## AC2.15 PHENOTYPIC CONSEQUENCES OF MUTATION ACCUMULATIONS ON DAPHNIA MITOCHONDRIA

**FRIDAY 6 JULY, 2018** **17:15**

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Mitochondria are essential organelles that generate ATP necessary to sustain life via the oxidative phosphorylation. The mitochondrial genome is known to be sensitive to the accumulation of deleterious mutations due to its highly mutagenic environment. Yet we lack a complete understanding of the impact of spontaneous DNA mutations on heritable damages within the germ line and how these affect mitochondrial functions. Exposure to mutagenic environmental contaminants can accelerate mutation accumulation. However, little is known about how mutagenic compounds affect the scope and extent of the phenotypic effects of spontaneous mutations on the mitochondria. The objective of our work was to assess the effects of mutation accumulations (MA) on mitochondrial traits in *Daphnia pulex* MA lines raised under mild copper and benign conditions. Ten *Daphnia* MA lines propagated for 123 generations under copper, 10 *Daphnia* lines propagated for the same number of generations under benign conditions, as well as 10 *Daphnia* individuals from a competitive (non MA) control were raised for three generations under standard conditions at 18°C prior to the measurements. Mitochondrial respiration rates at 27°C of both MA lines treatments were similarly reduced by 10% compared to controls. MA lines had a higher relative mtDNA copy number than controls at 18°C but did not differ from the control lines at 27°C. Our results are the first to empirically demonstrate the alleged sensitivity of mitochondria to mutational load and point at modulation of mtDNA content as an important mitigation mechanism of mutational impacts.

## AC2.16 OXIDATIVE STRESS IN COMMON CARP (*CYPRINUS CARPIO*) EXPOSED TO A MIXTURE OF METAL POLLUTANTS

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Aquatic environments are subjected to numerous anthropogenic stressors that are much more diverse and variable than those usually tested in laboratory conditions. Even if previous research indicated that strong interactive effects are observed in metal mixture scenarios that are not expected based on single metal exposures, the assessment of mixture stress remains a challenge. In order to investigate the impact of a mixture of metal pollutants (Cu, Zn and Cd) exposure on free radical processes and antioxidant defences in a model species, we exposed common carp, *Cyprinus carpio*, to concentrations representing 10% of their 96 h $LC_{50}$ ($Cu = 4.8 \mu g.L^{-1}$; $Cd = 2.9 \mu g.L^{-1}$ and $Zn = 206.8 \mu g.L^{-1}$) for 1 day, 3 days and one week. Indices of oxidative stress (malondialdehydes and hydrogen peroxide), genomic expression and activities of enzymes related to antioxidant defences (catalase, superoxide dismutase, glutathione peroxidase, glutathione reductase, glutathione S-transferase, xanthine oxidase) were analysed in liver to allow us to better understand the impact of mixture stress at different levels of oxidative processes and to apprehend their time course during an acute sublethal exposure in *C. carpio*.

## AC2.17 THE ENIGMA VARIATIONS: GENESIS AND EVOLUTION OF A PRIMORDIAL STRESS RESPONSE

**FRIDAY 6 JULY, 2018** **17:45**

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Survival of eukaryotic cells depends on their ability to integrate, process and respond to a multitude of stress signals emanating from endogenous and exogenous sources. As both a major intracellular source of stress signals and the main providers of the energy needed to respond to them, mitochondria are integral to this process, for which rapid and efficient communication between mitochondrial and the nuclear genomes is also vital. Regulated release of mitochondrial superoxide via voltage dependent anion channels (VDAC) serves this function at the most basal level. The Hepadnaviruses (Hepatitis B viruses; HBVs) comprise a family of small, ancient viruses that have infected all classes of vertebrates. Their recently-discovered evolutionary precursors, which infect fish, have been named Nakednaviruses. These ancient, enigmatic viruses have co-evolved with their hosts for at least 400 million years, during which time the tetrapod vertebrates have evolved a variety of respiratory innovations that have enabled metabolically demanding activities including endothermy, rapid locomotion and flight. HBVs replicate in the cytoplasm in close association with mitochondria. Replication is inducible by a variety of abiotic stressors. Infections are usually asymptomatic and self-limiting but may become chronic. Pathogenic effects of chronic infections are delayed and limited to mammals, the newest hosts. The evolutionary success and persistence of the Nakednavirus-Hepadnavirus lineage implies that infection with these viruses confers a survival advantage, which several lines of evidence identify as the ability to sense and respond more rapidly to stress. These data support the concept that viruses have been both drivers and passengers during evolution.