



NEWSLETTER

of the European Society for Animal Cell Technology

January 2007

A Word from the Chairman

Florian Wurm

Dear ESACT friend,

I hope you made it safely into the new year - and this wish shall of course be extended to anybody close to you and your family. For the wider ESACT community 2007 will be another exciting year since we will hopefully meet all together in June in the beautifully rebuild city of Dresden in the eastern part of Germany. Our friend Dr. Hansjörg Hauser speaks with so much enthusiasm about this place that I am extremely eager to see it myself (I admit not having visited Dresden ever). There are many architectural marvels to admire and also the setting with the mighty river Elbe is supposed to be extraordinary... but please check it out yourself. I know that Hansjörg and his team will do everything possible to make this event another memorable highlight in the history of ESACT.

This Newsletter is in a new format - also due to the fact that our friend Dr. Steve Oh from Singapore felt that after 5 years of service as Newsletter editor somebody else should carry forward this responsibility. You all have heard about the tremendous push for Biotech in that part of our globe and of course Steve has seen his non-ESACT responsibilities grow non-linearly, i.e. there was also a need to let go something. Steve - THANK YOU for a GREAT JOB done!

Our long-term friend from Geneva - Christophe Losberger (Merck Serono) agreed to help us to keep you all informed, not only about unpaid membership fees, as he had done with many of us in the past, but also now in his new function as the (Interim?) Editor of our Newsletter. So here you see the first result of his work and - I am very happy about this! Thank you Christophe!

Let me conclude by saying that I am eager to meet you all again, the latest in June and, also - as the Chairman of our Society - I want to point out how important your contributions will be to the society. Contributions in science and technology, but also, if you wish in helping us with keeping our society young and dynamic by joining forces in the "administration" and "organisation" of ESACT. So, if you feel that you could be more involved, come forward with your suggestions, propose yourself for election into the Executive Committee, help us to remain the leading cell culture science based organisation.

With the best wishes,

Florian

Contents January 2007

A Word from the Chairman	1
ESACT 2007 – Dresden	2
News from the Gene Therapy Front	3
Upcoming ACT related meetings	7
Membership	8
Nominations to the ESACT Executive Committee	9
Society	10

Contributions should be sent to eleter@esact.org
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ESACT 2007 – Dresden

Hansjörg Hauser

The next ESACT Meeting is approaching rapidly. The 20th ESACT-Meeting entitled “Cells & Culture” will be held in the city of Dresden in Germany between the 17th and 20th June 2007, is well on the way. The baroque city of Dresden on the Elbe river surrounded by the rolling hills of Saxony, represents a monument of European history and culture. It sets an inspirational stage for the high level scientific and social interactions in the spirit of ESACT.

A new feature of the upcoming meeting will be the format of the scientific program. As the diverse scientific disciplines related to cell culture technology are becoming more and more intertwined, integrative and systems approaches become essential. To address this the scientific committee has decided to shift the focus of the scientific sessions towards integration and systems biology and to give thus a “new face” to the scientific program. Six scientific sessions are planned for the 20th ESACT-Meeting. Each session will feature invited speakers as well as contributions from speakers chosen by the scientific committee from submitted abstracts.

SINGLE-CELL ANALYSIS AND ENGINEERING

(Chairs: Martin Fussenegger and Thomas Noll)

This session will cover all relevant aspects taking place at the single-cell level including miniaturization and characterization. Speed up of the quest for the highest producer cell line and the best-in-class metabolic/therapeutic engineering approaches require precise understanding of single-cell behaviour. Pioneering design and use of analysis hardware and innovative genetic engineering strategies will be presented.

APPLIED INTEGRATIVE PHYSIOLOGY

(Chairs: Simon Hoerstrup and Hansjörg Hauser)

This session will cover the exploitation of integrative physiological knowledge towards medical applications: Improvement of diagnostics, disease prevention and therapy.

SPEED AND INTENSIFICATION IN BIOPROCESS DEVELOPMENT

(Chairs: Manuel Carrondo, Barry Buckland and Peter Gray)

Pressure to reduce the development time for new biopharmaceuticals, coupled with the need to contain costs, requires more efficient product development. Presentations in this session will address all types of tools aiming at this desideratum, be they molecular, cellular or complex/engineering so long as a clear rationale is presented. Wherever appropriate or possible, integrative approaches will be sought after as well as tools/processes/technologies with predictive impact or predicted “outcome”.



The picture shows the Semper Opera building in the foreground. In the background, right, a crane marks the site of the Congress Center which was under construction at the time of this snapshot (2004).

SYSTEMS BIOTECHNOLOGY

(Chairs: Francis Stewart and Eleftherios Papoutsakis)

This session will cover system approaches applied to discovery of novel targets for therapy, the development of cell lines and cell therapy protocols, process identification and validation, clinical trials, the development of disease prognostic and diagnostic signatures, and to understanding cellular physiology at a systems molecular level. Emphasis will be placed on integrative approaches, the use of genomic tools and novel computational approaches.

COMPETING AND COMPLEMENTARY APPROACHES TO ANIMAL CELL TECHNOLOGIES (ACT)

(Chairs: John Aunins and Rolf Werner)

Processes or biological systems which compete with ACT for solutions as well as tools and technologies empowering ACT driven solutions will be covered by this session.

SOLUTIONS AND APPLICATIONS

(Chairs: Stefanos Grammatikos and Martin Gawlitzek)

Practical approaches for animal cell technologies will be covered in this session.

The submission deadline for the abstracts is over now, and the Scientific and ESACT Executive Committees will decide on the final scientific programme during a joint session in March 2007.

POSTER PRESENTATIONS

Posters of special quality will be recognized by 3 mechanisms: First of all, the scientific committee will select posters for the scientific sessions. Secondly, up to 15 posters will be selected in place for short oral presentations on Wednesday, June 20, 2007. Finally, the well known poster prizes will also be awarded this year.

The trade exhibition – the largest and best known of its kind – forms again an integral part of this ESACT meeting and promotes and underlines this interaction. As in previous meeting posters and coffee breaks will be mixed into the trade exhibition, providing excellent chances to interact. A significant part of the available space for the exhibition has been already given away and companies wanting to contribute should not lose time to contact the meeting organizer.

More information can be found on the web site of the 20th ESACT Meeting <http://www.esact2007.org/>. As usual during each meeting a general assembly will be organized. The general assembly will take place at the conference center in Dresden during lunch time on Tuesday, June 19, 2007. The agenda is not yet finalized.

The meeting organizer is DECHEMA. We will be looking forward to a well structured meeting with an interesting excursion and a party on the last meeting day's evening.

We are looking forward to an exciting and stimulating meeting and are inviting you wholeheartedly to join us at ESACT 2007 in Dresden/Germany!

On behalf of the Organizing, Scientific and Executive Committees

Hansjörg Hauser

News from the Gene Therapy Front

Otto Merten

Gene therapy clinical trials of rare diseases with DMD - Duchenne Muscular Dystrophy – as an example

For many inherited and acquired diseases gene therapy will be the only one or one of the few means for treating the patients and a lot of hope and expectations exist with respect to this form of treatments.

However, due to the complexity of this future molecular therapy, only one gene therapy treatment has been commercialized up to now (= treatment of head and neck squamous cell carcinoma with Gendicine (SiBono, Shenzhen, China), an adenovirus 5 vector transferring the p53 gene leading to apoptosis of the transduced cells due the expression of p53) (Peng 2005).

Despite considerable efforts performed since 20 years or so, this is a relatively poor result – with respect to the availability of regular gene therapy applications –

however, it clearly shows that gene therapy is a very complex undertaking with many problems still to be solved.

This only regular existing gene therapy application shall not indicate that the research and development performed since 20 years was in vain. In this context, the website of J. Gene Med. indicates the ongoing efforts in this sector and the fact that more than 1000 gene therapy trials have been performed of which only 26 are phase III clinical trials (= 2.2% of all trials)(for information: phase I: 62%, phase I/II: 20%, phase II: 14%, phase II/III: 1%).

For briefly resuming, 67% of all gene therapy trials are dealing with the treatment of cancer (797), 8.9% with vascular diseases (106), 8.6% with monogenic diseases (inherited diseases, 102), and 6.5% with infectious diseases. For these treatments, different viral and non-viral vectors have been used: 26% adenoviral vectors (305 trials), 24% retroviral vectors (288 trials), plasmid DNA in 17% of all trials (206), lipofection in 99 trials (8.3%), poxvirus in 82 trials (6.9%), vaccinia

virus in 78 trials (6.5%) and herpes virus and AAV in 40 trials (each) (= 3.4%), etc. The gene type transferred were cytokines (26%), antigens (16%), tumour suppressors (12%), suicide genes (7.9%), replacement genes for deficiencies (6%), etc.

However, these numbers indicate also that only 8.6% of all gene therapy trials were performed for the treatment of inherited diseases (rare diseases) and that most of the gene therapy trials were destined for the treatment of diseases of more commercial interest.

In the following, I will only stick to gene therapy of rare/inherited diseases; however, the reader is referred to the recent review by Young et al. (2006) for more information.

Gene therapy for the treatment of inherited disease for which no other treatments exist is of high importance for the patients; however, as already mentioned, only 8.6% of all clinical trials have been performed. Some of them had very spectacular results, as the trial which was conducted in Paris, in which the *ex vivo* treatment of CD34+ cells from patients with X-linked severe combined immunodeficiency could be 'repaired' via the delivery of the wild-type version of the gene encoding the common cytokine receptor gamma chain (γ_c) using MLV, thus proving important 'proof of principle' of the gene therapy approach. The development of T-cell proliferative disease in three of the patients as a consequence of retrovirus vector insertion highlights the need for safer gene transfer vectors but also the need for a better understanding of the underlying biology of the diseases to be treated (Cavazzana-Calvo et al. 2000, 2005, Hacein-Bey-Abina et al. 2003, Young et al. 2006).

As I am working in a research institute with the objective to develop new therapies for the treatment of inherited diseases, and in particular, of muscular diseases, the rest of this article is dealing with gene therapy treatments of these diseases and of DMD, as an example.

One of the first clinical gene therapy trials performed for the treatment of DMD (phase I trial) was promoted by Transgène and supported by the AFM (Association Française contre les Myopathies) and had for objective to evaluate the feasibility and tolerance of *iv* administration (use of overpressure) of a plasmid containing the full length human dystrophy gene into DMD patients (start: middle of 2006, end of study: end of 2006)(www.AFM-France.org). This assay is based on the results in terms of safety obtained with a previous clinical study (also phase I), in which plasmid DNA with the dystrophine gene was administered *im* (3 cohorts of 3 patients receiving either once 200 or 600 μ g of DNA or twice 600 μ g of DNA, 2 weeks

apart, end of study: 2003). In 6 out of 9 patients (DMD/BMD (Becker MD)) a consistent and moderate exogenous dystrophin expression was observed (up to 6% weak, but complete sarcolemmal dystrophin staining, and up to 26% partial sarcolemmal labelling) (Romero et al. 2004, Fardeau et al. 2005).

With respect to the *in vivo* application for direct or indirect muscle treatment (*im* or *iv*, respectively), AAV (adeno-associated virus) vectors represents another and more efficient means for gene transfer. In this context, only phase I clinical trials have been started up to now (start in March 2006), indicating that mainly the potential toxicity was evaluated practically without any benefit for the patient. This phase I trial which was based on the use of AAV transferring the mini-dystrophine gene for the treatment of Duchenne muscular dystrophy (DMD)(J. Mendell - Columbus Children's Res. Inst., Ohio State Univ.). In this trial, doses of 2×10^{10} vg/kg and 1×10^{11} vg/kg were injected into one biceps and empty AAV particles in the same excipient into the contra-lateral biceps of boys with a null-mutation (6 DMD subjects aged 5 years and more). In order to avoid any immuno-response against the AAV capsids the patients received a bolus of methylpred (2 mg/kg) at the time of AAV-injection. Biopsies were taken 6 and 12 weeks after AAV injection and were analysed for the presence of mini-dystrophy and for signs of immune-response. A phase IIa/IIb clinical trial is planned (Danos 2006).

Very recently, a similar trial (phase I/IIa) was started by Généthon for the treatment of gamma-sarcoglycanopathy (limb girdle dystrophy type 2C) (use of an AAV1 vector)(Press release by the AFM, 17th of November 2006).

These AAV based phase I or I/IIa trials are only trials with local administration. However, it is absolutely clear that a whole body treatment will be necessary for healing patients suffering from one or another form of muscular dystrophy, however, reasonably this cannot be performed via *im* injections of viral or non-viral vectors, wherefore other means have to be chosen.

Today, in principle several ways can be envisaged in order to get to a whole body treatment of patients. One way will be the systemic (*iv*) injection of viral vectors (AAV) which leads to the efficient transduction of muscle tissue (which is AAV serotype dependent), but also of many other tissues. Such approaches have been tested in pre-clinical studies in mice (whole body treatment via the injection of AAV vectors into the tail vein (Gregorevic et al. 2006)) and large animals (dog – systemic injection of AAV into the leg (Garcia & Danos 2005)). Although this approach seems to be successful when concluding animal studies, several major issues still have to be tackled, some of them are:

i) dose (which/dose per kg of patient)? This is of high importance for the transduction efficiency but also in view of potential immunoreactions against high vector loads (as recently shown for AAV injected im into dogs – anti-AAV immune-response, Wang et al. (2007)); and ii) the vector formulation (in order to assure a homogeneous and efficient distribution of the vector and efficient passage of the vector to the muscle tissue as well as an efficient transduction).

A completely different approach will be the use of myogenic stem cells derived from the patient. These cells (mesangioblastes or AC133+ cells) can be more or less easily amplified and injected into the vascular system and can thus be transported to every place in the body (muscles included). Recently, the group of G. Cossu in Milan (Press Release by the AFM, 15th of November 2006, Sampaolesi et al. (2006)) has shown that GRMD dogs (dog model of DMD in humans) can be treated with mesangioblastes either from healthy dogs (immune-suppression is necessary in this case) or from diseased dogs (in this case, autologous mesangioblastes are transduced in vitro with an LV vector for the expression of microdystrophine (ex vivo gene therapy)). Excellent results were obtained when mesangioblastes from donor dogs were used, however,

the results were much less spectacular when the dogs' own but 'repaired' mesangioblastes were used, probably due to the fact that the human microdystrophine gene was used and not the normal dystrophine gene of the dog. Further studies are in progress to clarify this issue.

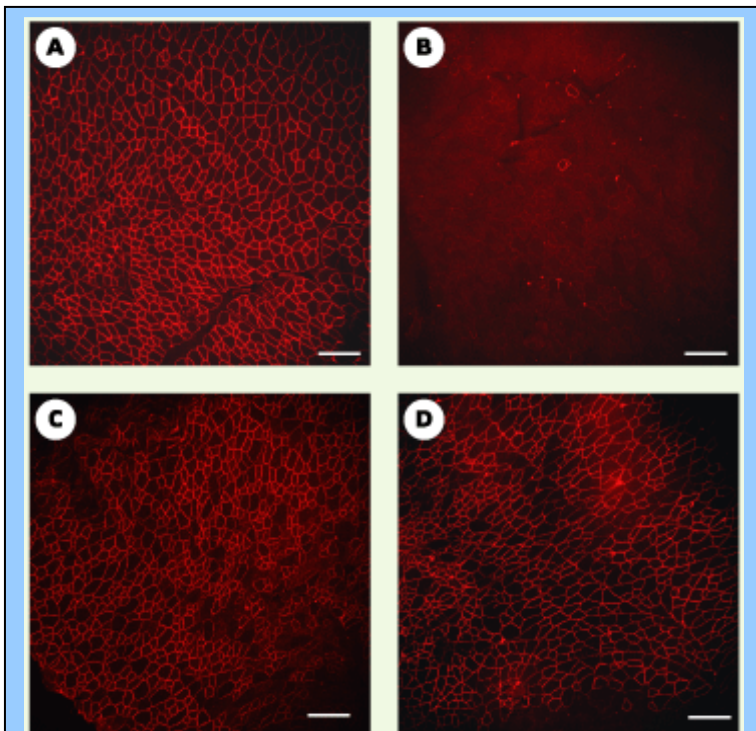
When fully developed this approach has the following advantages: The distribution through the whole body is relatively efficient and the cells can pass the blood tissue (muscles included) barrier without major problems. In addition, the cells are able to fuse with the muscle fibers and have a satellite like behaviour. This is essential for a successful gene therapy of muscular tissue.

Finally, the exon skipping approach seems to be of high interest, in particular for the treatment of DMD (but not only), because this form of therapy does not insert a new gene into the cellular genome but 'repairs' the expression of large genes via the skipping of one or several exons in order to repair a frameshift and/or the appearance of a stop codon due to a mutation. This approach leads to the expression of a protein which lack some of its exons but which keeps its functionality (this is of course dependent of the mutation). A further advantage is that the natural regulation of expression is preserved in this type of therapy.

However, it is also clear that not all forms of DMDs can be treated with this approach because it depends on the localisation of the genetic defect within the molecule.

The functionality of this approach was spectacularly confirmed by the group of L. Garcia (Généthon/Institut de Myologie, Goyenvalle et al. (2004)) for a mouse model of human DMD (MDX mouse) for which the skipping of the exon 23 was performed and the muscle fibres for which the exon 23 was skipped re-expressed functional dystrophine. This approach is highly efficient and more than 90% of the muscular fibers were dystrophine positive. The necessary oligonucleotides were transferred by an AAV1 vector, which was administrated im. Similar studies in large animals (dogs) are ongoing.

It is evident that this exon skipping approach can be applied via viral vectors (AAV is optimal for the treatment of muscular tissues in vivo), via myogenic stem cells which have been treated ex vivo (use of lentiviral vectors for the ex vivo treatment) for a later administration after amplification and quality control as well as via the systemic administration of antisense



Immunomarquage of dystrophine with a monoclonal mouse antibody (NCL-DYS2). Immunomarquage of muscle sections (tibialis anterior) from a normal mouse (C57Bl6) (A), from a non-treated mdx mouse (B), from a treated mdx mouse, one month after treatment (C) and 6 months after treatment (D) (the treatment was an injection of AAV-U7-SD23/BP22 (bar [A-D]: 0,5 mm) (from Goyenvalle et al. Med Sci 20:1163-1165.)

oligonucleotides (e.g. ESPRIT – Exon Skipping Pre-RNA Interference Technology – use of morpholinos) allowing in vivo skipping. However, there are two drawbacks of this latter approach: i) the treatment has to be repeated several times a year, because this is only a transient approach, ii) this type of treatment is very expensive, and iii) for unknown reasons it does not work on heart. However, due to the possibility of a rapid treatment high expectations are associated with this therapy and clinical trials (phase I) will start soon. In a proof-of-principle, controlled, dose-escalating trial, up to nine young boys with DMD will receive a single, intramuscular administration of the antisense oligonucleotide preparation which targets exon 51 (communication by AVI BioPharma (8th of December 2006), the clinical programme is a collaboration with the U.K. based MDEX Consortium). A similar trial ('smart therapy' – use of AONs based on the 2'-O-methyl phosphorothioate chemistry) will be started soon in the Netherlands (Press release by Prosensa, 10th of May 2006).

A commentary on the exon skipping approach for the treatment of DMD was published by van Deutekom (2005), and an overview is available from C. Dattola from the Duchenne Parent Project France (www.duchennefr.org).

As a last word, many different approaches can be applied for the performing of gene therapy of rare diseases and the present scientific knowledge and recent developments allow a much more optimistic view on the treatment of future diseases for which no treatment is available up to now.

O-W Merten, G n thon, Evry/France

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Upcoming ACT related meetings

Paula Alves

2007

WORKSHOP OF THE RESCUE SOCIETY (Research Experience of Stem Cells in European Society)
Basel, Switzerland, Feb 23-25
<http://www.rescuesociety.org/>

5TH HIC/RPC: BIOSEPARATION CONFERENCE
Interlaken, Switzerland, March 20-23
<http://www.hic-rpc.org/index.htm>

WORLD CONGRESS ON INDUSTRIAL BIOTECHNOLOGY AND BIOPROCESSING
Orlando, Florida, USA, March 21-24
<http://www.bio.org/worldcongress/>

2ND SINGAPORE BIOLOGICS MANUFACTURING CONFERENCE
Singapore, Mar 28-30
<http://www.sbmc.sg>

CBM7: 7TH CARBOHYDRATE BIOENGINEERING MEETING
Braunschweig/Germany, April 22 - 25
<http://events.dechema.de/CBM7-lang-en.html>

3RD PHARMACEUTICAL SCIENCES WORLD CONGRESS – OPTIMISING DRUG THERAPY: AN IMPERATIVE FOR WORLD HEALTH
Amsterdam, Netherlands, April 22-25
www.pswc2007.org

ASGT (AMERICAN SOCIETY OF GENE THERAPY) 10TH ANNUAL MEETING
Seattle, WA , USA, May 30 - June 3
<http://www.asgt.org/am07/>

ESACT 2007: 20th Meeting of the European Society for Animal Cell Technology
Dresden, Germany, June 17 - 20
<http://www.esact2007.org/>

BIOCHEMICAL ENGINEERING XV: Engineering Biology from Biomolecules to Complex Systems,
Québec City, Canada, July 15–19
<http://www.engconfintl.org/7am.html>

ENGINEERING CELL BIOLOGY II,
Massachusetts Institute of Technology, August 5-8, 2007
Cambridge, Massachusetts, USA
<http://www.engconfintl.org/7ak.html>

13TH EUROPEAN CONGRESS ON BIOTECHNOLOGY
Barcelona, Spain, 17-19 September
<http://www.ecb13.eu/>

8th PEACE
Angra dos Reis, Brasil, 16-20 September
<http://www.peace-conference.org/>

3RD WORLD CONGRESS ON REGENERATIVE MEDICINE
Leipzig, Germany, 17-19 October
<http://www.regmed.org/>

XVTH ANNUAL CONGRESS OF THE EUROPEAN SOCIETY OF GENE AND CELL THERAPY
Rotterdam, The Netherlands, 27-30 October 2007
<http://www.esgt.org/>

2008

- STEM CELL ENGINEERING (San Diego, California), Jan TBA
- CELL CULTURE ENGINEERING XI (Coolum, Australia), April 13-18
- 1ST EUROPEAN CONFERENCE ON PROCESS ANALYTICS AND CONTROL TECHNOLOGY (Frankfurt/Main) April 22 – 25
- VACCINE TECHNOLOGY II (Albufeira, Portugal), June 1-6
- METABOLIC ENGINEERING VII (Puerto Vallarta, Mexico) Sept 14-18



www.esact.org

Check our [website](http://www.esact.org) regularly to get the latest meeting listings.

Membership

Stefanos Grammatikos
Christophe Losberger

Membership Statistics & New Members

The ever growing number of ESACT Meetings' attendees confirms the attractiveness and popularity of the biannual Meeting and gives the impression that ESACT is a huge society supported by many members. Have you ever wondered how many members ESACT actually has? Try to take a guess before you read further.

While you are estimating, we are taking the opportunity to welcome the following new members to the society:

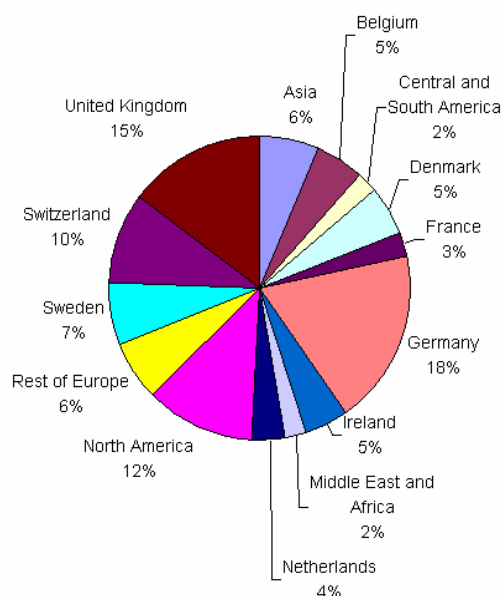
Bäckström Malin	Göteborg University
Barker Jenny	University of Kent
Barron Niall	Dublin City University
Boehm Daniela	University College Dublin
Bulthuis Ben	Centocor
Carvell John	Aber Instruments
Castillo José	ARTELIS SA
Comer Michael	Adelaide and Meath Hospital inc. National Children's Hospital, Ireland
David Carol	Teva Pharmaceuticals
Drugmand J-Christophe	Artelis
Durocher Yves	Biotechnology Research Institute, Canada
Essers Ruth	Siegfried Biologics GmbH
Ferreira Guilherme	University of Algarve
Hanke Petra	Roche Diagnostics GmbH
Hansen Jens Jacob	Novo Nordisk A/S
Hassin Shimon	InSight Biopharmaceuticals
Herrmann Andreas	Celonic GmbH
Ibarra Neysi	Univ of Oxford
Jelinek Nanni	BioGeneriX AG
Jordi Fernandez	Laboratorios Hipra, S.A.
Joseph Reg	Invitrogen Corporation
Kamen Amine	Biotechnology Research Institute/National Research Council, Canada
Kaufmann Hitto	Boehringer Ingelheim Pharma
Krampe Britta	University College Dublin
Marchant Rosalyn	University of Kent
Meleady Paula	Dublin City University
Molitor Chantal	GlaxoSmithKline Biologicals
Müller Dethardt	BOKU- University of Natural Resources and Applied Life Sciences, Austria
O'Dea Fergus	Wyeth Biotech
Pilaete Marie-Françoise	GlaxoSmithKline Biologicals
Pol, van der Leo	Netherlands Vaccine Institute
Rehberger Bernd	Rentschler Biotechnologie
Schiedner Gudrun	CEVEC Pharmaceuticals
Schwabe Jan-Oliver	F. Hoffmann La-Roche Ltd
Swiderek Halina	University College Dublin

Thomsson Elisabeth
Veraitch Farlan
Wieland Jürgen
Woolley John
Zeng An-Ping

Biomedicine
University College London
Merckle Biotec GmbH
University College Dublin
Technische Universität Hamburg-
Harburg

Now back to the statistics. ESACT currently has 189 active members. This figure includes Executive Committee Members (9), Honorary Members (12) and all Regular Members, as well as the new members mentioned above. Active members are defined to be those who have shown interest in the Society by at least paying the membership fee (20€/year) regularly. Actually there are about 800 individuals who have once been ESACT members in the last 15 years. Since the introduction of an electronic database and an online system for paying of membership fees, however, the "inactivity" of a large part of those on the list of members became transparent and the need for sorting out the records arose. All those who had outstanding membership fees were notified several times and all those who have remained inactive and did not respond to the notifications were permanently removed from the database.

The overwhelming majority of ESACT Members comes from Europe (147), mainly from Germany (35), the UK (28), Switzerland (18), Sweden (13) and Norway (10). Non-European Members are from North America (22), Asia (12), Central and South America (4) and the Middle East and Africa (4). The overall distribution is shown in the following Graph.



The ESACT Executive Committee regards the Society to be much more than an excellent Scientific Meeting every two years and strongly encourages a larger member involvement in ESACT matters and activities, through suggestions, ideas, feedback and various initiatives.

How to check / renew your membership ?

As the members database is managed with a web interface, subscriptions can only be renewed online as described here below:

- Logon the members page
<http://www.esact.org/amember/member.php>
with your username and password.

- Lost passwords can be retrieved by using the "Lost Password ?" feature at the bottom of the page.

Once you are logged:

- Select the subscription you wish
- Select the payment method and click "Subscribe"

Payment Methods

- Offline = bank to bank transfer. All the details for the transfer will be sent by email.
- Paypal = online direct transfer through paypal. Credit card payments can be processed even if you do not have a paypal account.

Subscriptions will be validated on reception of the fees: several days for bank transfer, immediately with a paypal payment.

If you have any question concerning your membership, please do not hesitate to send an email at admin@esact.org

Nominations to the ESACT Executive Committee

Stefanos Grammatikos

Dear Colleagues and fellow ESACT Members!

The current Executive Committee will conclude its biannual work by the 20th Meeting in Dresden and will report on its activities and on the status of the Society during the General Assembly to be held in Dresden on June 19, 2007.

A new Executive Committee will be elected for the forthcoming two-year period 2007-2009. The current Executive Committee is therefore inviting nominations for Ordinary Executive Committee Membership (officers are typically appointed by the incumbent Executive Committee). Two ESACT Members have to sign the nomination and the nominee must indicate acceptance. The form will be circulated by mid February 2007 to all members and the deadline for nominations will be the 31st of March, 2007. All Members will be given the chance to vote, from mid-April and up to the Meeting in Dresden. The election results will be announced during the General Assembly in Dresden.

Renewal keeps the Executive Committee on its toes. Active participation to ESACT matters keeps the Society alive.

On behalf of the Executive Committee

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