Answers correct on the whole have been directly reported (the name in brackets refers to who logged in, so it indicates the whole working group). Questions 1-3 have been addressed more or less correctly, at least by one group. By contrast, problems are clearly evident for questions 4 and 5.

QUESTION 1

Cicirò. The three main differences are the vascular changes, cell infiltrate presence and stromal changes. In the acute inflammation, the vascular changes are determined by a vasodilation, which increases permeability of the capillaries. Moreover, cell infiltration no infiltration during acute inflammation!! occurs, mainly due to the presence of granulocytes at the inflammation site. Alltogether these changes induce the formation of exudate, composed of salts, proteins and leukocytes, which exit from the vessels (diapedesis). The stromal changes in the acute inflammation are only marginal. In the chronic inflammation, marginal changes occur at the vascular level, the cell infiltrates are represented by macrophages, which proliferate at the inflammation site. Moreover, there is cell proliferation and fibrosis at the stromal level. The two conditions differ also in their duration: the acute inflammation has a rapid onset and resolution (few days-weeks), while the chronic inflammation is persistent and can evolve either from an acute inflammation or it can be chronic ab initio.

Two factors are lacking in this answer: 1) chemical mediators of (acute) inflammation; 2) acute phase reaction

QUESTION 2

Cicirò. Pro-inflammatory cytokines stimulate the immune system, downregulating the humoral response. Cytokines are the signal that triggers relocalization of cells of the inflammatory response from the circulation to the inflammatory sites, in the angioflogosis. This is due to an alteration of the cytokines homeostatic equilibrium. Proinflammatory cytokines are crucial also in the inflammaging process: there is a low and steady increase of pro-inflammation cytokines, that over time becomes chronic and produces damages. Inflammaging occurs both during aging and pathological situations such as Trisomia 21. In atherosclerosis initiation, LDL and oxidized LDL are picked up in the subendothelial layer by macrophages, which become activated and produce pro-inflammatory cytokines. Cytokines allow the recruitment of monocytes from circulation and stimulate lipolysis.

Also involved in the pathogenesis of amyloidosis (SAA hyperproduction by the liver). Contribute to neurodegeneration in protein aggregate-related diseases.

QUESTION 3

Ghibaudi. When there's a incorrect protein folding potential causes of altered protein folding are???, proteins can aggregate in cells or interstitial space, leading to dysfunction of the implicated tissues. For example in the AD the Amyloid Precursors Protein is processed in beta-sheet structure (instead of alpha-helix) leading to an accumulation in the interstitial space. Another example is the PD in which the alpha-Synuclein aggregate in the glial cytoplasm (instead being in random coil structure). Last, in the HD the Huntingtin is in a insoluble form in the nucleus (instead in trinucleotide repeats form) excess number of trinucleotide repeats is the cause of misfolding....

QUESTION 4

Cell degenerative processes take place when a nonlethal injury occurs, resulting in abnormalities of biochemical functions, structural changes, or a combination of both. Degeneration can be reversible but may progress to cell death if the damaging stimulus is persistent . Examples are: liver steatosis, hemochromatosis, enzymatic deficiencies,

neurodegenerations. I would not include aging among degeneration, that occurs only eventually.

QUESTION 5

Atherosclerosis affects large- and medium-sized arteries. Is characterized by atherosclerotic plaques showing a necrotic core, calcified regions, accumulated modified lipids, smooth muscle cells, endothelial cells, leukocytes, and foam cells. Modified LDL are the most important risk factor for atherosclerosis, inducing the formation of foam cells, the initiation of endothelial activation, and the expression of adhesion molecules that accelerate leukocyte homing to the site of lesion. Inflammatory cells, in particular are recruited and retained in the early atherosclerotic lesions, contributing to evolution of the plaque. Here they differentiate, activate and produce both pro- and anti-inflammatory cytokines. Indeed, both M1 and M2 macrophages are found in atherosclerotic lesions. Such a chronic inflammatory response also appear to have an immune component (anti-modified LDL antibodies). All phases of atherosclerosis (fatty streak, intermediate lesion, atheroma, fibroatheroma, complicated lesion) are regulated by inflammatory mechanisms that provide overlapping networks of pathways involved in the regulation of immune cell functions, activation of endothelium, and alteration of metabolic parameters. In addition to modified LDL, also ROS act as pro-inflammatory factors involved in the pathogenesis of atherosclerosis.