

## Investigating the Molecular Causes of Mild In-flight Hypoxia

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Received date: August 13, 2016; Accepted date: August 14, 2016; Published date: August 22, 2016

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### Editorial

In-flight hypoxia is a clinically and scientifically well-known problem when travelling in an airplane. Usually cabin altitude (i.e., the altitude corresponding to the cabin pressure) is equivalent to 7,000 ft or 2,400 m in many commercial aircrafts. In a healthy travelling passenger, exposure to this altitude results in a slightly decreased oxygen saturation (SpO<sub>2</sub>) of approximately 90-94%. From a clinical point of view, reduced cabin pressure results both in reduced arterial oxygen partial pressure (paO<sub>2</sub>) and oxygen saturation (SpO<sub>2</sub>) and can sometimes be the cause of in-flight medical emergencies (IFME) [1-3].

However, when having pre-existing diseases, resulting oxygenation can be lower and cause significant emergencies. Especially patients with pre-existing cardiac or pulmonary diseases are at a high risk of developing critical problems during flight [2]. Most common in-flight medical emergencies are syncope or presyncope (37.4%), respiratory symptoms (12.1%), nausea, or vomiting (9.5%) and cardiac symptoms (7.7%) [1]. At least cardiac or respiratory problems can be aggravated by reduced surrounding pressure and reduced SpO<sub>2</sub>. Since the partial pressure of oxygen is lower in a pressurized aircraft than at sea level, supplemental oxygen can be helpful in case of emergency [1].

On the other hand, it is well known that severe hypoxia leads to activation of different signalling cascades and pathways around the hypoxia-inducible factor (HIF) [4]. HIF regulates the expression of genes involved in angiogenesis, cellular energy metabolism, and cell survival and is active also during cancer development [5]. Genomic studies recently identified several genes that underlie high-altitude adaptive phenotypes, many of which are central components of the Hypoxia Inducible Factor (HIF) pathway [6].

However, very little is known on mild intermittent hypoxia. Whereas pathologies and clinical causes leading to emergencies are well-known today, molecular processes induced or inhibited by mild hypoxia in an airliner cabin are nearly unknown and not investigated

properly. To understand altered molecular pathways and signalling cascades during mild in-flight hypoxia, future research should focus on this topic and should use broad and unspecific methods to identify alterations in protein expression. Besides already known proteins, many yet unknown proteins may also be associated with cellular regulation processes during and after mild hypobaric hypoxia.

Today, proteomics using 2D-gel electrophoresis (2-DIGE), mass spectrometry (MALDI-TOF), and statistical bioinformatic methods are most suited to identify alterations in (cellular) protein expression and could give detailed insight into signalling pathways and affected cascades.

To our knowledge, no study yet analysed serum by proteomics of travelling passengers. Therefore, future research should use these methods to shed light into the dark of mild in-flight hypoxia and in-flight medical emergencies.

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