



## 10th Guide to German Biotech Companies

10th Guide to German Biotech Companies: German Biotechs 2008

### BIOCOM AG

RESprotect

RESprotect GmbH

\* RESprotect - Prevention of  
Chemoresistance - Overview

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~~Email Address~~

~~Internet Website~~

~~Number of Employees~~

~~Cancer chemotherapy;~~

~~Chemotherapy of infectious diseases~~

~~Areas of Activity~~

~~WITEGA/Berlin; Nycomed/Linz,~~

~~Austria; BIACOM/Budapest,~~

~~Hungary; Clinics Chemnitz;~~

~~University Leipzig; University~~

~~Munich; Technical University~~

~~Munich; University Vienna/~~

~~Austria; Avantogen/San Diego,~~

~~USA.~~

**Partnering: RESprotect is looking for the appropriate partner to develop its key project RP101 in Europe, South America and Asia worldwide.**

RESprotect GmbH is a privately



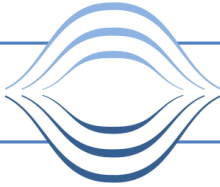
**RESprotect**

Prevention of Chemoresistance

owned biotechnology company located in Dresden Germany. RESprotect is focusing on the inhibition of chemoresistance and the enhancement of chemosensitivity. In contrast to the well known efforts to circumvent or decrease existing chemoresistance, this basic approach is unrivalled. RESprotect was founded in 2000. The founder is geneticist and came from the Fraunhofer-Institute for Toxicology in Hannover. At present clinical studies on RP101 are performed with pancreas cancer patients. The next generation of more efficient compounds is being explored, and a general broadening of the clinical indications is in process. RESprotect is in the position to enter a segment of the huge market of anticancer cytostatics. Use patents exist and an extension of the patent portfolio by substance patents was achieved in 2006. New chemical entities have been identified and introduced to the development pipeline.

**\* *Combating Chemoresistance - Chemogenomics joins the battleground***

In cancer model systems, chemoresistance is often mediated by a single gene, and, therefore, may in theory be inhibited by any drug that targets the product of that targets that gene. These drugs possess potency and specificity for only one of the several reasons for chemoresistance. For this reason, the chemogenomics approach focuses on small molecules, causing favorable phenotypic changes, and inhibiting or preventing the induction of chemoresistance. The drugs have to counteract the over-expression of apoptosis-antagonizing genes and to enhance the immune responses. By influencing not only one but a



number of different validated targets a new class of effective anti-cancer drugs will be developed. These compounds have to be administered in addition to standard chemotherapy. RP101 is the first drug that shows these effects in tumor cells in cultured tumor cells, in animals and in patients.

**\* RP101 improves the efficacy of chemotherapy treatment in pancreatic carcinoma cells and patients**

In pre-clinical studies, RP101 has shown strong antitumor effects due to inhibition of chemoresistance and the enforcement of apoptotic response upon drug treatment. RP101

affects numerous gene products related to chemoresistance and tumor immunity. In a Phase I study including five different tumor entities and 12 different cytostatic drugs, no enhancement of unwanted side effects had been observed. In a Phase II pilot study with 13 pancreas cancer patients, RP101 co-treatment enhanced remissions, survival and time to progression. The results of the pilot study were confirmed in a second study with 21 patients in similar stages of disease. The results were similar (Fahrig, et al., *Anti-Cancer Drugs* 17, 2006, 1045-56). Our two studies roughly showed the tendency of doubling the survival time. After one year, 4 to 5 times more patients with far metastases lived in the RP101 co-treatment group than in the chemotherapy alone group. In both studies, adverse events were consistent with those observed with the cytostatic drugs alone, or the underlying disease. The efficacy of RP101 exceeds all other regimens known to us. The data implicated that acquisition of chemoresistance was prevented and the antitumor efficacy of standard chemotherapy was improved. A double blind phase II/III study with 153 patients started in Q4 2007. 55 sites in Europe and America will participate in the study.

CEO &dash;

*Founder:*

Prof. Dr. Rudolf Fahrig

CSO &dash;

*Cell and molecular biology:*

Dr. Jörg-Christian Heinrich

CSO &dash;

*Pharmacy and chemistry:*

Dr. Dieter Lohmann

*COO/CFO - Marketing and Organisation:*

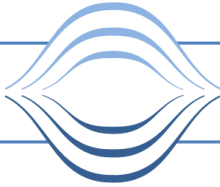
Dr. Torsten Fahrig

*Finances:*

BW Kerstin Jahn

**\* Financing**

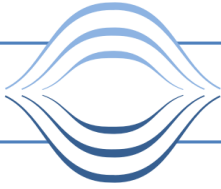
Nearly 7 million euros were raised by the company in the first round of financing. The next round was replaced by out-licensing the North-America rights of RP101. RESprotect has signed in September 2004 a license agreement with Australian Cancer Technology. The Australian Biotech company acquired the license for the use of the anti-cancer drug RP101 in North America. SciClone Pharmaceuticals Inc./ San Francisco acquired this license in 2007. SciClone finances the clinical development of RP101 in Europe and North America. RESprotect has free access to the data for approval of RP101 outside the USA and Canada.



The chemogenomics approach for preventing the induction of chemoresistance

- 1) Anti-recombinogenic effects  
Reason:  
Recombination leads to gene amplification and MDR-1 activation
- 2) Inhibition of the over-expression of STAT-3 and its target VEGF  
Reason:  
Over-expression leads to  
A) Prevention of apoptosis  
B) Blockade of the initiation of anti-tumor immunity  
C) Enhancement of tumor cell proliferation
- 3) Inhibition of the over-expression of other oncogenes or UPase  
Reason:  
Over-expression generally leads to poor prognosis for the patient
- 4) Inhibition of the over-expression of DNA-repair genes like APEX  
Reason:  
DNA-repair antagonises the effect of cytostatic drugs
- 5) Inhibition of the down-regulation of NQO1  
Reason:  
Often multifactorial multidrug resistant tumor cells show decreased NQO1 expression

All effects together cause induction of apoptosis and maintenance of chemosensitivity



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***RESprotect***  
Prevention of Chemoresistance

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