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# Link Between Ox-Stress and Lung Carcinoma

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## Perspective

Cancer is a group of various diseases with lowest survival rate in human population. Process of cancerogenesis is complex and include malignant transformations formed by mutations of genes caused by various cancerogens, tumor formation and contact inhibition, followed by spreading of malignant cels-metastasis, in blood stream, followed by death. Lung cancer survival rate after diagnosis is between 15 and 20%. Disease is asymptomatic, and deathly. In future prospect study/observation, we observe parafine samples of tumor, aortas, and tissue samples from patients that have been diagnosed with lung carcinoma, and pathological samples have been collected post-mortem. Lifestyle habits, such as diet, physical activity, smoking, drinking habits, and diet influence lifespan and death caused by cancer. Lung carcinoma is a common disease in a various human population, including population of active smokers. Tobacco smoke is recognised carcinogen and cause of death, not only because it contains various toxic compounds, nitrosamines and aromatical benzene rings compounds- 40 and more, but from the fact that reactive oxygen species that are part of tobacco smoke have devastating effect on cell membranes of myocytes and membranes of major arteries. It is a bold statement that cancerogenesis has in it occurrence besides gene mutations in Pten gen -tumor suppressor and Akt-PKB kinase genic pathway activation, same step-by step etiology as a atherosclerotic plaque progression, because there are many genes and genetic pathways included in human lung cancerogenesis and what heart failure-stroke human death and death caused by lung carcinoma have in common is atherosclerotic plaque progression and relapse. This statement comes from mechanistic occurrence of lung carcinoma, that as a final cause has relapse of lungs, caused by hypoxy. Trigger of death is physical blockade of air pathways that lead to death. It is needed to examine post-mortem samples of aortas of many patients died from Lung cancer. Oxidized LDL-has same effect on every aspect of CVS functional characteristic. This event has same background like plaque formation event, but the mechanistic background is controlled with key genes known like tumor/suppressors. We can observe here interplay of genetic pathways controlled by suppressors that influence tumor pathogenesis and progression and metabolic pathways controlled by interplay of immune system/adipose tissue, and these events are possible parallel, as a result of immune reaction of organisms' depletion.

Inactivation on PTEN gene by cancer forming mutations leads, as a sporadic event to formation of cholesterol esters, and these esters are incorporated in LDL particles. LDL particles are as a consequence of interaction with ROS (reactive oxygen species), oxidised and they take part in occurrence of atherosclerotic plaque formation in adipose tissue that is a part of pulmonary airways. Plaque formation is a multiple step process, that eventually leads to fibrosis and rupture of plaque, and lung relapse. This event takes place simultaneously as carcinogenesis, in which genetic pathways are common cause. This multiple step process complements cancerogenesis. In order to investigate lung cancer death occurrence, it is needed to do further research, complementary Elisa tests to determine Ox-LDL levels, and postmortem investigation on aortas of patients, with TEM. So, the possible order of events in lung cancerogenesis is:

1. PTEN tumor suppressor gene mutation.
2. Negative regulation of Akt kinase-PKB genetic pathway.
3. Accumulation of cholesterol.
4. Esterification of cholesterol esters.
5. Low grade inflammation.
6. Formation of Low-density lipoprotein particles.
7. Oxidation of LDL particles with Reactive oxidative species.
8. OX-LDL formation.
9. Accomulation of OX-LDL in adipocytes found in fat tissue of lungs.
10. Formation of plaque.
11. Plaque fibrosis.
12. Rupture of plaque.

Future prospects include further investigation of genomic libraries of genes in SCLC and non -SCLC and alternative ways of genetic regulation in different gene pathways and metabolic pathways also. Parrallel observation of TEM samples of pulmonary valves with tumor parafine samples will give a complete picture on the event of pathogenesis of lung carcinoma. TEM microscopy and genetic network analysis should be analyzed too.

Samples of tumor should be analyzed with DNA sequencing techniques, and OX/LDL samples and LDL status of patients should be analyzed with Kayman Ox/ldl species status protocol/kit. TEM microscopy would give a complete picture of lung tissue fibrosis. Western blot analyses should give complete picture on apoptosis/bcl diablo protein that is present in parafine tissue samples as a result of apoptotic death of pneumocytes.

What we can expect in future is a concrete evidence of pulmonary tissue fibrosis, Low/grade inflammation, disrupted Ox- LDL/status of samples from patients diagnosed with Lung Carcinoma as a possible cause of death in patients diagnosed with lung carcinoma. Positive Pten gene mutations, and consequent ptene gene inhibition, and simultaneously occurrence of positive regulation of ptene gene via oxidative LDL particles are common causes that are included in pathogenesis, and genetic regulation og genes that are common for lung cancer occurence. There is need for further investigation of ox-ldl status of lung tissue post mortem samples via ox-ldl Elisa test kit.

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