Emerging Challenges in Primary Care 2019

18th Annual Regional and Online CME Conference Series

Addressing the Missing Link in COPD: Alpha-1 Antitrypsin Deficiency

Final Live Outcome Report

Grifols Grant ID 3742 : February 4, 2020

NACE

Executive Summary

- This activity focused on improving the recognition, diagnosis and treatment of Alpha-1 Antitrypsin Deficiency (AATD).

- 712 attendees in multiple professional specialties were reached in this program.
- Improvement across all learning domains was noted ranging from 33% to 322%.
- Overall, the program improved the ability of learners to recognize how to diagnosis and manage AATD.

Persistent Educational Gaps

- Though improvements were observed, learners demonstrated score slippage on the PCA indicating persistent gaps in the several areas including:
  - Pathophysiology of AAT Deficiency
  - Genetic phenotyping in AATD and its impact on risk for COPD
  - AATD screening strategies
  - Criteria for initiation of AAT augmentation therapy

The post-test scores, and self reported confidence regarding the management of patients with Alpha-1 Antitrypsin Deficiency, signifies a clear gap in knowledge and an unmet need among clinicians. It continues to be an important area for future educational programs.

*These numbers represent the total number of attendees, irrespective of assessment participation

Learning Objectives

1. Discuss the pathophysiology of Alpha-1 antitrypsin deficiency (AATD) and its impact on chronic obstructive pulmonary disease (COPD) risk.
2. Interpret the clinical significance of laboratory test results for AATD.
3. Discuss treatment options for AATD and latest GOLD guideline recommendations.
4. Discuss strategies to enhance detection and treatment of AATD in clinical practice.

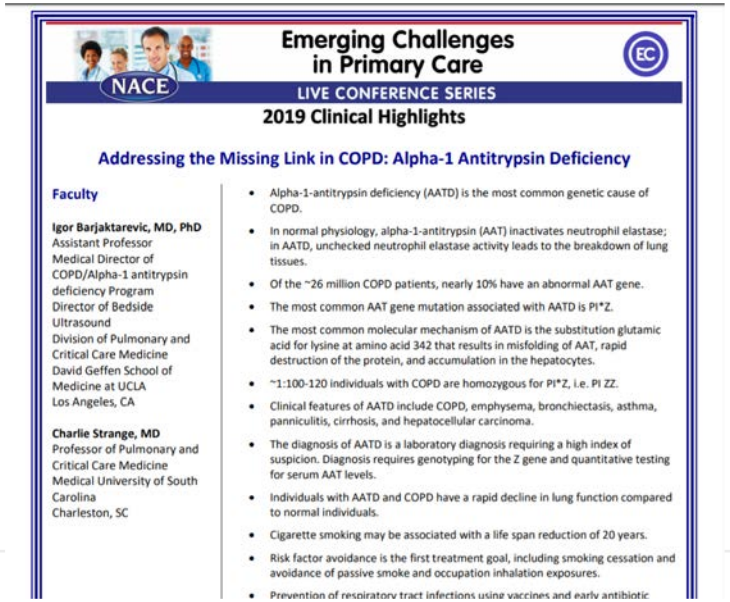
Live Curriculum Overview

2 Accredited Live Regional Symposiums 8/17/19 & 8/24/19

1 Accredited Live Virtual Simulcast: 8/24/2019

Clinical Highlights eMonograph

eMonograph, containing key teaching points from the CME activity was distributed 1 week after the meeting to all attendees.



Emerging Challenges in Primary Care

LIVE CONFERENCE SERIES

2019 Clinical Highlights

Addressing the Missing Link in COPD: Alpha-1 Antitrypsin Deficiency

Faculty

Igor Barjaktarevic, MD, PhD
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Medical Director of COPD/Alpha-1 antitrypsin deficiency Program
Director of Bedside Ultrasound
Division of Pulmonary and Critical Care Medicine
David Geffen School of Medicine at UCLA
Los Angeles, CA

Charlie Strange, MD
Professor of Pulmonary and Critical Care Medicine
Medical University of South Carolina
Charleston, SC

- Alpha-1-antitrypsin deficiency (AATD) is the most common genetic cause of COPD.
- In normal physiology, alpha-1-antitrypsin (AAT) inactivates neutrophil elastase; in AATD, unchecked neutrophil elastase activity leads to the breakdown of lung tissues.
- Of the ~26 million COPD patients, nearly 10% have an abnormal AAT gene.
- The most common AAT gene mutation associated with AATD is PI*Z.
- The most common molecular mechanism of AATD is the substitution glutamic acid for lysine at amino acid 342 that results in misfolding of AAT, rapid destruction of the protein, and accumulation in the hepatocytes.
- ~1:100-120 individuals with COPD are homozygous for PI*Z, i.e. PI ZZ.
- Clinical features of AATD include COPD, emphysema, bronchiectasis, asthma, panniculitis, cirrhosis, and hepatocellular carcinoma.
- The diagnosis of AATD is a laboratory diagnosis requiring a high index of suspicion. Diagnosis requires genotyping for the Z gene and quantitative testing for serum AAT levels.
- Individuals with AATD and COPD have a rapid decline in lung function compared to normal individuals.
- Cigarette smoking may be associated with a life span reduction of 20 years.
- Risk factor avoidance is the first treatment goal, including smoking cessation and avoidance of passive smoke and occupation inhalation exposures.
- Prevention of respiratory tract infections using vaccines and early antibiotic

Course Director

Franck Rahaghi, MD, MHS, FCCP
Director of Advanced Lung Disease Clinic
Director, Pulmonary Hypertension Clinic
Head of Alpha-1 Foundation Clinical Resource Center
Chairman, Dept. of Pulmonary and Critical Care
Cleveland Clinic Florida
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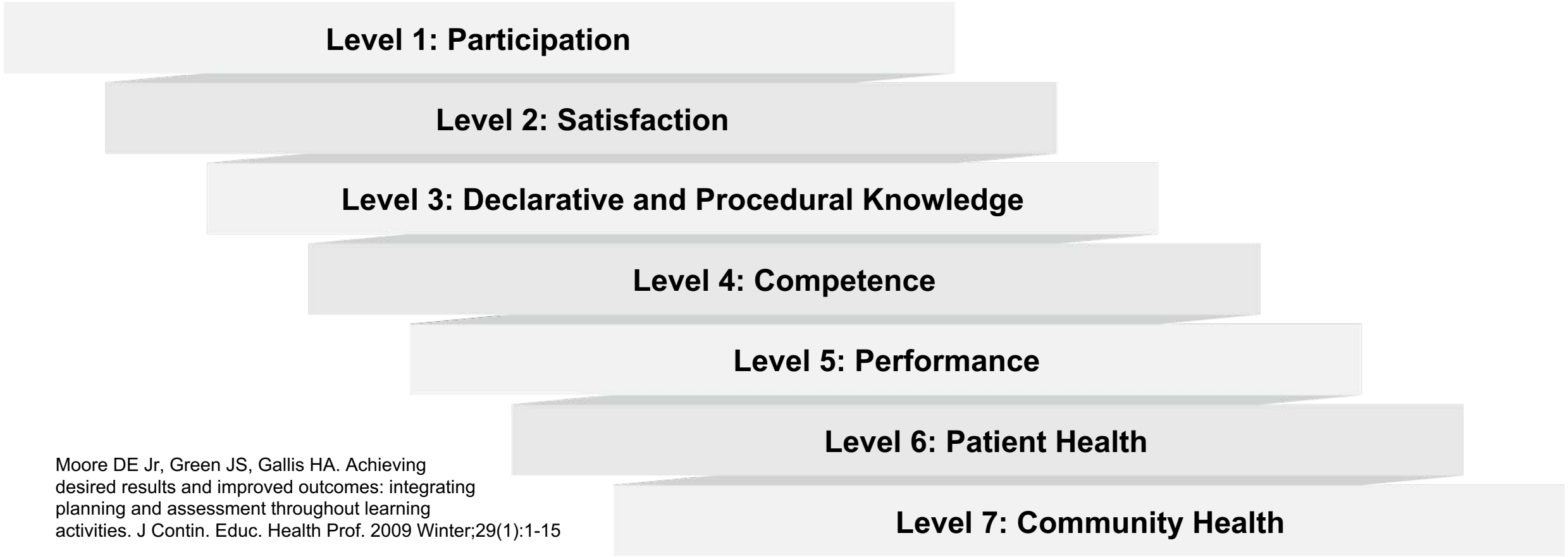
Commercial Support

The Emerging Challenges in Primary Care: 2019 series of CME activities were supported through educational grants or donations from the following companies:

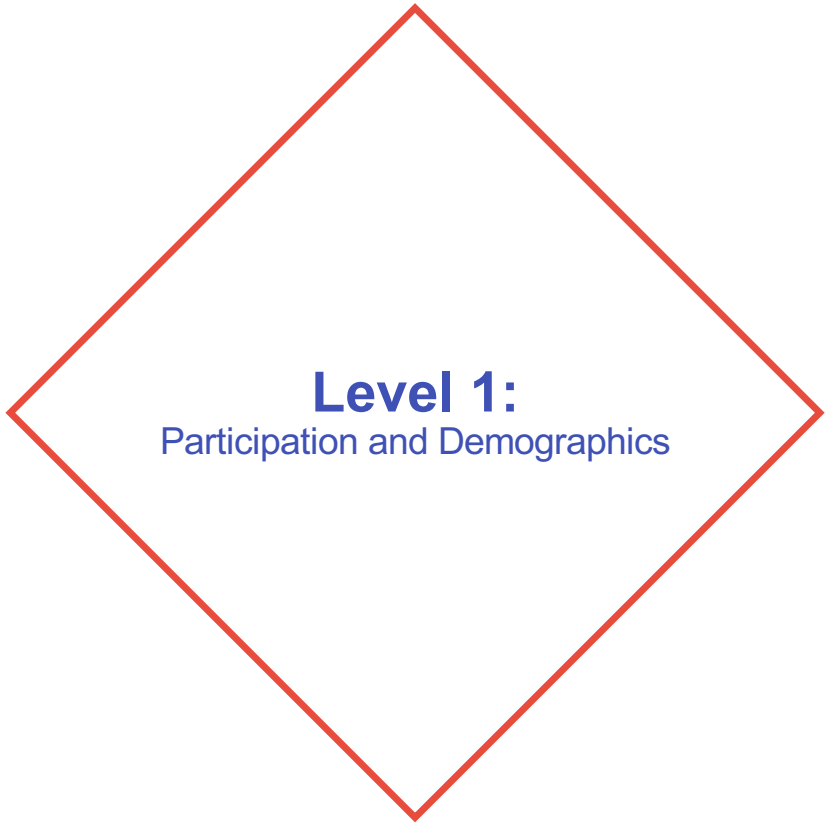
- Gilead Sciences, Inc.
- Amgen, Inc.
- AstraZeneca Pharmaceuticals LP
- Avanir Pharmaceuticals, Inc.
- Amarin
- Grifols
- Shire
- Novo Nordisk, Inc.
- Ferring Pharmaceuticals
- Sanofi US
- Med Learning Group

Levels of Evaluation

Consistent with the policies of the ACCME, NACE evaluates the effectiveness of all CME activities using a systematic process based on Moore's model. This outcome study reaches Level 5.



Moore DE Jr, Green JS, Gallis HA. Achieving desired results and improved outcomes: integrating planning and assessment throughout learning activities. J Contin. Educ. Health Prof. 2009 Winter;29(1):1-15


Level 1:
Participation and Demographics

Level 1:Participation

712 total attendees

2 cities: 328 attendees

1 live Virtual Simulcast: **384** attendees

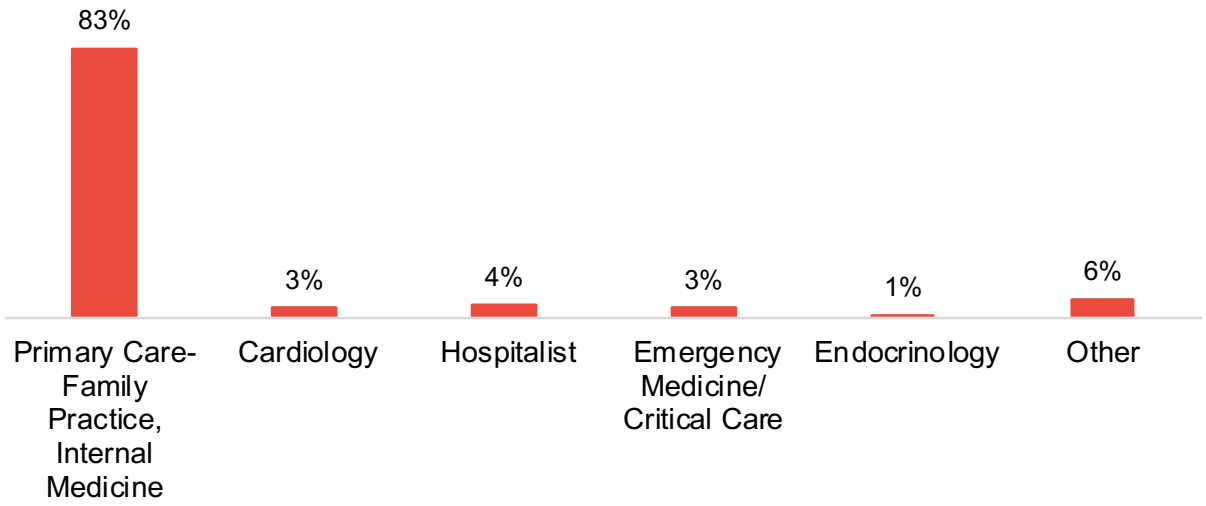
2018 Activity	Date	Attendees
Anaheim, CA	8/17/19	167
Troy, MI & Simulcast	8/24/19	161 & 384
Total		**712**

95%

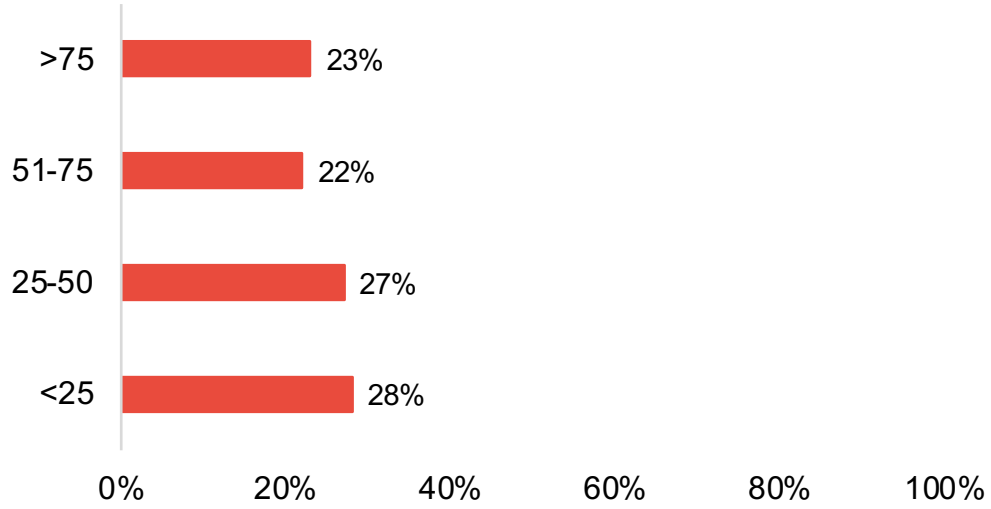
Provide direct patient care

Level 1: Demographics and Patient Reach

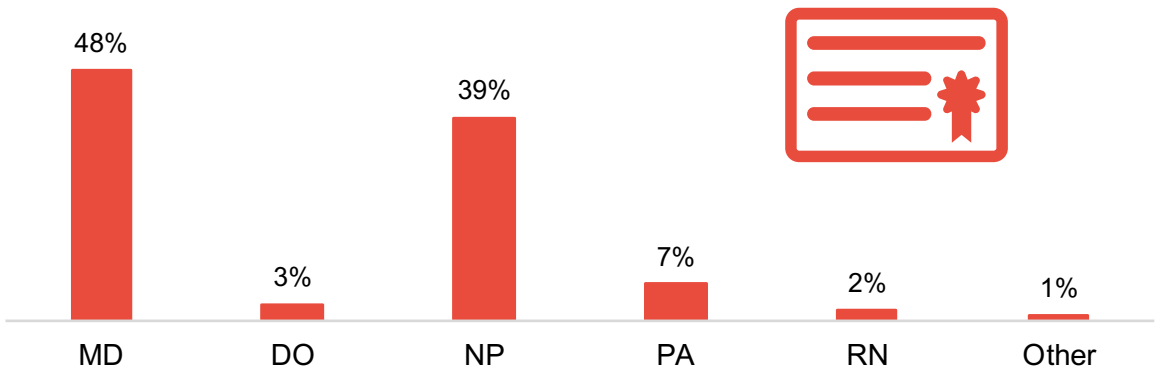
Specialty



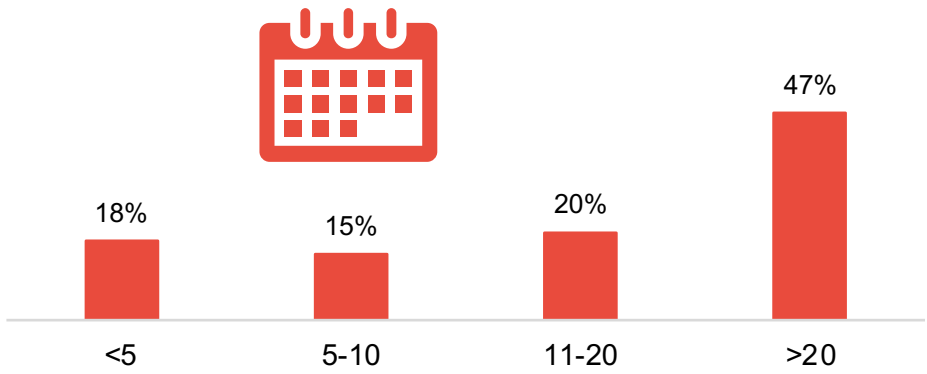
Patients seen each week, in any clinical setting:



Profession



Years in Practice




Level 2-5:
Outcomes Metrics

Level 2: Satisfaction

99% rated the activity as excellent

99% indicated the activity improved their knowledge

97% stated that they learned new and useful strategies for patient care

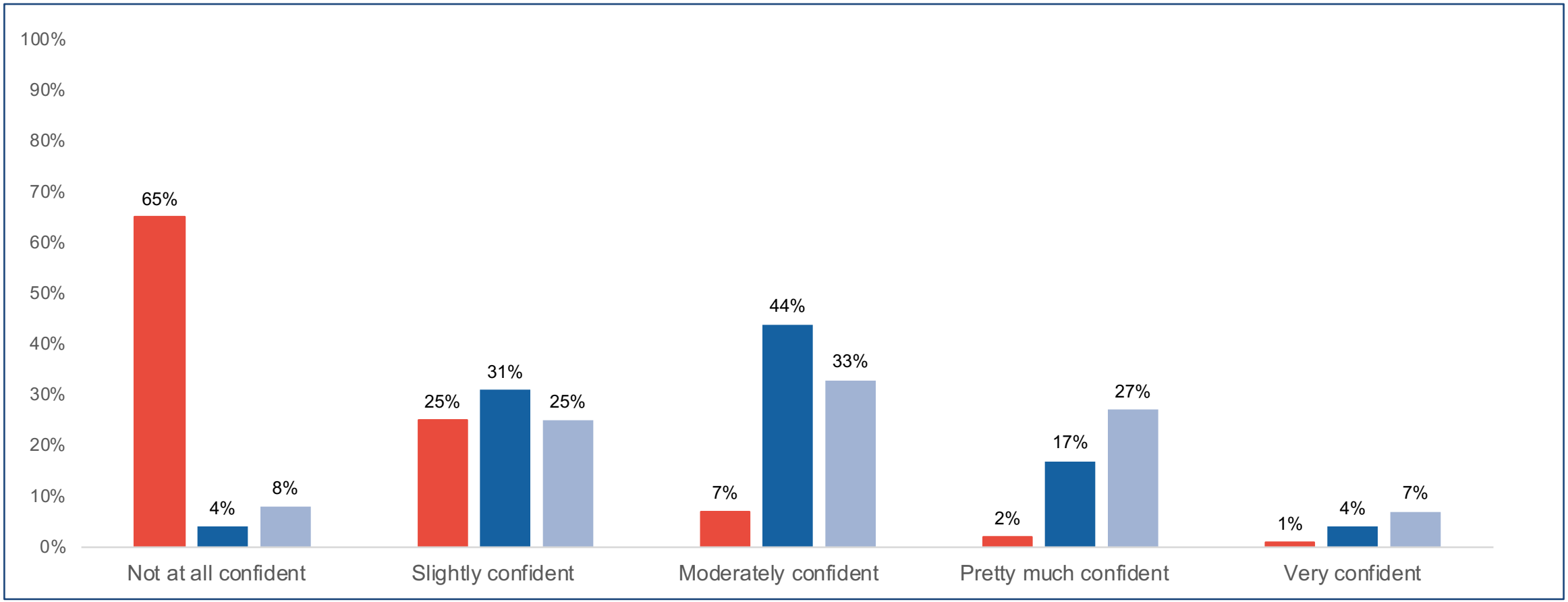
91% said they would implement new strategies that they learned

100% said the program was fair-balanced and unbiased

Confidence Assessment

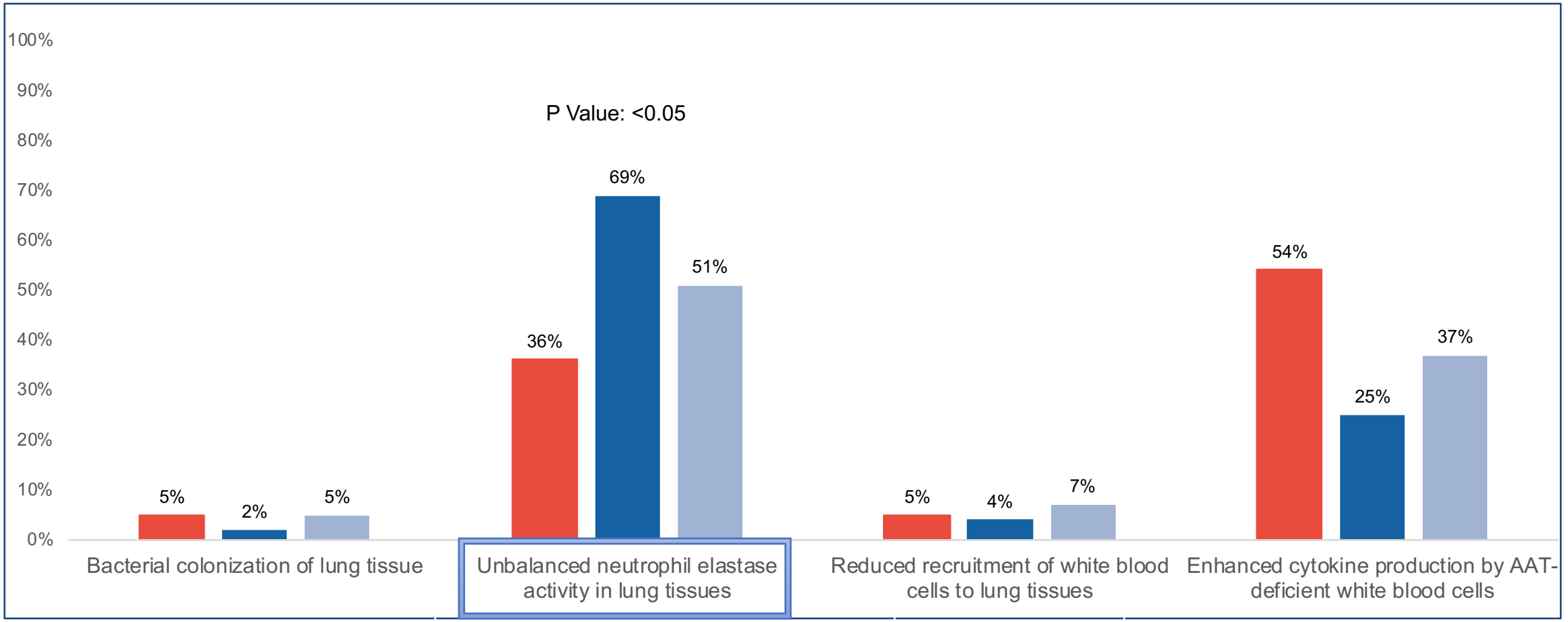
Please rate your confidence in your ability to integrate the assessment and management of AATD into the care of patients with COPD:

(Learning Objectives 1, 2, 3, and 4)



Pre: 401 Post: 412 PCA: 196

In patients with Alpha-1 antitrypsin deficiency (AATD), which of the following mechanisms contributes to breakdown of lung tissue? (Learning Objectives 1)

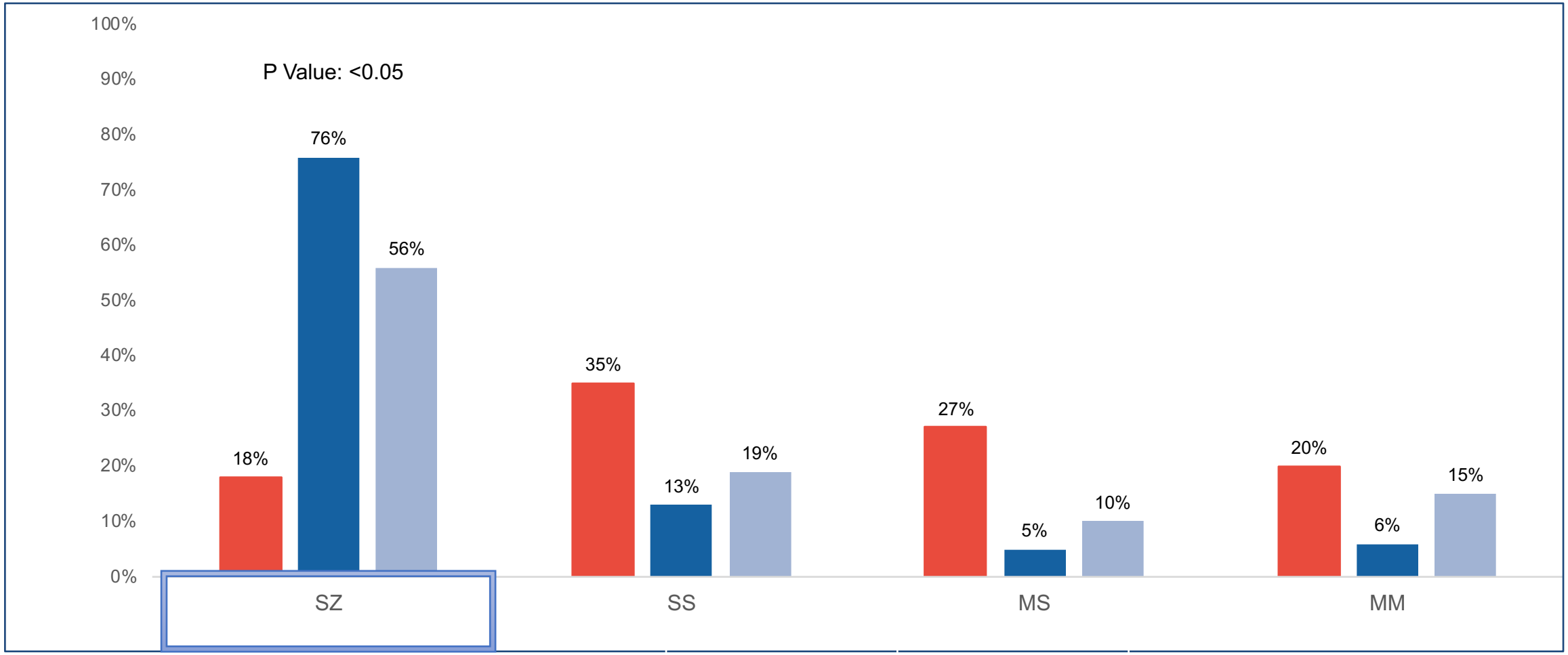


Pre: 364 Post: 354 PCA: 196

Pre-Post Change	**92%**
Pre-PCA Change	**42%**

Knowledge Assessment

On genetic testing for AATD, which of the following genotypes has the strongest predisposition for the development of emphysema? (Learning Objective 1, and 2)

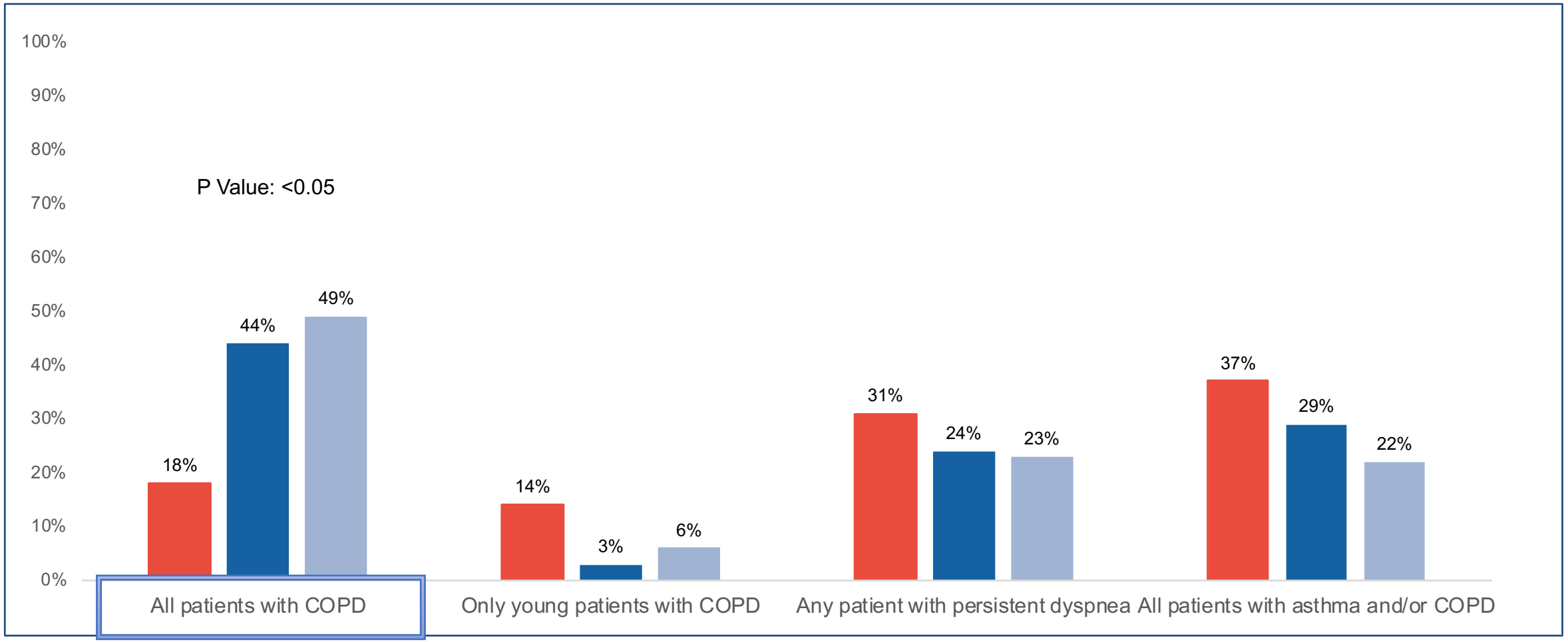


Pre: 365 Post: 403 PCA: 196

Pre-Post Change	**322%**
Pre-PCA Change	**211%**

Knowledge Assessment

According to current guidelines, which of the following groups should be screened for AATD? (Learning Objective 4)



Pre: 410 Post: 428 PCA: 196

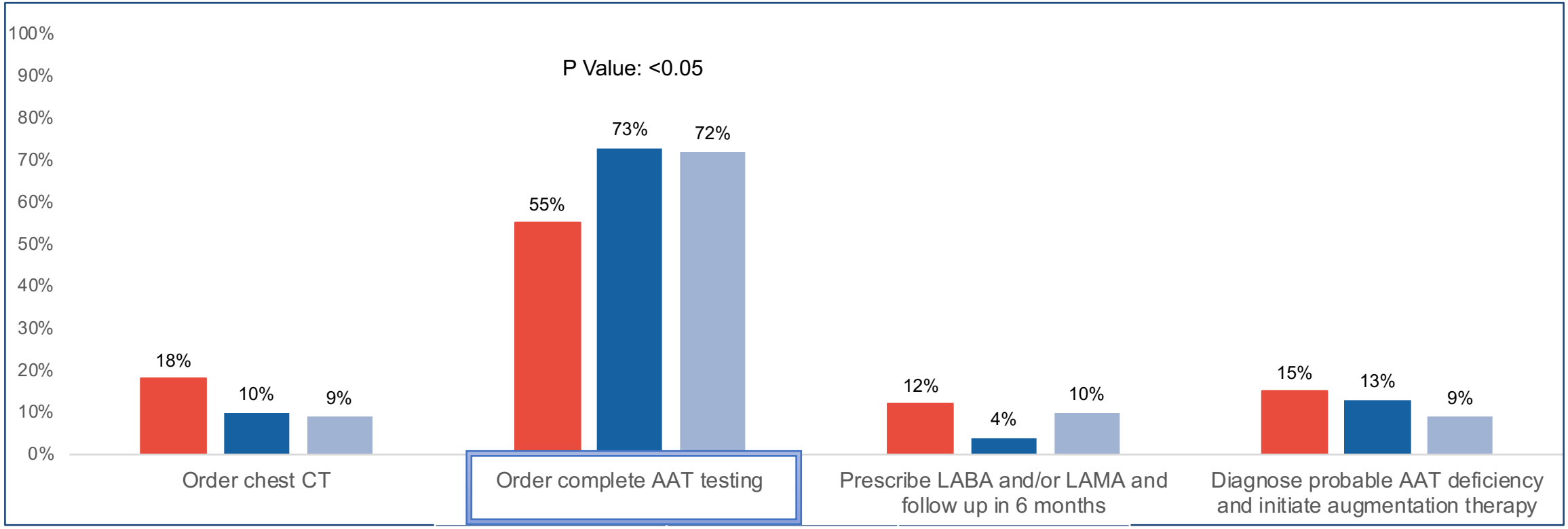
Pre-Post Change	**144%**
Pre-PCA Change	**172%**

Competence Assessment

A 62-y/o woman presents with progressive dyspnea and productive cough. She has no smoking history. Workup identifies FEV1/FVC 0.50 and FEV1 40% predicted. Chest X-ray shows mild emphysema with basilar predominance. Other findings are WNL.

Based on this information, what might be an appropriate next step?

(Learning Objective 2, 3, and 4)



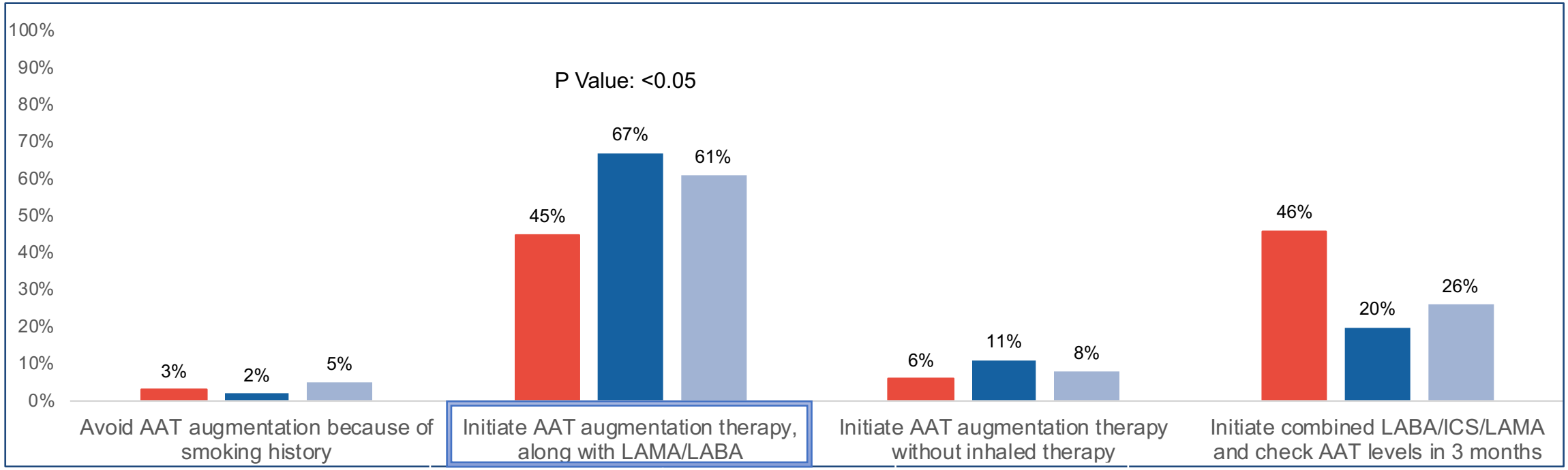
Pre: 407 Post: 415 PCA: 196

Pre-Post Change	**33%**
Pre-PCA Change	**31%**

Competence Assessment

A 51-y/o man who presents with progressive significant dyspnea is diagnosed with COPD on workup. He had one exacerbation and no hospitalizations. FEV1/FVC is 0.60 and FEV1 is 45% predicted. Testing for AAT deficiency identifies ZZ genotype and low serum AAT levels (7μM). He is a former smoker (10 pack-years, quit 20 years ago). Based on this information, what might be an appropriate next step?

(Learning Objective 2, and 3)



Pre: 390 Post: 386 PCA: 196

Pre-Post Change	**49%**
Pre-PCA Change	**36%**

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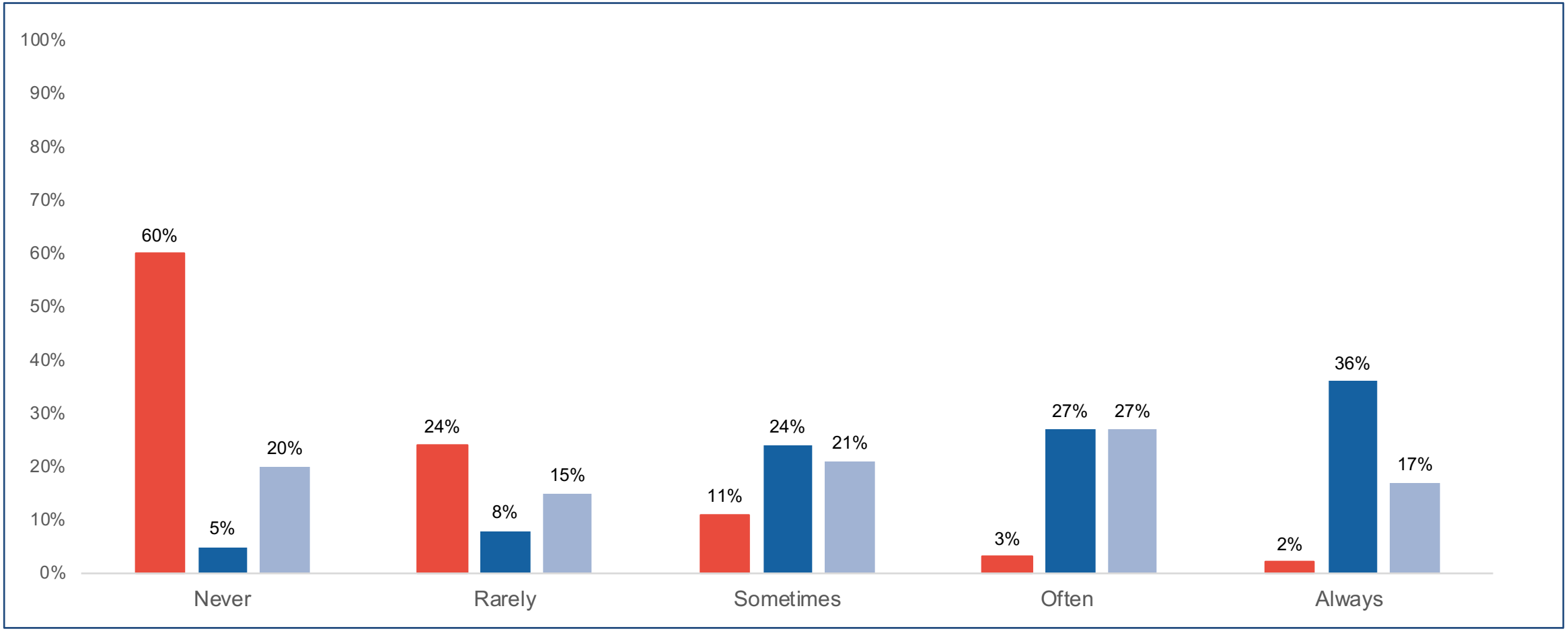
How often do you/will you order one-time AAT testing for your patients with COPD?

(Learning Objective 1, and 2)

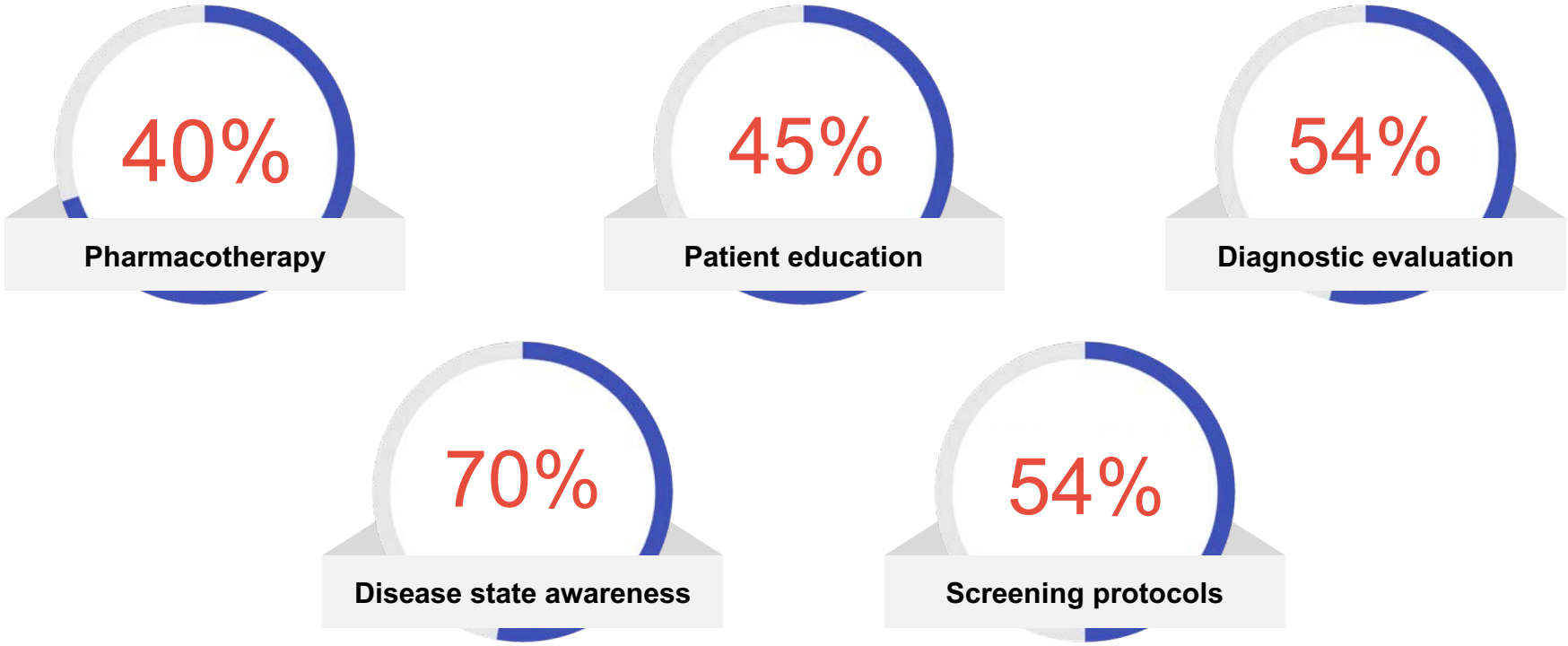


Pre: 439 Post: 422 PCA: 196

(4-week Post Assessment)

Please select the specific areas of *skills, or practice behaviors*, you have improved regarding the screening, diagnosis and treatment of AATD since this CME activity. (Select all that apply.)

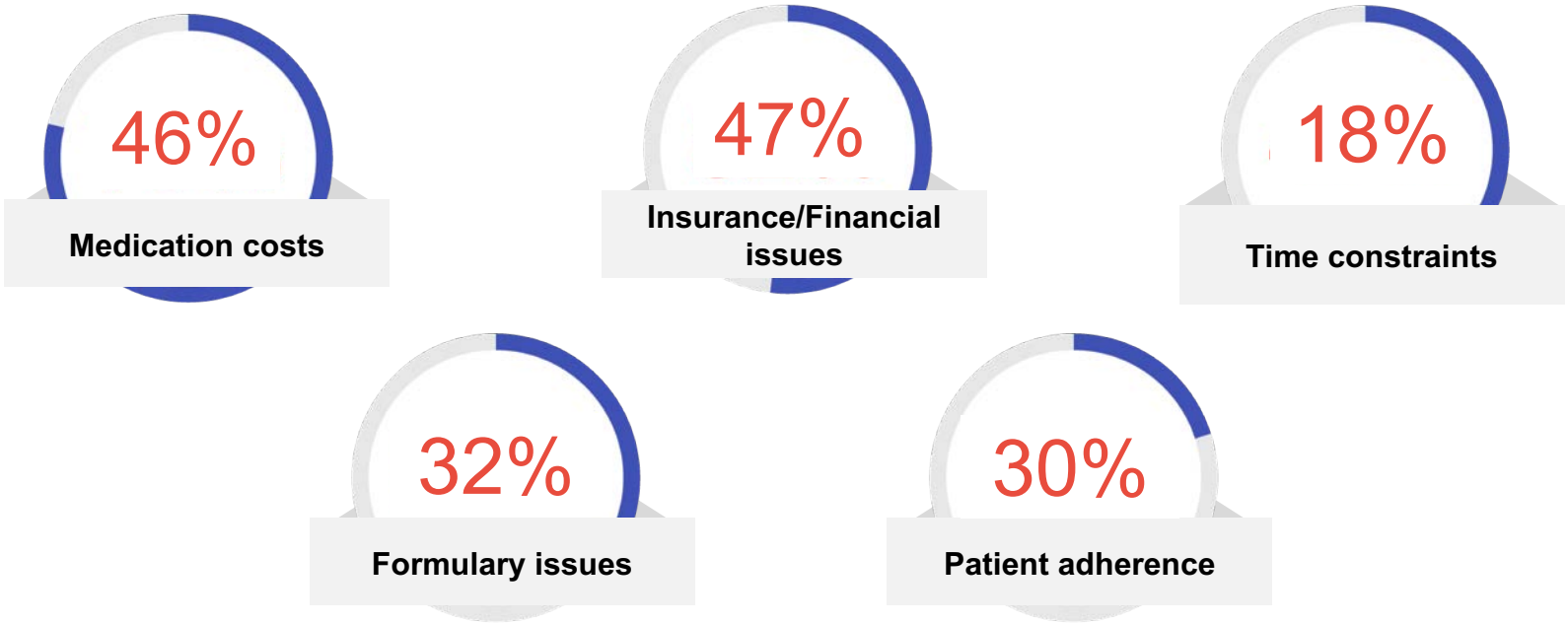
N=196



(4-week Post Assessment)

What specific *barriers* have you encountered that may have prevented you from successfully implementing screening, diagnosis and treatment of AATD strategies for patients with PAH since this CME activity? (Select all that apply)

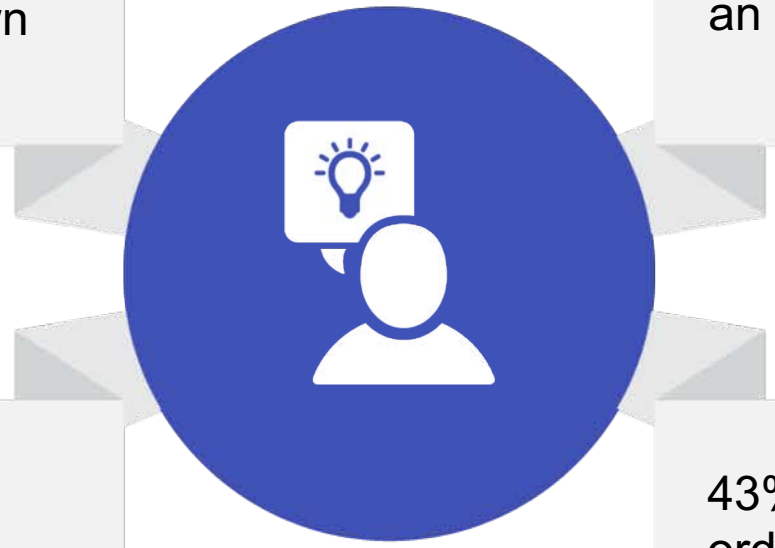
N=196



Participant Educational Gains

92% increased recognition of the mechanism by which AATD contributes to lung tissue breakdown

322% increased awareness of the AATD genotype most associated with an increased risk of COPD



144% increased recognition of the need to screen all patients with COPD for AAT Deficiency

43% increased competence in ordering appropriate quantitative and qualitative AAT tests for a patient with symptomatic COPD

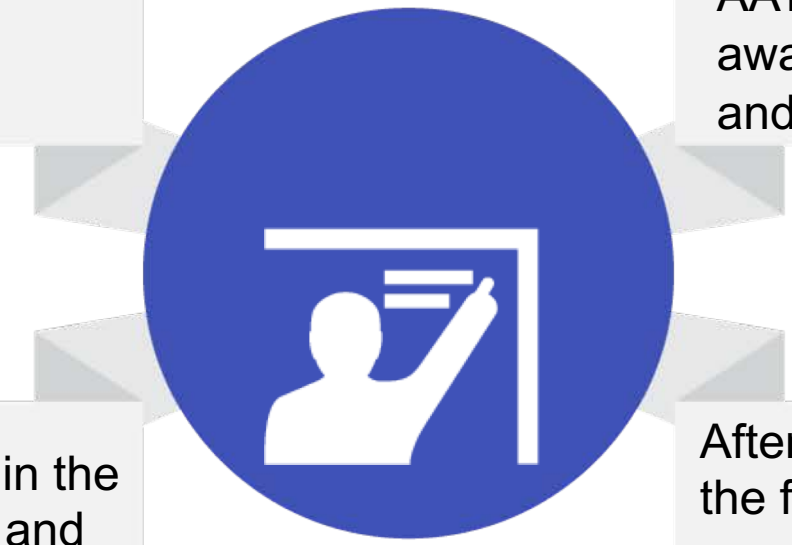
Persistent Educational Gaps After 4 Weeks

- Pathophysiology of AAT Deficiency
- Genetic phenotyping in AATD and its impact on risk for COPD
- AATD screening strategies
- Criteria for initiation of AAT augmentation therapy

Key Take-home Points

49% increased competence in recognizing appropriate patients for AAT augmentation therapy

After 4 weeks, participants reported the following improved skills regarding the screening, diagnosis and treatment of AATD: 70% disease state awareness,54% diagnostic evaluation, and 54% screening protocols



Significantly increased confidence in the ability to integrate the assessment and management of AATD into the care of patients with COPD

After 4 weeks, participants reported the following barriers regarding the screening, diagnosis and treatment of AATD: 47% insurance/financial issues, 46% medication costs, and 32% formulary issues

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