News Release

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FOR MORE INFORMATION, CONTACT:
Keely Savoie or Brian Kell
ksavoie@thoracic.org or bkell@thoracic.org
ATS Office 212-315-8620 or 212-315-6442 (until May 14)
Cell phones 917-860-5814 or 516-305-9251
ATS Press Room: 504-670-6926 (May 15 to 20)

Poster session time: 1:30-4:00 p.m. May 16
Location: CC-Room 295-296 (Second Level), Morial Convention Center

Children with Severe Asthma at Increased Risk of Developing COPD

ATS 2010, NEW ORLEANS— Children with severe asthma have more than 30 times the risk of developing adult chronic obstructive lung disease (COPD) as adults compared to children without asthma, according to a prospective longitudinal cohort study from the Royal Children's Hospital in Melbourne.

The results will be presented at the ATS 2010 International Conference in New Orleans.

"There is important epidemiological evidence to suggest that events in childhood that influence lung growth constitute a significant risk for COPD," explained lead author, Andrew Tai, MBBS, FRACP. "The aim of this study was to describe the association between the pattern of childhood asthma and the risk of developing adult COPD in a longitudinal cohort."

Subjects of the Melbourne Asthma Study were recruited at the age of seven, from a 1957 birth cohort and were assessed regularly until the age of 50. At recruitment, subjects were classified as having no history of wheeze, intermittent asthma (such as viral-induced wheezing), persistent asthma (in the absence of illness), or severe asthma. Of the surviving members of the original group, 197 answered a detailed questionnaire and underwent lung function testing for the current study.

Subjects who were classified as having severe asthma in childhood had an adjusted risk of COPD of 31.9 times that of children without asthma. Interestingly, children with mild asthma were not at increased risk of developing adult obstructive lung disease.

“At this stage, children with mild asthma are those who have symptoms of wheeze which are triggered primarily by respiratory infections. A majority [of children with mild asthma] remit by adolescence or adulthood,” explained Dr. Tai. “However, children with more severe asthma features tend to have predisposing risk factors (like atopy) and continue to have symptoms of wheeze well into adult life.”

“It is important to emphasize that the lung function decline in this group is not increased compared to those with mild or no asthma, as has been raised in some other studies,” Dr. Tai continued. “However, lung function in children with severe asthma are reduced in childhood years and decline in adult life to levels consistent with adult obstructive lung disease. Fundamentally, we believe that this severe asthma group start with a lesser baseline lung function and gradually deteriorate to the levels consistent with a diagnosis of COPD. At this stage, there is no data on when airway remodeling occurs in children and hence, its impact on lung function, but there is an emerging relationship between childhood severe asthma and adult obstructive lung disease.”

Importantly, this study was performed on a group of children recruited in the 1960s when anti-inflammatory treatment was not available. Studies to date suggest that anti-inflammatory medications do not alter the natural progression of mild childhood asthma, but there are no studies performed in those children with severe asthma.

“There should be greater emphasis on the surveillance and treatment of children with asthma, therefore potentially preventing the development of adult obstructive lung disease,” said Dr. Tai.

Researchers still do not fully understand the mechanisms that link severe childhood asthma with adult COPD, but these findings suggest that appropriate treatment strategies (and surveillance) should be instigated early in life to potentially minimize future risk.

“Early treatment to prevent airway remodeling in childhood may reduce the incidence of this long-term complication of childhood asthma,” concluded Dr. Tai. “Currently, there are more than 30 birth cohort studies of varying duration being conducted around the world. In particular, the long-term follow-up of the Tucson birth cohort into young adulthood has shown trends similar to our findings of airway obstruction originating from early life. Clearly, more research to understand the mechanisms and timing of changes within the airway wall, inflammation and function needs to be conducted, applying preferably non-invasive methods in determining potential contributing factors.”

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“Pediatric Origins of Adult Chronic Obstructive Pulmonary Disease (COPD): Childhood Asthma” (Session A95, Sunday, May 16, 1:30-4:00 p.m., CC-Room 295-296 (Second Level), Morial Convention Center; Abstract 2206)

**Please note that numbers in this release may differ slightly from those in the abstract. Many of these investigations are ongoing; the release represents the most up-to-date data available at press time.*

Pediatric origins of adult chronic obstructive pulmonary disease(COPD): Childhood asthma

A.S.N. Tai[1], H. Tran[2], M. Roberts[1], N. Clarke[2], J.W. Wilson[3], C.F. Robertson[1]
[1]Royal Children's Hospital Melbourne - Melbourne/AU, [2]Murdoch Children's Research Institute - Melbourne/AU, [3]The Alfred Hospital - Melbourne/AU

Rationale
There is important epidemiological evidence to suggest that events in childhood that influence lung growth constitute a significant risk for COPD. Impaired lung function and biopsy evidence of airway remodeling have been reported in children with asthma from the age of three years and provide a basis for subsequent development of COPD. The aim of this study was to describe the association between the pattern of childhood asthma and the risk of developing adult COPD in a longitudinal cohort.

Methods
The Melbourne Asthma Study was recruited from a 1957 birth cohort at the age of 7 years and reviewed regularly to the age of 50 years. At recruitment, the subjects were grouped as: Controls(C - no history of wheeze till age 7 years), Intermittent asthma (IA - viral induced wheezing), Persistent Asthma (PA - wheeze in the absence of a respiratory tract infection), and Severe Persistent Asthma (SPA). COPD at age 50 years was defined according to the GOLD criteria (Pre and Post bronchodilator $FEV_1/FVC < 70\%$ with no bronchodilator response). Participants completed a questionnaire and measures of lung function.

Results
346 of the surviving cohort participated in the current study (participation rate of 76%) of which 197 completed both questionnaires and lung function testing. COPD was identified in 28 (14%) subjects and was more common in males (OR 2.4; 95^{th} CI: 0.9-6.3). These COPD subjects were from the following recruitment groups, SPA: 15/28, PA: 8/28, IA: 4/28, C: 1/28. When compared to controls, the OR (95% CI) for developing COPD was 37.1 (4.6-301) for those with SPA, 9.1 (1.1-76.4) for PA and 3.0 (0.3-35.6) for IA.

At age 50 years, FEV_1/FVC (95^{th} CI) of the COPD group was 63% (61-65%) compared to 78% (76-80%) in the controls. At age 10 years, FEV_1/FVC of the COPD group was 75% (73-77%) compared to 89% (86-92%) in the controls. The rate of decline in lung function through adult years was not different between groups.

Conclusion
Children with severe persistent asthma are at increased risk of developing COPD. The fixed abnormalities in lung function in adult life are clearly established in childhood and track at lower values progressing to irreversible airways obstruction in adulthood. Early treatment to prevent airway remodeling in childhood may reduce the incidence of this long term complication of childhood asthma.