

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE PATENT TRIAL AND APPEAL BOARD

CURIUM US LLC,

Petitioner,

v.

**UNIVERSITÄT HEIDELBERG AND THE EUROPEAN ATOMIC
ENERGY COMMUNITY (EURATOM), REPRESENTED BY THE
EUROPEAN COMMISSION,**

Patent Owners

Case No. IPR2025-01582

Patent No. 10,980,901 B2

DECLARATION OF PROF. DR. ALFRED MORGENSTERN

1. I, Prof. Dr. Alfred Morgenstern, submit this declaration in the above-referenced proceeding in support of Patent Owners' Brief Requesting Discretionary Denial of Institution, Patent Owners' Preliminary Response Pursuant to 37 C.F.R. § 42.107, and any subsequent filings by Patent Owners in this matter.
2. I have personal knowledge of the matters set forth in this declaration unless otherwise indicated.
3. I am a named inventor on U.S. Patent No. 10,980,901 B2 ("the '901 patent") and was integrally involved in the development of the claimed invention. I am a full-time researcher at the European Commission Joint Research Centre (JRC) and collaboration partner of Heidelberg University Hospital, and my statements are based on my education, training, experience, and personal knowledge unless otherwise indicated.
4. I am not being separately compensated for my work preparing this declaration.

The statements in this declaration are true and correct to the best of my knowledge and belief and are based on my education, training, experience, and personal knowledge unless otherwise indicated.
5. My current contact information is:

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I. PERSONAL BACKGROUND

6. I am a research scientist with the European Commission's JRC in Karlsruhe, Germany, where I lead activities focused on radioisotopes for medical applications. Since 2017, I have served as Head of Sector for Radioisotopes in Medicine and Technology. My work centers on the development, preclinical evaluation, and clinical translation of targeted alpha therapy radiopharmaceuticals.
7. I earned a Diploma (Degree) in Chemistry from the Technical University of Munich in 1994 and subsequently received a Doctorate in Natural Sciences (a Ph.D. in Radiochemistry) from the Technical University of Munich in 1997. My early research training included positions as a Research Scientist at the Institute for Radiochemistry at the Technical University of Munich and postdoctoral research in the Department of Chemistry at Florida State University. I joined the European Commission's Joint Research Centre in 2000, first as a postdoctoral fellow in the Nuclear Chemistry Unit and, two years later, became a Research Scientist there.
8. Within the JRC, I have held leadership roles advancing nuclear science for health, including service as Action Leader for alpha-immunotherapy (2009–2013) and Project Leader for Targeted Alpha Therapy since 2014. My research team at JRC Karlsruhe has pioneered targeted alpha therapy approaches for

oncology, with an emphasis on actinium-225–labeled compounds. In 2013, our group, in conjunction with the other inventors on the '901 Patent, first developed ^{225}Ac -PSMA-617, which demonstrated remarkable efficacy in the treatment of prostate cancer and is undergoing clinical evaluation.

9. I maintain academic and professional affiliations supportive of translational research in nuclear medicine. Since 2018, I have held an extraordinary professorship at the Faculty of Health Sciences of the University of Pretoria. I am also a member of the European Commission Portfolio Leadership Team.
10. I am the author or co-author of more than 230 peer-reviewed journal publications in radiochemistry, nuclear medicine, and radiopharmaceutical sciences. My publications reflect sustained research leadership in the development and clinical translation of targeted alpha therapy and the broader application of medical radionuclides for oncology.

II. ORIGIN AND DIFFICULTIES OF THE '901 PATENT INVENTION

11. The invention described in the '901 Patent emerged from discussions that I had with Dr. Kratochwil in 2013. At that time, we were meeting regularly to discuss other collaborations for clinical treatments and additional ideas for treatments using radiopharmaceuticals. Dr. Kratochwil had told me about his work with ^{177}Lu -PSMA-617 and we discussed combining the PSMA-617 with ^{225}Ac during one of our regular discussions.

12. I knew at the time that work using ^{177}Lu -PSMA-617 was promising and we were interested in researching the clinical applications of the combination of ^{225}Ac with PSMA-617. We had previously added ^{225}Ac to DOTATOC for treating neuroendocrine cancer; however, the clinical results of that combination were, at best, only marginally better than using ^{177}Lu with the same carrier molecule (DOTATOC).
13. Adding ^{225}Ac was not just a matter of switching out one radionuclide for another. In order to use ^{225}Ac , we had to obtain the radionuclide with a high level of purity. Otherwise, metal impurities in the ^{225}Ac could combine with the carrier molecule and prevent the ^{225}Ac from binding. At the time of the invention, it was difficult to purify ^{225}Ac to the necessary grade. Moreover, although it was known at the time that ^{225}Ac could bind some DOTA chelators, the chelation step was often a low-yielding reaction (<5% yield).
14. The stable binding of ^{225}Ac with the chelator on the carrier molecule was also affected by the conditions of the chelation step, including the temperature, pH and time of reaction. The conditions for this step were unknown.
15. Even after the ^{225}Ac was complexed to the carrier molecule in a sufficient yield, the ^{225}Ac still needed to remain bound to the carrier molecule for a sufficient time and the new ^{225}Ac -carrier complex needed to be able to bind PSMA in cancer cells. Because different radionuclides could affect the functioning of the

carrier molecule, including its binding, replacing ^{177}Lu with ^{225}Ac could have resulted in different binding by PSMA-617. Therefore, my lab and I had to perform new *in vitro* binding experiments to confirm that the molecule could still stably bind PSMA in the target cancer cells.

16. There was also the concern that the high-energy alpha particles emitted by ^{225}Ac , which were intended to damage the DNA molecules of tumor cells, could damage the carrier molecules themselves, resulting in the rapid degradation of the molecule, including either the binding site or the chelator. This was of particular concern because PSMA-617 contains a peptide-like structure to bind PSMA, and peptides were known to be degraded by radiation. This damage could occur while the radiopharmaceutical was still in solution before even being given to the patient. We experimentally developed an approach to minimize the molecule's radiolytic degradation, including through the use of ascorbic acid.

17. Alpha-emitting radionuclides like ^{225}Ac can create a recoil effect, causing the daughter radionuclide to be ejected from the carrier. The resulting daughter radionuclide could then end up elsewhere in the body and cause unwanted damage to healthy tissue. To mitigate that risk, we wanted the radionuclide to be taken up by the tumor cells as quickly as possible. But, because the radionuclide affected the carrier molecule binding and could damage the carrier

molecule, we did not know if, or to what degree, the molecule would be taken up with the ^{225}Ac radionuclide.

18. At the time that we were working on the '901 Patent invention, there was little interest in using alpha emitters in radiopharmaceuticals. Other alpha emitters, like ^{211}At and ^{212}Pb , were being considered, but it was unknown what properties, like the decay rate, would be best for radiopharmaceuticals and which carrier molecules would be best for each radionuclide. When we conceived the invention, we believed that ^{225}Ac was the most promising. But there were only a small number of researchers even attempting this. The level of interest increased dramatically after the publication of our article, “ ^{225}Ac -PSMA-617 for PSMA-Targeted α -Radiation Therapy of Metastatic Castration-Resistant Prostate Cancer,” *Journal of Nuclear Medicine*, vol. 57, no. 12: 1941-1944 (2016), which I have been told the Petitioner refers to as “Kratochwil 2016.” In relation to the interest in using ^{225}Ac in radioligand therapy, there was a completely different period “before” and “after” the “Kratochwil 2016” paper was published.

19. In fact, at the time, I was told in no uncertain terms by my superiors at JRC that my work using ^{225}Ac as a radiopharmaceutical was a dead end and a waste of time and resources. Because of the failure of our previous clinical efforts, there was significant doubt in my department as to whether my work purifying and

making compounds with ^{225}Ac would ever be useful. I am convinced that had we not gotten the surprisingly extraordinary results we did with the '901 Patent invention, I would have been forced to give up entirely on ^{225}Ac radiopharmaceuticals.

20. I made the first ^{225}Ac -PSMA-617 molecule in 2013. Our clinical experiments began in 2014. I worked closely with Dr. Kratochwil during that period, spending about a third of my working time at Heidelberg Hospital discussing the dosages, approaches, and results with him.

21. Dr. Kratochwil and I led the efforts on the project. We were in constant communication discussing all aspects of the project together, but his specific responsibilities focused on dosing and caring for the patients and mine on creating the radiopharmaceutical. Dr. Kratochwil worked with Dr. Giesel and Haberkorn on methods of treating patients and determining the therapeutically effective dosage. Drs. Bruchertseifer and Apostolidis worked with me on purifying the ^{225}Ac to a grade that could be used with the PSMA-617 molecule and the complexation process for combining ^{225}Ac with PSMA-617 to create the radiopharmaceutical medicine.

22. Our work with ^{225}Ac -PSMA-617 was inspired by our hope that we could develop an improved treatment for cancer patients. The project was challenging, and, at times, I faced pressure from my department to give up on

²²⁵Ac, despite the significant effort I had made to develop methods for purifying the radionuclide for clinical applications. However, I never expected the astonishing results that we observed in our clinical studies, including patients with complete remission of their cancer.

23. Many others in the field were also excited by our clinical data disclosed in Kratochwil 2016 and whose data provided the cover image for that month's *Journal of Nuclear Medicine*. Our research was also featured that same month in an invited perspective by two peers from The Institute of Cancer Research in London ("Targeted α -Based Treatment of Metastatic Castration-Resistant Prostate Cancer: Revolutionizing Systemic Radiotherapy?"), which I have attached to this declaration as Annex 1. The authors envisioned the new molecule, which had produced complete PSA and imaging remissions with "remarkably mild" side effects, as a "new class of agents for the prostate cancer armamentarium." Based on the "impressive responses" with end-stage patients, the authors urged that this area of study "be given the highest priority" for future research.

24. We also received unsolicited praise directly from leaders in the field. For example, we received an email from Dr. Jonathan Simons, the President of the Prostate Cancer Foundation (attached as Annex 2), describing how he read our paper three times, took a walk around the block, and read it one more time

because he was so impressed by the significance of our work. He wrote that he considered our work to be “one of the most potentially important original papers in the last 20 years in our field of experimental therapeutics” and asked to meet with us. A few weeks later, he did come to University Hospital Heidelberg to meet with us to discuss our results and future steps.

25. Over the past decade, we have continued to perform clinical studies on ^{225}Ac -PSMA-617, investigating the molecule’s safety, dosage, and efficacy, among other properties. I am also co-leading new studies in South Africa, Poland, and Germany on ^{225}Ac -PSMA-617, ^{225}Ac -PSMA-I&T and other ^{225}Ac -PSMA radiopharmaceuticals.

III. THE ROLE OF NON-INVENTOR CO-AUTHORS IN KRATOCHWIL 2016

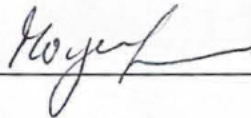
26. Although I did not interact directly with Drs. Frederik Verburg and Felix Mottaghy, we were included on the same email communications related to the drafting of the Kratochwil 2016 publication. Drs. Verburg and Mottaghy were included on the paper because they reached out to Dr. Kratochwil to have a patient treated in the already ongoing study, and they did the imaging for that patient. However, Drs. Verburg and Mottaghy played no role in the preparation of any ^{225}Ac -PSMA molecules or methods of using those molecules for the treatment of cancer.

27. Mirjam Weis was a radiobiological assistant and technician who worked in my lab. Her primary role in the project was administering the logistics of acquiring and transporting radioactive materials. She also helped with lab management, such as cleaning and maintaining the lab infrastructure that came into contact with radioactivity, and some small scientific experiments under my supervision, such as performing the cell-binding experiments that I designed and instructed her in. Ms. Weis did not create any of the molecules used in the study or suggest any dosages, radionuclides, treatment strategies, or otherwise contribute to the invention. Ms. Weis did not generate any of the data that appears in Kratochwil 2016 or the '901 Patent. However, we appreciated her efforts in the day-to-day management of our lab and the logistical support in sending the radioactive materials to the hospital where the patients were treated, and therefore included her as a co-author of the Kratochwil 2016 publication as a way of showing our appreciation.

28. I met Dr. Klaus Kopka only a few times. We included him as an author on the Kratochwil 2016 paper because he was one of the co-inventors of the PSMA-617 molecule and as a sign of respect for his previous work that we built upon. But Dr. Kopka did not work on the ^{225}Ac -PSMA-617 project. For example, Dr. Kopka did not suggest or assist in adding ^{225}Ac to PSMA-617, and I never saw him discussing the dosing or treatment parameters.

I declare under penalty of perjury under the laws of the United States of America
that the foregoing is true and correct.

Executed this 2 day of December, 2025.



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Dr. Alfred Morgenstern Declaration

Annex 1

AcPSMA Invited Perspective

Targeted α -Based Treatment of Metastatic Castration-Resistant Prostate Cancer: Revolutionizing Systemic Radiotherapy?

Wim J.G. Oyen¹ and Johann S. de Bono²

¹Division of Radiotherapy and Imaging, Institute of Cancer Research, London, United Kingdom; and ²Division of Clinical Studies, Institute of Cancer Research, London, United Kingdom

Nuclear medicine treatment of cancer exploits the targeting of radiation through transporters or receptors on tumor cells. Building on this principle, the complexity of therapeutic radiopharmaceuticals varies from simple radioactive elements such as ¹³¹I and ²²³Ra to conjugated carrier molecules that can subsequently be radiolabeled, such as antibodies, peptides, and small molecules. It is not simple to achieve the unique promise of targeted systemic therapeutic radiopharmaceuticals to deliver this cytotoxic payload to the tumor while sparing normal tissues. The success or failure of this approach is determined by a complex set of factors, including the affinity of the carrier molecule, the stability with which the radionuclide carrier complex is bound, pharmacokinetics, biodistribution, receptor expression, and the sensitivity of the tumor cells and normal organs to radiation (1).

For many decades, only β -emitting radionuclides were used for targeted systemic radiotherapy, starting with ¹³¹I treatment of

overcome. First, the radiopharmaceutical needs to be stable in vivo, so that the complex carries the radionuclide to the target. Thus, high-quality radiochemistry is mandatory. Second, preferential targeting of tumor cells with rapid blood clearance is needed to prevent long circulation times. Third, accumulation needs to be low in normal tissues in relation to targeted tumor cells, especially when the normal tissues are radiosensitive. Until recently, α -emitters were used only in an experimental setting, without achieving widespread clinical utility. The first α -emitter that proved to be successful was ²²³Ra, which is now globally used for the treatment of patients with bone metastases from castration-resistant prostate cancer (mCRPC) (3). Analogously to β -emitting radioiodine in thyroid cancer, the simple radioactive α -emitting element ²²³Ra preceded the application of more complex radiolabeled α -emitters. In parallel to the introduction of ²²³Ra treatment for mCRPC, a second major development occurred in the area of molecular imaging of prostate cancer. Prostate-specific membrane antigen (PSMA) binding ligands radiolabeled with ⁶⁸Ga for PET/CT proved to be successful in delineating prostate cancer metastases, showing high target to normal tissue uptake ratios as early as 1 h after injection (4). The use of ⁶⁸Ga-PSMA is being rapidly adopted, although sufficient evidence-based research to support its utility is lacking. The successful imaging of mCRPC by ⁶⁸Ga-PSMA immediately sparked research on the theranostic approach, selecting those patients with detectable metastases on ⁶⁸Ga-PSMA PET/CT for treatment with ¹⁷⁷Lu-PSMA (5). Although the data are preliminary, and conclusive trials with relevant clinical endpoints are pending, ¹⁷⁷Lu-PSMA holds the promise of success similar to that of ¹⁷⁷Lu-DOTA-octreotate, as 60%–70% of patients respond to ¹⁷⁷Lu-PSMA treatment. However, concerns about downregulation of androgen receptor signaling and PSMA have been raised, as well as about heterogeneous PSMA expression.

In this issue of *The Journal of Nuclear Medicine*, Kratochwil et al. report for the first time the results of compassionate-use treatment with the α -emitter ²²⁵Ac radiolabeled to the PSMA-617 molecule in two heavily pretreated patients with mCRPC for whom other options were no longer available (6). Despite being refractory to most of the currently available treatment options for this disease, including ¹⁷⁷Lu-PSMA-617, the patients went into a complete PSA remission and a complete imaging remission after treatment with this α -particle emitting radiopharmaceutical, described as ²²⁵Ac-PSMA-617. Despite previous chemotherapy exposure and extensive bone metastases, which can take up large amounts of radiopharmaceutical, side effects to bone marrow were remarkably mild, as no significant hematologic

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differentiated thyroid cancer more than 70 y ago. Many years later, a range of therapeutic radiopharmaceuticals was developed using small molecules, peptides, antibodies, and particles radiolabeled with ¹³¹I, ⁹⁰Y, and ¹⁷⁷Lu. Recently, the extraordinary clinical benefit of ¹⁷⁷Lu-labeled DOTA-octreotate in patients with neuroendocrine tumors was reported (2). As compared with the standard of care, ¹⁷⁷Lu-DOTA-octreotate resulted in an impressive gain in progression-free and overall survival for patients with metastatic neuroendocrine tumors.

α -emitting radionuclides have characteristics that are considered favorable for therapy. The ultrashort range and the high-linear-energy transfer resulting in double-strand DNA breaks in adjacent cells after a single hit are likely when the decay occurs nears the tumor cell nucleus. To stop this from happening in normal tissues sufficiently to prevent detrimental side effects, several challenges need to be

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toxicity was observed. The most significant side effect was severe xerostomia as the radiopharmaceutical was taken up by PSMA-expressing salivary glands, which are heavily targeted and subsequently become dysfunctional. Strategies to abrogate this toxicity are required.

Although Kratochwil et al. reported the treatment of only two patients, comparison comes to mind with the first report, in 2001, of the remission induced in a single gastrointestinal stromal tumor patient treated with imatinib (7). Development of imatinib and subsequent targeted therapies has had a major impact on the treatment of cancer. The article by Kratochwil et al. reports on the use of ^{225}Ac -PSMA-617 as last-resort salvage treatment, which is compliant with the updated Declaration of Helsinki on unproven interventions in clinical practice and with regulations in Germany, where these treatments were performed. Prospective studies are now urgently required to further investigate ^{225}Ac -PSMA-617 in mCRPC patients. The initial need is for dose-finding studies to further delineate tolerability, cumulative toxicity, recommended dosing and scheduling, optimal treatment duration, and safety in order to allow the rapid and seamless progression to phase II clinical studies with relevant oncologic endpoints that is needed to proceed beyond niche application in incidental patients. If the antitumor activity described to date is confirmed in a larger prospective trial, we envision this to be a new class of agents for the prostate cancer armamentarium, with potential for treating not only late-stage disease but also less advanced disease. ^{68}Ga -PSMA PET/CT, which could serve as the key predictive biomarker for patient selection for ^{225}Ac -PSMA-617, also requires further evaluation in these trials.

Obviously, there are many challenges ahead. For example, a guaranteed supply of the cyclotron product ^{225}Ac may be an issue. Legislation for clinical use of α -emitters may not be straightforward in every country, although with the approval of ^{223}Ra , much of the groundwork has been done. Another challenge is the significant financial investment that will be required to advance this clinical drug development program, although expedited approval may be feasible if antitumor activity is maintained. Protocol development will also require careful consideration. To make informed decisions, we need predictive biomarkers to evaluate in which subset of patients ^{225}Ac -PSMA-617 will work. Although the companion diagnostic ^{68}Ga -PSMA is likely to be important, tumor biology must also be considered; we will need an understanding of whether specific prostate cancer subtypes, such as those with DNA repair defects, are more sensitive to selective tumor cell killing by α -particle emitting radiopharmaceuticals. We also need to know the relevance of prostate cancer cells lacking PSMA expression. Because the path length of α -radiation is just a few micrometers, we will not be able to exploit the bystander effect from targeted PSMA-positive tumor cells toward PSMA-negative tumor cells as we do when using β -emitters. This will raise the question of whether to develop ^{225}Ac -PSMA-617 as a stand-alone agent or in combination with other (approved or novel) anticancer drugs. The fact that PSMA expression is driven

by activity of the folate-hydrolase 1 gene, which is controlled by androgen receptor signaling, will provide an option for combined treatment approaches. We need to evaluate whether the antitumor activity of such novel therapeutics will be increased or decreased by agents targeting androgen receptor signaling, particularly in light of concerns that some prostate cancer cells may be more likely to lose androgen receptor expression after next-generation endocrine treatments such as abiraterone and enzalutamide (8). Moreover, we need to evaluate the intratumoral inflammatory response induced in part by androgen deprivation and its impact on radiation effects, because studies indicate that myeloid-derived suppressor cells, for example, can protect prostate cancer cells from anticancer agent cytotoxicity (9).

In conclusion, Kratochwil et al. report preliminary but important results indicating that α -particle based systemic radiotherapy with ^{225}Ac -PSMA-617 may have substantial therapeutic potential with a favorable therapeutic window. Although it is too early to label ^{225}Ac -PSMA as a breakthrough for treatment of patients with mCRPC, the impressive responses in patients with end-stage disease should spark major efforts to perform further clinical studies with α -emitting radiopharmaceuticals. This area of study should be given the highest priority so that its full potential can be assessed and the evidence generated to enable rapid registration of this class of cancer therapeutics. Clinical studies on this topic must be accompanied by appropriate translational research so that we can better understand the effects of ^{225}Ac -PSMA-617 on a cellular level, maximize antitumor activity, and what is most important improve the clinical care of patients with this most common of male cancers.

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Dr. Alfred Morgenstern
Declaration

Annex 2

Email from
Dr. Jonathan Simons

MORGENSTERN Alfred (JRC-KARLSRUHE)

From: Kratochwil, Clemens <Clemens.Kratochwil@med.uni-heidelberg.de>
Sent: Wednesday, 20 July 2016 13:10
To: MORGENSTERN Alfred (JRC-KARLSRUHE)
Subject: WG: Urgent and Important Your Provocative Act225 PSMA Paper in Journal of Nuclear Medicine --Dringend und wichtig Ihre Provokative Act225 PSMA Papier in Journal of Nuclear Medicine

Importance: High

-----Ursprüngliche Nachricht-----

Von: Jonathan Simons (jsimons@pcf.org) [mailto:jsimons@pcf.org]

Gesendet: Mittwoch, 20. Juli 2016 07:23

An: Kratochwil, Clemens

Betreff: FW: Urgent and Important Your Provocative Act225 PSMA Paper in Journal of Nuclear Medicine --Dringend und wichtig Ihre Provokative Act225 PSMA Papier in Journal of Nuclear Medicine

From: Jonathan Simons <jsimons@pcf.org<mailto:jsimons@pcf.org>>

Date: Tuesday, July 19, 2016 at 9:56 PM

To: "clemens.kratochwil@med.uni.-heidelberg.de<mailto:clemens.kratochwil@med.uni.-heidelberg.de>"
<clemens.kratochwil@med.uni.-heidelberg.de<mailto:clemens.kratochwil@med.uni.-heidelberg.de>>

Cc: Steve Cho <scho@uwhealth.org<mailto:scho@uwhealth.org>>, Martin Pomper
<mpomper@jhmi.edu<mailto:mpomper@jhmi.edu>>

Subject: Urgent and Important Your Provocative Act225 PSMA Paper in Journal of Nuclear Medicine --Dringend und wichtig Ihre Provokative Act225 PSMA Papier in Journal of Nuclear Medicine

Dear Clemens

First could you kindly forward this email to Professor Morgenstern and your co authors on your brief report on July 8th in the Journal of Nuclear Medicine.

Congratulations!

I read your paper 3x then took a walk around the block here in Santa Monica California and read it one more time. I am not crazy, you can look me up.

For concept, execution, brevity and for cogency and innovation against one of the most promising and vexing surface targets PSMA in mCRPCA...it is one of the most potentially important original papers in the last 20 years in our field of experimental therapeutics -- potentially.

I am the CEO and President of the Prostate Cancer Foundation see www.pcf.org which is the largest foundation funder of prostate cancer in the world.

Could I give you and Professor Morgenstern a call in the next several days, I have a couple of questions technically on 225 Ac and tumor biology, and 3 in clinical research?

I copied my colleagues and friends and PCF funded investigators Dr Steven Cho at the University of Wisconsin and Dr Martin Pomper at Hopkins whose scholarship needs no introduction. I have to go to a meeting in Berlin in late September and if possible would like to come and visit with you in Heidelberg and Aachen and talk further and discuss funded collaboration.

Again many congratulations.

For the heck of it. given how poor my German is I ran this through Google Translate. That is below

My very best Jonathan

Erstens könnten Sie bitte diese E-Mail an Professor Morgenstern weiterleiten und Ihre Co Autoren auf Ihrem kurzen Bericht am 8. Juli im Journal of Nuclear Medicine.

Herzliche Glückwünsche!

Ich las das Papier 3x nahm dann einen Spaziergang um den Block hier in Santa Monica in Kalifornien und es noch einmal zu lesen.

Für Konzept, Ausführung, Kürze und Triftigkeit und Innovation gegen eine der vielversprechendsten und ärgerlichen Oberflächenziele PSMA it eine der potenziell wichtige Papiere in den letzten 20 Jahren in unserem Bereich der experimentellen Therapie potentiell ist.

Ich bin der CEO und Präsident der Prostate Cancer Foundation www.pcf.org die die größte Stiftung Funder von Prostatakrebs in der Welt ist.

Könnte ich Sie und Professor Morgenstern einen Anruf in den nächsten Tagen geben, habe ich ein paar Fragen technisch auf 225 Ac und Tumorbiologie und 3 in der klinischen Forschung.

Ich kopierte meinen Kollegen und Freunden und PCF finanzierte Forscher Dr. Steven Cho an der University of Wisconsin und Dr. Martin Pomper an Hopkins, deren Stipendium braucht keine Einführung. Ich habe Ende September zu einem Treffen in Berlin zu gehen und, wenn möglich, möchte mit Ihnen in Heidelberg und Aachen zu besuchen und sprechen weiter.

Nochmals vielen Glückwünsche.

Für das Heck von ihm gegeben, wie schlecht mein Deutsch ist ich dies durch Google lief übersetzen. Das ist unter

Meine besten Jonathan