



The Pezcoller
Foundation

Journal



Summary

- Editorial October 2008
- The Stanley J. Korsmeyer memorial lecture
- 2009 Pezcoller Foundation-ECCO Recognition for Contribution to Oncology
- 2010 Pezcoller Foundation-AACR International Award for Cancer Research
- 2009 Pezcoller Symposium "Unconventional therapeutic targets in cancer" 11 - 13 June

October 2008

We are glad to publish in this fall-issue of the Pezcoller Journal the third “Korsmeyer Lecture”. We are grateful to Dr. Axel Ullrich for his kindness to let us publish the lecture entitled “Novel Targeted Treatments for Cancer” given in Padua at VIMM the day before the Award ceremony in Trento on May 9. Axel Ullrich is the prestigious recipient of the 2008 Pezcoller Foundation-AACR International Award for Cancer Research.

I like to remind that the annual Stanley J. Korsmeyer Lectureship has been started by the Pezcoller Foundation in 2006 in accordance with the AACR American Association for Cancer Research and the VIMM Venetian Institute of Molecular Medicine in Padua. The goal of this event is to honour the fundamental contribution of the late S. Korsmeyer, an international leader in the field of cancer biology, whose pioneering observations opened the molecular era of programmed cell death.

S. Korsmeyer was the recipient of the Pezcoller Foundation-AACR International Award for Cancer Research in 2004. He presented his last European lecture by the VIMM in Padua (Italy) immediately before receiving the Pezcoller Award. Unluckily he passed away from cancer a few months later. Therefore we wish to remember Stanley Korsmeyer every year with this lecture given by the recipient of the Award.

We are also presenting on the last pages the call for the 2009 Pezcoller Foundation-ECCO Recognition for Contribution to Oncology and the call for the 2010 Pezcoller Foundation-AACR International Award for Cancer Research.

We are also glad to introduce the “focus and goals” of the next 21st Pezcoller Symposium which will be held in Trento on June 11-13,

2009 and will be entitled “Unconventional therapeutic targets in cancer”. This year the Symposium will offer an opportunity for a small, hand-picked group of European cancer scientists to become speakers in the Program and to be recognized at the same level as the more senior leaders of the field who are invited to speak. The specific aim of this initiative is to create a competition among such young people, the most able and accomplished of whom would be chosen to come to Trento at the expense of the Foundation and to present their work within the regular Program.

The last Symposium was the 20th of the series, therefore we gave particular prominence to the event. It took place in the headquarters of a research center of Trento last June and the usual five sessions were increased to six sessions: a complete three days symposium. The peculiar attractive, cutting edge nature of the Pezcoller Symposia has been recognized and underlined by the large group of prestigious speakers who attended the meeting from all over the world. Among this group we had the pleasure to include also some previous winners of the Pezcoller-AACR Award (see picture). We have been very glad to give the ‘Pezcoller Begnudelli Award’ for the best posters to three young deserving researchers: Olivia Crociani, University of Florence, Elena Quaglino, University of Turin, Andrea Lunardi, University of Trieste

Gios Bernardi MD

The Pezcoller Foundation President

Picture on front page:

speakers and past winners of the ‘Pezcoller Foundation-AACR Award’ (from the left) Lewis Cantley, Carlo Croce, Mina Bissell, the President Gios Bernardi, Tada Taniguchi

The Stanley J. Korsmeyer memorial lecture

Novel Targeted Treatments for Cancer

Prof. Axel Ullrich, MPI for Biochemistry, Martinsried - Germany

Abstract:

Thirty years ago, several genetic researchers had a seemingly exotic idea that has since revolutionized the treatment of one of the deadliest of human diseases: cancer. It appeared that cancer was an illness in which part of the genome “goes crazy”. The affected cells produce either too much or too little of some proteins and start to reproduce regardless of any other considerations. The vision at that time was to find drugs with which to target the molecular “Achilles’ heels” of tumors. Thanks to the rapid progress that has been made in genetic technology, this has now become possible. Two therapies based on this principle that were developed on the basis of research in laboratories at Genentech (South San Francisco) and the Max Planck Institute of Biochemistry (Martinsried) are now used successfully to treat cancer - and it is not by chance that the crucial impetus for the realization of this vision came not from industry, but from basic research.

Introduction

It is a mass killer, and it almost always approaches quietly: Cancer is still one of the most treacherous human diseases and the second most common cause of death after cardiovascular disease; throughout the world, around 7.6 million people die of the consequences of a tumor every year. There are numerous drugs with which to fight cancer nowadays, in addition to surgery and

radiotherapy, but scarcely any other disease is so difficult to come to grips with. Doctors not only distinguish between 230 types of cancer, the errant cells also manage to change constantly in one and the same tumor - avoiding attack from the immune system and from medicine, again and again. Only 30 to 40 percent of all patients are successfully treated today.

However, there has been a glimmer of hope for some time now. Thanks to the rapid progress in genetic research in recent decades, it has been possible to get to the heart of the problem: cancer, as it turns out, develops because parts of the genetic material goes “out of control”. Certain genes in the errant cells are so changed by mutations that they cannot communicate correctly with the surrounding tissue anymore. At some point, the control system in the diseased cells fails completely - and they start to divide uncontrollably, becoming spread to other parts of the body.

Exciting times at the first biotech company in the world

With hindsight, it is clear that my early work on the insulin gene opened the way for my subsequent research work on cancer. Firstly, in 1978 my publication about the cloning of the human insulin gene brought me to the newly-founded San Francisco company Genentech - the first biotech company in the world.

At that time, the company consisted of just eight employees and we were lucky that the management was able to ensure that sufficient money was available to allow us a great deal of freedom in pursuing our scientific interests. Secondly, my work on the insulin gene had started me along the path to an area of research that still fascinates me today: researching the ingenious messaging system

through which billions of cells in the human body communicate with each other. During every second of our lives, each one of these cells must remain in contact with others to receive instructions and also to transmit information to them.

As we know today, cells conduct incoming signals via specific antenna molecules - called receptors - through their membrane into their interior. Using various molecular switches in the cell, the communications system regulates many important biological processes, such as cell growth, -metabolism, survival and the specialization of immature cells into stomach, skin or blood cells.

However, the molecules that participate in this signal cascade also play a significant role in diseases like cancer and diabetes - either because they are defective or because they are being created in the wrong quantities, at the wrong time. In fact, the products of all known human "cancer genes", or oncogenes, are abnormal variants of molecules which, in some form, are involved in the signal chains that control important normal cell functions..

Genes that go astray

My colleagues and I discovered the first indication of this in the 1980s in the form of a receptor for a messenger substance called Epidermal Growth Factor Receptor, or EGFR. As it turned out, this antenna molecule was anything but unknown. It was, on the contrary, the normal version of an oncogene - v-erbB that Tadashi Yamamoto at Tokio University had discovered in the genetic material of a virus that causes tumors in infected chickens. The scientists Michael Bishop and Harold Varmus of UCSF had previously characterized another cancer gene "src" and decoded its elements; however the function of the corresponding "normal" gene had remained an unknown quantity. Nevertheless, they had proposed that mutated normal genes would play a crucial role in the development of cancer. It was only after we had isolated the gene for the EGF receptor from human cells, transferred it to the genetic material of bacteria

and compared its elements with genes that had already been characterized that the answer to this question became clearer: Only when stimulated normally by EGF the intact, not mutated EGF receptor is responsible for passing on growth signals from the surrounding tissues to the cell nucleus and, depending on other information the cell contains, it can then either divide into two daughter cells, change its function or give the cell signals that allow it to survive rather than enter the suicide program that is built into every cell.

This gets out of control in cancer cells. It was shown in the bird cells infected with the virus that the receptor is so changed in its structure that it constantly emits cell division signals.

In some human tumor tissues, the number of genes for the EGF receptor is so high that a similar effect arises. In comparison: a normal cell contains one EGF receptor gene per chromosome set. In cells that were isolated from a cervical carcinoma and then cultivated in the laboratory, we found 25 copies per tumor cell. These gene reproductions do not only lead to over-production of the EGF receptor protein but as we found later can cause a normal cell to change into a cancer cell.

This finding not only offered insight into the mechanisms of carcinogenesis. It also led to a discovery which resulted in the first medication that targeted a characteristic feature of certain cancer cells - and thus largely spared healthy cells.

As is so often the case in research, chance came to our aid. In the course of identifying sections of DNA that contained the genetic information for the EGF receptor, we found pieces of genetic information which had very similar sequences, but which clearly stemmed from another gene. We called the product of this still unknown gene HER2 (Human EGFR-related receptor).

Cells under permanent stimulation

In order to continue to explore the significance of oncogenes for the emergence of cancer in humans, we needed the cooperation of an oncologist who would give us

access to tumor biopsies, which would allow us to examine tumor cells in the laboratory for specific genetic changes. Fortunately, I succeeded in persuading the oncologist Dennis Slamon from the University of California in Los Angeles to join our project. The objective of the collaboration was to investigate which of the genes that we had isolated because of their importance for regulating cell growth, exhibited any abnormal changes in the cells of mammary tumors. In 1986, this investigation yielded a surprising finding: around 28 percent of all tumors of breast cancer patients contained an abnormally high number of HER2 genes, similar, therefore, to what we had found for the EGFR in the laboratory in the cells -A431- cultivated from a cervical tumor. The rate of reproduction varied, ranging from two to more than fifty gene copies per cell. And that was not all: the cells also produced excessive quantities of the HER2 protein. Indications that it was a receptor for an unknown growth factor and that Bishop and Varmus's oncogene hypothesis could also be relevant to human cancer were gaining ground.

However, there was still no proof of a clinically relevant connection between HER2 and cancer. In order to establish this, we looked more closely at the clinical development of breast cancer in the 200 patients at UCLA (D. Slamon). The result was clear and convincing: the more HER2 genes there were in the tumor cells, the more aggressive the tumor and the shorter the survival of the affected patient.

This made HER2 the first oncogene that could clearly be shown to be crucially involved in human cancer. Later it turned out that the abnormal signal function of this receptor is also significant in ovarian cancer and in other types of cancer. At the same time, the discoveries suggested a completely new classification for tumors: up to then, cancer had only been characterized by the anatomical location of the occurrence of the primary tumor. Today, we know that for the

treatment of the disease it is at least equally important to know which genes are defective in the errant cells, as this has a significant influence on how aggressive the tumor is and determine which therapy would be most effective.

Using antibodies against cancer

We soon found more proof that HER2 has key significance for the progress of breast cancer. In the mid-1970s, a German and an American scientist, Georges Köhler and César Milstein, developed a technology that opened up unsuspected opportunities in biomedical research. They had found a way to produce monoclonal antibodies against almost every conceivable protein. These are molecules that attach themselves with great specificity only to the type of protein for which they were previously "trained" in the laboratory.

We also used these antibodies for our investigations, including several against HER2, which we had generated. We tested these antibodies in mice into which we had previously implanted tissue from human breast tumors. Lo and behold: some of the monoclonal antibodies against HER2 inhibited the growth of tumors in the mice extremely effectively by blocking the uncontrolled over-function of the receptor - but only when the cancer cells were producing large quantities of HER2.

Resistance to genetic engineering

What could be more obvious than to develop a therapy against cancer that used antibodies? However, although this sounds quite plausible from today's perspective, in the 1980s it was initially too revolutionary even for Genentech, the first genetic engineering company in the world. At the time, company management did not recognize the potential in the new strategy against breast cancer and declined to pursue HER2 as the goal of a new tumor therapy.

There were several reasons for this. First of all, the situation had changed within the ten

years since I had joined Genentech. In 1988, the eight people employed by the company at the time I joined, had grown to 1,800. In such a large company it is very difficult to communicate and realize innovative ideas successfully - which was a sad realization for me.

Secondly, Genentech had suffered some bitter setbacks. In the first years, the company went all-out to find the anti-cancer medicine. However, this strategy proved to be a flop. Two of the substances that were initially considered likely candidates (the "tumor necrosis factor" and interferon) failed. After that, the management decided to stay away from potential anti-cancer medicines.

Antibodies also had a bad reputation at the time - shortly before this, another company had gotten into trouble as the result of a large project it had invested in a treatment for Sepsis. At Genentech, therefore, there was no enthusiasm to continue to support my HER2 project. For me, that was a crucial turning point. It became clear that I was probably too much of a scientist and not enough of a hard-nosed businessman to push through my ideas in a large, commercially-oriented company. Those in charge at Genentech found other projects more promising - and less risky.

Pharmaceutical companies' fear of innovation

Incidentally, this attitude was, and is not at all exclusive to Genentech. I encountered similar situations again and again later on: innovative concepts do not go down well with pharmaceutical companies or with risk capital investors, because most of them do not understand them. Many research managers say that they want something new, but they lack imagination, competence - and courage. It is therefore the case that the majority prefer approaches with which they are familiar. Most real innovation in the cancer field in recent years has come from academic research.

When I received the offer at this time from the Max Planck Society to build up my own department at the Institute for Biochemistry in Martinsried near Munich, I did not hesitate for long. It was clear to me that I would have great freedom to structure my scientific work and to define my scientific goals. For me that was primarily the plan to pursue projects that would lead to novel cancer treatments. *A Happy Ending with Herceptin.* The irony of the story is this: a few years after I returned to Germany, the management at Genentech recognized after all that using an antibody to attack HER2 was a promising prospect for the treatment for breast cancer patients. I had left the antibodies my laboratory and collaborators like Mike Shepard and Brian Fendley had developed there. In the years after that, successful antibodies were tested on breast cancer patients in wide-ranging clinical studies. Ten years later, in November 1998, the medicine was licensed in the USA under the name Herceptin. It has been on the market and available to breast cancer patients in the US since 1998 and in Germany since August 2000.

This area of research remained the focus of my work after my return from the USA to Germany. At that time, I continued the work I had begun at Genentech into receptor tyrosine kinases (RTKs), which also includes HER2. RTKs are proteins that are anchored in the cell membrane and with the appropriate stimulation cause the activation or deactivation of certain genes in the cell nucleus. Early in the 1990s, one of my doctoral students, Birgit Millauer, discovered a tyrosine kinase called Flk-1 for which we were able to show that it plays a key role in angiogenesis, the creation of new blood vessels. Normally, angiogenesis takes place mainly when organs and blood vessels emerge in the development of the embryo. In 1994, we proved that tumor growth also depends on the function of Flk-1. This defined Flk-1 as a target for cancer therapy development.

Shutting off the blood supply to the tumor

Without additional supply channels, a tumor will become at most the size of a pea. In order to continue to grow, cancer cells use a trick. As some point, they begin to secrete growth factors such as VEGF. For the surrounding blood vessels, in whose cells Flk-1 is located as an antenna molecule, this is the signal to allow new capillaries to grow in the direction of the tumor. In this way, it creates for itself its own network of blood vessels which supply its proliferating cells with abundant oxygen and nutrients.

My Ph.D. student Birgit Millauer and myself together with Werner Risau, who at that time was working in the neighboring Max Planck Institute for Neurobiology, discovered, in further experiments, that a deliberate and specific inhibition of the function of Flk-1 stopped the development of the blood vessel system in a tumor and halted its growth. This made it clear that Flk-1 had an important function in tumor angiogenesis - and that we wanted to search for a substance with which to block the activity of the receptor both non-invasively and deliberately in the body of patients.

After my experiences at Genentech, one thing was clear to me: if we wanted to make our discovery usable for medicine and develop a proper drug, we would either have to work together with a company or found one ourselves. For this reason, I founded SUGEN in California in 1991 with a colleague from New York University.

What today sounds so simple, back then took a lot of effort on my part to convince important people because a company “spin-out” was a novelty for the Max Planck Society. It was the first foundation of a biotechnology company from one of their institutes. There were a number of people that had serious reservations. However, with the help of the head of the Max Planck Technology Transfer office, Dr. Heinrich Kuhn, we soon found a way to suit everyone involved. Know-how and patents associated with Flk-1

and numerous other tyrosine kinases were licensed by the Max Planck Society to SUGEN in the following years.

SUGEN then developed numerous chemical substances that blocked the Flk-1/VEGFR2 receptor, among others. One of these proved to be particularly effective: the angiogenesis inhibitor SU11248. SUGEN was taken over in 1999 by the pharmaceutical company Pharmacia and bought by the US group Pfizer in 2003, which continued to develop the substances. To great success: at the beginning of 2006, SU11248 was approved by the FDA for the American market under the trade name SUTENT for the treatment of kidney cancer and a specific form of gastrointestinal cancer; a few months later, it was also approved in Europe for the same cancer indications.

Unlike Herceptin, which targets a single genetic defect in breast cancer cells for correction, it turned out that SUTENT attacks tumor cells with many different mechanisms at several points (termed targets) simultaneously. In fact, we now know that SUTENT is a “multi target medication”. It not only inhibits angiogenesis, but also blocks more than 70 kinases and prevents the progression of the disease on several fronts, including metastasis formation. In many cancer patients, it shrinks the tumors or practically kills them off. This is probably not just the case for the two forms of cancer for which it has been licensed so far, but also for others. In fact, it is now being clinically tested for application against other, more frequently occurring cancers.

The new strategy: multiple attacks instead of a single front war

The work on SUTENT also brought with it a surprise. Cancer researchers have long pursued the ideal goal that a tumor drug should only target one weak point of the diseased cells, so as to reduce the risk of causing severe side-effects. This is because, or so the theory goes, the fewer points a drug targets, the less likely it is that it will also

intervene in those areas that are required for normal body functions, thus reducing the risk of side-effects.

Amazingly, however, one of the recipes for success with SUTENT is that it does work on several targets in the tumor cell - a characteristic that we actually wanted to prevent. In a way, the success was forced upon us, because SUTENT's predecessor SU5416 was specific in tests, but not particularly effective. We searched, therefore, for a slightly different substance - and made a virtue of a necessity. It turned out that the active substance in SUTENT attacks cancer cells at several points simultaneously and is especially effective for precisely this reason. This yielded two crucial findings: first, it is possible to inhibit a whole series of important processes in tumor growth without causing serious side effects. This takes for granted, however, that all of these weak points are largely essential for the survival of cancer cells.

Secondly, we presumably have the best chances to develop improved cancer

therapies if we attack the cancer cells from several sides. One of the major characteristics of cancer cells is that their genetic material is not stable. This means not only that they change constantly, but also that different cells in the same tumor and in the metastases have different mechanisms to avoid the body's own immune system or the effect of certain medications.

I am therefore convinced that we will not conquer many types of cancer with a "single front" war. Tumors will only be successfully controlled when we combine several drugs that have different mechanisms of action. Furthermore, we must also look for strategies that activate the patient's immune system.

Even if that should prove successful, I doubt that we will really be able to cure major cancers within the space of a few years. However, in my view, we will have achieved a great deal if we can successfully hold most tumors in check for many years, turning cancer into a manageable, chronic disease.

2009 Pezcoller Foundation - ECCO Recognition for Contribution to Oncology

Call for Nominations

ECCO - the European CanCER Organisation and the Pezcoller Foundation are pleased to announce the **Call for Nominations for the 2009 Pezcoller Foundation - ECCO Recognition for Contribution to Oncology**.

For 2009 in collaboration with ECCO - the European CanCER Organisation, **the Pezcoller Foundation - ECCO Recognition for Contribution to Oncology will be awarded to a single individual for his/her professional life dedication to the improvement of cancer treatment, care and research. The award is open to all professions and specialties within the oncology field.**

Nominations for the 2009 Pezcoller Foundation - ECCO Recognition for Contribution to Oncology will be accepted for candidates irrespective of race, sex or nationality. Institutions, groups or associations are not eligible. Self-nominations will not be considered. Candidates must be nominated on the official form by one who is, or has been, affiliated with a university or medical institution.

A curriculum vitae and description of the professional contribution to the field of oncology of the candidate should be included with the application form.

Nominators are requested to keep their nomination confidential and to refrain from informing the nominee. The awardee will be selected by an International Committee appointed by ECCO's President with the agreement of the Council of the Pezcoller Foundation. The decision concerning the 2009 winner will be taken in April 2009.

The award consists of a prize of 30.000 Euros and a commemorative plaque. The award ceremonies will be held in Rovereto (Italy), on Friday 11 September 2009 and in Berlin, during the joint ECCO 15 – 34th ESMO Multidisciplinary Congress as a Plenary Lecture to be delivered during the Presidential Session on Tuesday 22 September 2009.

More Information:

For more information about the nomination process and to submit a completed form and support documentation please contact Carine Lecoq:
Tel: + 32 2 775 29 31, Fax: +32 2 775 02 00,
Email: carine@ecco-org.eu

Nomination forms can be downloaded at: www.ecco-org.eu (select "Education" > "Awards") and must be completed and received by **31 January 2009**.

Award Committee

Chair: Alexander M.M. Eggermont (NL),
President of ECCO - Chair

Gios Bernardi (IT), President of the Pezcoller Foundation
Franco Cavalli (CH), Foundation for Research and Cure of Lymphomas, Tessin

Raffaella Giavazzi (IT), "Mario Negri" Institute for Pharmacological Research, Bergamo

Jacques Pouyssegur (F), Institute of Signalling, Developmental Biology and Cancer Research, Nice

Ulrik Ringborg (SE), Karolinska Institute, Stockholm

Ex-officio members:

Michel Ballieu (BE), Chief Executive Officer of ECCO

Alberto Costa (IT), Director of ESO

About the Pezcoller Foundation

The Pezcoller Foundation was established in 1980 through a most generous donation from Professor Alessio Pezcoller, a dedicated Italian surgeon who devoted his life to his profession. Professor Pezcoller not only made important contributions to medicine but through his generosity and foresight, provided his lifetime's savings for others to do the same.

Formerly, until 1997, the Pezcoller Foundation presented an award in collaboration with the European School of Oncology (ESO). The Pezcoller Foundation - ECCO Recognition for Contribution to Oncology build on this tradition.



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2010 Pezcoller Foundation-AACR International Award for Cancer Research

The prestigious Pezcoller Foundation-AACR International Award for Cancer Research was established in 1997 to annually recognize a scientist:

- who has made a major scientific discovery in basic cancer research or who has made significant contributions to translational cancer research;
- who continues to be active in cancer research and has a record of recent, noteworthy publications;
- whose ongoing work holds promise for continued substantive contributions to progress in the field of cancer.

The Award is intended to honor an individual scientist. However, more than one scientist may be co-nominated and selected to share the Award when their investigations are closely related in subject matter and have resulted in work that is worthy of the Award. In the rare event that there are dual winners of the Award, the cash award will be shared equally between them, and the AACR Executive Committee will determine which of the two co-recipients will present the Pezcoller-AACR Award Lecture at the AACR Annual Meeting.

Candidates for the Award will be considered by a prestigious international Selection Committee of renowned cancer leaders appointed by the President of the AACR and the Council of the Pezcoller Foundation. The Committee will consider all nominations as they have been submitted; the Committee may not combine submitted nominations, add a new candidate to a submitted nomination, or otherwise make alterations to the submitted nominations. After careful deliberations by the Committee, its recommendations will be forwarded to the Executive Committee of the AACR and the

Council of the Pezcoller Foundation for final consideration and determination.

Selection of the Award winner will be made on the basis of the candidate's scientific accomplishments. No regard will be given to race, gender, nationality, or religious or political view.

The Pezcoller Foundation was established in 1980 by Professor Alessio Pezcoller, a dedicated Italian surgeon who made important contributions to medicine during his career and who, through his foresight, vision and generous gift in support of the formation of the Foundation, stimulated others to make significant advances in cancer research. Previously the Pezcoller Foundation, gave a major biennial award for outstanding contributions to cancer and cancer-related biomedical science, in collaboration with the ESO-European School of Oncology. The American Association for Cancer Research (AACR) was founded in 1907 by eleven physicians and scientists dedicated to the conquest of cancer and now has over 25,000 laboratory, translational, clinical and epidemiological scientists engaged in all areas of cancer research in the United States and in more than 60 other countries around the world.

The AACR is dedicated to its mission of preventing and curing cancer through the communication of important scientific results in a variety of forums including publications, meetings and training and educational programs. Because of the commitment of the Pezcoller Foundation and the AACR to scientific excellence in cancer research, these organizations are now collaborating annually on the presentation of the Award. This will strengthen international collaborations and will be a catalyst for advancements in cancer research internationally.

The winner of the Pezcoller Foundation-AACR International Award for Cancer Research will give an award lecture during the AACR Annual Meeting (April 2010), and the memorial Korsmeyer lecture at the VIMM in Padua and will receive the award in a ceremony at the Foundation's headquarters in Trento, Italy (May, 2010). The award consists of a prize of € 75.000 and a commemorative plaque.

Nomination Deadline: September, 2009

Questions about the nomination process: Monique P. Eversley, Staff Associate - American Association for Cancer Research, 17th Floor, 615 Chestnut Street, Philadelphia, PA 19106-4404 - Tel. +1 (267) 646-0576; E.mail: eversley@aacr.org - www.aacr.org

2009 Pezcoller Symposium Unconventional therapeutic targets in cancer

The 21st annual Pezcoller Symposium entitled, "Unconventional therapeutic targets in cancer", will be held in Trento, Italy, on June 11–13, 2009. The Symposium will be co-chaired by Pier-Paolo Pandolfi and David Livingston (Dana Farber Cancer Institute, Boston, MA), and its Program Committee will include Enrico Mihich and William Kaelin. The focus of the Symposium will be on identifying new targets for anticancer drugs action, and on utilizing new concepts and methods for drug design and development. The topics to be discussed will include protein/protein interactions, targeting transcription factors, targeting death pathways, new chemical screening of novel molecular targets, valid alternatives to known target proteins, and unexpected effects

of disrupted angiogenesis. Each talk will be followed by an equal time for discussion thus providing ample opportunities for interactions and cross-fertilization among participants. A session of posters in the areas discussed at the Symposium will be included. Poster abstracts should be submitted to Enrico Mihich (Roswell Park Cancer Institute, Buffalo, NY, Enrico.mihich@roswellpark.org) on or before April 15, 2009 and will be reviewed for acceptance by the Co-chairmen and E. Mihich. For scientific matters contact Dr. Pandolfi [ppandolf@bidmc.harvard.edu] or Dr Livingston [david_livingston@dfci.harvard.edu]; for local arrangements and travel matters contact Pezcoller Foundation, secretariat office, Trento, Italy [Pezcoller@pezcoller.it].

Save the date!

21st Pezcoller Symposium

June 11 - 13, 2009
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Unconventional therapeutic targets in cancer



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