



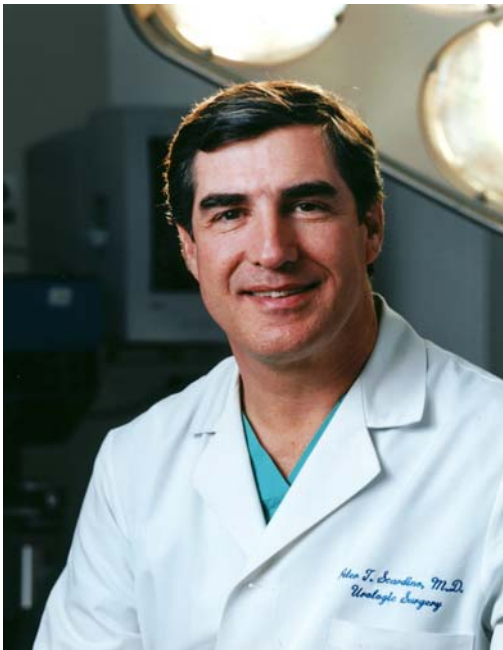
Newsletter

Prostate Cancer 101, Inc.

<http://prostatecancer101.org>
November, 2005

The Prostate Cancer Information and Support Group of the Mid-Hudson

Our November 15 Guest Speaker is Dr. Peter T. Scardino



Peter Scardino, MD, Chair, Urology Dept. and Head, Prostate Cancer Program, Memorial Sloan-Kettering Cancer Center, NY, NY will be the November 15, 2005 speaker in our Distinguished Lecture Series. The topic of his presentation will be "The Treatment of Early Stage Prostate Cancer. A 25 Year Perspective".

In 1967 Dr. Scardino did his undergraduate work at Yale Univer-

sity in Religious Studies and obtained his MD at Duke University and his Residency in Urology in UCLA School of Medicine. Since 1998 he has been in his present position at MSKCC in addition to being a Professor of Urology at Weill Medical College of Cornell University, NY, NY.

Dr Scardino is internationally recognized for his work in urologic oncology, particularly with regard to the natural history, early detection, prognosis and treatment of prostate cancer. Among his major research interests are markers of prognosis in prostate cancer, informatics tools for predicting prognosis and response to therapy, imaging studies to stage prostate cancer, and surgical methods for improved outcome. Dr Scardino is Principal Investigator of MSKCC's Specialized Program of Research Excellence

(SPORE) in Prostate Cancer, funded by the National Cancer Institute, and heads a team of investigators in collaboration with Cancer Research UK, studying the natural history of prostate cancer.

Recognized for his excellent teaching, Dr Scardino has held visiting professorships at academic institutions in the US, Canada, Europe, Japan and Australia. Among his numerous honors and awards are the Fuller Triennial Prostate Award from the American Urological Association, the Presidential Citation from the American Foundation for Urologic Disease, being the Keynote Speaker at the American Association for Cancer Research conference in 2001 and the Folke Edsmyr Memorial Lecturer at the Karolinska Institute in Stockholm in 2003. He has been named one of the "Best Doctors in New York City" by New York magazine every year since 2000.

HER2/Neu Gene Associated With Breast Cancer May Play Major Role In Prostate Cancer Recurrence Herceptin may not be the key therapy

April 20; updated Oct 24, 2005-- The HER2/neu gene, associated with breast cancer, may also play a major role in the recurrence of prostate cancer, according to research from the University of North Carolina at Chapel Hill School of Medicine and UNC Lineberger Comprehensive Cancer Center.

Herceptin, a drug which targets HER2/neu and has "stunning" effects for some women with breast cancer. Another drug called lapatinib (GlaxoSmithKline) is now in clinical trial to see if it blocks HER2/neu's effects in men with recurrent or metastatic prostate cancer. This trial is running at several Canadian centers and at Fox Chase Cancer Center, Philadelphia, Pennsylvania (see [link](#) below).

The most common cancer in men, prostate cancer can be effectively treated with surgery or radiation when detected early. But advanced prostate cancer is usually treated by drugs or surgery aimed at reducing the level of testosterone and other male hormones, or androgens, that stimulate cancer cell growth. While the disease usually regresses after such treatment, prostate cancer invariably comes back, although it's not clear why it recurs and progresses.

The UNC study, published last April 15 in the journal *Cancer Research*, indicates that the gene HER-2 is a key culprit in prostate cancer recurrence. The findings also suggest a new treatment strategy for targeting HER-2 in patients with advanced prostate cancer.

HER-2 refers to human epidermal growth factor receptor 2. The gene helps control how

cells grow, divide and repair themselves, and directs the production of a special protein called HER-2 tyrosine kinase. This protein acts as receptors on the cell membrane, and when activated by external hormones, it promotes cell growth and division.

In about one in four breast cancers, a genetic mutation creates too many HER-2 receptors. This helps spur rapid cancer cell growth. While treatment with the antibody drug Herceptin can be effective in slowing breast cancer growth, this is not the case in prostate cancer, researchers said.

"The treatment with the antibody has been a uniform failure in prostate cancer because the gene is not over-expressed in this disease. We need a different approach to attack HER-2 in prostate cancer," said the study's senior author, Dr. Young Whang. He is an assistant professor of medicine and medical oncologist at UNC and a member of UNC Lineberger.

"We believe that the driving force for recurrence of prostate cancer is the reactivation of the androgen receptor, which normally requires the presence of androgen, and this reactivation of the androgen receptor underlies tumor progression of prostate cancer despite hormonal therapy. Exactly how this occurs, we're not sure, but our hypothesis is that activation of HER-2 tyrosine kinase leads to activation of the androgen receptor."

In testing their hypothesis, Whang and his co-authors inhibited HER-2 activity in two laboratory experiments involving human cancer cells. In the first, they used an artificial antibody to HER-2 delivered directly into the cells via a modified virus. In the second, they used an experimental drug that specifically inhibits HER-2 tyrosine kinase activity. In both experiments, tyrosine kinase activity and androgen receptor function were largely derailed.

"We discovered that inhibition of HER-2 strongly inhibits proliferation of prostate cancer cells and the function

of androgen receptor," Whang said.

To properly carry out its function, the androgen receptor protein binds specifically to the regulatory DNA sequence of the genes regulated by androgens such as testosterone, he said. "And we have shown that inhibition of HER-2 impairs the androgen receptor function at this step of binding to the DNA sequence of critical genes such as prostate specific antigen."

The implication of this work, he added, is that HER-2 is important and necessary for prostate cancer viability and progression.

"This provides the rationale for initiating a clinical trial of this novel drug inhibiting HER-2, which is being planned for patients within several months," Whang said. "I envision this drug becoming one of several that could be used in combination with other specifically targeted drugs to prolong the lives of prostate

cancer patients."

UNC co-authors with Whang include postdoctoral researchers Drs. Yuanbo Liu and Samarpan Majumder; Wesley McCall, research technician; Dr. Carolyn Sartor, assistant professor of radiation oncology; Dr. James Mohler, professor of surgery; and Dr. Shelton Earp, director, UNC Lineberger. Dr. Christopher Gregory, former UNC assistant professor of pathology and another co-author, is now with Voyager Pharmaceutical Corp. in Raleigh.

The research was supported by grants from the U.S. Army Medical Research and Materiel Command and the National Cancer Institute, a component of the National Institutes of Health.

Original news release by LESLIE H. LANG, UNC School of Medicine. Updated by J. Strax, psa-rising.com

psa rising

Findings identify likely origins of prostate cancer

By Liz Szabo, USA TODAY

Researchers have found a set of genes that may play a key role in prostate cancer — a discovery that doctors are hailing as a major breakthrough that changes the way they think about the genetic roots of the disease.

If further research confirms these findings, published Friday in the journal *Science*, the discovery eventually might lead to better tests for prostate cancer as well as targeted therapies, says one of the study's authors, Mark Rubin, chief of urologic pathology at Brigham and Women's Hospital in Boston.

"This is amazing," says Michael Heinrich, a professor at the Oregon Health & Science University Cancer Institute, who was not involved in

the study. "This is the Rosetta stone of prostate cancer. Cracking the code lets you read the whole library. The implications of this are huge in a lot of different ways."

About 232,000 men a year are diagnosed with prostate cancer.

Until now, doctors thought it was the result of lots of random genetic mutations, Heinrich says. This study, however, suggests for the first time that prostate cancer begins after specific genes fuse.

Doctors found these merged genes in nearly 80% of 29 prostate cancer samples, says Arul Chinnaiyan, a professor at the University of Michigan Medical School who directed the study. None of the 50 samples of non-cancerous tissue had the genes, he says.

This may allow doctors to begin to divide prostate cancer — which is now treated as a single disease — into different types. Doctors have been treating breast cancer this way for years: They prescribe the drug Herceptin to women whose tumors make too much of certain protein, and they give the drug tamoxifen to those whose tumors respond to hormones.

So far, Chinnaiyan and his colleagues have found fused genes only in prostate tissue. They are trying to see whether they can detect the genes in blood or urine, which could allow them to develop a more accurate diagnostic test for prostate cancer.

Chinnaiyan also hopes the genes will tell doctors which tumors are deadly and require aggressive treatment. That could allow men whose tumors are relatively harmless to avoid treatment and its side ef-

Bostwick Laboratories Announces uPM3(TM) Test, First Genetic Test for Prostate Cancer

fects. Doctors now have few good ways to tell these men apart, leading about half to undergo unnecessary therapy, says Otis Brawley, medical director of Grady Health System's Georgia Cancer Center for Excellence.

Chinnaiyan says his discovery may allow doctors to develop new treatments. Chronic myeloid leukemia patients can live for years without serious side effects thanks to the drug Gleevec, which was developed after scientists discovered the cancer's genetic roots.

Brian Druker, the scientist who developed Gleevec, says it could take years or even decades to develop a targeted therapy for prostate cancer. But these genes at least give scientists a target, a critical first step. "This is incredibly important," Druker said in an e-mail. "Finding the cause gives us hope for finding a cure."

Source: USA Today

RICHMOND, Va., Sep. 23 /WIREDBIRD/ -- Bostwick Laboratories of Richmond, VA announced today its introduction of the uPM3(TM) test, the first-ever urine-based genetic test for prostate cancer.

Supportive clinical data are so compelling that Bostwick Laboratories has decided to offer the test for their patients immediately. uPM3(TM) is licensed from DiagnoCure Inc. of Quebec, Canada, who holds worldwide patent rights for the diagnostic and therapeutic application of the PCA3 gene. uPM3(TM) is based on PCA3, a specific gene that is profusely expressed in prostate cancer tissue (on average, 34 times greater than in benign prostate tissue). No other human tissues have ever been shown to produce PCA3.

The uPM3(TM) test predicts

cancer in prostate biopsy with 81% accuracy, compared to 47% accuracy for prostate specific antigen (PSA). Multiple studies reported at several urology meetings throughout 2002 and 2003 have confirmed that the test helps urologists solve the significant diagnostic dilemma of men who have an elevated PSA and a negative biopsy, but who are strongly suspected of having prostate cancer.

Patients who receive the uPM3(TM) undergo a thorough digital rectal prostate examination by a urologist, a standard procedure in prostate cancer detection. This exam causes cells from the patient's prostate to be shed into the urine, and the urine sample, containing the released cells, is sent to Bostwick Laboratories to be tested for genetic ex-

pression of the PCA3 gene. If the sample is positive for PCA3, then the patient has a very high likelihood of having prostatic adenocarcinoma. The uPM3(TM) is currently available in the U.S. exclusively from Bostwick Laboratories.

According to Dr. Bostwick, Medical Director and CEO of Bostwick Laboratories: "The sensitivity and specificity of uPM3(TM) surpasses PSA and all other existing prostate cancer detection tests other than biopsy. Introduction of this new and exciting test by our laboratory underscores our commitment to provide breakthrough technology to our physician clients and the patients we serve."

Pierre Desy, President and CEO of DiagnoCure says: "We are very excited to work with Bostwick Labora-

tories to bring this important new test to the market. Dr. Bostwick is one of the most respected pathologists in the world specializing in urologic diseases. Bostwick Laboratories' ability to offer the test is a significant endorsement of the importance of measuring the PCA3 gene as part of a prostate cancer detection protocol."

Bostwick Laboratories is a leading private anatomic pathology reference laboratory based in Richmond, VA. Its mission is to help physicians and patients make informed treatment decisions when faced with the diagnosis of cancer by providing the best in diagnostic accuracy and technical and scientific expertise. Bostwick Laboratories focuses on the diagnosis of cancer of urologic organs (prostate, urinary bladder, kidney, and testis), with a special interest in prostate cancer.

DiagnoCure Inc. specializes in the development and production of diagnostic tests

for the detection of cancer. DiagnoCure's mission is to become a leading developer of highly accurate immunoassay and molecular tests enabling the early detection of cancer. The company is traded on the Toronto Stock Exchange under the symbol CUR.

For more information about Bostwick Laboratories or uPM3(TM), contact Bostwick Laboratories at 1-804-288-6564.

Bostwick Laboratories
CONTACT: Bostwick Laboratories, +1-804-288-6564

W e b s i t e :
<http://www.bostwicklaboratories.com/>

DiagnoCure
<http://www.diagnocure.com/anglais/section3/section33.asp>
psa rising

Nanotechnology May Help Treat Cancer

By EMMA ROSS, AP Medical Writer Tue Nov 1, 5:18 PM ET

Experiments on mice have shown promise for the future of nanotechnology in treating cancer — research that could bring doctors a step closer to using the technology to release cancer-killing drugs inside tumors while leaving the rest of the body unscathed.

After seeing how some mice were cured of human prostate cancer with the technology, cancer specialists at the European Cancer Conference in Paris said Tuesday they had high hopes for its future application.

"There are a lot of candidates for intelligent carriers and these nanoparticles are among them," said Dr. Gordon McVie, a professor at the European Institute of Oncology in Milan, Italy, who was not involved with the research.

"This is a new system, and

the more systems we have, the better, because we'll probably be lucky if we get one system to work out of 10," he said. "It looks as if it could be quite good."

Dr. David Kerr, a professor of clinical pharmacology and cancer therapeutics at Oxford University in England who also was not connected with the research, said the approach may have the edge on others. Previous designs of nanoparticles have used antibodies to zone in on cancer cells.

"The body's immunodefense system can create antibodies to the therapeutic antibodies, deactivate them and prevent the antibody binding to the right cancer cells. This looks like a step forward," Kerr said.

Nanotechnology is the science of manipulating matter smaller than 100 nanometers and taking advantage of

properties that are present only at that level, such as conductivity. A nanometer is one-billionth of a meter, or about one-millionth the size of a pin head. The prefix comes from "nanos," the Greek word for dwarf.

Nanotech has been around for several decades, but only now is its potential starting to be realized. Medicine is expected to be one of the fields to benefit most from the technology. In cancer, it is hoped the technology will allow for more precisely targeted drugs and surgery and less toxic chemotherapy.

The study, done by scientists at Harvard Medical School and the Massachusetts Institute of Technology, involved engineering nanoparticles embedded with the cancer drug Taxotere. The particles were then injected into human tumors created from prostate cancer cell lines and implanted into the flanks of

mice. The mice were watched for 100 days.

The technology being tested involves a nanoparticle made of a hydrogen and carbon polymer with bits of drug bound up in its fabric and attached to a substance that homes in on cancer cells. The polymer gradually dissolves, exposing the nuggets of drug little by little.

The mice were divided into five groups, including one that had their tumors injected with ineffective saltwater. A second group died after injections of a nanoparticle containing no drugs.

Another group was given one shot of the drug, experienced an initial decrease in tumor size and then suffered a strong rebound. They also died.

Other mice were injected with a nanoparticle-encased drug, but one that was not designed to specifically target cancer cells.

"What happens here is the lymphatic system of the tumor can take it up and wash it away, because the nanoparticle is not targeted to the cancer cells," said the study's presenter, Dr. Omid Farokhzad of Harvard Medical School. The tumor initially shrank to half its original size, but then rebounded.

In a final group of mice, scientists injected the targeted nanoparticles containing the drug.

"The tumor completely disappeared," Farokhzad said. Injecting targeted nanoparticles into the bloodstream and having them seek out tumors and get inside on their own is the ultimate goal, but direct injection is also promising for cancers where the tumor is accessible and hasn't spread, such as early prostate cancer, Farokhzad said. He said his group hopes to test the approach in prostate cancer patients within two years.

Kerr said he doubted that di-

rect injection of tumors would turn out to be a useful treatment in itself. "Cancer tends, almost from the outset, to be a systemic disease," he said. "This is only one design step toward what ultimately must be a systemic treatment."

He said there may be a use for the direct injection of nanoparticles to deliver vaccines to tumors. The idea is that the cancer can be vaccinated against itself, meaning the immune system would then destroy the cancer in other parts of the body.

Yahoo!News

Past Year's DVDs?

If you would like a DVD or video tape of any of this past year's lectures, get in touch with Yavuz Birturk and specify which lecture and format you would like. The cost for each is \$15.00 and includes mailing.

His address is PO Box 142, Cottkill, NY 12419.

A Wake Up Call!

In order for this group to continue, we need a few more good people to help do some of the everyday work to keep us going. We are not talking about more than a half hour to one hour of your time a month; not an onerous commitment. We need help with advertising, programs and lectures. Most of this is already in a format, you simple have to follow it.

Next year, we will have a much more limited lecture series, mostly due to lack of attendance for the amount of work involved. Help is needed to contact willing lecturers, set up a time schedule (spring or fall) and make the complete arrangements. Are you willing to help, even a little bit?

The newsletter will be available on line and will only be published four times next year. The hours spent each month in compiling information, getting it ready for printing and mailing and distribution is not cost effective, especially when so few show up for a lecture.

We also need to know if any of you have an interest in serving on the Board of Directors or as an Officer of PCa101. Meetings of the Board will also be cut back, as there will be fewer lectures and items to discuss.

We not only need your feedback, we need your interest and help. It is time to stand up and be counted! Will you be one of the people who care?

Diane Sutkowski, Secretary Treasurer

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What is Phase Zero?

Companies embrace preclinical microdosing studies to try to save millions in development costs By [Randall C. Willis](#)

At one time, the drug development process had three phases. Then, Phase IV, post-marketing surveillance, became part of the picture. But that has done nothing to prevent late-stage Phase III failures and recent high-profile, postrelease Phase IV failures of drugs such as Vioxx because of adverse side effects.

"It is well known that the approval rate for innovative new drugs is declining," says Dr. David Jacobson-Kram, associate director for Pharmacology and Toxicology at the Food and Drug Administration's Center for Drug Evaluation and Research (CDER) in Silver Spring, Md. "Additionally, development costs for new drugs are rising dramatically. A large factor in the increased costs is that many drugs are failing late in development [Phase III trials]."

Some of these failures can be attributed to poor or poorly understood pharmacokinetic (PK) parameters and the fact that regardless of how well characterized a compound's behavior is in vitro or in animal models, these systems are imperfect representatives of human physiology. Some 30%–40% of new drugs fail

due to poor performance at the transition from animal to human trials, according to a November 2004 presentation to the FDA by Michael Chansler, vice president of business development for Accium Biosciences, an analytical service company in Seattle. To mitigate the risks and costs associated with late-stage failures, companies have recently looked to a new method of testing compounds earlier in humans: Phase Zero.

SMALL DOSES

These microdosing studies involve the administration of subpharmacologic or subtherapeutic doses (on the order of micrograms) of a drug candidate to humans, who are monitored to generate a preliminary ADME or PK profile (see Sidebar). It is hoped that giving companies earlier, safer data on how the drug is processed in the body will dramatically accelerate the more expensive clinical testing phase.

"Although the Phase Zero approach is not appropriate for all compounds, when thoughtfully applied, Phase 0 techniques help developers select only the most promising drug candidates for further development by mitigating the risk of failure due to poor PK and bioavailability character-

istics in humans," says Chansler. "For early-stage pharma and biotech firms, Phase Zero testing is a cost-effective way to increase value by providing first-in-human data earlier in the development/investment cycle."

Others are less convinced. At the June 2005 meeting of the Canadian Society for Pharmaceutical Sciences held in Toronto, several participants expressed concern about the validity or use of Phase Zero data, considering that typical microdosing experiments rely on 1% or less of the final therapeutic dose. "One must take into account possible differences in the PK of a subpharmacological dose compared to a full pharmacological dose," says Chansler. "Compounds that have transporter-mediated metabolism, high first-pass metabolism, or are tightly bound to plasma proteins or target-binding sites may not have comparable PK properties between a microdose and a full dose."

To some extent, the recent CREAM trial – the Consortium for Resourcing and Evaluating AMS (accelerator mass spectrometry) Microdosing – addressed these concerns. Companies such as Eli Lilly and Company, Schering-Plough, and Roche sponsored the trial. The CREAM

trial compared the micro- and pharmacologic-dose PK profiles of five compounds considered to be representative of the type of compounds that present researchers with preclinical PK problems: warfarin, the anti-estrogen ZK253, diazepam, midazolam, and erythromycin.

Three of these drugs showed microdose-PK results reflective of pharmacologic doses, while the other two provided "useful insights into the properties of the drugs," according to the CREAM researchers.

REGULATING PHASE ZERO

The Europeans took the early lead in presenting an opinion on the efficacy of Phase 0 analysis when the European Agency for the Evaluation of Medicinal Products (EMA) put out a position paper in early 2003. The paper supported the use of microdosing as nonclinical safety studies in support of further clinical studies, and it defined a microdose as 1/100th the dose required to present a pharmacologic effect, and no more than 100 grams. This past April, the FDA went a step beyond the EMA paper by issuing a draft guidance document relating to exploratory Investigational New Drug (IND) applications and which included reference to the use of microdosing as part of this process.

The guidance was designed to

give sponsors the opportunity to test drugs in humans earlier in development so that the most promising new chemical entities can be selected and advanced, while those destined to fail can be eliminated early, according to CDER's Jacobson-Kram. "The EMA position paper deals only with microdose studies," he explains. "These allow only single, nonpharmacologic doses and provide information only on pharmacokinetics. The FDA guidance also discusses the option of performing repeat-dose clinical studies using doses designed to induce pharmacological effects. These latter types of studies provide much more information regarding potential efficacy."

When implemented, says Chansler, the new guidance should save companies millions of dollars in development costs in short order. In the traditional IND, he explains, preclinical toxicology and safety requirements cost more than \$650,000 and can take as long as six months to perform. Based on the abbreviated toxicology and safety requirement described in the Exploratory IND draft guidance, however, a human microdosing experiment can be initiated with less than \$150,000 in preclinical toxicology and safety testing, which can be completed within one month.

"Another important financial consideration is the savings in test-compound synthesis," he adds. "To fulfill CMC requirements for pilot, batch, and manufacturing scale synthesis of a test compound using the traditional IND approach, a company will spend approximately \$1.2 million over a 12-month period. A microdosing study requires only a pilot-scale synthesis run, which can often be achieved for below \$500,000 in less than six



THREE ROADS TO PHASE ZERO

In many respects, Phase Zero studies are possible only because of the technical advances in detection instrumentation – accelerator mass spectrometry (AMS), positron emission tomography (PET), and liquid chromatography-tandem mass spectrometry (LC-MS/MS) – that allows almost single-molecule detection. Here, Ali Arjomand, president and chief operating officer of Accium Biosciences in Seattle, outlines each technique's strengths and weaknesses.

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"And don't forget the significant opportunity cost of scientific development teams remaining preoccupied with lead compounds that could have been eliminated already by Phase Zero study," says Royce Morrison, director of medical affairs at Northwest Kinetics in Tacoma, Wash., a company that conducts clinical research studies on behalf of clients.

NOT A PANACEA

David Shoultz, vice president of business development at Ockham Development Group, a drug development company in Tacoma, Wash., warns that companies shouldn't see the Exploratory IND as a development shortcut. "We believe that it is imperative for pharmaceutical and biotechnology companies to be certain to choose the appropriate type of IND [exploratory vs. traditional] to file based on their objectives, candidates, and research and development objectives," he says.

"Regulatory issues could arise if a company tries to conduct studies under an exploratory IND that would be more appropriate under a traditional IND. We are concerned that companies not try to use the exploratory IND process as a shortcut without understanding the full implications."

At the same time, Shoultz adds, Phase Zero and microdosing studies represent an area in which forward-thinking service organizations can help to differentiate themselves, as well as help to provide guidance and expertise to their clients in the process. He compares the current opportunities to those of the industry in electronic data capture (EDC) five to seven years ago.

"Those organizations that have made investments in understanding and defining the in-

dustry landscape with respect to EDC systems, implementation, and electronic submissions to the FDA are really able to serve their clients more adeptly and completely," he says. "As such, many of these organizations have been rewarded by earning the trust and business of their pharmaceutical and biotechnology clients. By leveraging expertise in bridging from preclinical to clinical development, pharmacokinetics, and drug development, a CRO focused on the early stages of clinical development can really provide a valuable service to clients in providing guidance on when to conduct Phase Zero and microdosing studies, how to conduct them, and how to use the data gathered from these studies."

Microdosing won't be a panacea for what ails the industry, says Jacobson-Kram. "It provides early information on pharmacokinetics and can eliminate further development of drugs with poor bioavailability. This will by no means identify drugs that fail later in development because of safety or efficacy issues. It's a tool, not a magic wand."

The Scientist

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