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The pulmonary rehabilitation program

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Pulmonary rehabilitation is new in the diversity of respiratory therapy. What is pulmonary rehabilitation and how does it work? Why are outcomes so important? These are questions we know the answers to. Most importantly what sets the reactive respiratory therapist into the realm of pulmonary rehabilitation/ prophylactics? We are advancing the face of the respiratory field; from the hospitalist to the critical end stage patient. Why we as practitioners have to advance the education of pulmonary rehabilitation in educational institutions for only 2% of all institution show the dynamics of pulmonary rehabilitation and are sent as practitioners to successfully make it in the world of pulmonary rehabilitation. I work at one of the top facilities for pulmonary rehabilitation. I have successfully made a starting program from people to try and make a program work, to making it one of the best in the field. I will discuss what we need to do, to take this to the next level. To set ourselves as the heart of the cure of COPD and restrictive lung disease. I have increased lung efficiently dramatically but it takes motivation from therapist. Adding other diversity's to work as a team includes physical therapy, to make the outcome numbers and prognoses a reality. I have increased an average of 70% of my patients FEV1% by more than 42%. Reversing COPD diagnosis from stage 4 to stage 1 to normal spirometry. To walking from admission 20 feet on a 6 MW because of work of breathing problems and muscle disease or fatigue to an average of 425 Foot increase in someone's 6MW. The numbers are validated. People want to know how we do it. Doctors call me all the time to ask how you have increased my patient's quality of life so dramatically. I understood that I must speak out about the autonomy of this diversity to my fellow therapists. We are now making a huge difference in the realm of respiratory therapy. I have gone to most of the hospitals in Maryland and find out how we are lacking in education required to keep patients home. A total of 98% of my patients say they have never received so much education about how to manage WOB, Medication administration, proper deposition of medication, and importance of exercise, staying prophylactic and how to, and more.

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Beta-nicotinamide adenine dinucleotide (β-NAD) reverses the LPS-induced endothelial barrier dysfunction in sickle cell mice

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Background: Recent studies have demonstrated that sickle cell disease (SCD) is characterized by weakening of the endothelial barrier, which predisposes the lung to acute loss of barrier function, reminiscent of acute chest syndrome (ACS) with high morbidity and mortality. Our previous studies demonstrated the protection of β-NAD against LPS-induced EC barrier dysfunction in an acute lung injury. However, the potential protective role of β-NAD in ACS has not been explored. The purpose of the present study was to determine the protective effects of β-NAD against LPS-induced EC barrier dysfunction in freshly isolated EC from lungs SCD mice.

Methods: Freshly isolated endothelial cells from lungs of transgenic sickle cell mice were used in this study. The trans-endothelial electrical resistance (TER) was carried out using an electrical cell-substrate impedance sensing (ECIS) instrument. Immunofluorescence studies and Western immunoblotting were done according to standard laboratory protocol. All the reagents were obtained from Sigma unless otherwise stated. The β-NAD was purchased from Calbiochem.

Results: Our preliminary results suggest that EC isolated from sickle cell mice are more susceptible to LPS (100ng/ml) compared to their heterozygote littermates based on the TER analysis using ECIS. We observed a strikingly enhanced barrier function with β-NAD (100 μM) in EC from sickle cell mice and the heterozygotes. In addition, β-NAD significantly attenuates the LPS response as evidenced by attenuated actin stress fibers and VE-cadherin at the cell-cell junctions.

Conclusions: Our data suggests that EC from sickle cell mice are more susceptible to LPS and β-NAD reverses the LPS- induced EC barrier dysfunction.

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