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講題摘要：

Pediatric pulmonary hypertension (PedPH) is a heterogeneous group of pulmonary vascular disorders that differs fundamentally from adult pulmonary hypertension in epidemiology, etiology, developmental biology, genetics, clinical presentation, and therapeutic approaches. Rather than representing a smaller form of adult pulmonary arterial hypertension (PAH), PedPH encompasses unique developmental disorders that begin during fetal life and continue throughout postnatal lung growth. Recent advances in molecular biology, genetics, and targeted therapies have significantly improved our understanding of disease mechanisms and clinical management.

The pathogenesis of PAH is characterized by three major pathological processes: pulmonary vasoconstriction, pulmonary vascular remodeling, and vascular pruning. These changes result from dysregulation of three principal signaling pathways: endothelin-1 (ET-1), nitric oxide (NO)-cGMP, and prostacyclin (PGI₂). Reduced BMPR2 signaling together with activation of TGF- β , PDGF, HIF-1 α , and glycolytic pathways promotes excessive proliferation of pulmonary vascular cells. These molecular abnormalities resemble the hallmarks of cancer, including sustained proliferation, resistance to apoptosis, and progressive structural remodeling.

Unlike adults, pediatric PH is predominantly associated with developmental disorders. The major causes include persistent pulmonary hypertension of the newborn (PPHN), congenital heart disease (CHD)-associated PAH, developmental lung diseases such as bronchopulmonary dysplasia (BPD), congenital diaphragmatic hernia (CDH), childhood interstitial lung disease (chILD), and heritable PAH. Registry data demonstrate that Group 1 PAH and Group 3 PH account for most pediatric cases. Children commonly present with exertional dyspnea, fatigue, syncope, cyanosis, and progressive right heart failure, while infants may exhibit poor feeding, tachypnea, or failure to thrive, often leading to delayed diagnosis. In CHD-associated PAH, careful assessment of pulmonary vascular resistance remains critical for determining operability.

Developmental biology is central to pediatric disease. Increasing evidence indicates that abnormal pulmonary vascular and alveolar development begins prenatally, making PedPH fundamentally distinct from adult PH. Therefore, future therapies should aim not only to dilate pulmonary vessels but also to restore normal lung growth and vascular development. Genetic factors play an increasingly important role in pediatric PAH. Although BMPR2 remains the most common disease-causing gene, numerous additional pathogenic and candidate genes have been identified. Expanded genetic testing is particularly valuable in children with early-onset disease, syndromic features, or systemic comorbidities and may facilitate precision medicine and discovery of novel molecular pathways.

Current targeted therapies focus on the three major signaling pathways. Endothelin receptor antagonists reduce vascular remodeling, phosphodiesterase-5 inhibitors and

soluble guanylate cyclase stimulators enhance NO signaling, and prostacyclin analogues improve vasodilation while suppressing inflammation and platelet activation. Sotatercept, targeting the TGF- β superfamily, represents a novel therapeutic strategy that restores signaling imbalance rather than acting solely as a vasodilator.

Emerging evidence suggests that PAH can be categorized into therapeutic endotypes based on the predominant molecular pathway. Endothelin-dominant disease responds preferentially to endothelin receptor antagonists, NO-deficient disease to PDE5 inhibitors or riociguat, and advanced prostacyclin-deficient disease to parenteral prostacyclin therapy. However, because more than 80% of patients exhibit dysregulation of all three pathways, initial combination therapy is increasingly recommended for appropriate patients. In conclusion, pediatric pulmonary hypertension is a complex developmental pulmonary vascular disease with diverse etiologies and molecular mechanisms. Continued advances in developmental biology, genomics, molecular endotyping, and pathway-directed therapies are expected to improve diagnostic precision, individualized treatment, and long-term outcomes for children with pulmonary hypertension.

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講題摘要：

肺動脈高壓 (PAH) 是一種嚴重且影響兒童預後的血管疾病。雖然目前的標靶治療主要集中在降低肺血管阻力，但心腎軸的相互作用在疾病進展中同樣扮演關鍵角色。本綜述從腎臟視角出發，探討兒童肺動脈高壓的病理生理機制與治療策略。我們重點分析了標靶藥物對體液平衡、腎臟灌流以及腎素-血管收縮素-醛固酮系統 (RAAS) 的影響。此外，本文也討論了右心衰竭與腎功能不全之間的密切聯繫，並評估了利尿劑與 RAAS 抑制劑在維持兒童患者容量平衡中的臨床應用。深入理解這類「心-肺-腎」三者間的複雜互動，有助於優化精準臨床決策，進而改善兒童肺動脈高壓患者的長期預後。

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講題摘要：

核心免疫病理機轉（三大核心免疫失衡）先天性免疫細胞周圍縱火：巨噬細胞與肥大細胞受到內皮趨化因子（如 CCL2）召喚，大量浸潤於肺小動脈外膜層，持續釋放促發炎細胞激素（IL-1 β 、IL-6）與 TGF- β ，直接刺激血管壁不正常增生。補體系統的毀滅性活化：科羅拉多兒童醫院等前沿團隊研究證實，補體級聯（如 C5a、C5b-9 膜攻擊複合物）在肺血管異常沉積，誘發內皮細胞發炎性死亡，徹底撕裂第一道血管屏障。獲得性免疫防線崩潰（自體免疫自殘）：兒童調節性 T 細胞（Treg）功能缺陷，無法抑止 Th17 細胞，導致 IL-17 加劇血管壁纖維化。局部更會形成「三級淋巴結構」產生抗內皮細胞自體抗體（AECA），使小血管發生不可逆的進行性縮窄。臨床轉型：從血管擴張到免疫調控傳統標靶藥物（如 Sildenafil 威而鋼、Bosentan 波生坦）主要聚焦在傳統內皮素或一氧化氮的「血管擴張」途徑。然而，新世代臨床思維已著重於抗炎與逆轉血管重塑：早期免疫抑制治療：針對自體免疫疾病（如 SLE 紅斑性狼瘡、結締組織病）相關的兒童 PAH，早期合併免疫抑制劑的成效顯著。反轉重塑標靶新藥：臨床一線新星 Sotatercept（活化素訊號抑制劑），正是透過調控 TGF- β 免疫超家族訊息路徑，重新恢復血管壁細胞的凋亡與增殖平衡，成為對抗肺血管重塑的劃時代突破。

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講題摘要：

Role of pulmonary scintigraphy and functional imaging in the evaluation of pediatric pulmonary hypertension

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Pediatric pulmonary hypertension is a pathophysiological condition via vascular remodeling and increased pulmonary vascular resistance result in blood pressure in the lungs' arteries is higher than normal in children. This causes the right side of heart to work harder to push

blood through the lungs, consequences of right heart failure, which can lead to symptoms and heart-related complications over time. There are two major etiologies of pediatric pulmonary hypertension: primary pulmonary hypertension (idiopathic & heritable forms, genetic mutation) and secondary pulmonary hypertension (congenital heart disease, lung disease, hematologic disorders, chronic hypoxia and other factors). The different etiology between pediatric and adult pulmonary hypertension. Adult pulmonary hypertension is often linked to left heart disease, lung disease (COPD, ILD), thromboembolic disease, connective tissue disease and idiopathic cases.

The lungs are respiratory organ, like a pair of silent bellows in the chest, day and night playing an uninterrupted tune for life. Each inhalation is a letter delivered from earth and sky; each exhalation is the body's returned reply. However, we live in earth with gravity. West's zones illustrate how gravity affects blood flow in the lungs and its impact on respiration efficiency. They underscore the importance of matching ventilation and perfusion for optimal gas exchange. Collective changes in ventilation and perfusion in the lungs are measured clinically using the ratio of ventilation to perfusion (V/Q). Changes in the V/Q ratio can affect gas exchange and can contribute to hypoxemia. In tradition nuclear medicine, Tc99m MAA pulmonary perfusion and Tc99m DTPA aerosol ventilation scintigraphy provide the detailed functional V/Q mismatch and match dysfunction in pediatric pulmonary hypertension condition. Another important functional imaging as FDG PETCT is able to disclose the alternative metabolism of right heart failure and predict the outcome. However, the other traditional MPI SPECT is very helpful to show the heart function while pulmonary hypertension.

Although radiation safety is always an issue in pediatrics, we believe Nuclear Medicine functional imaging is the most important technology to evaluate sub nanometer fields of medicine: As light traverses spacetime by powers of ten, it records the interactions of living molecules. Through optoelectronics, the microscopic is magnified into macroscopic—this the true essence of nuclear medicine.

Key Words: pediatric pulmonary hypertension, nuclear medicine, Tc99m MAA pulmonary perfusion scintigraphy. Tc99m DTPA aerosol ventilation scintigraphy. FDG PETCT. MPI.

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講題摘要：

核心營養挑戰高代謝需求：呼吸困難與右心室高度負荷，使病童燃燒比常人更多的熱量。餵食困難與疲勞：嬰幼兒常因吮吸、吞嚥時呼吸不順而極易疲勞，導致餵食中斷及進食量不足。腸胃道耐受力差：心臟功能不全可能引發腸道淤血，導致消化吸收不良、噁心或腹脹。微量營養素缺乏：病童普遍缺乏維生素 D、維生素 C 及鐵質，這會進一步削弱呼吸肌力與血管健康。

支持與管理策略高密度熱量補充：提供高熱量密度、高蛋白的配方奶或食物（如利用油脂或強化劑），以在有限的進食量中極大化營養攝取。嚴格限制鈉與液體：採取低鈉飲食並精準監控水分。避免多餘

液體加重肺部淤血與右心負擔，導致呼吸更加困難。少量多餐與管灌輔助：透過少量多餐減少胃部壓力；對於嚴重疲勞或吸吮困難的嬰兒，應考慮及時使用鼻胃管灌食以確保基本熱量達標。精準微量營養素支持：定期監測並適時補充鐵質、維生素 D 及維生素 C，以支持肌肉強度、免疫力及血管完整性。體重與水腫監測：每日定時測量體重。短期內體重驟增通常是心衰竭引起水腫的惡化警訊，而非真正的肌肉生長。

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講題摘要：

兒科肺高血壓 (Pediatric Pulmonary Hypertension, PH) 的臨床照護已從「單純疾病管理」走向「以家庭為中心」的跨領域心理社會全面護理。由於兒科 PH 具有高度不可預測性、需長期依賴複雜藥物 (如連續靜脈注射前列環素) 且預後極具挑戰，病童及其照顧者正面臨嚴重的多重心理社會壓力。本文摘要其核心心理社會衝擊、以家庭為中心的護理實務及全人照護策略。

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講題摘要：

睡眠呼吸障礙 (Sleep-Disordered Breathing, SDB) 與夜間低氧血症 (Nocturnal Hypoxemia) 現已被證實是驅動兒童肺動脈高壓 (Pediatric PAH) 惡化的關鍵隱性誘因 [1, 2]。由於兒童在睡眠期間的生理反應具有特異性，夜間長期的反覆缺氧與氣道壓力變化，會在神經體液與血管結構上對肺循環造成持續性打擊 [1, 2]。本文摘要其核心病理機轉、臨床評估診斷及整合性治療調控策略。