intra-amniotic infection
- The presence of inflammation within the amniotic cavity without the presence of microorganisms
- Interestingly, sterile intra-amniotic inflammation is associated with acute histologic chorioamnionitis (40-60% of cases)
- Non-microbial origin example oxidative Stress (OS) is a major pathophysiologic factor →p38MAPK activation & accelerate senescence-associated secretory phenotype (SASP) amnion epithelial cells
- cells undergoing stress, necrosis, or senescence →release alarmins: Damage-Associated Molecular Patterns (DAMPs) →activating the innate immune system
- non-viable microorganisms which may release chemotactic factors leading to inflammation. These organisms may have invaded the amniotic cavity and been cleared by the immune system



FREQUENCY OF CHORIOAMNIONITIS ACCORDING TO GESTATIONAL AGE

Weeks of gestation	Chorioamnionitis (n)	Total number of patients	Percent (%)
21–24	17	18	94.4
25–28	19	48	39.6
29–32	34	96	35.4
33–36	53	497	10.7
37–40	233	6139	3.8
41–44	36	707	5.1
Total	392	7505	5.2

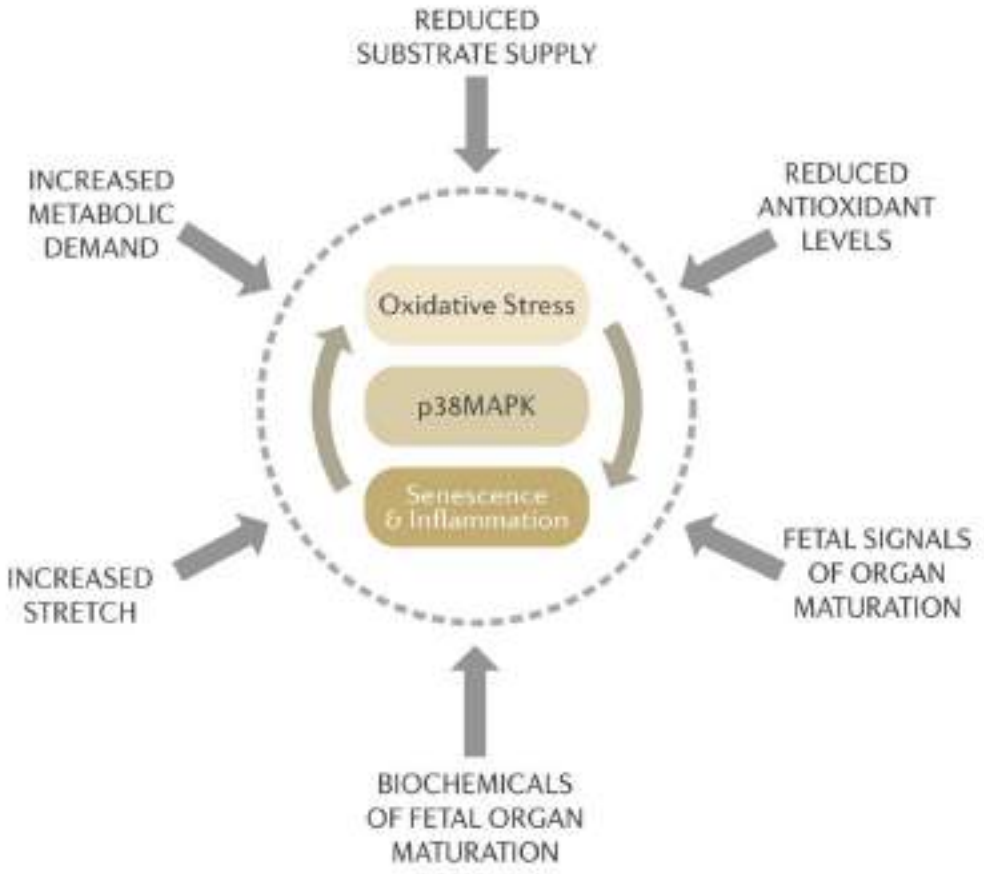
Modified from Russell P, Inflammatory lesions of the human placenta I, The American journal of diagnostic gynecology and obstetrics 1979; 1: 127-137

Membrane Streching

- the amnion is tougher than the chorion
- The extracellular matrix of the amnion consists of fibrillin, elastin cross-linking enzymes, lysyl oxidases, and lysyl oxidase-like (LOXL) enzymes
- Fetal membranes undergo mechanical stretching during fetal growth and development →induces cellular stress and inflammatory responses in the fetal membranes
- stretching lead to increased levels of the pro-inflammatory factor NF-κB, signifcant increase in infammatory cytokines (IL-1β, IL-6, IL-8, TNF-α, CCL2) and prostaglandins (prostaglandin E2, prostaglandin F2α)

Oxidative Stress

- Antioxidant nutrient defciency, high-altitude pregnancies, or underlying microvascular diseases, can lead to oxidative stress (OS)
- OS increases placental mitochondrial activity and production of ROS when the energy demands of the fetoplacental unit are high
- Reactive Oxygen Species (ROS) damage collagen matrix and deplete antioxidant defenses
- significantly lower vitamin C levels in women with PPROM compared to controls, suggesting a role for antioxidant deficiencies
- NAC inhibits MMP9 and NF-κB activated pathway and pro-inflammatory cytokine release, and protease activity in human–fetal membranes



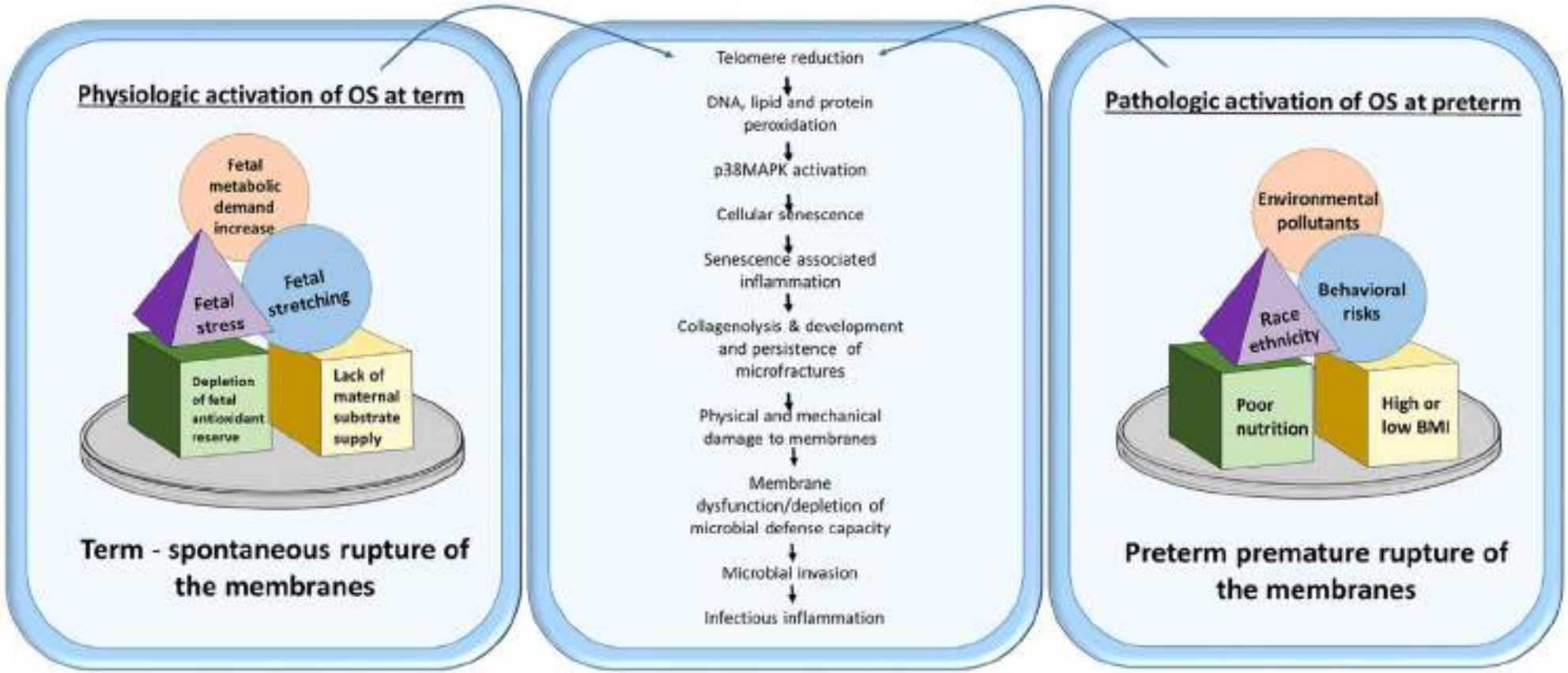
Senescence (aging) of the fetal membranes

- Premature aging of membranes (senescence) releasing inflammatory markers and matrix-degrading enzymes
- activates p38 Mitogen-Activated Protein Kinase (MAPK→promotes cell cycle arrest and induces senescence in amniotic membrane cells
- Reduced remodeling and healing capacity formation and persistence of Microfractures (being wider and deeper)→ channels for amniotic fluid leakage, and for the infiltration of inflammatory cells and microbes
- Evidence of senescence in pPROM includes shorter fetal membrane and cord blood DNA telomere lengths

External Influences, Individuals cirmcustances & Medical procedures

- Behavioral factors (such as smoking, drug abuse, and alcohol consumption) contribute to the buildup of OS
- History of PPROM in a previous pregnancy, history of previous preterm delivery, cervical incompetence, antepartum hemorrhage, poor nutritional status associated with pPROM
- Invasive diagnostic procedures such as amniocentesis, fetoscopy, chorionic villous sampling (CVS), or cordocentesis → 1-2% fluid leak

Factors Affecting Fetal Membrane Homeostasis Leading to Dysfunction and Rupture



Components of membrane dysfunction related to term spontaneous rupture of the membranes.

Components of membrane dysfunction related to Preterm premature rupture of the membranes.

The Fetal Inflammatory Response Syndrome (FIRS)

The ports of entry for bacteria into the fetus include the respiratory tract (fetal breathing), gastrointestinal tract (swallowing), skin, and ear.

Once microorganisms gain access to the fetal mucosa, they are recognized by pattern recognition receptors such as Toll-like receptors (TLRs)

induce the production of transcription factors such as NFκB

microorganisms reaching the fetal circulation could lead to a systemic inflammatory response

Maternal risk score for the prediction of fetal inflammatory response syndrome after preterm premature rupture of membranes

Mariko Nakahara[1], Shunji Goto[2], Eiji Kato[2], Shuko Nojiri[3], Atsuo Itakura[1] and Satoru Takeda[1]

[1]*Department of Obstetrics and Gynecology, Juntendo University Faculty of Medicine, Bunkyo-ku, Tokyo, Japan*
[2]*Perinatal Center for Maternity and Neonate, Japan Community Health Care Organization Funabashi Central Hospital, Funabashi, Japan*
[3]*Medical Technology Innovation Center, Clinical Research and Trial Center Juntendo University Faculty of Medicine, Tokyo, Japan*

Table 3 The point scoring system of FIRS score

Maternal factors	Cut-off value	Point
Expected delivery weeks	≤30 weeks	6
Maternal CRP	≥1.2 mg/dL	7
Maternal WBC	≥13 000/μL	3
Corticosteroid use	None	1
PROM latency period	≥3 days	5

CRP, C-reactive protein; PROM, premature rupture of membrane; WBC, white blood cell count.

Table 4 Correlation between FIRS score (the total point score) and other characteristics (n = 158)

Category	FIRS score	n	FIRS (%)	Umbilical cord blood IL-6 (pg/mL)	HCA (%)	Funisitis (%)	Maternal CRP (mg/mL)	Maternal WBC (/μL)	Delivery weeks (weeks)
Low	0–7	65	7 (11)	3.3 (1.7, 5.9)	7 (11)	4 (6)	0.35 (0.22, 0.71)	9800 (8500, 11 400)	34 (32,34)
Intermediate	8–15	60	30 (50)	10.7 (4.0, 39.8)	26 (43)	13 (22)	0.98 (0.23, 2.24)	13 650 (10 675, 16 675)	30 (28,32)
High	16–22	33	29 (88)	72 (27, 269)	24 (75)	18 (55)	2.29 (1.79, 4.04)	16 400 (14 200,18 500)	28 (26,30)

Values are presented as numbers (%) or medians (interquartile range). CRP, C-reactive protein; FIRS, foetal inflammatory response syndrome; HCA, histological chorioamnionitis; IL-6, interleukin-6; PROM, premature rupture of membrane; WBC, white blood cell count.

Can Premature Rupture of Membranes Heal Spontaneously?

- self-repair capacity of pPROM is limited due to the lack of vascular tissue in fetal membranes
- The main self-healing modes of fetal membranes are: sliding of amnion and chorionic villus segments against each other, which reduces the size of functional defects
- The fetal membrane lacks vascular tissue, and a gap of more than 2 mm exceeds the limit of the self-repairing ability of the fetal membrane

Reproductive Sciences (2024) 31:917–931 https://doi.org/10.1007/s43032-023-01411-9

Management during the Latency Period

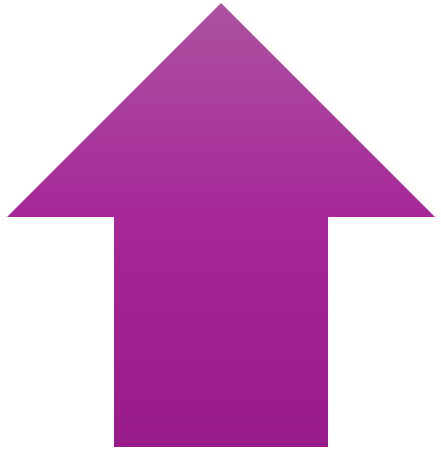
The latency period in pPROM refers to the time between the rupture of the fetal membranes and the onset of labor

The approach to managing pPROM during the latency period largely depends on the gestational age, the presence of any maternal or fetal contraindications to expectant management & neonatal survival rate

- Careful monitoring
- Antibiotic
- Probiotic
- Tocolysis
- Corticosteroid
- Neuroprotection
- Amnioinfusion

the risk of intra-amniotic infection and its consequences for the mother and infant

benefits of prolongation of the pregnancy



Careful Monitoring for Infection and Other Complications

clinical assessment (pulse, blood pressure, temperature and symptoms), maternal blood tests (C-reactive protein and white cell count) and fetal heart rate using cardiotocography, should be employed to diagnose clinical infection

Inpatient: vital signs, including pulse, blood pressure, respiratory rate and temperature, recorded on an obstetric early warning chart.

Outpatient: blood tests [white cell count and C-reactive protein], clinical recordings and fetal heart rate monitoring) 1 – 2 times per week

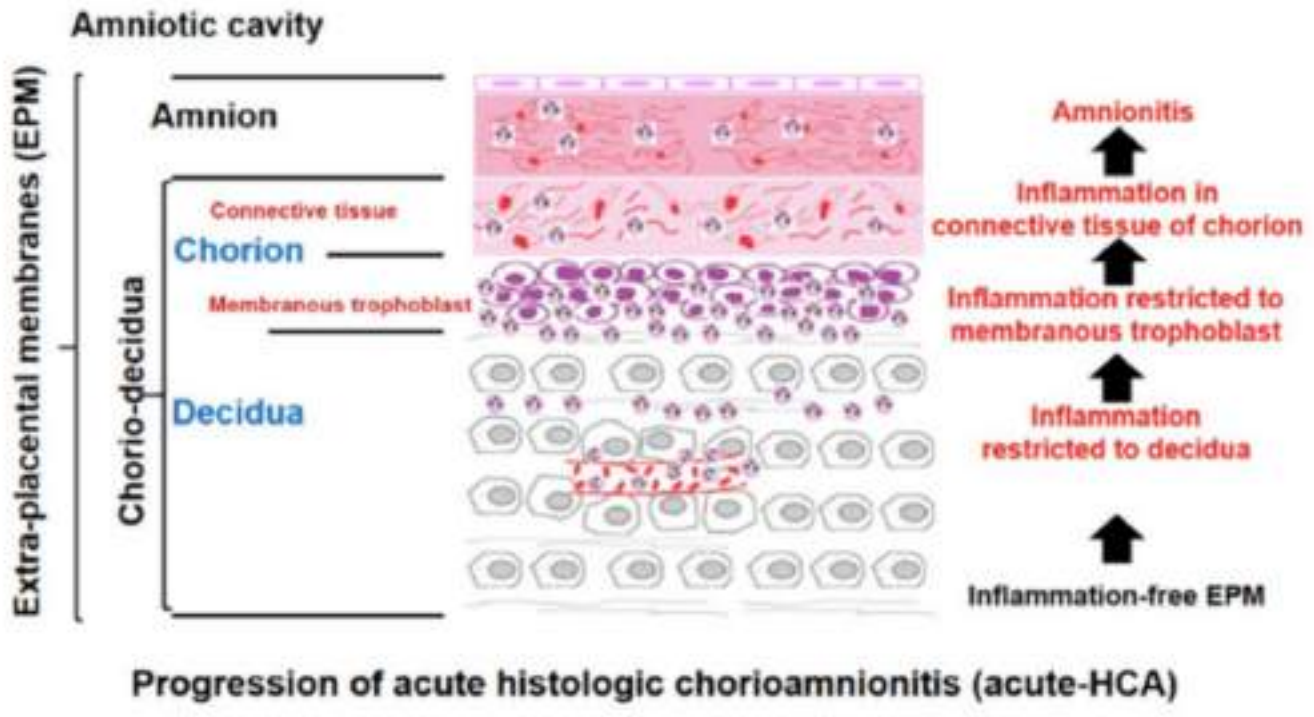
C-reactive protein has a sensitivity of only 68.7% and specificity of 77.1% in diagnosing histological chorioamnionitis

The **Neutrophil/Lymphocyte Ratio (NLR)** in maternal blood has been shown to progressively increase with the progression of acute histological chorioamnionitis. Increased maternal NLR (≥7.75) is an independent risk factor for amnionitis

NLR AS INDEPENDENT RISK FACTOR FOR HC

	Sensitivity	Specificity	Positive Predictive Value	Negative Predictive Value	Positive LR (95% CI)	Negative LR (95% CI)
NLR ≥ 7.75	80.0% (12/15)	59.0% (69/117)	20.0% (12/60)	95.8% (69/72)	2.9487 (1.0597–8.2047)	0.5128 (0.3674–0.7158)

CI, confidence interval; LR, likelihood ratio; NLR, neutrophil to lymphocyte ratio.

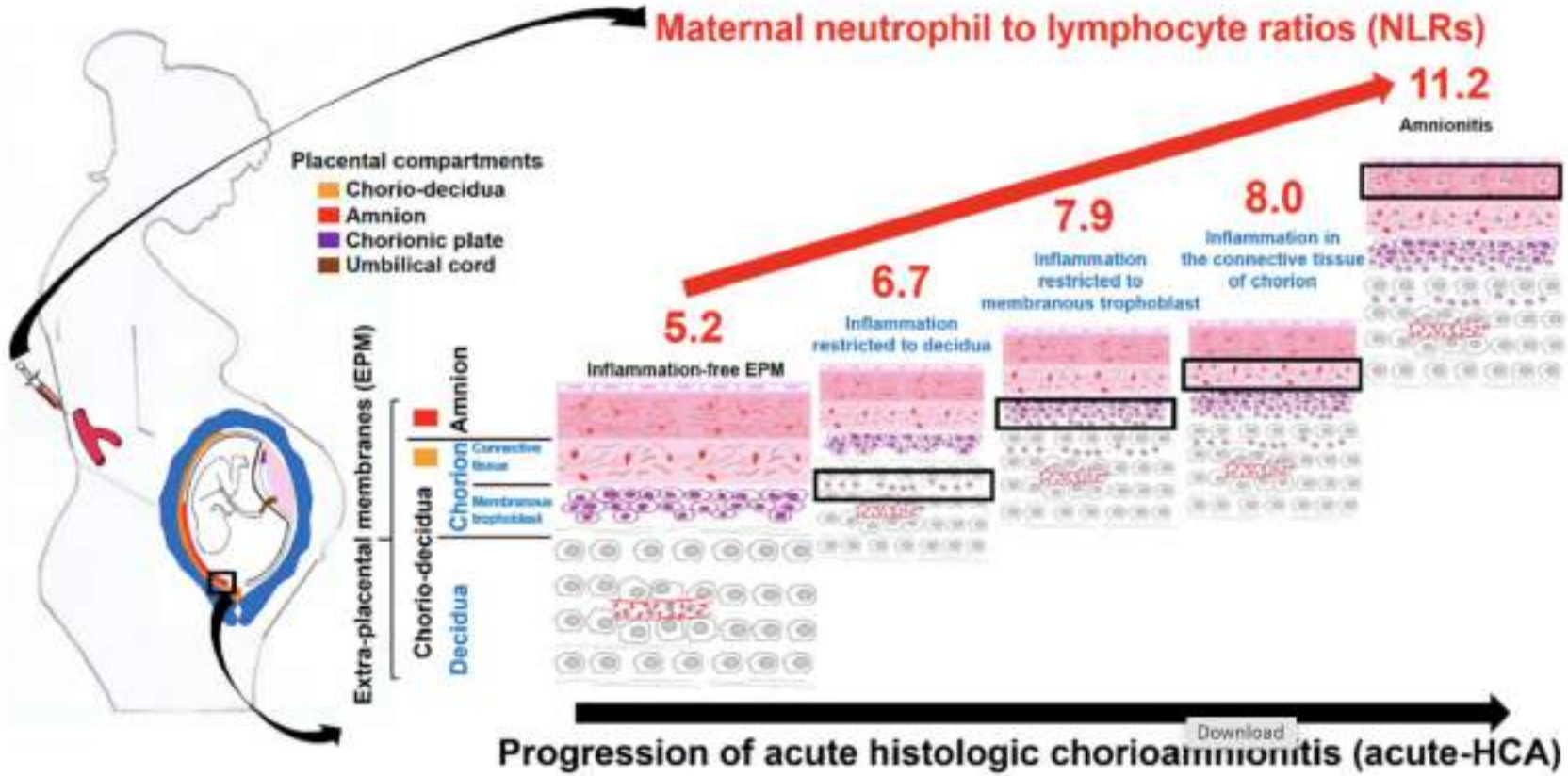


J. Clin. Med. 2021, 10, 2673. https://doi.org/10.3390/jcm10122673

Prediction of Histological chorioamnionitis

Blood inflammatory markers	Cut-off value	Sensitivity % (95 % CI)	Specificity % (95 % CI)	PPV % (95 % CI)	NPV % (95 % CI)
C-reactive protein	8.5	84 (74–91)	77 (63–87)	74 (60–85)	86 (76–92)
White blood cells	12.62×10^9	75 (65–84)	75 (61–86)	65 (65–77)	83 (73–91)
Neutrophil–lymphocyte ratio	5.97	77 (63–97)	95 (88–99)	91 (78–97)	87 (79–93)

Balciuniene G, Kvederaite-Budre G, Gulbiniene V, Dumalakiene I, Viliene R, Pilypiene I, Drasutiene GS, Ramasauskaite D.
Neutrophil–lymphocyte ratio for the prediction of histological chorioamnionitis in cases of preterm premature rupture of membranes: a case-control study. BMC Pregnancy and Childbirth. 2021 Dec;21:1-7.



J. Clin. Med. 2021, 10, 2673. https://doi.org/10.3390/jcm10122673

Antibiotics

- statistically significant reduction in chorioamnionitis (RR 0.66, 95% CI 0.46–0.96). reduction in the numbers of babies born within 48 hours (RR 0.71, 95% CI 0.58–0.87) and 7 days (RR 0.79, 95% CI 0.71–0.89). Neonatal infection, use of surfactant, oxygen therapy and abnormal cerebral ultrasound prior to discharge from hospital was also reduced.
- pPROM at **<34 weeks of gestation**, a common regimen involves **intravenous ampicillin and erythromycin for 48 hours, followed by oral amoxicillin and erythromycin for an additional 5 days**. Erythromycin is generally recommended for 10 days or until the woman is in established labor.
- **Co-amoxiclav should be avoided** due to an increased risk of neonatal necrotizing enterocolitis and may be associated with an increased risk of cerebral palsy or epilepsy in children born to women who received them, but further research is needed
- The combination of **ceftriaxone, clarithromycin, and metronidazole** can treat and prevent intra-amniotic inflammation/infection in patients with preterm PROM.

J Matern Fetal Neonatal Med. Author manuscript; available in PMC: 2017 Nov 27.
Published in final edited form as: J Matern Fetal Neonatal Med. 2015 Dec 2;29(17):2727–2737. doi: 10.3109/14767058.2015.1103729

Antenatal corticosteroids

Administration of corticosteroids to women with PPROM reduces the risks of respiratory distress syndrome (RR 0.81, 95% CI 0.67–0.98) and intraventricular haemorrhage (RR 0.49, 95% CI 0.25–0.96).

No increased risk of chorioamnionitis or neonatal sepsis with maternal steroid use.

Recommended for pregnancies between **24+0 and 33+6 weeks of gestation**.

They may also be considered as early as 23 0/7 weeks and up to 35+6 weeks.

An additional course of corticosteroids may be considered if several weeks have passed since the initial dose and a new episode of pPROM or threatened preterm labor occurs at an early gestational age (<33 weeks and at least 14 days after the first therapy), but more than two courses should be avoided

Magnesium sulfate for neuroprotection

- reduces cerebral palsy (RR 0.69, 95% CI 0.55–0.88) and motor dysfunction in the offspring (RR 0.6, 95% CI 0.43–0.83).24–26 The benefit is greatest before 30+0 weeks of gestation.
- recommend offering magnesium sulfate to women at risk of giving birth before 30+0 weeks of gestation.
- recommends that magnesium sulfate should be considered when preterm birth is anticipated between 30+0 and 33+6 weeks
- established labour or having a planned preterm birth within 24 hours, intravenous magnesium sulfate should be offered between 24+0 and 29+6 weeks of gestation

tocolytic agents

- A Cochrane review found that tocolysis does not significantly improve perinatal outcome and might be associated with an increased risk of chorioamnionitis

- Insufficient evidence to support the use of tocolysis in women with PPROM, as there is an increase in maternal chorioamnionitis without significant benefits to the neonate

Amnioinfusion in PPROM

A Cochrane systematic review of five trials (using the data from four) found that amnioinfusion is associated with: improved fetal umbilical artery pH at delivery, reduced variable decelerations in labour, neonatal death, neonatal sepsis, pulmonary hypoplasia and puerperal sepsis.→ further evaluation needed

Inpatient Criteria (Hospitalization Recommended/Required)

- Early Gestational Age: Patients with pPROM at less than 34 weeks of gestation are typically admitted to the hospital for expectant management, unless there are contraindications. For PPROM in the periviable period (20 0/7 to 25 6/7 weeks), initial hospital observation is recommended to ensure stability before considering discharge.
- Concerning Maternal or Fetal Conditions:
  - Nonreassuring fetal status
  - Clinical Chorioamnionitis
  - Significant Vaginal Bleeding
  - Imminent Labor
- Administration of Interventions Requiring Close Monitoring:
  - Antenatal corticosteroids for fetal lung maturation
  - Magnesium sulfate for fetal
  - Initial intravenous antibiotics

Criteria for outpatient care

Selection criteria prior to discharge were:

- Singleton pregnancy
- Cephalic presentation
- Patient is clinically stable for at least 5–7 d, no additional pregnancy risks such as preeclampsia, fetal growth retardation, placenta previa
- Unremarkable CTG, no fetal tachycardia, no contractions
- No signs of infection/Triple I (no fever, lab test results are unremarkable)
- Cervical opening < 2 cm, cervical length > 20 mm
- No vaginal bleeding
- No persistent anhydramnios (except < 22 GW), no green amniotic fluid
- Proximity to a perinatal center (< 30–40 min)
- Requested by the pregnant woman
- Compliance of the pregnant woman, informed consent, no language barrier
- Bacterial cultures: culture tests to detect Group B Streptococcus (GBS) and multi-resistant gram-negative bacilli (MRGN) are essential to allow stratification by the Neonatology Department

Comparison of international guidelines on the outpatient approach for pregnant women with preterm premature rupture of membranes (PPROM)

Guideline	Recommendation	Monitoring
ACOG 17 (USA)	Inpatient monitoring as soon as the fetus has achieved viability	Not applicable
RCOG 18 (UK)	Individual decision depending on risk factors for a shortened latency period (evidence level 3)	No optimal monitoring method capable of predicting adverse fetal outcome
GNCOF 19 (France)	If the patient remains clinically stable over 48 h, outpatient care possible (professional consensus)	Clinical signs of infection and laboratory tests No information about the frequency of testing
SOGC 20 (Canada)	Inpatient: 72 h Outpatient care if: • > 23 GW • Lives near the hospital • No contractions, no signs of infection, no maternal or fetal risk factors, fetus is well, singleton pregnancy	Perinatal center: 1×/week
Queensland 21 (Australia)	Individual decision; if the patient is suitable, consider outpatient care	Self-monitoring 1–2×/d CTG: 1×/week US: every 2 weeks Lab tests: if indicated

Geburtshilfe Frauenheilkd. 2024 Jan 3;84(1):43–47. doi: 10.1055/a-2205-1725

Monitoring during outpatient care (follow-up strategy)

From (22+0) 24+0–33+6 weeks of gestation:

- between 22+0–23+6 GW: if maximum therapy is requested
- admission as an inpatient for at least 5–7 d
- interdisciplinary counselling by Obstetrics and Neonatology departments
- antenatal corticosteroids
- antibiotic prophylaxis (e.g., IV administration of ampicillin for 2 d, followed by 5 d of oral amoxicillin plus a single dose of oral azithromycin at the start)
- Group B Streptococcus status has been investigated and result is available (if status is known or test was done more than 5 weeks ago)
- If the patient is clinically stable for at least 5–7 d: outpatient management based on strict selection criteria is possible (s. above)

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Monitoring during outpatient care (follow-up strategy)

> 34+0–36+6 weeks of gestation:

- admission as an inpatient for at least 5 d
- antibiotic prophylaxis (e.g., IV administration of ampicillin for 2 d, followed by 5 d of oral amoxicillin plus a single dose of oral azithromycin at the start)
- Group B Streptococcus status has been investigated and result is available (if status is known or test was done more than 5 weeks ago)
- if the patient is clinically stable for 5–7 d: outpatient management based on strict selection criteria is possible (s. above)

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Option for outpatient monitoring:

- 2× day: temperature measurement with recording of results
- 2× week: lab tests (leukocytes)
- 2× week: clinical evaluation including CTG
- 1× week: ultrasound checkup

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Appropriate time to deliver the baby

- Women whose pregnancy is complicated by PPROM after 24+0 weeks' gestation and who have no contraindications to continuing the pregnancy should be offered **expectant management until 37+0 weeks**
- timing of birth should be discussed with each woman on an individual basis with careful consideration of patient preference and ongoing clinical assessment
- women with PPROM 'with no contraindications to continuing the pregnancy, expectant management with careful monitoring is associated with better outcomes for the mother and baby'.

Thank You