target was developed. Confocal microscopy studies confirmed the localization of the probe in the mitochondria. We aim to use both probes to study the redox response of the cell to hypoxia and reoxygenation in both cellular compartments.

NC5
Performance evaluation tests of an auto-fluorescence observation system for non-invasive biological measurements

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Conventional oxidative stress markers, such as lipid-derivers free radicals have been studied to cause damages to cell membranes, proteins and other biomolecules. Decompositions of lipid hydroperoxides are known to release excited triplet states of biomolecules composed with carbonyl groups. The previous study suggested that ultra-weak photon emissions of the carbonyl groups composed with various wave lengths. On the other hand, more detailed investigations on *in vivo* redox status are needed to elucidate the mechanisms contributing to damage caused by stress. Recently, glycative stress related to accumulation of advanced glycation products (AGEs) might be important to monitor in the redox status because AGEs have been used as biomarkers for non-invasive measurement techniques. However, a skin condition marked by an overgrowth of layers of horny skin and distance from skin surface to high moisture stage layer might affect measuring auto-fluorescence *in vivo*. To elucidate mechanisms of photon emissions from human fingers including fluorescent oxidation products, we executed performance tests of an auto-fluorescence observation system. Our findings provide that auto-fluorescence intensities of human fingers changed during COVID-19 related crisis. The auto-fluorescence observation system for non-invasive biological measurements might be a lifestyle habit improvement support device.

NC6
Redox control of the transcriptional circadian rhythmicity by SOD2

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Endogenous control of circadian rhythmicity is mainly governed by clock genes at the genomic level. Furthermore, both clock genes and redox regulation also modulate cell metabolism, thus showing a closed and strong interconnection between both regulatory pathways.
Mitochondrial superoxide dismutase (SOD2/MnSOD) is a key antioxidant and redox-regulating enzyme, depurating superoxide anion mainly generated in the electron transport chain. The increase in the activity of this enzyme has been postulated as a major event during the antioxidant defense response; on the other hand, loss of SOD2 function constitutes a significant oxidative stress factor. With this premise, the work presented here used two transgenic murine models for SOD2 characterized by either a reduced function in hemizygous ($SOD2^{+/-}$) and by a SOD2 overexpressing mice ($SOD2^{+/++}$) in order to study the role of oxidative stress and redox regulation on the physiological circadian transcriptional rhythmicity.
Interestingly, both transgenic models, $SOD2^{+/-}$ and $SOD2^{+/++}$, displayed a similar transcriptional profile which differed to WT, sharing some transcriptional changes regarding cytokeratin, calcium binding, and kallikrein serine proteases. Both genotypes presented a significant loss of rhythmic metabolic transcripts, when compared to WT, resulting in a lower number of rhythmic transcripts. However, the changes in circadian rhythmic transcripts in heterozygous $SOD2^{+/-}$ mice were greater than those observed in $SOD2^{+/++}$ overexpressing mice. Therefore, a change in the expression pattern of ARNTL/BMAL1, the transcriptional decrease in NADH dehydrogenase or the pro-inflammatory transcript profile were among the major features observed in $SOD2^{+/-}$ mice.
The study points that deregulation of SOD2 expression accounts for important changes in the metabolic transcriptional machinery, especially in a situation of oxidative stress, in which clock genes and the metabolic activity appear to be highly compromised.
Keywords**:** Superoxide dismutase 2, Circadian rhythm, Metabolism, ARNTL, Electron transport chain.
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