IOC61. *Mitochondrial Physiology Network* 16.01: 1-12 (2011)

61st International Workshop on HRR

2011 April 26 – May 01 Schröcken, Vorarlberg, Austria

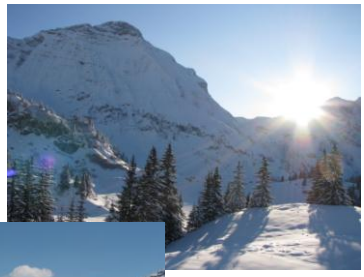
The **61st Workshop on High-Resolution Respirometry (HRR)** is the 25th international Oxygraph Course held in Schröcken since 1988. The workshop includes experiments with biological samples, providing a practical overview of the **Oxygraph-2k**, with integrated on-line analysis by **DatLab 4.3**, applications of the **TIP2k**, and perspectives of HRR in mitochondrial physiology. Parallel to the introductory workshop, a group of advanced users will focus on **O2k-MultiSensor** applications of the TPP+ electrode (**ISE**) for measurement of mt-membrane potential and the application of ISE for measurement of Ca^{2+}, and acidification rate (pH).

An international team of experienced tutors guide small working groups step-by-step through the approach of HRR. Five Oxygraph-2k (10 chambers) are available for do-it-yourself applications of both hardware and software. Combined with an introduction and demo experiment, it is best to put the O2k into action yourself.

Lunch breaks provide an opportunity for relaxing walks and talks, to enjoy the refreshing scenery of the secluded alpine environment, or use the spare time for specific tutorials. With DatLab 4.3 we accomplish data analysis on-line during the experiment, providing final results and their graphical presentation by the end of an experimental run. Thus we gain sufficient time to see the Titration-Injection microPump TIP2k with feedback-control in action and practice its simple and automatic operation.

Tutors

Erich Gnaiger, AT
Mario Fasching, AT
Dominik Pesta, AT
Suzana Sumbalová, SK/AT



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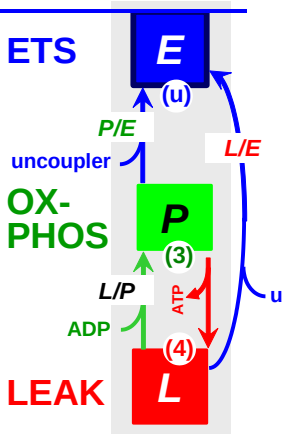
Programme IOC61

Day 1 Tuesday, April 26



15:00 **Arrival in Bregenz:** Meeting point Bregenz train station at 3:00 pm; 1.1 hour bus drive to Schröcken and Hochtannberg (Salober). Transfer to Hotel Körbersee.

18:30 Welcome reception at Hotel Körbersee.

19:00 *Dinner*

21:00 **Introductions of participants and their research interests.**

Day 2 Wednesday, April 27

08:30 - 9:00 **Erich Gnaiger:** General introduction.

09:00 - 9:30 **Mario Fasching:** Introduction to measurement of mitochondrial membrane potential with a TPP-electrode.

09:30 - 10:00

Intro-Group	TPP-Group
Erich Gnaiger: Principles of HRR - from switching on the Oxygraph-2k to the experimental result - with a little help from a friend: **the O2k-Manual.**	**Mario Fasching:** Planning and performing a TPP Experiment: Introduction. **MiPNet14.05**

10:00 *Coffee break*

10:30 - 12:30

The O2k-System: Introduction and oxygen calibration of the polarographic oxygen sensors (OROBoPOS). **Hands-on:** Oxygen calibration with DatLab. **MiPNet12.08**	**Hands-on:** Assembly and maintenance of TPP and reference electrodes; Set up of the instrument with TPP and reference electrodes. **MiPNet15.03**

12:30 - 14:00 *Lunch break*

14:00 - 14:30 **Erich Gnaiger:** DatLab 4.3 – An overview. **MiPNet12.07**

14:30- 16:00

Intro-Group	TPP-Group
14:30 - 15:00 Erich Gnaiger: Introduction: Instrumental background **MiPNet14.06**	**Hands-on:** Instrumental background oxygen flux in the presence of the TPP electrode.

15:00 – 16:30 Hands-on (3 groups): Instrumental background test for experiments with cells and isolated mitochondria, from air saturation to zero oxygen concentration or for permeabilized muscle fibres, in the high-oxygen range of 500 - 200 µM. A. Instrumental background test, **with automatic TIP2k** titration protocol. B. Instrumental background test, **with manual titrations**.	**Hands-on:** *continued*

16:30 *Coffee break*

17:00 – 18:30

Hands-on: *continued* Background analysis and summary.	**Hands-on:** *continued*

19:00 *Dinner*

21:00 - 21:30 **Hot topics: MiPNet Session 1 (2 x 10+5 min)**

Day 3 Thursday, April 28

08:30 - 10:00

Erich Gnaiger: Experimental protocols Talk and parallel hands-on with DatLab demo files: a) Basic protocol with isolated mitochondria: LEAK, OXPHOS, ETS, ROX. **MiPNet12.11** b) Phosphorylation Control Protocol (PCP) with intact cells: ROUTINE, LEAK, ETS, ROX. **MiPNet08.09** **Hands-on:** Start air calibration	**Hands-on:** TPP calibration and extending the TPP method for the determination of mitochondrial membrane potential beyond isolated mitochondria: TPP calibration and experiment with biological sample.

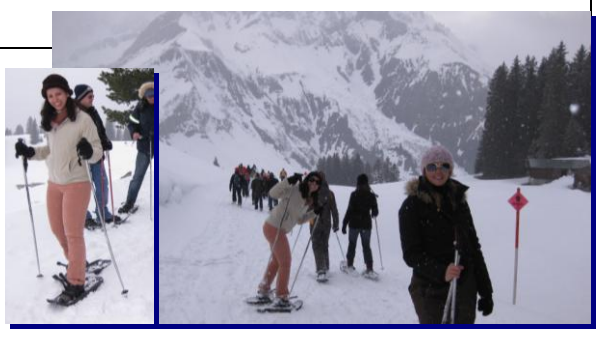
10:00 *Coffee break*

10:30 - 12:00

Hands-on: Experiment with biological sample in the Oxygraph-2k: SUIT protocol and on-line DatLab analysis.	**Hands-on:** *continued*

12:00 - 16:00 *Lunch break*

16:00 *Coffee, tea*



16:30 – 19:00

Intro-Group	TPP-Group
Hands on: *continued* DatLab analysis and discussion of results. **Design of SUIT-protocols**: Questions from participants and discussion.	**Mario Fasching:** From the TPP+ signal to mitochondrial membrane potential - Guide through the Excel templates. **Hands-on:** Data evaluation.

19:00 *Dinner*

20:30 – 21:30

Dominik Pesta: Permeabilized muscle fibres – preparation and HRR.	**Hands-on:** *continued* and discussion.

Day 4 Friday, April 29

08:30 – 10:30

Parallel group sessions: Hands-on with the Oxygraph-2k

	Setup	POS Service	Dat Lab Analysis
08:30 – 09:30	Gr.1	Gr.2	Gr.3
09:30 – 10:30	Gr.3	Gr.1	Gr.2

Hands-on: TPP chemical background

10:30 *Coffee break*

11:00 – 12:00

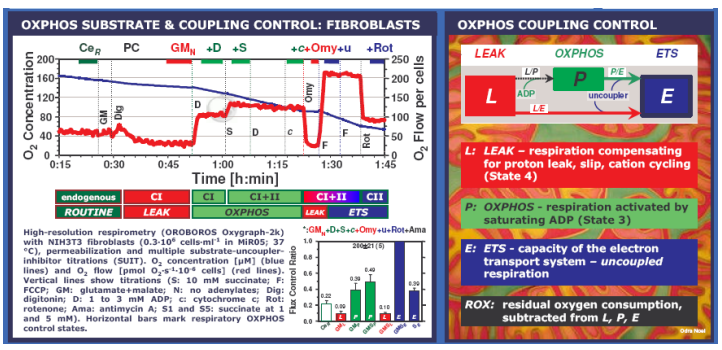
Parallel group sessions: *continued*

	Setup	POS Service	Dat Lab Analysis
11:00 – 12:00	Gr.2	Gr.3	Gr.1

Hands-on: Evaluation of data *continued*

12:00 - 16:00 *Lunch break - exercise*

16:00 *Coffee, tea*



16:30 – 17:30 Erich Gnaiger: MitoPathways: Respiratory States and Coupling Control Ratios. **MiPNet12.15**

17:30 – 18:30 Erich Gnaiger: MitoPathways: through Complexes I+II: Convergent Electron transfer at the Q-junction and Additive Effect of Substrate Combination. **MiPNet12.12**

19:00 *Dinner*

20:30 - 21:00 **Hot topics: MiPNet Session 2 (2 x 10+5 min)**

Day 5 Saturday, April 30

08:30 – 10:00 **Special interest groups - Parallel sessions**

A. **Hands-on Permeabilized fibres:** Fibre preparation and permeabilization; Respiration of permeabilized fibres.
B. **Hands-on with TPP-electrodes** and membrane potential of permeabilized fibres.
C. **Hands-on Repeats:** Intact cells experiment or Instrumental background test (in the high oxygen range), with/without TIP2k.
D. **Erich Gnaiger and Mario Fasching:** Oxygen kinetics - O2k-MultiSensor overview: NO, pH.

10:00 *Coffee break*

10:30 – 12:00 **Special interest groups:** *continued*

12:00 - 14:00 *Lunch break*

14:00 *Coffee, tea*

14:30 – 15:15 **Mario Fasching:** Introduction to trouble shooting.

15:15 – 17:15

Intro-Group	TPP-Group
Working groups: Elaborate answers to the 'Questions for the O2k-Course' Discussion of 'Answers'.	Analysis of TPP experiment from **Day 4**. Discussion of results and final discussion and conclusions for the TPP special interest group.

17:15 – 18:00 **Panel Discussion – Feedback IOC61**
Marelsson Sigurdur
Le Catherine
Angelin Alessia
Watala Cezary

18:30 *Dinner*

20:00 **Snowshoe walk** (rental of snowshoes) **to the Alpmuseum**: Guided tour and reception: € 15

Alpmuseum uf m Tannberg, Batzen www.alpmuseum.at

Day 6 Sunday, May 01

Early morning: Departure

MiPNet Abstracts–

Hot topics in Mitochondrial Physiology

Retrieved from: http://wiki.oroboros.at/index.php/IOC61_Abstracts

MiPNet 61.1. Nuskova H, Pecina P, Kovarova N, Dell'Agnello C, Zeviani M, Houstek J (2011) Cytochrome c oxidase with decreased H+/e− ratio in SURF1 knockout mice

Leigh syndrome is most frequently caused by mutations in SURF1 gene which encodes cytochrome c oxidase (COX) specific assembly factor. Our previous studies focused on fibroblasts from patients harbouring SURF1 mutations [1, 2]. To further characterize the mitochondrial energetics affected by SURF1 mutation, we used immortalized fibroblasts originated from SURF1 knockout (KO) mice (3).

The COX content was decreased to 58 % of the control, which was in accordance with 38% decline of COX activity measured spectrophotometrically. However, there was no change in the rate of endogenous respiration or in the rate of ascorbate+TMPD-dependent respiration of permeabilized cells. In contrast, mitochondrial membrane potential generated by COX achieved 92 % of maximal membrane potential in the control cells, but only 73 % in the KO cells. Furthermore using ascorbate and TMPD, the decrease of membrane potential at state 3 (ADP) compared to state 4 (oligomycin) was more profound in the KO fibroblasts. Therefore, the proton-pumping activity of COX was partially impaired unlike the electron-transporting activity, suggesting a decrease in the H+/e− ratio. Since the p50 value of the KO cells was approximately 2-fold increased in all metabolic states measured, the oxygen affinity of COX was also decreased.

Taken together, the KO cells from mice showed similar but milder functional manifestations of COX impairment than the cells of Surf1-deficient patients, which indicates that the Surf1 protein is not as essential for mouse as for human.

(1) Pecina P, Capkova M, Chowdhury SKR, Drahota Z, Dubot A, Vojtiskova A, Hansikova H, Houstkova H, Zeman J, Godinot C, Houstek J (2003) Functional alteration of cytochrome c oxidase by SURF1 mutations in Leigh syndrome. BBA 1639: 53-63.

(2) Pecina P, Gnaiger E, Zeman J, Pronicka E, Houstek J (2004) Decreased affinity for oxygen of cytochrome-c oxidase in Leigh syndrome caused by SURF1 mutations. Am. J. Physiol. Cell. Physiol. 287: C1384-C1388.

(3) Dell'Agnello C, Leo S, Agostino A, Szabadkai G, Tiveron C, Zulian A, Prelle A, Roubertoux P, Rizzuto R, Zeviani M (2007) Increased longevity and refractoriness to Ca2+-dependent neurodegeneration in Surf1 knockout mice. Hum. Mol. Gen. 16: 431-444.

MiPNet 61.2. Gabrielova E[1], Jaburek M[2], Gazak R[3], Vostalova J[1], Jezek J[2], Kren V[3], Modriansky M[1] (2011) Evaluation of an oxygen consumption in rat heart mitochondria treatment by polyphenol compounds

[1]Institute of Medical Chemistry and Biochemistry, Faculty of Medicine and Dentistry, Palacký University, Olomouc, Czech Republic ; [2]Institute of Physiology, Dept. 75, Academy of Sciences of the Czech Republic, Prague, Czech Republic; [3]Institute of Microbiology, Academy of Sciences of the Czech Republic, Prague, Czech Republic; eva.gabriel@email.cz

Reactive oxygen species (ROS) originating from mitochondria are perceived as a factor contributing to cell aging and means have been sought to attenuate ROS formation with the aim of extending the cell lifespan. Silybin and dehydrosilybin, two polyphenolic compounds, display a plethora of biological effects generally ascribed to their known antioxidant capacity.

Experiments evaluating oxygen consumption and membrane potential revealed that dehydrosilybin uncouples the respiration of isolated rat heart mitochondria albeit with a much lower potency than synthetic uncouplers. Furthermore, dehydrosilybin revealed a

very high potency in suppressing ROS formation in isolated rat heart mitochondria with IC50=0.15 µM.

We infer that the apparent uncoupler-like activity of dehydrosilybin is the basis of its ROS modulation effect in neonatal rat cardiomyocytes and leads us to propose a hypothesis on natural ischemia preconditioning by dietary polyphenols.

MiPNet 61.3. Marelsson S (2011) Mitochondrial dysfunction in children with unknown encephalopathy

Introduction: Mitochondrial disorders are extremely heterogeneous and can involve single tissue, such as the optic nerve to widespread pathologies including muscle disorders, peripheral neuropathies, encephalopathy, cardiomyopathies or complex multisystem disorders. The age at onset ranges from neonatal to adult life. Mitochondrial dysfunction is a relatively common disorder but the clinical and genetic variability makes it difficult to diagnose.

Our primary hypothesis is that disturbance in mitochondrial respiratory chain can be diagnosed with blood test and skin biopsy, by combining structural (Blue native page) and functional information, with high resolution respirometry of the respiratory chain in blood cells. This rapid diagnostic method can be used to diagnose the flora of undiagnosed and unknown encephalopathy in children today.

Methods: Our aim is to 1) Establish reference material for mitochondrial normal function in children through high resolution respirometry by diagnosing thrombocytes and fibroblasts. We also want to establish reference material for structural information with Blue Native PAGE (BNP) in fibroblasts. 2) We want to use these methods in children with known mitochondrial disease to confirm that our methods are useful. 3) We want to compare our methods to known methods today for diagnoses of mitochondrial disease (muscle biopsy). 4) We want to see the benefits of treatment by comparing results through BNP and respirometry before and after treatment. 5) We want to use these methods for diagnosis of unknown encephalopathy in children.

Results: We have started collecting reference material from children from 0-17 years old. We collect blood and skin biopsy from healthy children that are having a small operation at the University Hospital in Lund. Our aim is to collect reference material from 60 children in different age groups. We also collect blood and skin biopsy from 30 newborn babies from the umbilical cord. We have also done respirometry on children that have both suspected mitochondrial disease and children with known mitochondrial disease. The results are promising. We have also taken skin biopsy from these children but we do not know the outcome yet. We have also started using our methods to look at children with autism and other encephalopathy.

Conclusion: Mitochondrial dysfunction has been difficult to diagnose. Our methods give us the opportunity to diagnose mitochondrial dysfunction in unknown encephalopathy in children by a more rapid and simple way than before.

MiPNet 61.4. Magnifico MC[1], Arese M[1,2], Mastronicola D[3], Forte E[1,2], Giuffre A[3], Testa F[1], Sarti P[1,2] (2011) NO-signalling and cell bioenergetics

[1]Department of Biochemical Sciences, Sapienza University of Rome; [2]The second Faculty of Medicine, S. Andrea Hospital, Sapienza University of Rome; [3]CNR-Institute of Molecular Biology and Pathology, Rome.

Nitric oxide (NO) is a biological messenger which regulates several physiological responses including relaxation of smooth muscle, neurotransmission, inhibition of platelet aggregation, cell migration and mitochondrial respiration. In mammals NO is synthesized by three different gene-encoded NO synthase (NOS), the neuronal NOS (nNOS or NOS1), the inducible NOS (iNOS or NOS2), the endothelial NOS (eNOS or NOS3) and possibly a mitochondrial NOS. Made available exogenously or endogenously, NO reacts with heamoproteins such as guanylate cyclase, haemoglobin, myoglobin and cytochrome c oxidase (CcOX).

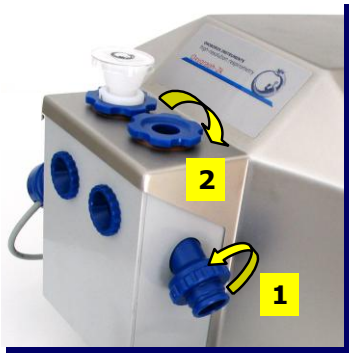
The NO-CcOX interaction is of particular interest, being rapid and reversible and leading to changes of the ATP synthesis (1). Inhibition may or may not occur in competition with O2, particularly depending on substrates availability (e-, O2) (2). Experimental evidence suggests that NO might be a physiological regulator of cell respiration turning to pathological under circumstances (3-4). Many effectors have been shown to control the enzymatic activity of the NOSs, thus the cell bioavailability of NO. We have focused our attention on the effects of compounds likely involved in the regulation of the level of NO endogenously produced in the cells, such as morphine, melatonin, hydrocortisone (5). Under a number of conditions we have measured the NOSs expression and tentatively correlated the observation to nitrate-nitrite accumulation and parameters of mitochondrial efficiency.

(1) Brown G.C. & Cooper C.E. (1994) Nanomolar concentrations of nitric oxide reversibly inhibit synaptosomal respiration by competing with oxygen at cytochrome oxidase. FEBS Lett., 356: 295-298.
(2) Sarti P. et al. (2000) Nitric oxide and cytochrome c oxidase: mechanisms of inhibition and NO degradation. Biochim. Biophis. Res. Com. 274:183.
(3) Sarti P. et al. (2003) Nitric oxide and cytochrome oxidase: reaction mechanisms from the enzyme to the cell. Free Radic. Biol. Med. 34(5):509-20.
(4) Mason M.G. et al. (2006) Nitric oxide inhibition of respiration involves both competitive (heme) and noncompetitive (copper) binding to cytochrome c oxidase. Proc. Natl. Acad. Sci. USA. 103: 708.
(5) Mastronicola D. et al. (2004) Morphine but not fentanyl and methadone affects mitochondrial membrane potential by inducing nitric oxide release in glioma cells. Cell. Mol. Life Sci. 61: 2991-7.

Questions for the O2k-Workshop

The **O2k-Manual** and **Protocols** provides answers to many of these questions [MiPNet numbers in the O2k-Compendium on the CD] – and you find more information on www.oroboros.at and wiki.oroboros.at

1. Oxygraph-2k assembly [MiPNet12.06]



1.1. What is the most important consideration for positioning the glass chamber during assembly of the O2k?
1.2. How do you detect an oxygen leak in the chamber?

2. Polarographic oxygen sensor (POS)

2.1. Why is it important to check the non-calibrated raw signal (voltage, after current-to-voltage conversion) of the polarographic oxygen sensor, and how can you quickly see the raw signal on-line?
2.2. The sensor voltage is 9.9 V. What should you do?
2.3. Why is it important to maintain an extremely constant temperature in and around the O2k-chamber?
2.4. Does the POS respond to oxygen concentration, cO2 [µmol·dm-3 = µM], or partial oxygen pressure p_{O2} [kPa]?

3. POS calibration [MiPNet12.08]

3.1. How many calibration points are required for proper calibration of the polarographic oxygen sensor (POS)?
3.2. Should the chamber be open or closed during air calibration?
3.3. What is an acceptable voltage (raw signal) of the POS at (a) air calibration, and (b) zero oxygen calibration, and how are these raw signals affected by the gain setting?
3.4. Why should you check the raw voltage during calibration?
3.5. How do you perform a zero oxygen calibration?
3.6. The oxygen solubility, S_{O2} [µM·kPa^{-1}], relates oxygen concentration to partial pressure. How is S_{O2} related to the solubility factor, F_M? Which variables need to be considered for estimation of the oxygen solubility of an aqeous solution, for example of mitochondrial respiration medium MiR06? [MiPNet06.03]
3.7. When is the oxygen calibration of a POS preferentially performed?

3.8. How long does it take approximately (5, 15, 30 or 45 min) to perform an oxygen calibration at air saturation, after the O2k is switched on (at experimental temperature in the range of 20 to 37 °C)?
3.9. Do you have to consider the instrumental background when performing an oxygen calibration of the POS at zero oxygen concentration?
3.10. Do you need to consider the instrumental background when performing an oxygen calibration of the POS at air saturation?
3.11. Does the oxygen signal have to be stable for an oxygen calibration of the POS?
3.12. How do you define POS signal stability? [MiPNet06.05]
3.13. Do you have to perform a zero oxygen calibration of the POS before air calibration?
3.14. Can you calibrate the POS with biological sample and respiratory activity in the aqueous solution, when equilibration is performed with a gas phase in the chamber and stability of the signal is observed?
3.15. What is the difference between static calibration [MiPNet12.08] and dynamic sensor calibration (time constant – for advanced users)? How can you use a dynamic calibration (stirrer test) as a quick sensor test? [MiPNet02.04]

4. POS Service [MiPNet08.04]

4.1. What should be done if the sensor connector threads appear dark and dirty?
4.2. The POS membrane box appears to have two types of membranes, which one should be applied to the sensor?
4.3. How can you avoid creating bubbles when filling the electrolyte reservoir of the POS?
4.4. Can the ammonia treatment be applied repeatedly?
4.5. How can you check sensor performance?
4.6. What precautions should be taken when handling the sensor connector?

5. Cleaning of the Chamber [MiPNet06.03]

5.1. Which solution should be placed in the chamber when the O2k is not in use (i.e. overnight, for a few days)?
5.2. Can detergents be used to clean the chamber and the PVDF stoppers?
5.3. What is the recommended cleaning procedure between experimental runs?
5.4. The glass chambers appear to have surface residue. Can this be removed, what is the procedure?
5.5. The stirring bar gets stuck. What can be done?

6. Instrumental background test [MiPNet12.09; MiPNet14.06]

6.1. Does the oxygen signal have to be stable (constant) for setting a mark in an instrumental background test?
6.2. Does the oxygen flux have to be constant for setting a mark in an instrumental background test?
6.3. How do you define flux stability? Is a flat horizontal red line always an indication of a stable flux?
6.4. Do you need to determine instrumental background flux at air saturation and zero oxygen concentration?
6.5. Do you need to calibrate the POS before performing an instrumental background calibration?
6.6. We use the symbol *a*° for the intercept at zero oxygen concentration, and the symbol *b*° for the slope of background oxygen flux as a function of oxygen concentration. In the analysis of instrumental background, we have obtained 0.022 and -1.7. Which value is *a*° and *b*°, respectively?
6.7. Does the background-corrected flux have to be zero when the oxygen signal is stable?
6.8. How often do you have to check the instrumental background?

Literature

Boushel R, Gnaiger E, Calbet JA, Gonzalez-Alonso J, Wright-Paradis C, Sondergaard H, Ara I, Helge JW, Saltin B (2011) Muscle mitochondrial capacity exceeds maximal oxygen delivery in humans. Mitochondrion 11: 303-307.

Pesta D, Gnaiger E (2010) High-Resolution Respirometry. OXPHOS protocols for human cell cultures and permeabilized fibres from small biopisies of human muscle. In: Mitochondrial bioenergetics: methods and protocols (Series Editor: Sir John Walker), edited by Carlos Palmeira and António Moreno. In press.

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Gnaiger E, Kuznetsov AV, Schneeberger S, Seiler R, Brandacher G, Steurer W, Margreiter R (2000) Mitochondria in the cold. In: *Life in the Cold* (Heldmaier G, Klingenspor M, eds) Springer, Heidelberg, Berlin, New York: 431-442. – *Isolated mitochondria and permeabilized muscle fibres, MiR06.*

Renner K, Amberger A, Konwalinka G, Kofler R, Gnaiger E (2003) Changes of mitochondrial respiration, mitochondrial content and cell size after induction of apoptosis in leukemia cells. *Biochim. Biophys. Acta* 1642: 115-123. – *Intact cells, cytochrome c oxidase, cytochrome c test, respiration per million cells, per citrate synthase, per mg protein, or per cytochrome c oxidase activity.*

Further information: Introductory course material is available on our homepage www.oroboros.at, within the following sections:

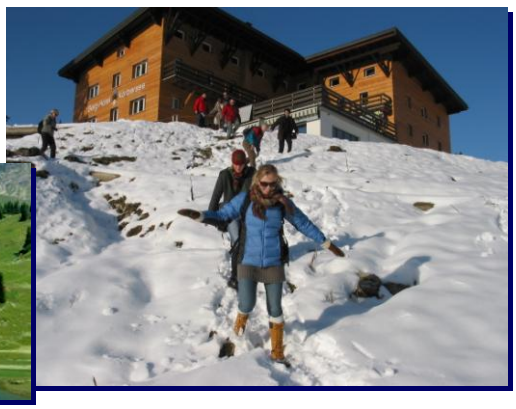
O2k-Manual - www.oroboros.at/index.php?o2k-manual
Protocols - www.oroboros.at/index.php?mipnet-protocols

wiki.oroboros.at - the *information synthase* for Mitochondrial Physiology and high-resolution respirometry:

Publications – http://wiki.oroboros.at/index.php/O2k-Publications
Continue the discussion - http://wiki.oroboros.at/index.php/Talk:IOC61

Accomodation and Location

Hotel Körbersee www.koerbersee.at;
Tel +43 5519 265; hotel@koerbersee.at



Participants and Areas of Interest

Alexeyev Mikhail, Department of Cell Biology and Neuroscience, University of South Alabama, USA. malexeye@jaguar1.usouthal.edu (*mouse modeling of mitochondrial disorders caused by mtDNA mutations*)

Angelin Alessia, Center for Mitochondrial and Epigenomic Medicine (CMEM), The Children's Hospital of Philadelphia |Research Institute, Colket Translational Research Building, Philadelphia, USA. agenlina@email.chop.edu (*function in muscle tissue; in particular human muscle disorder - muscular dystrophy*)

Balboa Castillo Elisa, Pontifical Catholic University of Chile, Santiago, CL. silvana@med.puc.cl (*increase in mitochondrial cholesterol and dysfunction in NPC*)

Castelein Natascha, Department of Biology, Gent University, BE. Natascha.Castelein@ugent.be (*the role of mitochondria in dietary restriction induced lifespan extension in C. elegans*)

Fasching Mario, OROBOROS INSTRUMENTS, Innsbruck, AT. mario.fasching@oroboros.at (*lecturer, tutor*)

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Guadaloupe Grau Amelia, Physical Education Department, Faculty of Sport Sciences, Campus Universitario de Tarifa, Las Palmas de Gran Canaria, ES. amelia.guadalupe@gmail.com (*mitochondrial physiology in human skeletal muscle; role of mitochondria in obesity and exercise*)

Hals Ingrid, Department of Cancer Research and Molecular Medicine, Norwegian University of Science and Technology, Trondheim, NO. Ingrid.hals@ntnu.no (*effects of hypoxia in pancreatic beta-cells on mitochondrial functions*)

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Kasal Daniel A., Chagas Filho Biophysics Institute (IBCCF), Federal University of Rio de Janeiro, BR. kasald@netscape.net (*modulation of cardiomyocyte respiration by bone marrow stem cells*)

Kilic Ana, Institut für Pharmakologie und Toxikologie, Universität Bonn, DE. akilic@uni-bonn.de (*respiration in isolated murine tissues; uncoupled respiration in mitochondria*)

Kovalcikova Jana, Department of Cell Biology, Faculty of Science, Charles University, Prague, CZ. indi1@centrum.cz (*respiration and OXPHOS regulation in heart mitochondrial populations under oxidative stress*)

Labieniec-Watala Magdalena, Department of General Biophysics, University of Lodz, PL. magdalab@bio.uni.lodz.pl (*mitochondrial respiratory capacity; mitochondrial membrane potential; calcium homeostasis; mitochondria in health and pathology*)

Le Catherine, Health and Exercise Science, Colorado State University, Fort Collins, USA. aaralyn.mail@gmail.com (*implications of mitochondrial dysfunction in heart disease*)

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