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Lung Health after Preterm Birth: Knowledge Gaps and Respiratory Care Across the Lifespan

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Impact Statement: This expert consensus statement from the BPD Collaborative outlines proposed standards for the outpatient care of individuals with prematurity-associated lung disease (PLD) across the lifespan. The goal of this statement is to stimulate the field to develop stronger evidence to care for patients at risk for late and underrecognized morbidities, prioritize the gaps in knowledge, and encourage the development of strategies to improve long-term PLD outcomes through greater awareness and continuity of care among pediatric and adult providers.

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ABSTRACT

Prematurity affects approximately 10% of live births worldwide and contributes to substantial short and long-term morbidity, particularly respiratory disease. Approximately 35% of infants born before 32 weeks gestation are diagnosed with bronchopulmonary dysplasia (BPD). Many preterm infants regardless of BPD diagnosis also develop respiratory problems in both childhood and adulthood, more recently termed as prematurity-associated lung disease (PLD). As survival of preterm infants continues to improve, the lifetime burden of PLD and related health conditions remains high. Optimizing lung growth, minimizing the impact of comorbidities, and improving quality of life are ongoing challenges. Individuals born preterm are also at higher risk for being diagnosed with early-onset chronic obstructive pulmonary disease in adulthood. Despite these public health implications, outpatient management is often variable, and PLD remains underrecognized and heterogeneous across clinical settings. In this expert consensus statement from the BPD Collaborative, we summarize key knowledge gaps and provide pragmatic recommendations to help standardize outpatient care for PLD across the lifespan. Recommendations address models of care delivery, approaches to disease monitoring and management, and the evaluation and treatment of common comorbidities. Given limited high-quality evidence in several domains, many recommendations are necessarily based on expert opinion. Our objectives are to highlight priorities for future research, promote continuity between pediatric and adult care, and support development of evidence-based strategies to reduce underdiagnosis and undertreatment and to improve long-term outcomes for individuals born preterm.

INTRODUCTION

Prematurity and its Consequences: Prematurity, defined as birth before 37 weeks-gestation,(1) accounts for approximately 10% of live births, with more than 13 million preterm infants born globally in 2020.(2) Despite advances in obstetric care and fetal surveillance, the prevalence of prematurity has remained largely unchanged over the past decade. Compared with term-born peers, children born preterm experience substantially higher morbidity and mortality, and prematurity remains a leading cause of death in children under 5 years of age worldwide.(3, 4) Lower gestational age at birth strongly predicts mortality and short-term complications, including prolonged respiratory support, retinopathy of prematurity, intraventricular hemorrhage, necrotizing enterocolitis, and other neonatal morbidities.(5, 6) Importantly, the consequences of preterm birth often extend beyond NICU discharge and persist across the life course, with increased risks of respiratory and cardiovascular disease, adverse neurodevelopmental outcomes, and higher all-cause mortality.(7-11) These long-term risks underscore the need for longitudinal follow-up, especially in those children with prematurity-associated lung disease (PLD). To optimize respiratory health outcomes and quality of life, health-care providers need to implement preventive care strategies to identify emerging problems early and support timely intervention. Education for individuals born preterm and their families should emphasize seeking evaluation for respiratory symptoms irrespective of gestational age at birth.(12, 13)

Prematurity-associated Lung Disease (PLD): PLD represents the most common morbidity of preterm birth, with bronchopulmonary dysplasia (BPD) as its archetype.(14) Globally, 21–35% of infants born before 32 weeks' gestation are diagnosed with BPD, with rates varying by gestational age, birth weight, and region.(15, 16) As survival of extremely preterm infants has improved, BPD remains a major cause of ongoing respiratory morbidity, and is associated with increased risk of chronic respiratory symptoms that may persist into adulthood.(17, 18) However, commonly used BPD definitions that are largely

based on the need for respiratory support at 36 weeks postmenstrual age may not reliably predict lifelong respiratory health.(19-21) Importantly, preterm birth itself has been associated with obstructive lung disease in later life,(11, 22, 23) and persistent respiratory morbidity has been observed in preterm children and adults both with and without a prior BPD diagnosis.(24) Accordingly, we define PLD as chronic or recurrent respiratory symptoms, physiologic impairment, and/or structural lung disease attributable, at least in part, to preterm birth, irrespective of BPD status. PLD may be conceptualized as altered lung-function trajectories beginning in early life, with heterogeneous patterns including catch-up growth, persistent impairment, or accelerated decline.

Historically, research has emphasized BPD prevention and treatment, with comparatively less attention to other respiratory manifestations of prematurity (e.g., airway, voice, and chest wall abnormalities). Improved risk stratification and precise phenotyping could enable personalized management, support phenotype-informed clinical trials, and promote earlier intervention for individuals at greatest risk of adverse long-term outcomes.(25) PLD likely reflects early impaired lung growth and injury with variable regenerative capacity, further shaped by cumulative environmental exposures (e.g., infections, nutrition, growth) and genetic influences.(26, 27) Emerging data also suggest distinct immune endophenotypes among extremely preterm children (impaired vaccine responses and altered innate immune responses to lower respiratory tract illnesses),(28, 29) underscoring the need for targeted, phenotype-specific approaches to optimize lifelong respiratory health.

Prenatal and Postnatal factors and Prematurity-associated Lung Disease: Children born prematurely have underdeveloped lungs, with fewer alveoli and distal pulmonary vessels compared to term infants.(30) Although earlier gestational ages correlate with a higher likelihood of chronic lung disease, including BPD, other factors during pregnancy can influence lung outcomes. Placental insufficiency and antenatal stress, such as preeclampsia, premature rupture of membranes, maternal smoking, and

chorioamnionitis can contribute to poor fetal growth, lung hypoplasia and pulmonary hypertension, postnatal small airway dysfunction, and metabolic dysregulation (**Table 1**).⁽³¹⁻³⁹⁾ Close follow-up of such high risk pregnancies and education regarding modifiable factors, such as maternal smoking can help to mitigate some of these risk factors in the preterm child.

Postnatally, other early-life exposures can adversely affect lung health in preterm infants. These can include ventilator-related barotrauma, hyperoxia, pulmonary over-circulation, necrotizing enterocolitis, sepsis, corticosteroids, maternal smoking, and inadequate nutrition.⁽⁴⁰⁻⁴³⁾ Additional factors include lower respiratory tract infections (viral and bacterial), aspiration, and microbiome dysbiosis (i.e., antibiotic exposure). These postnatal insults can contribute to alveolar simplification, trachea-/bronchomalacia, subglottic stenosis, small airway disease, chronic respiratory failure, and pulmonary hypertension. Furthermore, these early-life exposures can have long-term impacts on cardiorespiratory health across the lifespan, including potentially increasing the risk for COPD in later life.⁽⁴⁴⁻⁴⁶⁾

Social and Economic Aspects of Prematurity: Direct and indirect economic costs for children born prematurely are substantial. This underscores the importance of minimizing morbidities associated with prematurity that often drives up these costs. In 2016, the estimated direct cost excess (discounted present value) for the lifetime care of children born prematurely in the US was estimated to be \$25.2 billion.⁽⁴⁷⁾ Predicted direct costs are especially high in the extremely preterm children during the first five years of life.⁽⁴⁸⁾ Both before and after initial hospital discharge, the indirect costs of ongoing care of preterm children can lead to financial hardships for caregivers and families. Additionally, social isolation due to child health concerns can lead to lower quality of life in primary caregivers.^(49, 50)

Developmental and behavioral problems are common in children born preterm,⁽⁵¹⁾ with additional impacts on caregiver quality of life.^(49, 52) After initial hospital discharge, supportive care for children and their families is needed to optimize health and developmental outcomes.

Social work screening can identify familial stress, transportation, housing insecurity, utility bill issues, food insecurity, limited parental leave, and other economic hardships that impede the welfare of the child and their family.(53, 54) Since, preterm births are more prevalent among different racial and underserved populations there is a need to address social inequities to improve health outcomes. In general, patient, parent and family important outcomes are understudied. Studies of family wellbeing after the NICU highlight that respiratory themes related to NICU admissions and/or long-term respiratory health were more often impacting the child (e.g., exercise limitations) or the family (e.g., readmissions) than BPD severity.(55, 56) In addition, diverse medical, psychological, and social issues can persist that are often under-recognized by health care professionals throughout life, leading to lost opportunities to deliver optimal care and support.

Consequences of Prematurity on Adult Lung Health: Respiratory morbidity associated with preterm birth may persist well beyond childhood, with survivors of bronchopulmonary dysplasia (BPD) representing a particularly vulnerable population. In a systematic review of studies examining lung health among adult survivors of BPD, Gough et al. reported a higher prevalence of dyspnea, wheeze, and cough compared with term-born control populations. Across studies, adults with a history of BPD also demonstrated evidence of persistent airflow limitation, including reduced FEV₁ and forced expiratory flow between 25% and 75% of vital capacity (FEF₂₅₋₇₅), as well as structural and/or parenchymal abnormalities on chest imaging. (57) Findings from the New Zealand 1986 Very Low Birth Weight (VLBW) cohort similarly demonstrated lower FEV₁, FEV₁/FVC, and FEF₂₅₋₇₅, among VLBW adults compared to term-born controls; with these impairments greatest among adults with a history of BPD. Reduced single-breath diffusing capacity for carbon monoxide (DLCO) was also observed, suggesting persistent abnormalities in gas transfer.(58) In a separate cohort of adults born preterm during the post-surfactant era, airflow obstruction, respiratory symptoms, and radiographic abnormalities were more common

than in term-born controls. Childhood respiratory hospitalization was a risk for lower adult lung function, with the strongest associations observed among survivors of BPD.(59) Collectively, these findings indicate that BPD is associated with an increased risk of persistent respiratory symptoms, airflow limitation, impaired gas exchange, and structural lung abnormalities into adult life. Longitudinal follow-up of this growing population is therefore important to define trajectories of lung function decline and identify opportunities for preventive and therapeutic intervention (**Figure 1**).

Our Goals: The BPD Collaborative recognizes that manifestations of PLD extend well beyond early childhood and that there is a paucity of guidance currently available to healthcare professionals, families, and patients regarding best practices. As such, we seek to outline recommendations on 10 respiratory topics related to Models of Delivery (Disease Risk Recognition, Medical Home and Preventative Care, and Subspecialty Consultation and Follow Up), Management (Lung Function Testing, Lung Imaging, and Therapies), and Co-Morbidities (Aerodigestive Disease, Cardiovascular Disease, Nutrition/Growth, and Other). Items that may be more difficulty to implement due to access or practice context are labeled as recommended or to be considered.

Methodology: A multidisciplinary expert panel was convened to develop consensus-based recommendations for the outpatient care of infants and children with prematurity-associated lung disease. Panelists were selected based on clinical expertise and/or research experience in neonatology, pediatric pulmonology, and other disciplines relevant to the longitudinal management of children with PLD as well as patient advocates. A literature review was conducted to identify available background evidence and inform an initial set of clinical topic areas as listed above and draft key gaps in knowledge and recommendations addressing key domains of outpatient BPD care. Consensus was established using review of aggregated panel feedback and comments. Additionally, 31 members of the outpatient committee of the BPD Collaborative reviewed the recommendations to provide feedback. The entire

process was intended to integrate available evidence with multidisciplinary clinical expertise, patient and family considerations, and the feasibility of implementing recommendations across outpatient care settings. AI was used to generate Figures 1 and 4, but no other figures or text.

Limitations: These recommendations are largely based on expert consensus since published data are limited regarding whether the recommended testing and follow-up alter long-term outcomes. Existing data often do not address many domains of health, related to prematurity. Implementation may be challenging owing to both available resources and costs to both the healthcare system and patients and their families. These recommendations for testing and follow-up attempt to strike a balance between early detection of disease, timely titration of therapies, thoughtful assessment of complications versus increased costs and resource utilization, burden on families and providers, and oversurveillance. Not all testing and follow-up may be available in resource-limited settings and for patients and families where some care costs may not be covered by national healthcare or insurers; therefore, advocacy for policy and system-level changes is essential to improve the health of the millions of individuals born preterm.

CARE DELIVERY: Disease Risk Management

Background and Evidence:

Establishing optimal long-term care for individuals with PLD requires early identification of those at risk for later cardiopulmonary morbidity based on perinatal history. Common definitions of BPD, the archetype of PLD, rely on respiratory support at a single time point (36 weeks postmenstrual age) and do not capture heterogeneous disease phenotypes.(19-21) In retrospective single-center data (n = 83), Wu and colleagues described three contributing phenotype domains, parenchymal lung disease, pulmonary vascular disease, and airway disease, which in combinations were associated with severe BPD; however, these categories remain broad, inconsistently operationalized, insufficiently studied

beyond the NICU, and may omit key modulators (e.g., large airway malacia, chest wall mechanics).(60) Accordingly, scalable phenotypic definitions applicable across the lifespan are needed, integrating clinical features, imaging, family priorities and spirometry.(24, 55) Notably, PLD is associated with impaired pulmonary function in preterm-born individuals even without a formal BPD diagnosis. (61) Although ICD-10/ICD-11 include codes for BPD and related perinatal chronic lung diseases, respiratory symptoms in preterm-born individuals are often coded as asthma or obstructive lung disease with increasing age, irrespective of etiology, contributing to variable treatment responses.(62) Spirometry-based phenotyping can identify several distinct patterns, including bronchodilator-responsive and nonresponsive obstruction, prematurity-associated preserved ratio impaired spirometry (pPRISm) and dysanapsis and direct therapy (**Figure 2**).(63) For example, bronchodilator non-responsiveness may reflect fixed small-airway narrowing despite alveolar catch-up growth (**Figure 3**). Maintaining PLD documentation may prompt consideration of prematurity-related mechanisms when symptoms arise. Future revisions to the International Classification of Diseases should support phenotype-specific coding for prematurity-associated lung disease, while in the interim, longitudinal assessment may be strengthened by tracking functionally defined outcomes (e.g., wheeze, feeding difficulties) and using validated, family-centered outcome instruments.(49, 64-66) Despite increasing recognition of PLD, its natural history and modifiable drivers remain incompletely defined.

Gaps in Knowledge:

- Are there long-term phenotypes of PLD that follow predictable trajectories? Are phenotypic transitions common?
- Are there elements of disease that track together (e.g., vascular, airway, immune endotypes etc.)?
- How do genetics and family history influence long-term trajectories?

- What are the modifiable factors that can improve lung function trajectories over time (including both prenatal and postnatal exposures, selective therapeutic interventions, and caregiver involved interventions, etc.)?
- What other outcomes should be followed long-term in PLD (including ones important to patients and their families) such as disability, limitation of activities, persistent symptoms, severe acute respiratory exacerbations/illnesses, health-related quality of life, systemic issues of cardiovascular, renal and metabolic functions?

Recommendations:

- All providers should document prematurity/PLD lifelong: Record history of prematurity and PLD (with/without BPD) using ICD-10 (BPD: P27.1; PLD without BPD: P27.8)/ICD-11 (BPD: KB29.0; PLD without BPD: KB29.Y) codes and carry it forward across the lifespan.
- Educate families: PLD clinics are recommended to counsel patients/caregivers on potential long-term respiratory risks after prematurity/PLD.
- Research: Define PLD endophenotypes: Convene consensus panels to standardize PLD endophenotypes in children and adults.
- Research: Validate and operationalize endophenotypes: Confirm endophenotypes in diverse, prospective, longitudinal multicenter cohorts (ideally birth-to-adulthood), then use them to update ICD coding, identify high-risk trajectories (including pre-COPD/COPD per GOLD), and enable targeted therapies.
- Research: Track modifiers and exacerbations: Systematically document infectious/noninfectious exacerbations, environmental exposures (e.g., smoke/vape, air pollution), and early-life acute care use to identify modifiable risk factors and therapeutic targets.

- Research: Assess extra-pulmonary phenotypes: Study and monitor non-lung comorbidities (e.g., cardiac, upper airway) across the lifespan.

CARE DELIVERY: Medical Home and Preventative Care

Background and Evidence: Prior expert consensus has advocated for a “medical home” model for individuals with bronchopulmonary dysplasia (BPD).(67) Although evidence that this model improves outcomes is limited, a medical home can support accessible, family-centered preventive care, co-management of active cardiorespiratory disease, and early identification of individuals at elevated risk for comorbidities associated with preterm birth. Because most postnatal lung growth occurs in the first two years of life, with continued alveolar development through adolescence, early childhood represents a potential window to promote favorable lung-growth trajectories.(68, 69) (70) Infant pulmonary function testing in preterm-born infants, even without BPD, demonstrates early physiologic patterns consistent with airway and parenchymal disease, underscoring the importance of early risk stratification.(70) However, intervention studies targeting long-term trajectories are scarce. Extended use of continuous positive airway pressure (CPAP) in the NICU has been associated with higher lung volumes at 6 months, but long-term clinical significance is unknown.(71) Care models that increase caregiver participation (e.g., home oxygen weaning) may be beneficial and warrant broader implementation and evaluation.(72)

Recurrent respiratory insults during the preschool years may further impair lung development. In term-born cohorts, childhood pneumonia is associated with reduced adult lung function and chronic obstructive pulmonary disease (COPD).(73-75) In preterm-born children, lower respiratory tract infections (LRTIs) are a leading cause of rehospitalization and may worsen underlying lung and airway disease or limit alveolar catch-up growth; alternatively, higher infection rates may reflect greater baseline disease susceptibility.(76) Regardless of directionality, reducing the frequency and severity of

LRTIs is a plausible strategy to support lung growth and reduce later respiratory morbidity.

Immunoprophylaxis and vaccination against respiratory syncytial virus (RSV), influenza, and pneumococcal disease can prevent illness or attenuate severity.(77) RSV infection is associated with increased hospitalization, intensive care use, respiratory support, and mortality in severe BPD, and RSV prophylaxis can mitigate these risks.(78) Other viruses (e.g., rhinovirus, human metapneumovirus, adenovirus, parainfluenza) also contribute substantially to morbidity.(79-82) Trials of trained-immunity prophylaxis are ongoing.(83)

Environmental and social factors further modify risk. Daycare exposure, household crowding, and indoor/outdoor air pollution are associated with increased respiratory symptoms and acute care use in preterm-born preschool children.(84-87) Policies such as paid family leave may improve outcomes, but optimal duration is uncertain.(70) Avoiding secondhand smoke and traffic-related air pollution and ensuring vaccine access across the lifespan may reduce short- and long-term morbidity. Social determinants of health are associated with acute care utilization among individuals with BPD, supporting routine screening and timely referral to appropriate resources; however, insurance coverage may limit access to recommended therapies and diagnostic testing.(88) (86, 89, 90) Although PLD may improve in infancy or early childhood, clinically quiescent disease can re-emerge in adolescence or early adulthood, and preterm birth is associated with adverse adult lung-function trajectories.(23) Coordinated lifespan care, including structured transition to adult providers, is needed; however, few centers currently provide PLD-specific follow-up into school age or adulthood.(91)

Gaps in Knowledge:

- Do PLD-focused clinics improve respiratory outcomes versus usual primary care or non-PLD pediatric pulmonary care?
- Does shifting from BPD-centric metrics to functional outcomes (e.g., spirometry) enable earlier risk stratification and prevention?

- What is the impact of parental leave on infection rates, rehospitalization, and long-term respiratory outcomes in PLD?
- Which immunoprophylaxis strategies (including additional vaccines) most improve outcomes across childhood and adulthood?
- What are the best models for pediatric-to-adult transition planning and shared accountability across providers?
- What are the most effective ways to disseminate PLD education to pediatric and adult clinicians (primary care and subspecialists)?

Recommendations:

- Education: PLD clinics should consider providing targeted education for pediatric and adult clinicians on expected respiratory trajectories and monitoring needs after prematurity/PLD. PLD specialists should also be prepared to counsel families on long-term PLD trajectories throughout the lifespan.
- Coordinated care: Primary care providers are recommended to partner with PLD clinics (neonatology- or pulmonology-based) to coordinate longitudinal management.
- Clinic handoffs: Neonatology-based programs are recommended to ensure timely referral to pediatric pulmonology PLD clinics when patients age out.
- Transition planning: Pediatric PLD clinics are recommended to support care into young adulthood when appropriate and feasible and implement structured transition pathways with adult pulmonology.
- Immunoprophylaxis: All providers should promote and facilitate immunoprophylaxis for key respiratory pathogens (e.g., RSV, influenza, pneumococcus) per current guidance for all preterm individuals.

- Prevention counseling: Providers are recommended to counsel families on modifiable exposures (secondhand smoke/vape, daycare infection risk, indoor/outdoor air pollution) for all preterm individuals.
- Activity/procedures: PLD clinics are recommended to provide anticipatory guidance on exercise tolerance and peri-procedural respiratory risk (sedation/anesthesia).

CARE DELIVERY: Subspecialty Consultation and Follow-Up

Background and Evidence: Pulmonary consultation facilitates disease phenotyping of parenchymal, cardiovascular, and airway disease through expertise in lung imaging, lung function testing, and coordinated care in aerodigestive and PH clinics. The care of children requiring chronic mechanical ventilation via tracheostomy or non-invasive positive pressure ventilation is an area of additional medical complexity. More advanced strategies for managing respiratory disease are frequently available only in subspecialty clinics. Appropriate and timely subspecialty follow-up is thus necessary for optimal management of PLD and early recognition of potential complications (**Table 2**). It is recognized that local or regional limitations may not permit evaluation as outlined below; however, delayed evaluation at a later age remains valuable. We would encourage the development of interprofessional clinics to better promote holistic care and support efficiencies to reduce appointment burden. We acknowledge that the follow-up timepoints discuss in this section are arbitrary, but represent an attempt to recognize complications of disease early, titrate or wean therapies in a timely manner, and provide up-to-date preventative guidance.

Gaps in Knowledge:

- Who should receive pulmonary consultations, either in the inpatient setting or for chronic preventative care?

- How should individuals with tracheostomies and home ventilators be followed longitudinally, including best practices for titration and weaning?
- How often should routine assessments occur in largely asymptomatic individuals to detect subtle adverse changes in health or early markers of progressive disease?

Recommendations:

- Inpatient: Before initial discharge, it is recommended to obtain pediatric pulmonary consultation for preterm infants expected to have active PH at discharge, tracheostomy, invasive/noninvasive positive-pressure ventilation, or chronic home systemic corticosteroids. Provide care via an interdisciplinary, standardized pathway with structured family communication on discharge planning, care coordination, system navigation, and transition to outpatient care.
- Outpatient: Within 2 months of discharge, it is recommended to be evaluated by a pediatric pulmonologist (or PLD-experienced clinician), prioritizing infants on any respiratory support (including oxygen), with active lung disease-related PH, or receiving chronic daily respiratory medications (e.g., diuretics, bronchodilators, inhaled/systemic corticosteroids, PH therapies). Prefer a dedicated PLD clinic; assess earlier if no inpatient consult. Consider higher oxygen use at high altitude when setting referral thresholds.
- Long-term: It is recommended to continue pulmonology follow-up while home respiratory support persists, respiratory admissions occurred within 12 months, PH remains active, pulmonary medications are needed (chronic/intermittent), or spirometry/imaging is abnormal.
- Re-evaluation: Regardless of symptoms or therapies, repeat subspecialty assessment at 6, 12, and 18 years should be considered, including spirometry when feasible, prioritizing those discharged on respiratory support or with recurrent admissions; use validated instruments for broader respiratory health.

- Adult care: It is recommended to transition to adult pulmonology for ongoing support, recent admissions, active PH, respiratory medications, abnormal testing, or persistent childhood-onset respiratory morbidity.

EVALUATION AND TREATMENT: Lung Function Testing

Background and Evidence: Preterm-born children often have normal pulmonary function; however when abnormalities occur, airflow obstruction predominates, particularly among those with BPD and/or low birth weight.(92) Because some children underrecognize or underreport symptoms, objective assessment (e.g., spirometry) is important to identify small-airway disease.(93) However, cognitive, motor, or behavioral limitations may preclude reliable testing in a subset, necessitating careful clinical assessment. Exercise intolerance is a common marker and may reflect reduced cardiorespiratory fitness.(94) Spirometry identifies distinct prematurity-associated phenotypes.(95) Dysanapsis reflects disproportionate airway caliber relative to lung size and is typically defined by preserved FVC and FEV₁ (both $\geq$ LLN) with reduced FEV₁/FVC ($<$ LLN).(96) The development of dysanapsis is likely multifactorial, and may arise from lung growth arrested by prematurity,(97) intrauterine inflammation,(98) and/or early accelerated somatic growth.(99) Individuals with dysanapsis may be at increased risk for prematurity-associated COPD in adulthood, independent of smoking.(95) Additional phenotypes include prematurity-associated obstructive lung disease (POLD: FEV₁ $<$ LLN with FEV₁/FVC $<$ LLN), which may be bronchodilator responsive or nonresponsive, and prematurity-associated preserved ratio impaired spirometry (pPRISM: FEV₁ $<$ LLN with FEV₁/FVC $\geq$ LLN) which is associated with increased all-cause mortality and rapid decline in lung function.(100, 101) Given prognostic implications of these patterns, serial lung-function testing should be prioritized and interpreted longitudinally to guide timely modification of risk factors and optimize respiratory trajectories (**Table 3**). We acknowledge that the spirometry timepoints discuss in this section are arbitrary, but represent a

balance between detecting disease and oversurveillance. We also note that at this time there is no available evidence that early detection of lung disease will alter long-term trajectories.

Gaps in Knowledge:

- What are the optimal timing and frequency of serial spirometry to define PLD phenotypes, detect early decline, and guide interventions that improve long-term lung function trajectories?
- What are the diagnostic performance and clinical utility of impulse oscillometry for early-childhood assessment (before reliable spirometry) and for patients unable to perform spirometry?
- What are indications for adding non-spirometric testing (e.g., plethysmography, DLCO, FeNO) and/or lung imaging to detect occult disease and track longitudinal change?
- What is the role of exercise-based assessments in characterizing exercise intolerance and informing management?
- What are the validity and usefulness of respiratory symptom/functional questionnaires (e.g., Liverpool Respiratory Symptom Questionnaire) in children born preterm?(65)

Recommendations:

- Spirometry: It is recommended based on available resources that baseline spirometry be performed using race-neutral Global Lung Function Initiative reference equations at ages 6, 12, and 18 years for individuals born <34 weeks' gestation regardless of PLD diagnosis, prioritizing those discharged on home respiratory support and/or with recurrent respiratory readmissions. Repeat spirometry every 1–2 years for patients with abnormal results, and sooner with clinical deterioration or management changes. It is recommended to obtain spirometry at transition to adult care if transition occurs after age 18.
- Additional testing: Consider plethysmography with DLCO at ages 12 and 18 years in patients with abnormal spirometry suggestive of restriction and/or active pulmonary hypertension, with

interval tailored to clinical need. Consider a 6-minute walk test at 12 and 18 years, including for patients unable to perform spirometry. Consider cardiopulmonary exercise testing and high-altitude simulation testing on an individual basis. Further testing based on these results is at the discretion of the treating provider (e.g., assessing pulmonary artery pressures with abnormal DLCO testing).(102)

EVALUATION AND TREATMENT: Lung Imaging

Background and Evidence: Given increasing recognition of phenotypic heterogeneity in PLD, imaging is likely to play an expanding role in longitudinal phenotyping with age. Chest imaging can identify individuals with alveolar simplification, hyperinflation, cystic change, regionally heterogeneous atelectasis, and enlarged central pulmonary arteries; features associated with higher risk of cardiopulmonary complications and age-related declines in lung function. While chest radiography and computed tomography remain important for evaluating acute presentations or severe structural abnormalities, emerging modalities may better define disease patterns and trajectories with lower risk. Magnetic resonance imaging can delineate regional heterogeneity in parenchymal and airway disease without ionizing radiation.(103) Lung ultrasound is a portable, radiation-free option for repeated bedside assessment of aeration, interstitial findings, and regional abnormalities.(104) However, optimal imaging frequency remains uncertain; prospective studies are needed to establish standardized protocols and link imaging phenotypes to longitudinal outcomes and treatment response.

Gaps in Knowledge:

- There is an urgent need to develop standardized imaging to minimize exposure to ionizing radiation.
- Further data are required to determine optimal type and timing of follow-up imaging.

- Research is needed to determine the utility of newer MRI modalities, electrical impedance tomography, and lung ultrasound as radiation-free testing for longitudinally following lung growth and disease.

Recommendations:

- Routine (“follow-up”) imaging during childhood is not recommended and should be performed only when clinically indicated. A chest radiograph may be considered at transition to adult pulmonary care for individuals with PLD if recent imaging is unavailable.
- Chest radiography remains first-line recommended imaging for acute respiratory status changes, but is less sensitive than computed tomography (CT) for detecting parenchymal and airway abnormalities in patients with persistent symptoms or for longitudinal assessment of PLD.
- Chest CT should be considered when clinical concerns cannot be adequately evaluated by radiography, including persistent supplemental oxygen requirement beyond 2 years of age (after excluding sleep-disordered breathing), recurrent lower respiratory infections, or severe spirometric impairment/decline. Non-contrast CT is typically preferred; intravenous contrast may be added when vascular evaluation is required.

EVALUATION AND TREATMENT: Therapies

Background and Evidence: Few clinical trials in PLD have evaluated treatment efficacy for symptom control and respiratory outcomes, with most evidence derived from single-center retrospective studies. Inhaled corticosteroids (ICS), alone or combined with long-acting beta-agonists (ICS/LABA), are frequently used in outpatient management of bronchopulmonary dysplasia (BPD) to reduce airway resistance and work of breathing during chronic or acute symptoms, but responses are variable and likely phenotype dependent.^(105, 106) Heterogeneity may reflect distinct mechanisms (e.g., type 2 inflammation, neutrophilic inflammation, dysanapsis, fixed airflow obstruction, or combinations

thereof). Translating frameworks from asthma and chronic obstructive pulmonary disease (COPD) may facilitate PLD endotyping and enable phenotype-directed therapies, including use of biologics.

Immunomodulatory strategies are particularly promising for selected endophenotypes. Term and preterm infants have immature adaptive immunity, requiring repeated early-life immunizations secondary to lower vaccine responses (e.g., to the pneumococcal conjugate vaccine).(107, 108) Waning pneumococcal antibody titers and inadequate immune responses have been associated with recurrent or chronic respiratory symptoms.(109) However, prolonged or severe infections are not uniformly attributable to immune dysfunction. Impaired mucociliary clearance or chronic aspiration can produce similar presentations and may limit response to standard outpatient antibiotic courses for pathogen-related lower respiratory tract infection.

Prematurity and prenatal/postnatal exposures may also alter gene-expression programs governing lung development and repair. Adverse stimuli can increase expression of inflammatory genes (e.g., IL6, IL8). Higher pro-inflammatory expression and lower expression of growth/angiogenesis genes (e.g., VEGF) has correlated with BPD risk.(110, 111) Genetic susceptibility may contribute to respiratory morbidity. Higher COPD polygenic risk scores have been associated with reduced lung function in children born preterm,(112) and single-nucleotide polymorphisms linked to bronchopulmonary dysplasia have been associated with asthma, COPD, and lower FEV₁/FVC.(113) Familial asthma history and early-life asthma gene signatures have further been linked to wheeze and BPD, supporting shared pathways and potentially overlapping therapeutic targets.(114, 115)

Gaps in Knowledge:

- What is the efficacy of current symptom-based therapies across specific PLD/BPD endophenotypes (e.g., short- and long-acting beta-agonists, inhaled corticosteroids (ICS), ICS/LABA, anticholinergics/long-acting muscarinic antagonists)?

- When should genetic testing and detailed laboratory/immune phenotyping (endotyping) be applied to optimize lung function trajectories and enable targeted/personalized therapy?
- What role, if any, do biologics have in PLD management?
- Do procedural interventions (e.g., bronchial thermoplasty, lung volume reduction) have a role in select patients?
- How do gut / lung dysbiosis influence immune responses and chronic inflammation in PLD?

Recommendations:

- PLD clinics are recommended to support school/daycare respiratory care plans and advocate for adequate insurance coverage of commonly used PLD/BPD medications.
- Consider therapeutic trials of bronchodilators and ICS (alone or in combination), particularly in children with obstructive phenotypes by symptoms and/or spirometry.
- Consider immune evaluation (CBC with differential, serum immunoglobulins, vaccine titers) in any preterm child with recurrent bacterial infections or severe viral illnesses; refer to Immunology if abnormal or clinically indicated. Consider pneumococcal booster vaccination for ages 2–18 years with documented low titers.
- Consider bronchoalveolar lavage for lower-airway microbial testing for persistent symptoms refractory to standard therapy, especially with acute or prolonged lower respiratory tract illness.
- Consider genetic consultation/testing when PLD severity exceeds that predicted by gestational age and recognized risk factors.

COMORBIDITIES: Cardiovascular Disease

Background and Evidence: Pulmonary hypertension (PH) occurs in up to 20-40% of infants with

BPD.(116) Disrupted alveolar growth and impaired angiogenesis due to preterm birth can contribute to

PH development. Several risk factors are associated with BPD-PH in preterm children including a history of intrauterine growth restriction (IUGR), the presence of severe BPD, and intracardiac shunts (patent ductus arteriosus (PDA), particularly PDAs that are open for prolonged periods of time,(117) ventricular and atrial septal defects, and other congenital cardiac conditions). With improved lung growth over time, PH due to preterm birth tends to resolve,(118) but may recur during periods of rapid growth (e.g., adolescence) or physiologic stress such as acute respiratory illness, supporting selective screening and longitudinal follow-up (**Table 3**). Prematurity with severe BPD is also a major risk factor for pulmonary vein stenosis (PVS) with an incidence of 1.2%.(119) PVS and PH (PVS-PH) in infants with BPD is associated with higher morbidity and mortality than PH alone.(120)

Gaps in Knowledge:

- Improved early-life risk stratification is needed to identify preterm children at highest risk for incident PH, persistent PH, or late recurrence across childhood and adulthood.
- Long-term, comparative outcome studies of therapies for BPD-associated PH and PVS-associated PH are needed, including morbidity, mortality, and cardiopulmonary function.
- Additional data are required on the long-term impact of potentially shunt-producing lesions (e.g., PDA, PFO, ASD, VSD) and on optimal timing/selection of interventions.

Recommendations:

- Inpatient consultation: Preterm infants expected to be discharged on PH-directed therapy are recommended to receive evaluation by a PH specialist clinician comfortable with PH features and management, and resourced to escalate care as needed, before initial discharge.
- Outpatient Consultation: Infants discharged on PH medications are recommended to be established with a PH specialty team prior to discharge, with planned follow-up ideally within 2 months.

- Surveillance: For PH persisting beyond 36 weeks postmenstrual age, we recommend serial echocardiography until resolution and discontinuation of PH therapy.
- Targeted screening: We recommend obtaining an ECHO in older patients with prior PH or PLD with unexplained hypoxemia, dyspnea, or exertional limitation. Refer to PH/cardiology if abnormalities found.
- Transition of care: Arrange adult subspecialty follow-up for individuals with active PH or elevated cardiovascular risk at transition.

COMORBIDITIES: Aerodigestive Disease

Background and Evidence: Commonly acquired upper airway lesions in preterm infants include subglottic stenosis or cyst, vocal cord paresis/paralysis, voice quality abnormalities, and tracheobronchomalacia. (121) (122) These conditions can present as stridor, obstructive sleep apnea, recurrent croup, and/or chronic respiratory symptoms often caused by recurrent trauma from endotracheal tube insertion or from prolonged intubation. Severe disease may require tracheostomy and/or airway reconstruction. Causes of vocal cord paresis or paralysis, (unilateral or bilateral), may include endotracheal intubation-related injury or recurrent laryngeal nerve damage following cardiac surgery. Tracheomalacia and/or bronchomalacia can develop in preterm infants requiring prolonged mechanical ventilation, resulting in excessive dynamic collapse of the trachea and major bronchi and is associated with increased respiratory morbidity in children with bronchopulmonary dysplasia.(123, 124) Dysphagia and inadequately controlled gastroesophageal reflux (GER) can worsen subglottic disease, impair postoperative healing, and increase risk of aspiration and sleep-disordered breathing. Gastrointestinal complications associated with prematurity can include structural and motility disorders (e.g., dysmotility, short bowel syndrome) and feeding/reflux-related disorders (dysphagia, gastroesophageal reflux (GER), avoidant/restrictive food intake disorder (ARFID), eosinophilic

esophagitis EOE)). Medical or surgical necrotizing enterocolitis can cause intestinal strictures, abnormal gut motility, gut dysbiosis and short gut syndrome (especially when large sections of small bowel are resected). Exposure to antibiotics can lead to intestinal dysbiosis and altered GI motility, which can affect somatic growth. GER and aspiration can worsen pulmonary symptoms and lead to dysphagia and ARFID in children born preterm. Children with dysphagia or ARFID may require outpatient therapy with occupational therapy and speech pathologists to help with advancement of oral feeding and may require long-term tube feedings. EOE has also been shown to be increasingly associated with preterm birth and can contribute to ongoing feeding difficulties and poor weight gain.(125)

Gaps in Knowledge:

- The natural history of airway and voice disorders after prematurity (time to resolution; late sequelae) is unclear.
- The best methods to diagnose and grade airway malacia and to titrate respiratory support (e.g., imaging vs dynamic bronchoscopy) remain uncertain.
- Thresholds and timing for evaluation of suspected silent aspiration are not well defined.
- Evidence-based feeding strategies that optimize safety while supporting lung growth and repair are limited.
- What are the links between feeding outcomes and respiratory disease need study, including indications for nutrition referral, enteral tube use, and gastrostomy timing?
- The long-term effects of lung–gut axis perturbations on outcomes in prematurity-associated lung disease (PLD) are incompletely understood.

Recommendations:

- Consultation: Consider aerodigestive referral when indicated for all preterm individuals; targeted evaluation may include laryngoscopy, EGD, and/or bronchoscopy with BAL to define airway–GI contributors.

- Upper-airway obstruction: Consider overnight polysomnography when symptoms suggest obstructive sleep apnea or abnormal gas exchange for all preterm individuals.
- Aspiration: We recommend periodically assessing for dysphagia/GERD for all preterm individuals, recognizing aspiration may be silent; if suspected, obtain swallow evaluation and implement aspiration-mitigation strategies with GI referral as needed .
- Voice/airway symptoms: Consider referring for suspected voice abnormalities or upper-airway pathology to airway/voice specialists for evaluation and management for all preterm individuals.

COMORBIDITIES: Growth and Nutritional Deficiencies

Background and Evidence: Proper nutritional management can enhance lung and systemic growth and promote injury repair in preterm infants. Providing adequate calories during early life can be challenging particularly in those preterm infants that require fluid restriction. Early preterm and low birth weight children have been shown to be shorter and lighter than term children at 12 years of age,(126) and reduced linear growth during puberty has also been shown to correlate with lower lung growth as measured by FVC and FEV₁.(127) Thus, optimizing linear growth and BMI early in life is needed to help improve long-term lung growth. In contrast, overfeeding or rapid weight gain in older infants with BPD may be detrimental and is associated with a dysanapsis lung phenotype in later life.(95) Limited evidence suggests adults born at very low birth weights may have poorer nutritional intake, which may be a risk factor for developing osteoporosis and cardiovascular disease.(128) In preterm infants, vitamin A injections have been shown to reduce progression to BPD or death, but supplementation is most effective in lower risk preterm infants.(129) Adequate vitamin D levels may lower inflammation and zinc supplementation may enhance lung repair and immune regulation in preterm infants.(130) Feeding with human milk is associated with a decreased risk of necrotizing enterocolitis (NEC) particularly in very-low birth weight infants.(131)

Gaps in Knowledge:

- What are appropriate nutritional targets for preterm individuals at different ages?
- What are the optimal strategies for managing faltering weight?
- In addition to somatic growth, what are other important nutritional outcomes?

Recommendations:

- Screening: Nutrition should be optimized in early childhood to support optimal lung growth for all individuals born preterm. Primary care providers are recommended to monitor somatic growth closely in childhood and be prepared to discuss respiratory risks of faltering weight and obesity. Nutrition referrals should be considered for patients below the 3rd percentile or above the 97th percentile at 2-3 years of age or significant percentile changes despite usual counselling.
- Outpatient Consultation: We recommend that any preterm infant discharged home with a feeding tube receive an outpatient consultation by a nutritionist or pediatric gastroenterologist, ideally within 2 months of discharge.

COMORBIDITIES: Other Comorbidities

Background and Evidence: There are several other common comorbidities associated with prematurity that can affect respiratory health and lung development, including immune deficiencies, renal insufficiency, endocrine conditions, and others. In addition, the gut and airway microbiome in preterm infants may influence lung health outcomes. Microbial dysbiosis of the gut and airways can influence disease progression of preterm infants with BPD. In infants who developed BPD, elevated serum levels of IL1- β , IL4, IL6, TNF α and IL8 during the first month of life was associated with higher intestinal levels of *Proteobacteria*.⁽¹³²⁾ High levels of *Proteobacteria* in the gut have been associated with intestinal inflammation. In a systematic review, airway dysbiosis has also been found to be positively associated

with disease progression in infants with BPD.(133) Also, while the presence of PH is closely followed for many preterm individuals, systemic hypertension may not be as closely tracked as a sequelae of prematurity,(134) and should be assessed regularly per American Academy of Pediatrics guidelines.(135, 136) Furthermore, mental health and quality of life of children and their caregivers can influence or be influenced by respiratory status and overall health in PLD, and should be assessed regularly per American Academy of Pediatrics guidelines.(49, 137) Long-term corticosteroid use (systemic and high-dose inhaled) may require endocrinology follow-up per expert consensus and published endocrinology guidance owing to the risk of associated complications.(138, 139)

Gaps in Knowledge:

- Does renal function require periodic assessment through blood pressure assessments and/or blood or urine testing?
- What are optimal screening timepoints for developmental, cognitive and behavioral issues for preterm individuals?
- How can we design follow-up and improve mental health and quality of life for individuals and their families?

Recommendations:

- Screening: Primary care providers are recommended to obtain blood pressure annual visits for all preterm individuals and refer to an appropriate hypertension specialist when indicated.
- Screening: Primary care providers are recommended to screen for mental/cognitive/behavioral health issues at annual visits and as needed in any school age and adolescent child with a history of prematurity.
- Endocrinology consultation: Recommended for all PLD patients on chronic systemic steroids and considered for patients on long-term high-dose ICS.

CONCLUSION

This effort represents an important step towards standardizing outpatient management to improve outcomes for a disease that remains both underrecognized and highly heterogeneous (**Figure 4**). We recognize that most of our recommendations are based on limited, and often retrospective, observational data and expert opinions. We also acknowledge that many of our recommendations emphasize identifying patients at risk for complications; however, there remains an urgent need to identify strategies aimed at preventing or slowing disease progression. We also are acutely aware that shortages of pediatric specialists make it challenging to institute many of the recommendations in resource limited areas nationally and globally. Nevertheless, we hope that both primary care providers and subspecialists will be spurred to take up the challenge in coordinating care for individuals with PLD throughout the lifespan. We also hope that these efforts continue to reinforce studying the gaps in knowledge as priorities for both domestic and international funding agencies and foundations, with emphasis on the dearth of knowledge regarding phenotype differences, treatments, and long-term outcomes.

Table 1. Pathogenesis of Prematurity-associated lung disease (PLD)

Prenatal Factors	Postnatal Factors	Post-NICU Factors
Prematurity	Hyperoxia	Infection (daycare, etc.)
Chorioamnionitis	Infection	Secondhand smoke
Pre-eclampsia	Inflammation	Vaping emissions
IUGR	Ventilator barotrauma	Air pollution
Fetal Infection	Nutrition	Racial/ethnic disparities
Smoking/Substance Use		Socioeconomic status
Genetic Factors		Non-adherence
		Nutrition
		Immunoprophylaxis

Table 2. Indications for Pulmonary Subspecialty Care

	Respiratory Support	Pulmonary Hypertension	Daily Medications	Respiratory Admissions	Other
Inpatient (Prior to NICU discharge)	Tracheostomy IPPV NIPPV (All anticipated to be active at discharge)	Anticipated to be active at discharge	SCS PH therapy (All anticipated to be active at discharge)		
Outpatient (After NICU discharge; initial visit)	NC oxygen Tracheostomy IPPV NIPPV	Active	Diuretics Any ICS SCS PH therapy		
Outpatient (Pediatric; ongoing care)	NC oxygen Tracheostomy IPPV NIPPV	Active	Diuretics Any ICS ICS/LABA SCS PH therapy	Within the past 12 months	Abnormal imaging or spirometry; exercise limitations; slow resolution from or recurrent respiratory tract infections
Outpatient (Pediatric; screening visit at 6, 12, and 18yo)	Any prior history of outpatient respiratory support			Recurrent admissions	
Outpatient (Adult; ongoing care)	NC oxygen Tracheostomy IPPV NIPPV	Active	Diuretics High-dose ICS ICS/LABA SCS PH therapy	Within the past 12 months	Abnormal imaging or spirometry; exercise limitations; slow resolution from or recurrent respiratory tract infections

ICS: Inhaled corticosteroids; IPPV: Invasive positive pressure ventilation; LABA: Long-acting beta-agonist; NC: Nasal cannula; NIPPV: Non-invasive positive pressure ventilation; PH: Pulmonary hypertension; SCS: Systemic corticosteroids

Table 3. Recommended Screening Tests*

	Screening Tests			
	Spirometry	Plethysmography and DLCO	6 Minute Walk Test	Echocardiography
6yo	Yes			Serial echocardiography until resolved and off of any PH medications. Screening for any patient with a history of PH or severe BPD who presents with unexplained ongoing hypoxia, shortness of breath, or dyspnea with exertion.
12yo	Yes	If spirometry abnormal	Consider, particularly if spirometry not feasible	
18yo**	Yes	If spirometry abnormal	Consider, particularly if spirometry not feasible	

*See Table 2 for indications for screening.

**Testing should also be considered when transitioning to adult pulmonology care if this does not occur at 18yo.

PH: Pulmonary hypertension

FIGURE LEGENDS

Figure 1. Adult respiratory outcomes after prematurity and bronchopulmonary dysplasia

Figure 2. Spirometry patterns depicting the spectrum of common lung phenotypes in formerly preterm children

Figure 3. Persistent small airway narrowing in preterm children with bronchopulmonary dysplasia. **A.** Narrow small airways with fewer alveoli in infant with BPD. **B.** Catch-up alveolar growth with age in BPD child but persistent small airway narrowing. (Adapted from Journal of Perinatology, 2026.)

Figure 4. Recommendations for PLD care across the lifespan

REFERENCES

1. Ward VC, Lee AC, Hawken S, Otieno NA, Mujuru HA, Chimhini G, Wilson K, Darmstadt GL. Overview of the Global and US Burden of Preterm Birth. *Clin Perinatol* 2024; 51: 301-311.
2. Ohuma EO, Moller AB, Bradley E, Chakwera S, Hussain-Alkhateeb L, Lewin A, Okwaraji YB, Mahanani WR, Johansson EW, Lavin T, Fernandez DE, Dominguez GG, de Costa A, Cresswell JA, Krasevec J, Lawn JE, Blencowe H, Requejo J, Moran AC. National, regional, and global estimates of preterm birth in 2020, with trends from 2010: a systematic analysis. *Lancet* 2023; 402: 1261-1271.
3. Walani SR. Global burden of preterm birth. *Int J Gynaecol Obstet* 2020; 150: 31-33.
4. Cao G, Liu J, Liu M. Global, Regional, and National Incidence and Mortality of Neonatal Preterm Birth, 1990-2019. *JAMA pediatrics* 2022; 176: 787-796.
5. Myrhaug HT, Brurberg KG, Hov L, Markestad T. Survival and Impairment of Extremely Premature Infants: A Meta-analysis. *Pediatrics* 2019; 143.
6. Cao X, Li S, Gu X, Chen H, Yang C, Qian M, Tian X, Xu F, Yang Z, Wang Y, Guo J, Lee SK, Jiang S, Cao Y. Predicting death and survival without major morbidity for extremely preterm infants using information on hospital admission: a multicenter cohort study. *Translational pediatrics* 2025; 14: 927-938.
7. Ahmed AM, Grandi SM, Pullenayegum E, McDonald SD, Beltempo M, Premji SS, Pole JD, Bacchini F, Shah PS, Pechlivanoglou P. Short-Term and Long-Term Mortality Risk After Preterm Birth. *JAMA Netw Open* 2024; 7: e2445871.
8. Goss KN, Beshish AG, Barton GP, Haraldsdottir K, Levin TS, Tetri LH, Battiola TJ, Mulchrone AM, Pegelow DF, Palta M, Lamers LJ, Watson AM, Chesler NC, Eldridge MW. Early Pulmonary Vascular Disease in Young Adults Born Preterm. *American journal of respiratory and critical care medicine* 2018; 198: 1549-1558.
9. McGrath-Morrow SA, Collaco JM. Bronchopulmonary dysplasia: what are its links to COPD? *Ther Adv Respir Dis* 2019; 13: 1753466619892492.
10. Simpson SJ, Du Berry C, Evans DJ, Gibbons JTD, Vollaeter M, Halvorsen T, Gruber K, Lombardi E, Stanojevic S, Hurst JR, Um-Bergstrom P, Hallberg J, Doyle LW, Kotecha S, Pelican. Unravelling the respiratory health path across the lifespan for survivors of preterm birth. *Lancet Respir Med* 2024; 12: 167-180.
11. Du Berry C, Gray DM, Bates A, Salaam-Geydien D, Doyle LW, Simpson SJ. Trajectories of prematurity-associated lung disease: lifelong lung health. *Lancet Respir Med* 2026; 14: 72-84.
12. Rivera-Sanchez Y, Blair ZW, Wall TJ, Goss KN. Growing Up With Developmental Lung Diseases: A Review for the Adult Pulmonologist. *Chest* 2025; 168: 95-105.
13. Berkelhamer SK, Helman JM, Gugino SF, Leigh NJ, Lakshminrusimha S, Goniewicz ML. In Vitro Consequences of Electronic-Cigarette Flavoring Exposure on the Immature Lung. *International journal of environmental research and public health* 2019; 16.
14. Northway WH, Jr., Rosan RC, Porter DY. Pulmonary disease following respirator therapy of hyaline-membrane disease. Bronchopulmonary dysplasia. *The New England journal of medicine* 1967; 276: 357-368.

15. Siffel C, Kistler KD, Lewis JFM, Sarda SP. Global incidence of bronchopulmonary dysplasia among extremely preterm infants: a systematic literature review. *The journal of maternal-fetal & neonatal medicine : the official journal of the European Association of Perinatal Medicine, the Federation of Asia and Oceania Perinatal Societies, the International Society of Perinatal Obstet* 2021; 34: 1721-1731.
16. Moreira A, Noronha M, Joy J, Bierwirth N, Tarriela A, Naqvi A, Zoretic S, Jones M, Marotta A, Valadie T, Brick J, Winter C, Porter M, Decker I, Bruschetti M, Ahuja SK. Rates of bronchopulmonary dysplasia in very low birth weight neonates: a systematic review and meta-analysis. *Respiratory research* 2024; 25: 219.
17. Collaco JM, McGrath-Morrow SA. Respiratory Phenotypes for Preterm Infants, Children, and Adults: Bronchopulmonary Dysplasia and More. *Ann Am Thorac Soc* 2018; 15: 530-538.
18. Collaco JM, McGrath-Morrow SA. Bronchopulmonary dysplasia as a determinant of respiratory outcomes in adult life. *Pediatr Pulmonol* 2021; 56: 3464-3471.
19. Higgins RD, Jobe AH, Koso-Thomas M, Bancalari E, Viscardi RM, Hartert TV, Ryan RM, Kallapur SG, Steinhorn RH, Konduri GG, Davis SD, Thebaud B, Clyman RI, Collaco JM, Martin CR, Woods JC, Finer NN, Raju TNK. Bronchopulmonary Dysplasia: Executive Summary of a Workshop. *The Journal of pediatrics* 2018; 197: 300-308.
20. Jobe AH, Bancalari E. Bronchopulmonary dysplasia. *American journal of respiratory and critical care medicine* 2001; 163: 1723-1729.
21. Jensen EA, Dysart K, Gantz MG, McDonald S, Bamat NA, Keszler M, Kirpalani H, Laughon MM, Poindexter BB, Duncan AF, Yoder BA, Eichenwald EC, DeMauro SB. The Diagnosis of Bronchopulmonary Dysplasia in Very Preterm Infants. An Evidence-based Approach. *American journal of respiratory and critical care medicine* 2019; 200: 751-759.
22. Lillebø HL, Engeset MS, Clemm HH, Halvorsen T, Røksund OD, Potrebny T, Vollsæter M. Expiratory airflow limitation in adults born extremely preterm: A systematic review and meta-analysis. *Paediatr Respir Rev* 2024; 50: 2-22.
23. Wang G, Hallberg J, Faner R, Koefoed HJ, Kebede Merid S, Klevebro S, Björkander S, Gruzieva O, Pershagen G, van Hage M, Guerra S, Bottai M, Georgelis A, Gehring U, Bergström A, Vonk JM, Kull I, Koppelman GH, Agusti A, Melén E. Plasticity of Individual Lung Function States from Childhood to Adulthood. *American journal of respiratory and critical care medicine* 2023; 207: 406-415.
24. Course CW, Bush A, Kotecha S. Looking beyond bronchopulmonary dysplasia: prematurity-associated lung disease and its phenotypes. *Lancet Respir Med* 2026; 14: 60-71.
25. Duijts L, Um Bergstrom P, Kotecha SJ, Kotecha S, Pijnenburg MW. Management of prematurity-associated lung disease from infancy through to adulthood. *Lancet Respir Med* 2026; 14: 85-98.
26. Collaco JM, Aoyama BC, Rice JL, McGrath-Morrow SA. Influences of environmental exposures on preterm lung disease. *Expert Rev Respir Med* 2021; 15: 1271-1279.
27. Aoyama BC, McGrath-Morrow SA, Psoter KJ, Collaco JM. Patterns of early life somatic growth in infants and children with a history of chronic lung disease of prematurity. *Pediatric pulmonology* 2023; 58: 2592-2599.

28. Sadeck L, Kfoury RA. An update on vaccination in preterm infants. *J Pediatr (Rio J)* 2023; 99 Suppl 1: S81-S86.
29. Townsi N, Laing IA, Hall GL, Simpson SJ. The impact of respiratory viruses on lung health after preterm birth. *Eur Clin Respir J* 2018; 5: 1487214.
30. Langston C, Kida K, Reed M, Thurlbeck WM. Human lung growth in late gestation and in the neonate. *Am Rev Respir Dis* 1984; 129: 607-613.
31. Ji X, Wu C, Chen M, Wu L, Li T, Miao Z, Lv Y, Ding H. Analysis of risk factors related to extremely and very preterm birth: a retrospective study. *BMC Pregnancy Childbirth* 2022; 22: 818.
32. Hart K, Cousins M, Watkins WJ, Kotecha SJ, Henderson AJ, Kotecha S. Association of early-life factors with prematurity-associated lung disease: prospective cohort study. *Eur Respir J* 2022; 59.
33. Kuiper-Makris C, Selle J, Nüsken E, Dötsch J, Alejandre Alcazar MA. Perinatal Nutritional and Metabolic Pathways: Early Origins of Chronic Lung Diseases. *Front Med (Lausanne)* 2021; 8: 667315.
34. Morrow LA, Wagner BD, Ingram DA, Poindexter BB, Schibler K, Cotten CM, Dagle J, Sontag MK, Mourani PM, Abman SH. Antenatal Determinants of Bronchopulmonary Dysplasia and Late Respiratory Disease in Preterm Infants. *Am J Respir Crit Care Med* 2017; 196: 364-374.
35. Spiroski AM, Oliver MH, Harding JE, Bloomfield FH. Intrauterine Intervention for the Treatment of Fetal Growth Restriction. *Curr Pediatr Rev* 2016; 12: 168-178.
36. Hirata K, Nakanishi H, Namba F, Mochizuki N, Hirano S, Wada K, Fujimura M. Perinatal risk factors and lung function at 8 years of age in extremely preterm infants: Insights from a new Japanese bronchopulmonary dysplasia classification. *Pediatr Neonatol* 2025.
37. Costa S, Fattore S, De Santis M, Lanzzone A, Spanu T, Arena V, Tana M, Trapani M, Sanguinetti M, Barnea ER, Vento G. Effect of acute histologic chorioamnionitis on bronchopulmonary dysplasia and mortality rate among extremely low gestational age neonates: A retrospective case-control study. *Int J Gynaecol Obstet* 2024; 165: 1040-1046.
38. Gilliland FD, Berhane K, McConnell R, Gauderman WJ, Vora H, Rappaport EB, Avol E, Peters JM. Maternal smoking during pregnancy, environmental tobacco smoke exposure and childhood lung function. *Thorax* 2000; 55: 271-276.
39. Aguilera J, Konvinse K, Lee A, Maecker H, Prunicki M, Mahalingaiah S, Sampath V, Utz PJ, Yang E, Nadeau KC. Air pollution and pregnancy. *Seminars in perinatology* 2023; 47: 151838.
40. Cicalese E, Howell H, Nuzum TA, Mavrogiannis N, Kaur G, Pierce K, Fleishaker S, Desai PV. Continuous positive airway pressure vs. high velocity nasal cannula for weaning respiratory support of preterm infants. *Journal of perinatal medicine* 2025; 53: 783-787.
41. Bussmann N, Smith A, Breatnach CR, McCallion N, Cleary B, Franklin O, McNamara PJ, El-Khuffash A. Patent ductus arteriosus shunt elimination results in a reduction in adverse outcomes: a post hoc analysis of the PDA RCT cohort. *Journal of perinatology : official journal of the California Perinatal Association* 2021; 41: 1134-1141.

42. Briana DD, Malamitsi-Puchner A. An Update on Lung Function of Extremely and Very Preterm Infants in Later Life: The Role of Early Nutritional Interventions. *Nutrients* 2023; 15.
43. Rojas-Quintero J, Faner R, Chiu CY, Kue JT, Zhang Y, Sanz Rubio D, Colborg AS, Jakwerth CA, Schmidt-Weber CB, Maitland-van der Zee AH, Abdel-Aziz MI, Pilon AL, Owen CA, Plosa EJ, Kinney GL, Mc-Grath Morrow S, Gazzaneo MG, Cortes Santiago N, Lingappan K, Ledford JG, Spence J, Sucre JM, Sauler M, Wu TD, Agusti A, Wheelock A, Illi S, von Mutius E, Bowler RP, Celli B, Abman SH, Wells JM, Polverino F. Maternal smoking and CC-16: implications for lung development and COPD across the lifespan. *American journal of respiratory and critical care medicine* 2026; 212: 591-604.
44. Sun BZ, Gaffin JM. Recent Insights into the Environmental Determinants of Childhood Asthma. *Curr Allergy Asthma Rep* 2024; 24: 253-260.
45. Bui DS, Perret JL, Walters EH, Lodge CJ, Bowatte G, Hamilton GS, Thompson BR, Frith P, Erbas B, Thomas PS, Johns DP, Wood-Baker R, Hopper JL, Davis PG, Abramson MJ, Lowe AJ, Dharmage SC. Association between very to moderate preterm births, lung function deficits, and COPD at age 53 years: analysis of a prospective cohort study. *Lancet Respir Med* 2022; 10: 478-484.
46. Jussinniemi L, Benum SD, Aakvik KAD, Kulmala MK, Jorgensen APM, Andersson S, Hovi P, Leppanen HK, Steinshamn S, Evensen KAI, Kajantie E. Lung function in adults born with very low birth weight from young to mid-adulthood. *Pediatr Res* 2026; 99: 215-223.
47. Waitzman NJ, Jalali A, Grosse SD. Preterm birth lifetime costs in the United States in 2016: An update. *Semin Perinatol* 2021; 45: 151390.
48. Yu S, Lui K, Fiebig DG, Travadi J, Homer CSE, Sinclair L, Scarf V, Viney R. Preterm Birth and Total Health Care Use and Costs in the First 5 Years of Life: A Population-based Study. *J Pediatr* 2023; 258: 113327.
49. McGrath-Morrow SA, Ryan T, Riekert K, Lefton-Greif MA, Eakin M, Collaco JM. The impact of bronchopulmonary dysplasia on caregiver health related quality of life during the first 2 years of life. *Pediatric pulmonology* 2013; 48: 579-586.
50. Peralta GP, Piatti R, Haile SR, Adams M, Bassler D, Moeller A, Natalucci G, Kriemler S. Respiratory morbidity in preschool and school-age children born very preterm and its association with parents' health-related quality of life and family functioning. *European journal of pediatrics* 2023; 182: 1201-1210.
51. Twilhaar ES, Wade RM, de Kieviet JF, van Goudoever JB, van Elburg RM, Oosterlaan J. Cognitive Outcomes of Children Born Extremely or Very Preterm Since the 1990s and Associated Risk Factors: A Meta-analysis and Meta-regression. *JAMA Pediatr* 2018; 172: 361-367.
52. Berlin KEK, Scott W, Dawson S, Brousseau D, Lagatta JM. Health-Related Quality of Life for Parents of Infants with Bronchopulmonary Dysplasia. *The Journal of pediatrics* 2024; 264: 113773.
53. Carlin D, Martin A, VanCliff C, Baldwin N, Aoyama BC, Collaco JM, McGrath-Morrow SA. Caregiver Social Needs and Outpatient Respiratory Outcomes in a Cohort of Children With Bronchopulmonary Dysplasia. *Pediatr Pulmonol* 2025; 60: e71286.

54. Harris M, Cusatis R, Malnory M, Dawson S, Konduri GG, Lagatta J. Identifying barriers and facilitators to care for infants with bronchopulmonary dysplasia after NICU discharge: a prospective study of parents and clinical stakeholders. *Journal of perinatology : official journal of the California Perinatal Association* 2024; 44: 970-978.
55. Thivierge E, Luu TM, Bourque CJ, Barrington KJ, Pearce R, Jaworski M, Janvier A. Pulmonary important outcomes after extremely preterm birth: Parental perspectives. *Acta paediatrica* 2023; 112: 970-976.
56. McAndrew S, Acharya K, Westerdahl J, Brousseau DC, Panepinto JA, Simpson P, Leuthner J, Lagatta JM. A Prospective Study of Parent Health-Related Quality of Life before and after Discharge from the Neonatal Intensive Care Unit. *The Journal of pediatrics* 2019; 213: 38-45 e33.
57. Gough A, Spence D, Linden M, Halliday HL, McGarvey L. General and Respiratory Health Outcomes in Adult Survivors of Bronchopulmonary Dysplasia: A Systematic Review. *Chest* 2011.
58. Yang J, Kingsford RA, Horwood J, Epton MJ, Swanney MP, Stanton J, Darlow BA. Lung Function of Adults Born at Very Low Birth Weight. *Pediatrics* 2020; 145.
59. Smith EF, Hemy NR, Hall GL, Wilson AC, Murray CP, Simpson SJ. Risk factors for poorer respiratory outcomes in adolescents and young adults born preterm. *Thorax* 2023; 78: 1223-1232.
60. Wu KY, Jensen EA, White AM, Wang Y, Biko DM, Nilan K, Fraga MV, Mercer-Rosa L, Zhang H, Kirpalani H. Characterization of Disease Phenotype in Very Preterm Infants with Severe Bronchopulmonary Dysplasia. *American journal of respiratory and critical care medicine* 2020; 201: 1398-1406.
61. Kotecha SJ, Watkins WJ, Paranjothy S, Dunstan FD, Henderson AJ, Kotecha S. Effect of late preterm birth on longitudinal lung spirometry in school age children and adolescents. *Thorax* 2012; 67: 54-61.
62. Harju M, Keski-Nisula L, Georgiadis L, Raisanen S, Gissler M, Heinonen S. The burden of childhood asthma and late preterm and early term births. *The Journal of pediatrics* 2014; 164: 295-299 e291.
63. Bamat NA, Nelin TD, Eichenwald EC, Kirpalani H, Laughon MM, Jackson WM, Jensen EA, Gibbs KA, Lorch SA. Loop Diuretics in Severe Bronchopulmonary Dysplasia: Cumulative Use and Associations with Mortality and Age at Discharge. *The Journal of pediatrics* 2021; 231: 43-49 e43.
64. Synnes A, Lam MM, Ricci MF, Church P, Simard MN, Zwicker JG, Luu TM. How to measure patient and family important outcomes in extremely preterm infants: A scoping review. *Acta paediatrica* 2024; 113: 1228-1235.
65. Powell CV, McNamara P, Solis A, Shaw NJ. A parent completed questionnaire to describe the patterns of wheezing and other respiratory symptoms in infants and preschool children. *Archives of disease in childhood* 2002; 87: 376-379.
66. Trinick R, Southern KW, McNamara PS. Assessing the Liverpool Respiratory Symptom Questionnaire in children with cystic fibrosis. *The European respiratory journal* 2012; 39: 899-905.
67. Abman SH, Collaco JM, Shepherd EG, Keszler M, Cuevas-Guaman M, Welty SE, Truog WE, McGrath-Morrow SA, Moore PE, Rhein LM, Kirpalani H, Zhang H, Gratny LL, Lynch SK, Curtiss J, Stonestreet BS, McKinney RL, Dysart KC, Gien

- J, Baker CD, Donohue PK, Austin E, Fike C, Nelin LD, Bronchopulmonary Dysplasia C. Interdisciplinary Care of Children with Severe Bronchopulmonary Dysplasia. *The Journal of pediatrics* 2017; 181: 12-28 e11.
68. Thurlbeck WM. Postnatal human lung growth. *Thorax* 1982; 37: 564-571.
 69. Herring MJ, Putney LF, Wyatt G, Finkbeiner WE, Hyde DM. Growth of alveoli during postnatal development in humans based on stereological estimation. *Am J Physiol Lung Cell Mol Physiol* 2014; 307: L338-L344.
 70. Stein ES, Hevroni A, Zarfati M, Erlichman I, Boursheh L, Gileles-Hillel A. Comprehensive pulmonary function analysis identifies predominant obstructive phenotype in former premature infants around one year of age. *Pediatric pulmonology* 2024; 59: 3667-3676.
 71. McEvoy CT, MacDonald KD, Go MA, Milner K, Harris J, Schilling D, Olson M, Tiller C, Slaven JE, Bjerregaard J, Vu A, Martin A, Mamidi R, Schelonka RL, Morris CD, Tepper RS. Extended Continuous Positive Airway Pressure in Preterm Infants Increases Lung Growth at 6 Months: A Randomized Controlled Trial. *Am J Respir Crit Care Med* 2025; 211: 610-618.
 72. Rhein L, White H, Simoneau T, Traeger N, Lahiri T, Kremer T, Sheils C, Meyer K, Rosenkrantz T, Krishnan S, Hartman T, Feldman HA, Abu Jawdeh EG. Transmitted Home Oximetry and Duration of Home Oxygen in Premature Infants. *Pediatrics* 2020; 146.
 73. Chan JY, Stern DA, Guerra S, Wright AL, Morgan WJ, Martinez FD. Pneumonia in childhood and impaired lung function in adults: a longitudinal study. *Pediatrics* 2015; 135: 607-616.
 74. Martinez FD. Early-Life Origins of Chronic Obstructive Pulmonary Disease. *N Engl J Med* 2016; 375: 871-878.
 75. Hayden LP, Hobbs BD, Cohen RT, Wise RA, Checkley W, Crapo JD, Hersh CP, Investigators CO. Childhood pneumonia increases risk for chronic obstructive pulmonary disease: the COPDGene study. *Respiratory research* 2015; 16: 115.
 76. Wang X, Li Y, Shi T, Bont LJ, Chu HY, Zar HJ, Wahi-Singh B, Ma Y, Cong B, Sharland E, Riley RD, Deng J, Figueras-Aloy J, Heikkinen T, Jones MH, Liese JG, Markić J, Mejias A, Nunes MC, Resch B, Satav A, Yeo KT, Simões EAF, Nair H. Global disease burden of and risk factors for acute lower respiratory infections caused by respiratory syncytial virus in preterm infants and young children in 2019: a systematic review and meta-analysis of aggregated and individual participant data. *Lancet* 2024; 403: 1241-1253.
 77. Graham RJ, Enriquez LF, Elmi AF, Zavadoski JV, Bielak LG, Baker CD, Mansbach JM. Influenza and COVID-19 Vaccination Rates Among Children Receiving Long-Term Ventilation. *JAMA Netw Open* 2024; 7: e2430989.
 78. Chaw PS, Hua L, Cunningham S, Campbell H, Mikolajczyk R, Nair H, Investigators R. Respiratory Syncytial Virus-Associated Acute Lower Respiratory Infections in Children With Bronchopulmonary Dysplasia: Systematic Review and Meta-Analysis. *J Infect Dis* 2019.
 79. Costa LF, Queiroz DA, Lopes da Silveira H, Bernardino Neto M, de Paula NT, Oliveira TF, Tolardo AL, Yokosawa J. Human rhinovirus and disease severity in children. *Pediatrics* 2014; 133: e312-321.

80. Principi N, Esposito S. Paediatric human metapneumovirus infection: epidemiology, prevention and therapy. *J Clin Virol* 2014; 59: 141-147.
81. Pancham K, Sami I, Perez GF, Huseni S, Kurdi B, Rose MC, Rodriguez-Martinez CE, Nino G. Human Metapneumovirus Infection is Associated with Severe Respiratory Disease in Preschool Children with History of Prematurity. *Pediatrics and neonatology* 2016; 57: 27-34.
82. Cohen S, Dabaja-Younis H, Etshtein L, Gnatt I, Szwarcwort-Cohen M, Hadash A, Kassis I, Halberthal M, Shachor-Meyouhas Y. Burden of viral respiratory infections in the pediatric intensive care unit: age, virus distribution, and the impact of the COVID-19 pandemic. *European journal of pediatrics* 2024; 184: 88.
83. Kotecha SJ, Lowe J, Gillespie D, Perez-Alijas M, Aboklaish AF, Mahachi TL, Cumming OS, Harris D, Hubbard M, Thomas-Jones E, Jones T, Ladell K, Moore C, Humphreys I, Grigg J, Berrington J, Kotecha S. Randomised, double-blind, parallel group, placebo-controlled, trial of Bactek for the prevention of lower respiratory tract infections in preterm infants in the UK: BALLOON study - study protocol. *BMJ Open* 2026; 16: e107929.
84. McGrath-Morrow SA, Agarwal A, Alexiou S, Austin ED, Fierro JL, Hayden LP, Lai K, Levin JC, Manimtim WM, Moore PE, Rhein LM, Rice JL, Sheils CA, Tracy MC, Bansal M, Baker CD, Cristea AI, Popova AP, Siddaiah R, Villafranco N, Nelin LD, Collaco JM. Daycare Attendance is Linked to Increased Risk of Respiratory Morbidities in Children Born Preterm with Bronchopulmonary Dysplasia. *The Journal of pediatrics* 2022.
85. Rice JL, Collaco JM, Tracy MC, Sheils CA, Rhein LM, Popova AP, Moore PE, Miller AN, Manimtim WM, Lai K, Kaslow JA, Hayden LP, Fierro JL, Bansal M, Austin ED, Aoyama B, Alexiou S, Akangire G, Agarwal A, Villafranco N, Siddaiah R, Lagatta JM, Abul MH, Cristea AI, Baker CD, Abman SH, McGrath-Morrow SA. Parental Report of Indoor Air Pollution Is Associated with Respiratory Morbidities in Bronchopulmonary Dysplasia. *The Journal of pediatrics* 2024; 275: 114241.
86. Collaco JM, Morrow M, Rice JL, McGrath-Morrow SA. Impact of road proximity on infants and children with bronchopulmonary dysplasia. *Pediatric pulmonology* 2020; 55: 369-375.
87. Collaco JM, Tsukahara KR, Tracy MC, Sheils CA, Rice JL, Rhein LM, Popova AP, Nelin L, Miller AN, Manimtim WM, Levin JC, Lai K, Kaslow JA, Hayden LP, Bansal M, Austin ED, Aoyama B, Akangire G, Agarwal A, Villafranco N, McGrath-Morrow SA. Number of children in the household influences respiratory morbidities in children with bronchopulmonary dysplasia in the outpatient setting. *Pediatric pulmonology* 2023.
88. Banwell E, Collaco JM, Oates GR, Rice JL, Juarez LD, Young LR, McGrath-Morrow SA. Area deprivation and respiratory morbidities in children with bronchopulmonary dysplasia. *Pediatric pulmonology* 2022; 57: 2053-2059.
89. Szeto JJ, Radack JK, DeMauro SB, Jensen EA, Gibbs K, Novick NP, Scott KA, Murosko DC, Burris HH, Nelin TD. Environmental Determinants of Post-Discharge Acute Respiratory Illness among Preterm Infants with Bronchopulmonary Dysplasia. *International journal of environmental research and public health* 2024; 21.

90. Urbach H, Ostfeld-Johns S, McCollum S, Weiss P, Kandil S, Brumer E. Social Determinants of Health and Healthcare Utilization in Infants With Bronchopulmonary Dysplasia. *Pediatric pulmonology* 2025; 60: e71153.
91. Moore PE, Hayden LP, Villafranco NM, Toprak D, Rice JL, Rhein LM, Popova AP, McKinney RL, Manimtim WM, Levin JC, Lai KV, Gage SC, Bansal M, Baker CD, Austin ED, Agarwal A, McGrath-Morrow SA, Collaco JM. Outpatient clinical care for bronchopulmonary dysplasia: A survey of the BPD collaborative. *Pediatric pulmonology* 2025; 60: e27296.
92. Kotecha SJ, Edwards MO, Watkins WJ, Henderson AJ, Paranjothy S, Dunstan FD, Kotecha S. Effect of preterm birth on later FEV1: a systematic review and meta-analysis. *Thorax* 2013; 68: 760-766.
93. Koefoed HJL, Wang G, Gehring U, Ekstrom S, Kull I, Vermeulen R, Boer JMA, Bergstrom A, Koppelman GH, Melén E, Vonk JM, Hallberg J. Clinical implications of airway obstruction with normal or low FEV(1) in childhood and adolescence. *Thorax* 2024; 79: 573-580.
94. Martinez-Zamora MD, Valenzuela PL, Esteban Díez I, Martínez-de-Quel Ó. Influence of preterm birth on physical fitness in early childhood. *Eur J Sport Sci* 2023; 23: 2129-2138.
95. Cousins M, Hart K, Kotecha SJ, Henderson AJ, Watkins WJ, Bush A, Kotecha S. Characterising airway obstructive, dysanaptic and PRISm phenotypes of prematurity-associated lung disease. *Thorax* 2023; 78: 895-903.
96. McGinn EA, Mandell EW, Smith BJ, Duke JW, Bush A, Abman SH. Dysanapsis as a Determinant of Lung Function in Development and Disease. *American journal of respiratory and critical care medicine* 2023; 208: 956-963.
97. Morty RE. Recent advances in the pathogenesis of BPD. *Seminars in perinatology* 2018; 42: 404-412.
98. McGinn EA, Sosa A, Corrigan M, Seedorf G, Mecklenburg J, Veress LA, Abman SH, Smith BJ, Mandell EW. Early dysanapsis in experimental bronchopulmonary dysplasia: implications for lifelong lung disease. *American journal of physiology Lung cellular and molecular physiology* 2026; 331: L165-L177.
99. Peralta GP, Abellan A, Montazeri P, Basterrechea M, Esplugues A, Gonzalez-Palacios S, Roda C, Santa-Marina L, Sunyer J, Vrijheid M, Casas M, Garcia-Aymerich J. Early childhood growth is associated with lung function at 7 years: a prospective population-based study. *The European respiratory journal* 2020; 56.
100. Cadham CJ, Oh H, Han MK, Mannino D, Cook S, Meza R, Levy DT, Sánchez-Romero LM. The prevalence and mortality risks of PRISm and COPD in the United States from NHANES 2007-2012. *Respiratory research* 2024; 25: 208.
101. Dai C, Wu F, Tian J, Cui J, Deng Z, Tian H, Huang P, Yang H, Zheng Y, Wang Z, Xiao S, Wen X, Lu L, Zhao N, Yang C, Yu S, Liu S, Wang D, Wang X, Lu J, Zhong N, Zhou Y, Ran P. Lung Function Decline and Airflow Limitation Risk in Preserved Ratio Impaired Spirometry Subtypes by Smoking Status. *Chest* 2025.
102. Harris C, Jenkinson A, Zivanovic S, Pushparajah K, Dassios T, Abman SH, Greenough A. Diffusion of carbon monoxide in the lungs and pulmonary artery pressures in school aged children who were born preterm. *Pediatr Res* 2026.
103. Narayanan M, Beardsmore CS, Owers-Bradley J, Dogaru CM, Mada M, Ball I, Garipov RR, Kuehni CE, Spycher BD, Silverman M. Catch-up alveolarization in

- ex-preterm children: evidence from (3)He magnetic resonance. *Am J Respir Crit Care Med* 2013; 187: 1104-1109.
104. Raimondi F, Migliaro F, Corsini I, Meneghin F, Dolce P, Pierri L, Perri A, Aversa S, Nobile S, Lama S, Varano S, Savoia M, Gatto S, Leonardi V, Capasso L, Carnielli VP, Mosca F, Dani C, Vento G, Lista G. Lung Ultrasound Score Progress in Neonatal Respiratory Distress Syndrome. *Pediatrics* 2021; 147.
 105. Leon C, Martin A, Young LR, Aoyama BC, Rice JL, Kelchtermans J, Collaco JM, McGrath-Morrow SA. Outpatient inhaled corticosteroid use in bronchopulmonary dysplasia. *Pediatr Pulmonol* 2024; 59: 2815-2822.
 106. Goulden N, Cousins M, Hart K, Jenkins A, Willetts G, Yendle L, Doull I, Williams EM, Hoare Z, Kotecha S. Inhaled Corticosteroids Alone and in Combination With Long-Acting β 2 Receptor Agonists to Treat Reduced Lung Function in Preterm-Born Children: A Randomized Clinical Trial. *JAMA Pediatr* 2022; 176: 133-141.
 107. D'Angio CT, Heyne RJ, O'Shea TM, Schelonka RL, Shankaran S, Duara S, Goldberg RN, Stoll BJ, Van Meurs KP, Vohr BR, Das A, Li L, Burton RL, Hastings B, Phelps DL, Sanchez PJ, Carlo WA, Stevenson DK, Higgins RD. Heptavalent pneumococcal conjugate vaccine immunogenicity in very-low-birth-weight, premature infants. *Pediatr Infect Dis J* 2010; 29: 600-606.
 108. Martínón-Torres F, Wysocki J, Center KJ, Czajka H, Majda-Stanislawski E, Omeñaca F, Concheiro-Guisan A, Gimenez-Sanchez F, Szenborn L, Blázquez-Gamero D, Moreno-Galarraga L, Giardina PC, Sun G, Gruber WC, Scott DA, Gurtman A. Circulating Antibody 1 and 2 Years After Vaccination With the 13-Valent Pneumococcal Conjugate Vaccine in Preterm Compared With Term Infants. *Pediatr Infect Dis J* 2017; 36: 326-332.
 109. Song CH, Estevez D, Chernikova D, Hernandez F, Sakai-Bizmark R, Stiehm R. Low Baseline Pneumococcal Antibody Titers Predict Specific Antibody Deficiency, Increased Upper Respiratory Infections, and Allergy Sensitization. *Allergy Rhinol (Providence)* 2020; 11: 2152656719900338.
 110. Eldredge LC, Creasy RS, Presnell S, Debley JS, Juul SE, Mayock DE, Ziegler SF. Infants with evolving bronchopulmonary dysplasia demonstrate monocyte-specific expression of IL-1 in tracheal aspirates. *Am J Physiol Lung Cell Mol Physiol* 2019; 317: L49-L56.
 111. Collaco JM, McGrath-Morrow SA, Griffiths M, Chavez-Valdez R, Parkinson C, Zhu J, Northington FJ, Graham EM, Everett AD. Perinatal Inflammatory Biomarkers and Respiratory Disease in Preterm Infants. *J Pediatr* 2022; 246: 34-39.e33.
 112. Nissen G, Hinsienbrock S, Rausch TK, Stichtenoth G, Ricklefs I, Weckmann M, Franke A, Herting E, König IR, Kopp MV, Rabe KF, Göpel W. Lung Function of Preterm Children Parsed by a Polygenic Risk Score for Adult COPD. *NEJM Evid* 2023; 2: EVIDoA2200279.
 113. Kelchtermans J, March ME, Hakonarson H, McGrath-Morrow SA. Phenotype wide association study links bronchopulmonary dysplasia with eosinophilia in children. *Scientific reports* 2024; 14: 21391.
 114. Moreira A, Coleman M, Aziz K, Triviño V, Randolph L, Bierwirth NC, Valadie CT, Arya S, Meunier JA, McOmber B, Winter C, Lee GC, Decker I, Smith AM, Petershack M, Blanco CL, Kwinta P, Ahuja SK. Bronchopulmonary dysplasia in

- preterm neonates: Th2-Eosinophilic inflammation and asthma-like features. *Pediatr Res* 2025.
115. McGlynn JR, Aoyama BC, Collaco JM, McGrath-Morrow SA. Family history of asthma influences outpatient respiratory outcomes in children with BPD. *Pediatric pulmonology* 2021; 56: 3265-3272.
 116. Hansmann G, Sallmon H, Roehr CC, Kourembanas S, Austin ED, Koestenberger M. Pulmonary hypertension in bronchopulmonary dysplasia. *Pediatr Res* 2021; 89: 446-455.
 117. Gentle SJ, Travers CP, Clark M, Carlo WA, Ambalavanan N. Patent Ductus Arteriosus and Development of Bronchopulmonary Dysplasia-associated Pulmonary Hypertension. *Am J Respir Crit Care Med* 2023; 207: 921-928.
 118. Aoyama BC, McGrath MSA, Collaco JM. Characteristics of Children with Bronchopulmonary Dysplasia with Prolonged and/or Later-Onset Pulmonary Hypertension. *Ann Am Thorac Soc* 2021; 18: 1746-1748.
 119. McArthur E, Murthy K, Zaniletti I, Sharma M, Lagatta J, Ball M, Porta N, Grover T, Levy P, Padula M, Hamrick S, Vyas-Read S, BPD CS, Cardiology Focus G. Neonatal Risk Factors for Pulmonary Vein Stenosis in Infants Born Preterm with Severe Bronchopulmonary Dysplasia. *The Journal of pediatrics* 2024; 275: 114252.
 120. Swier NL, Richards B, Cua CL, Lynch SK, Yin H, Nelin LD, Smith CV, Backes CH. Pulmonary Vein Stenosis in Neonates with Severe Bronchopulmonary Dysplasia. *Am J Perinatol* 2016; 33: 671-677.
 121. Cristea AI, Ren CL, Amin R, Eldredge LC, Levin JC, Majmudar PP, May AE, Rose RS, Tracy MC, Watters KF, Allen J, Austin ED, Cataletto ME, Collaco JM, Fleck RJ, Gelfand A, Hayes D, Jr., Jones MH, Kun SS, Mandell EW, McGrath-Morrow SA, Panitch HB, Popatia R, Rhein LM, Teper A, Woods JC, Iyer N, Baker CD. Outpatient Respiratory Management of Infants, Children, and Adolescents with Post-Prematurity Respiratory Disease: An Official American Thoracic Society Clinical Practice Guideline. *American journal of respiratory and critical care medicine* 2021; 204: e115-e133.
 122. Reynolds V, Meldrum S, Simmer K, Vijayasekaran S, French N. Dysphonia in very preterm children: a review of the evidence. *Neonatology* 2014; 106: 69-73.
 123. Hysinger EB. Central airway issues in bronchopulmonary dysplasia. *Pediatr Pulmonol* 2021; 56: 3518-3526.
 124. Hysinger EB, Friedman NL, Padula MA, Shinohara RT, Zhang H, Panitch HB, Kawut SM. Tracheobronchomalacia Is Associated with Increased Morbidity in Bronchopulmonary Dysplasia. *Ann Am Thorac Soc* 2017.
 125. Kurt G, Svane HML, Erichsen R, Heide-Jørgensen U, Sørensen HT, Dellon ES, Jensen ET. Prenatal, Intrapartum, and Neonatal Factors Increase the Risk of Eosinophilic Esophagitis. *Am J Gastroenterol* 2023; 118: 1558-1565.
 126. Morkuniene R, Cole TJ, Levuliene R, Suchomlinov A, Tutkuvienė J. The associations of preterm birth and low birth weight with childhood growth curves between birth and 12 years: a SITAR-based longitudinal analysis. *Ann Hum Biol* 2025; 52: 2472757.
 127. Mahmoud O, Granell R, Tilling K, Minelli C, Garcia-Aymerich J, Holloway JW, Custovic A, Jarvis D, Sterne J, Henderson J. Association of Height Growth in

- Puberty with Lung Function. A Longitudinal Study. *Am J Respir Crit Care Med* 2018; 198: 1539-1548.
128. Kaseva N, Wehkalampi K, Hemio K, Hovi P, Jarvenpaa AL, Andersson S, Eriksson JG, Lindstrom J, Kajantie E. Diet and nutrient intake in young adults born preterm at very low birth weight. *The Journal of pediatrics* 2013; 163: 43-48.
 129. Rysavy MA, Li L, Tyson JE, Jensen EA, Das A, Ambalavanan N, Laughon MM, Greenberg RG, Patel RM, Pedroza C, Bell EF. Should Vitamin A Injections to Prevent Bronchopulmonary Dysplasia or Death Be Reserved for High-Risk Infants? Reanalysis of the National Institute of Child Health and Human Development Neonatal Research Network Randomized Trial. *J Pediatr* 2021; 236: 78-85.e75.
 130. Chen CY, Lai MY, Lee CH, Chiang MC. Nutritional Management for Preterm Infants with Common Comorbidities: A Narrative Review. *Nutrients* 2025; 17.
 131. Quigley M, Embleton ND, Meader N, McGuire W. Donor human milk for preventing necrotising enterocolitis in very preterm or very low-birthweight infants. *The Cochrane database of systematic reviews* 2024; 9: Cd002971.
 132. Zhang Z, Jiang J, Li Z, Wan W. The Change of Cytokines and Gut Microbiome in Preterm Infants for Bronchopulmonary Dysplasia. *Frontiers in microbiology* 2022; 13: 804887.
 133. Pammi M, Lal CV, Wagner BD, Mourani PM, Lohmann P, Luna RA, Sisson A, Shivanna B, Hollister EB, Abman SH, Versalovic J, Connett GJ, Bhandari V, Ambalavanan N. Airway Microbiome and Development of Bronchopulmonary Dysplasia in Preterm Infants: A Systematic Review. *J Pediatr* 2019; 204: 126-133.e122.
 134. Juonala M, Cheung MM, Sabin MA, Burgner D, Skilton MR, Kahonen M, Hutri-Kahonen N, Lehtimäki T, Jula A, Laitinen T, Jokinen E, Taittonen L, Tossavainen P, Viikari JS, Magnussen CG, Raitakari OT. Effect of birth weight on life-course blood pressure levels among children born premature: the Cardiovascular Risk in Young Finns Study. *J Hypertens* 2015; 33: 1542-1548.
 135. Kelly MM, Tobias J. Recommendations to optimize life-long health and wellbeing for people born preterm. *Early Hum Dev* 2021; 162: 105458.
 136. Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, de Ferranti SD, Dionne JM, Falkner B, Flinn SK, Gidding SS, Goodwin C, Leu MG, Powers ME, Rea C, Samuels J, Simasek M, Thaker VV, Urbina EM, Subcommittee On S, Management Of High Blood Pressure In C. Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents. *Pediatrics* 2017; 140.
 137. Peralta GP, Piatti R, Haile SR, Adams M, Bassler D, Moeller A, Natalucci G, Kriemler S. Correction to: Respiratory morbidity in preschool and school-age children born very preterm and its association with parents' health-related quality of life and family functioning. *Eur J Pediatr* 2023; 182: 1211-1212.
 138. Liu D, Ahmet A, Ward L, Krishnamoorthy P, Mandelcorn ED, Leigh R, Brown JP, Cohen A, Kim H. A practical guide to the monitoring and management of the complications of systemic corticosteroid therapy. *Allergy Asthma Clin Immunol* 2013; 9: 30.

139. Kapadia CR, Nebesio TD, Myers SE, Willi S, Miller BS, Allen DB, Jacobson-Dickman E, Drugs, Therapeutics Committee of the Pediatric Endocrine S. Endocrine Effects of Inhaled Corticosteroids in Children. *JAMA pediatrics* 2016; 170: 163-170.

ACCEPTED MANUSCRIPT

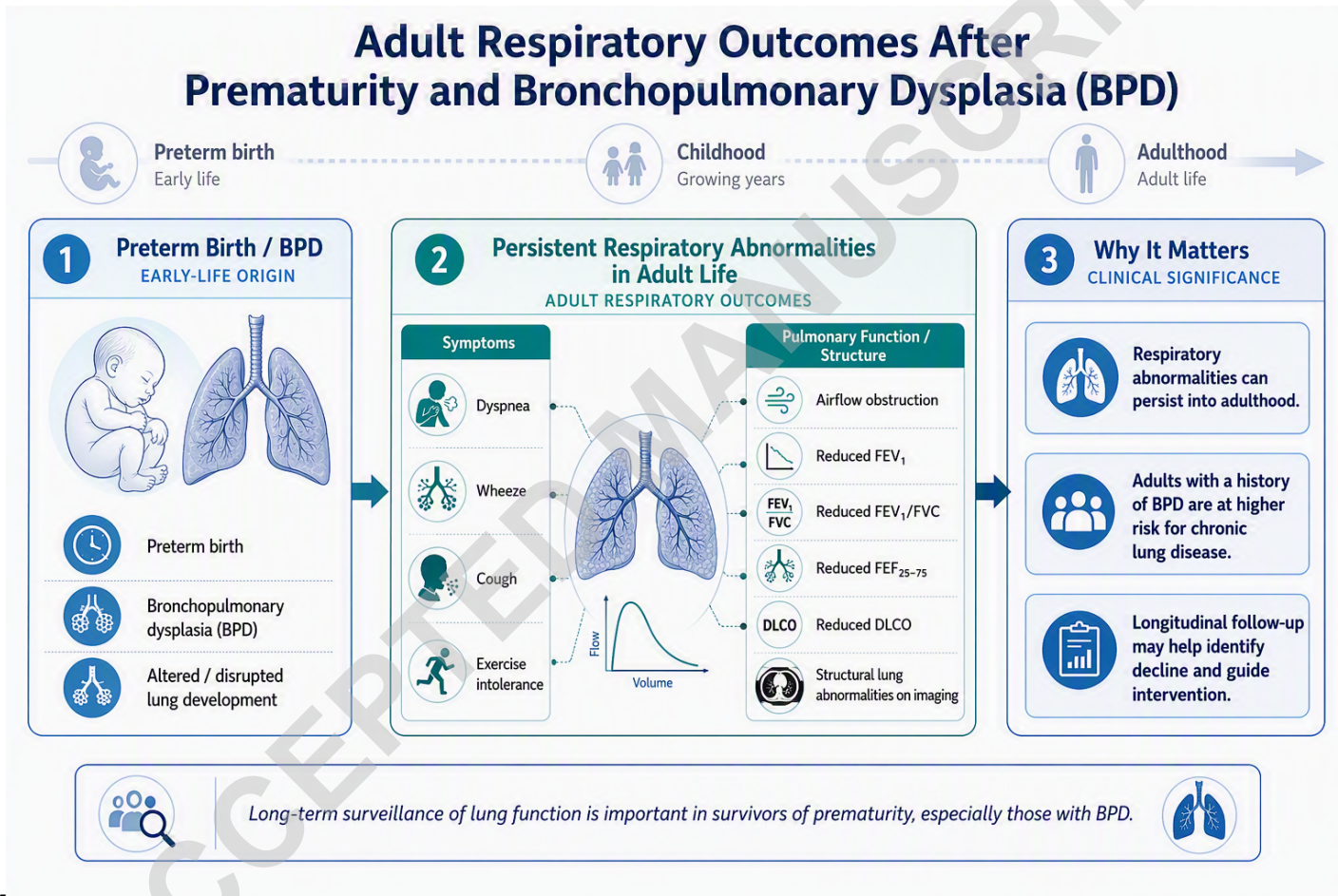


Figure 1

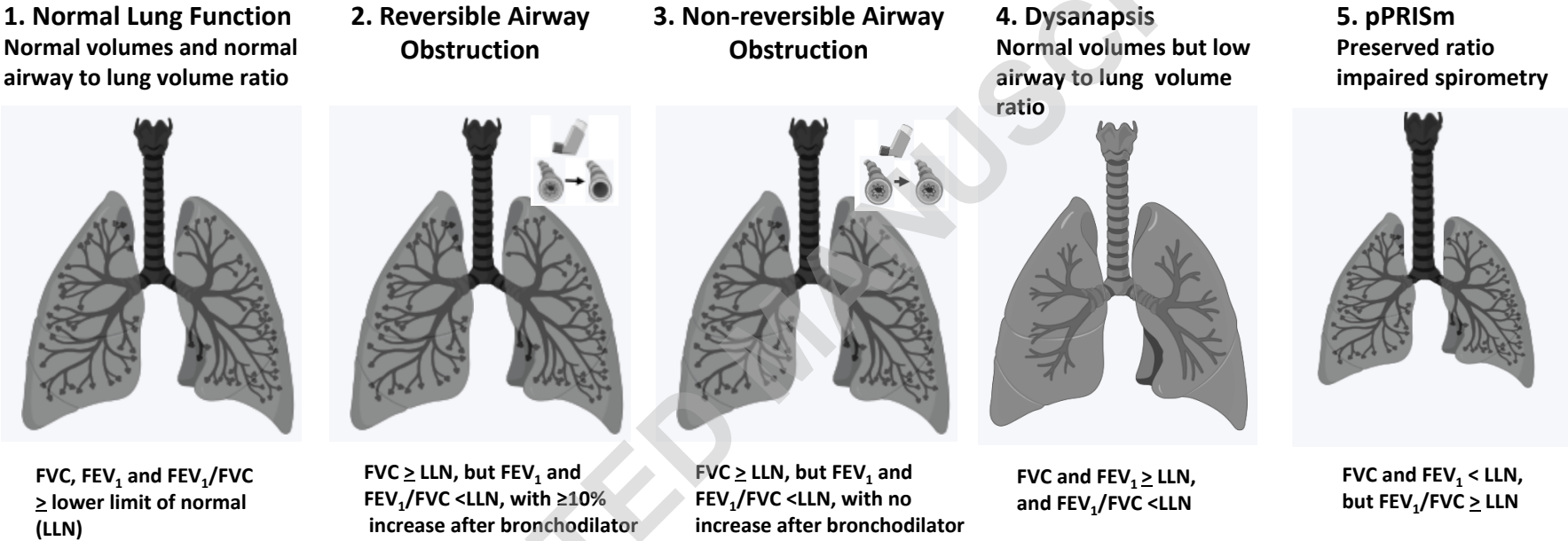


Figure 2

Fewer alveoli and narrow airways in BPD infant with airflow obstruction

With increasing age, catch-up alveolar growth with persistent airway narrowing in formerly preterm child

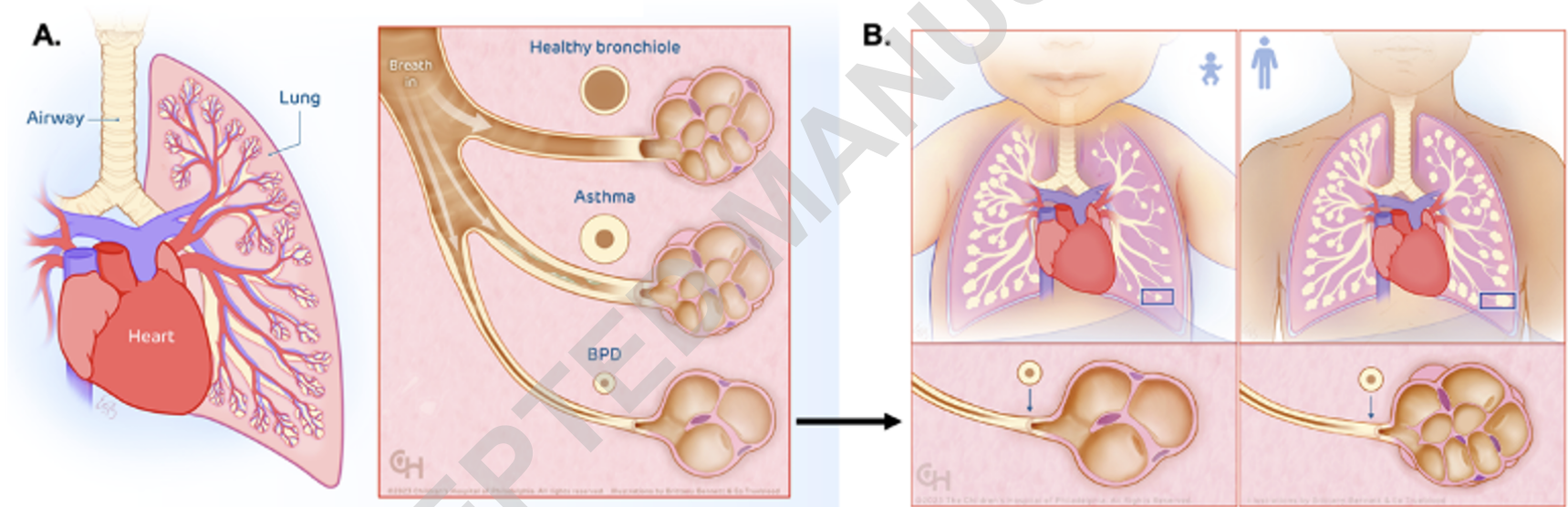


Figure 3

Figure 4. Prematurity-Associated Lung Disease (PLD): Lifespan Follow-up Framework
Consensus Recommendations Across the Lifespan

Target population: Individuals born <34 weeks' gestation, prioritizing those with BPD diagnosis, home respiratory support at discharge, and/or recurrent respiratory hospitalizations.

= RECOMMENDED

= CONSIDER

