Varian Biopharmaceuticals

Developing a first-in-class atypical Protein Kinase C iota inhibitor
Targeting Gli-1, KRAS, HH Driven Cancers

Confidential / February 2022

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Forward Looking Statements

Certain statements in this Presentation may be considered forward looking statements. Forward looking statements generally relate to future events or SPK's or the Company's future financial or operating performance. For example, statements concerning the following include forward looking statements: the growth of the Company's business and its ability to realize expected results; the viability of its growth and commercial strategy; financial projections; the success, cost and timing of its product development activities; the advantages and potential of its technology and products, including in comparison to competing technologies and products; trends and developments in the industry; changes to federal and state laws and regulations; changes to reimbursement rates; the impact of the COVID 19 pandemic; its total addressable market; and the potential effects of the Business Combination on the Company. In some cases, you can identify for war d looking statements by terminology such as "may", "should", "expect", "intend", "will", "estimate", "anticipate", "believe", "predict", "potential" or "continue", or the negatives of these terms or variations of them or similar terminology. Forward looking statements are based upon estimates and assumptions that, while considered reasonable by SPK and its management, and the Company and its management, as the case may be, are inherently uncertain. All forward looking statements are subject to risks, uncertainties, and other factors which could cause actual results to differ materially from those expressed or implied by such forward looking statements. For further information, please see the Risk Factors set forth in this Presentation as well as the sections entitled "Risk Factors" and "Cautionary Note Regarding Forward Looking Statements" in SPK's final prospectus relating to its initial public offering, dated June 7, 2021, and other filings that SPK or the Company will make with the Securities and Exchange Commission (SEC). New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. You should not place undue reliance on forward looking statements in this Presentation, which speak only as of the date they are made and are qualified in their entirety by reference to the cautionary statements herein. Neither SPK nor the Company undertakes any duty to update these forward looking statements.

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Investment Overview

Precision Oncology – a novel protein kinase inhibitor

- **VAR-101/102** is being developed as a potential high-potency, specific, atypical ***Protein Kinase C iota*** (aPKCi) inhibitor
  - Recent discoveries characterized ***aPKCi as a potentially promising oncogene*** – PKCi is required for growth of multiple forms of human cancer including basal cell carcinoma (BCC), cutaneous T-cell lymphoma (CTCL), pancreatic, non small cell lung cancer (NSCLC), acute myeloid leukemia (AML) and others
  - To date, the active compound in VAR-101/102 has demonstrated dose responsive characteristics in murine and human BCC cell lines, as well as non-small cell lung cancer (NSCLC squamous cell carcinoma) mice models
- ***VAR-101 (topical)*** for BCC could potentially offer clinical utility as a surgical neoadjuvant or adjuvant; a significant medical and commercial opportunity
- ***VAR-102 (oral)*** could lend itself to broader aggregation strategy in multiple tumor types, both as a single agent and in combination therapy
- Experienced management team – ***focused on getting VAR-101 into first-in-man clinical trial in BCC***

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Management & Consultants

Our Team	
Jeffrey Davis - *President & CEO*	• Abeona Therapeutics, Access Pharmaceuticals, Bioenvision, Deutsche Bank, Philips NV, AT&T Bell Laboratories, Wharton Business School – University of Pennsylvania
Paul Mann - *Chairman*	• Highbridge Capital, Soros Fund Management, Morgan Stanley, Procter and Gamble, PolarityTE, Cambridge University
Jonathan Lewis, MD, PhD - *CMO & SAB*	• BOD, Co-Founder, Samus, Ziopharm, Genentech, Roche, Memorial Sloan-Kettering Cancer Center, Yale University, Cambridge University, Wits University.
Robert Morgan, JD - *Regulatory Advisor*	• DuPont Pharmaceuticals Company, Genzyme Corp. PAREXEL Int'l, Ziopharm, Samus Therapeutics, Theseus Imaging Corp, Northeastern University
John Amedio, PhD - *CMC & Tech Ops*	• Arqule (acquired by Merck), Samus, Seaside, Ziopharma, Epix, Novartis, Sandoz
Colleen Johnson - *Non-Clinical Ops*	• 20+ years Experience in Toxicology, Development of Non-clinical / Toxicology Testing programs, Spinifex Pharmaceuticals Pty Ltd, University of Arizona
Stacey Channing, JD - *Legal Advisor & IP*	• Samus, Proscript, Immulogic, WR Grace, Kenway and Jenney

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Scientific Advisory Board

SAB - Leaders in aPKCi Research and Precision Oncology Development	
Jonathan Lewis, MD, PhD - *SAB Chair*	• BOD, Co-Founder, Samus, Ziopharm, Genentech, Roche, Memorial Sloan-Kettering Cancer Center, Yale University, Cambridge University, Wits University.
James O. Armitage, MD - *SAB*	• BOD Tesaro (acquired by GSK), BOD MGI Pharma (acquired by Eisai), Emeritus Dean, University of Nebraska Medical Center, The Joe Shapiro Professor of Medicine Division of Oncology & Hematology, Former ASCO President
Peter Parker, PhD, FRS - *SAB*	• Professor, Senior Group Leader, Francis Crick Institute, Director – CRUK KHP Centre, King's College London, Royal Society (2006), European Academy of Cancer Sciences (2011)
William Matsui, MD - *SAB*	• University of Texas Health Austin's LiveStrong Cancer Institute, Professor of Oncology and Internal Medicine, Dell Medical School, ASCO, AACR, ASH
Todd Wider, MD - *Director & SAB*	• Emendo Biotherapeutics, Arya, Mount Sinai Medical Center, Columbia University, Princeton University.

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PKCi Inhibition - *Therapeutic Rationale, Disease Linkage, Competitive Landscape*

aPKCi Inhibitor Description

- **Novel and emerging oncogenic target**
  - **Atypical PKC iota isozyme – a potentially unique mechanism of action**
  - **First PKC isozyme identified as oncogene in 2005**

- **PKCi appears involved in regulation of polarity, epithelial-mesenchymal transition (EMT) and initiation of metastasis, cancer stem cells, therapeutic resistance**
  - **Couples to major survival pathways in cancer cells – apoptosis and autophagy**
  - **Regulates chemotaxis and invasive activities in tumor cells**
  - **Regulates expansion and self-renewal properties of cancer stem cells**
  - **Essential for downstream signaling (e.g. c-Met, EGFR, HER2)**

- **Advanced non-clinical studies and pre-clinical data package** suggest a wide window of physiological activity in BCC via Gli-1, and via K-RAS-related NSCLC, pancreatic, and colon cancers

- **Competitive Landscape: Potential NCE opportunity with defined cancer patient populations**
  - **Novel target, MoA**
  - **Favorable drug-like profile with no known competitors**

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PKCi Inhibition - *Personalized Medicine based on Cancer Pharmacogenomics and Biomarkers*

- PKCi – gene amplification and protein activation in specific tumors (patient stratification)
  - Gene amplification or somatic mutations in 36% of NSCL (SCC– 70%), 44% of serous high grade ovarian, 50% of esophageal SCC, majority of triple-negative breast
  - Pharmacogenomic linkage of PKCi activation and tumor stage, grade, prognosis and patient survival
  - Pre-clinical evidence of anti-tumor activity
- Biomarker strategy for patient identification and PD response; collaborating with leading KOLs
  - PKCi gene copy number and/or increased PKCi expression; correlation with sensitivity to Varian PKCi inhibitors in pre-clinical models
  - Potential of predictive biomarker for patient stratification
  - PKCi activation resulted in increased pEct2, pSOX-2, pMEK and pERK and increased levels of MMP10 in pre-clinical models
  - Potential surrogates for PKCi inhibition in tumors

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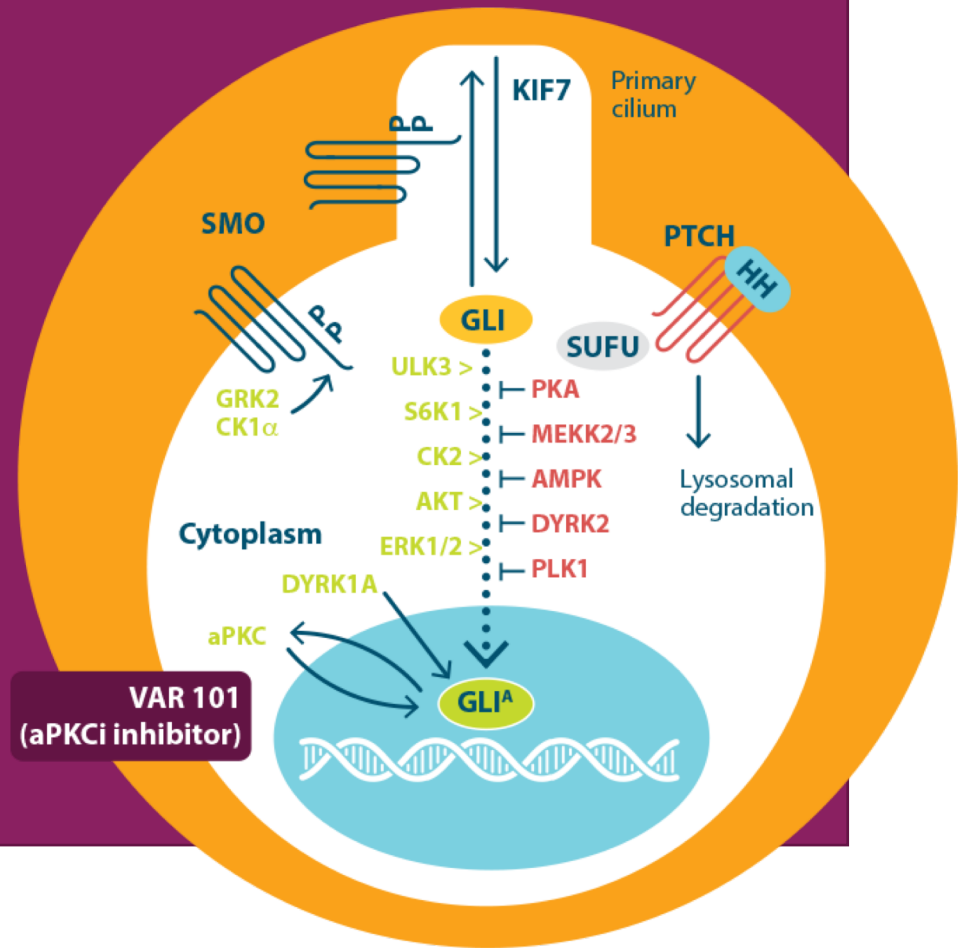
VAR-101 was Observed in Mouse Models to Inhibit aPKCi and Blocks Gli-1 Transcription

BCC must express GLI1 to have cancer phenotype



Potentially Having Better Efficacy and Fewer Adverse Events Compared to the Traditional Hedgehog Pathway Blockade

- Vismodegib (*Erivedge*, Genentech) and Erismodegib (*Odomzo*, Novartis/Sun Pharma), both inhibitors of up-stream Smoothened (SMO), are being used for the treatment of basal cell carcinoma
- Direct targeting of a aPKCi/Gli-1 may offer the potential to ***limit toxicity*** and ***resistance*** that is observed with other SMO/Hh inhibitors where de novo resistance of >50% and poor side effects have proved limiting
- Increasing body of scientific evidence supporting ***aPKCi/K-RAS*** activation in multiple solid malignancies
- No potent, selective aPKCi inhibitors are currently known to being tested in the clinic

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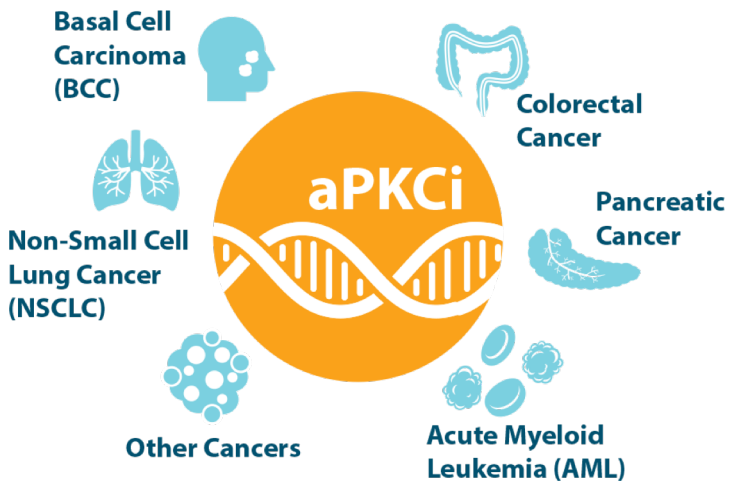
VAR-101 Pre-clinical Attributes

Value Attributable To CRUK/CRT and Prior Collaborative Efforts

Key VAR-101 Attributes	
Rational Design	✓Molecules designed through med chem, SAR, and in vitro and in vivo testing
Potential Selectivity	✓Specific downstream targeting of aPKCi effects *and* evidence of resistance
Biomarker Activity	✓Associated biomarkers: aPKCi, Gli-1 mRNA, other phosphorylation components
Potency	✓Strong cytoplasmic and nuclear activity observed in line with published tool compounds
Low Toxicity	✓Low toxicity with high and repeat dosing observed in murine and large animal models
Therapeutic Index	✓Dose dependent potential and potential biomarker activity observed across *in vitro* murine and human BCC cells lines and in explanted human BCC cells from Moh's sections



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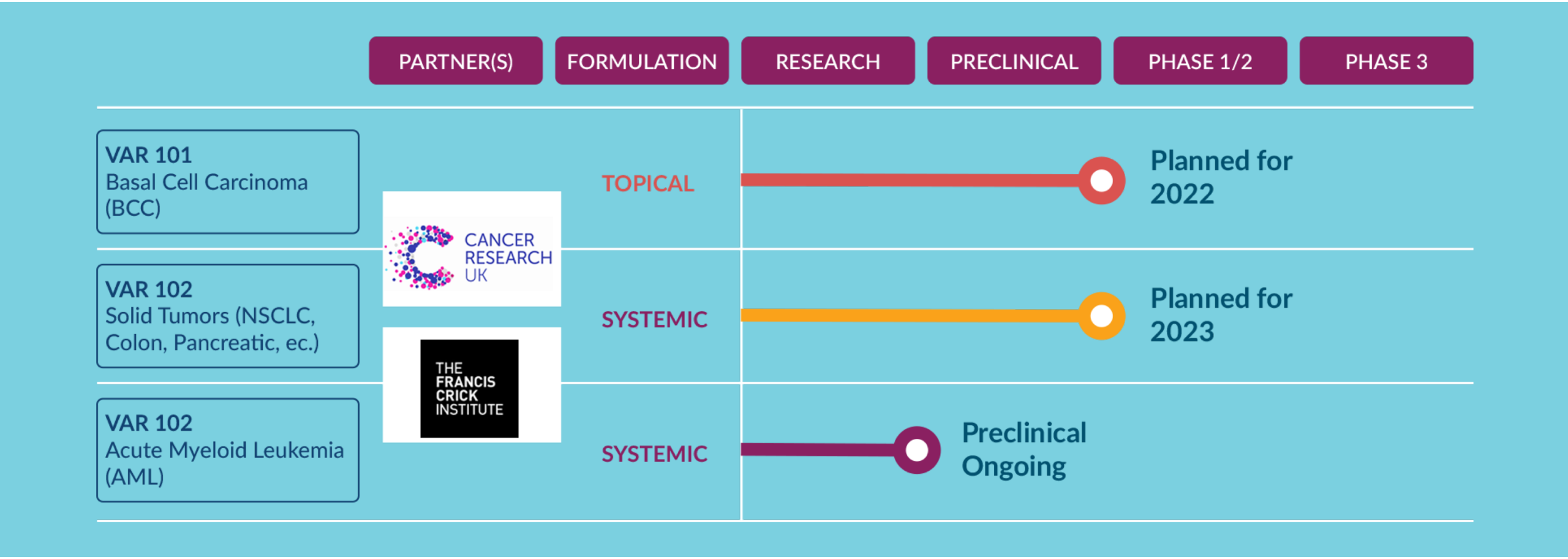
Clinical Development Pipeline



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Review of Pre-Clinical Evidence

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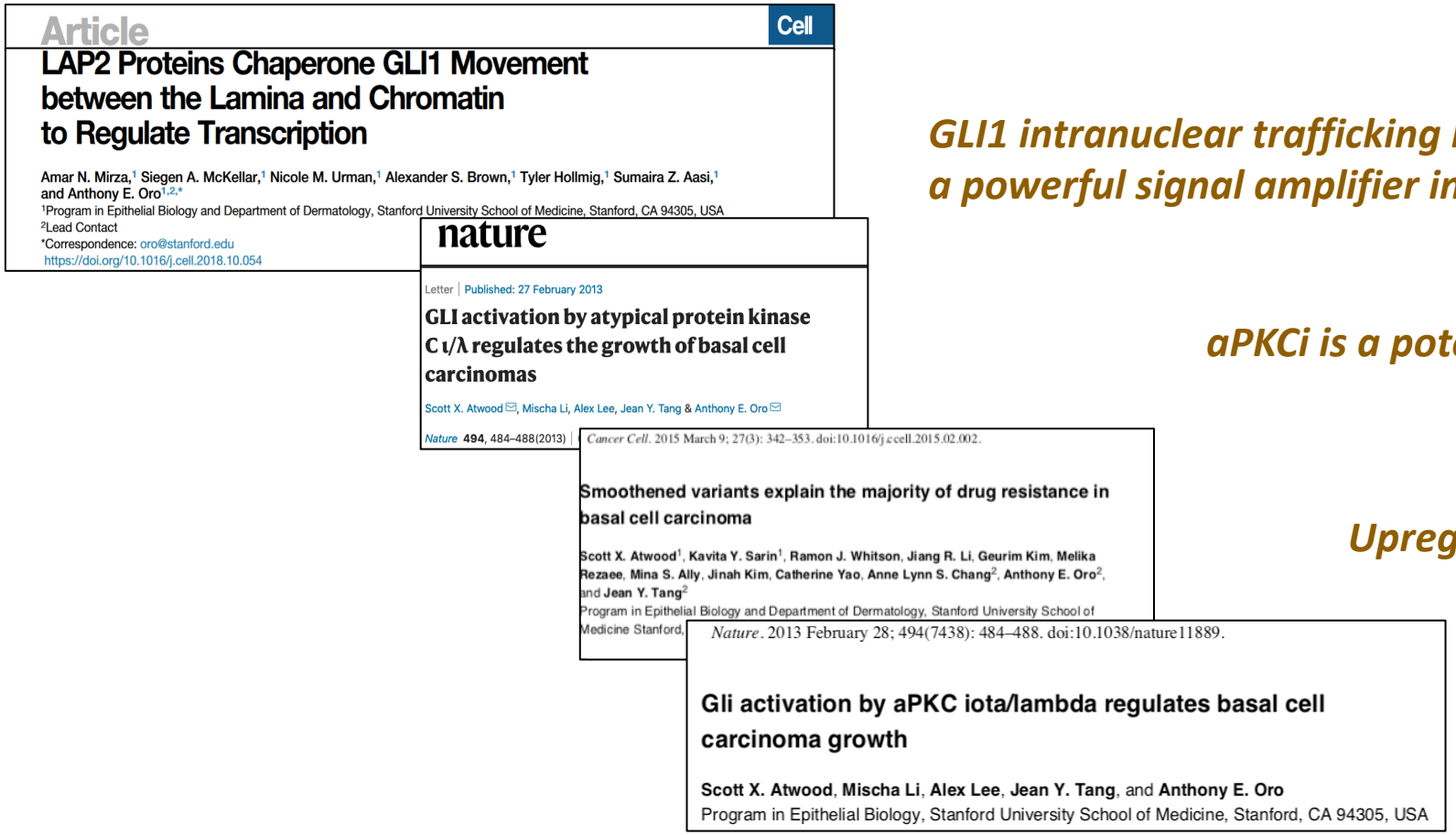
VAR-101 – *aPKCi inhibition in BCC Animal Models*

Increasing Body of Scientific Evidence Supporting aPKCi and GLI-1 Inhibition in BCC



Article

Cell

LAP2 Proteins Chaperone GLI1 Movement between the Lamina and Chromatin to Regulate Transcription

Amar N. Mirza,[1] Siegen A. McKellar,[1] Nicole M. Urman,[1] Alexander S. Brown,[1] Tyler Hollmig,[1] Sumaira Z. Aasi,[1] and Anthony E. Oro[1,2,*]

[1]Program in Epithelial Biology and Department of Dermatology, Stanford University School of Medicine, Stanford, CA 94305, USA

[2]Lead Contact

*Correspondence: oro@stanford.edu

https://doi.org/10.1016/j.cell.2018.10.054

nature

Letter | Published: 27 February 2013

GLI activation by atypical protein kinase C ι/λ regulates the growth of basal cell carcinomas

Scott X. Atwood, Mischa Li, Alex Lee, Jean Y. Tang & Anthony E. Oro

Nature **494**, 484–488(2013)

Cancer Cell. 2015 March 9; 27(3): 342–353. doi:10.1016/j.ccell.2015.02.002.

Smoothened variants explain the majority of drug resistance in basal cell carcinoma

Scott X. Atwood[1], Kavita Y. Sarin[1], Ramon J. Whitson, Jiang R. Li, Geurim Kim, Melika Rezaee, Mina S. Ally, Jinah Kim, Catherine Yao, Anne Lynn S. Chang[2], Anthony E. Oro[2], and Jean Y. Tang[2]

Program in Epithelial Biology and Department of Dermatology, Stanford University School of Medicine Stanford,

Nature. 2013 February 28; 494(7438): 484–488. doi:10.1038/nature11889.

Gli activation by aPKC iota/lambda regulates basal cell carcinoma growth

Scott X. Atwood, Mischa Li, Alex Lee, Jean Y. Tang, and Anthony E. Oro

Program in Epithelial Biology, Stanford University School of Medicine, Stanford, CA 94305, USA

GLI1 intranuclear trafficking by LAP2 appears to be a powerful signal amplifier in BCCs

aPKCi is a potential GLI regulator

Upregulation of GLI occurs in resistance

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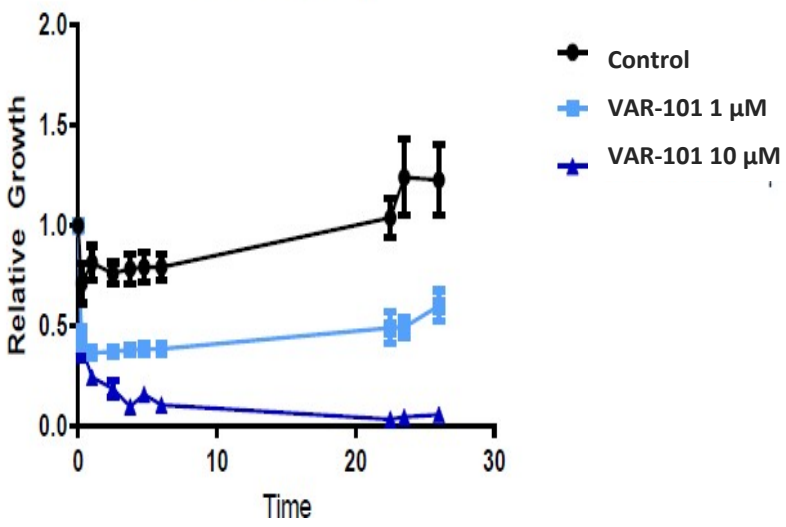
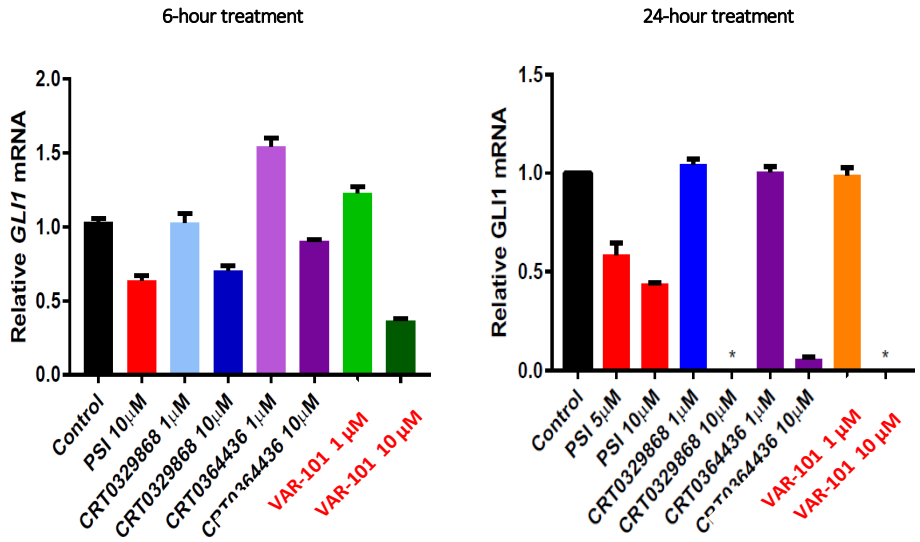
VAR-101 – *Active in BCC Cell Lines*

VAR-101: Evidence of Dose-dependent Modulation of BCC Cell Viability and Gli-1 mRNA, in vitro

VAR-101 BSC1 Viability



Gli-1 mRNA



- **Relatively consistent profile in panel of murine and human BCC cell lines**
- **All BCCs express Gli-1 and aPKC**

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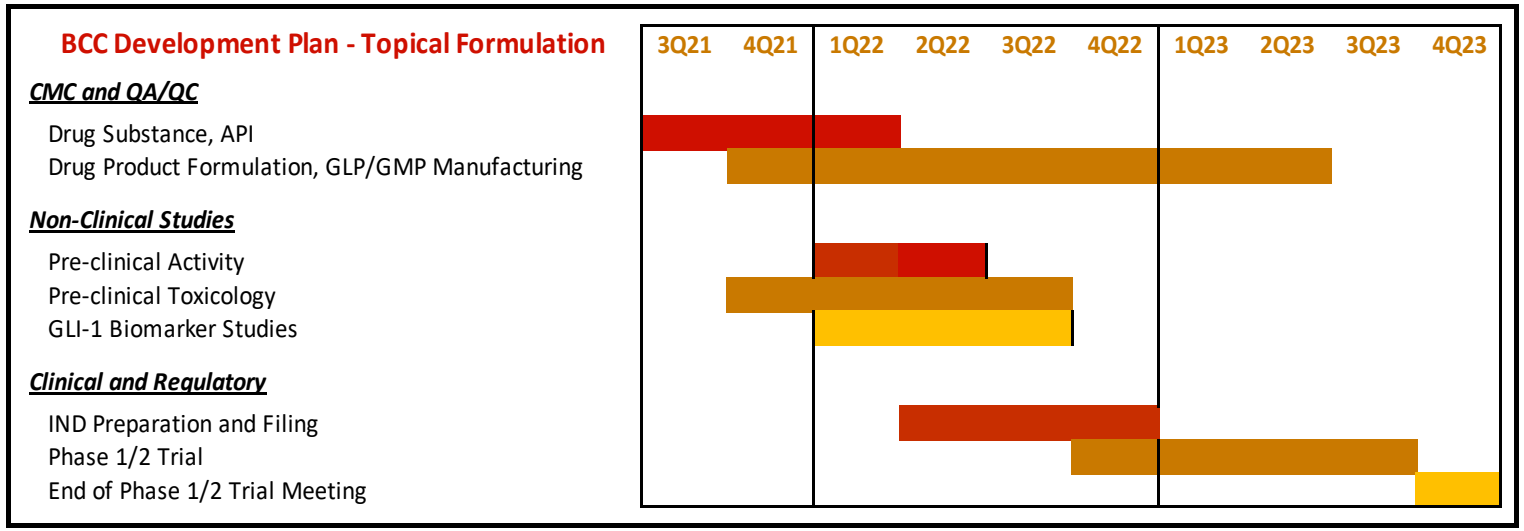
VAR-101 for BCC - *Development Timeline*

Plan to develop POC data in humans

- VAR 101 API and formulation work in progress; IND enabling nonclinical studies to follow
- Target IND filing by year-end 2022
- POC human clinical study (n= 15 to 30 patients) within 24 months
- Sufficient drug substance to explore pre-clinical studies in solid tumor models



VAR-101 Commercial Opportunity

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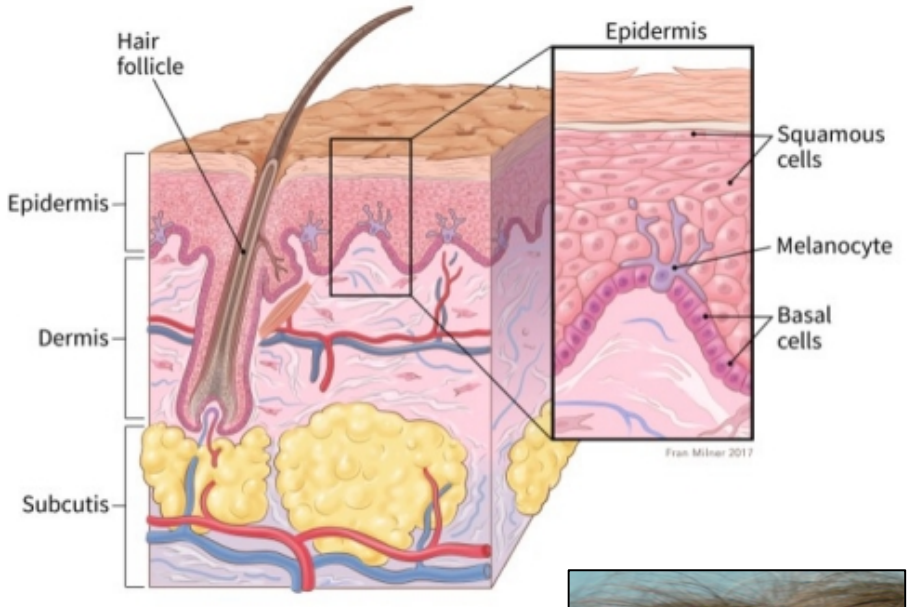
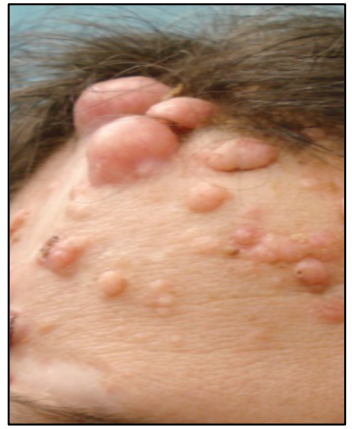
Basal Cell Carcinoma (BCC)

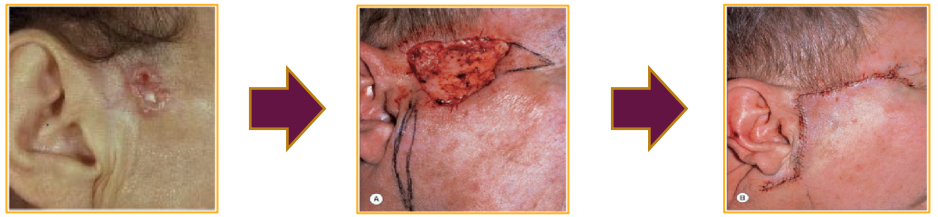
- Skin cancer that arises from basal cells, small round cells found in the epidermis.
- Most common cancer in humans in the US, Europe, and Australia.
  - ~80% of all skin cancers are BCC
  - 4.3 million cases of BCC are diagnosed in the U.S. each year, resulting in more than 2,000 deaths
  - Recurrent and disfiguring, with a rare risk of metastasis: 0.003% – 0.5%
- Greater than $5 billion in annual costs – *5th most costly cancer for US Medicare*
- Risk of secondary BCC is 44% in 3 years





- *American Cancer Society (ACS) www.cancer.org*
- *Curr Dermatol Rep. 2014; 3(1): 40–45*
- *Ridky, 2007; ACS, 2012; Rubin et al, 2005; Housman et al, 2003; Christenson et al, 2005; Marcil et al, 2000.*

BCC Clinical Need



Mohs Surgery

- SOC for higher risk/aggressive BCC – with >1 million Mohs performed annually[1]
- While Mohs offers a low recurrence rate postoperatively, issues with a surgical approach include:
  - ✓ *Cosmetic Damage* - Surgery can result in large ablative defects which require complex reconstructive surgery
  - ✓ *Time consuming* - Primary Mohs typically takes four hours to remove 2 layers (range of 1-10 layers), with follow up reconstructive surgery
  - ✓ *Highs Costs* - $5 billion in annual costs and 5th most costly cancer for Medicare[2]

Drug Therapy

- Non-compliance issues with current drug tx - a follow-up study of ~500 BCC patients found that oral Erivedge (vismodegib) treatment was discontinued in 80% patients
- 36% due to AEs; 14% due to progressive disease; 10% due to patient decision[3]

Images courtesy of Jean Y. Tang, MD, PhD.

1 Adamson JAMA. 2016
2 Goppner et al, 2011. www.cancer.org
*3 Lancet Oncol. **2015**,16(6):729-36*

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VAR-102 Science & Pre-clinical Evidence

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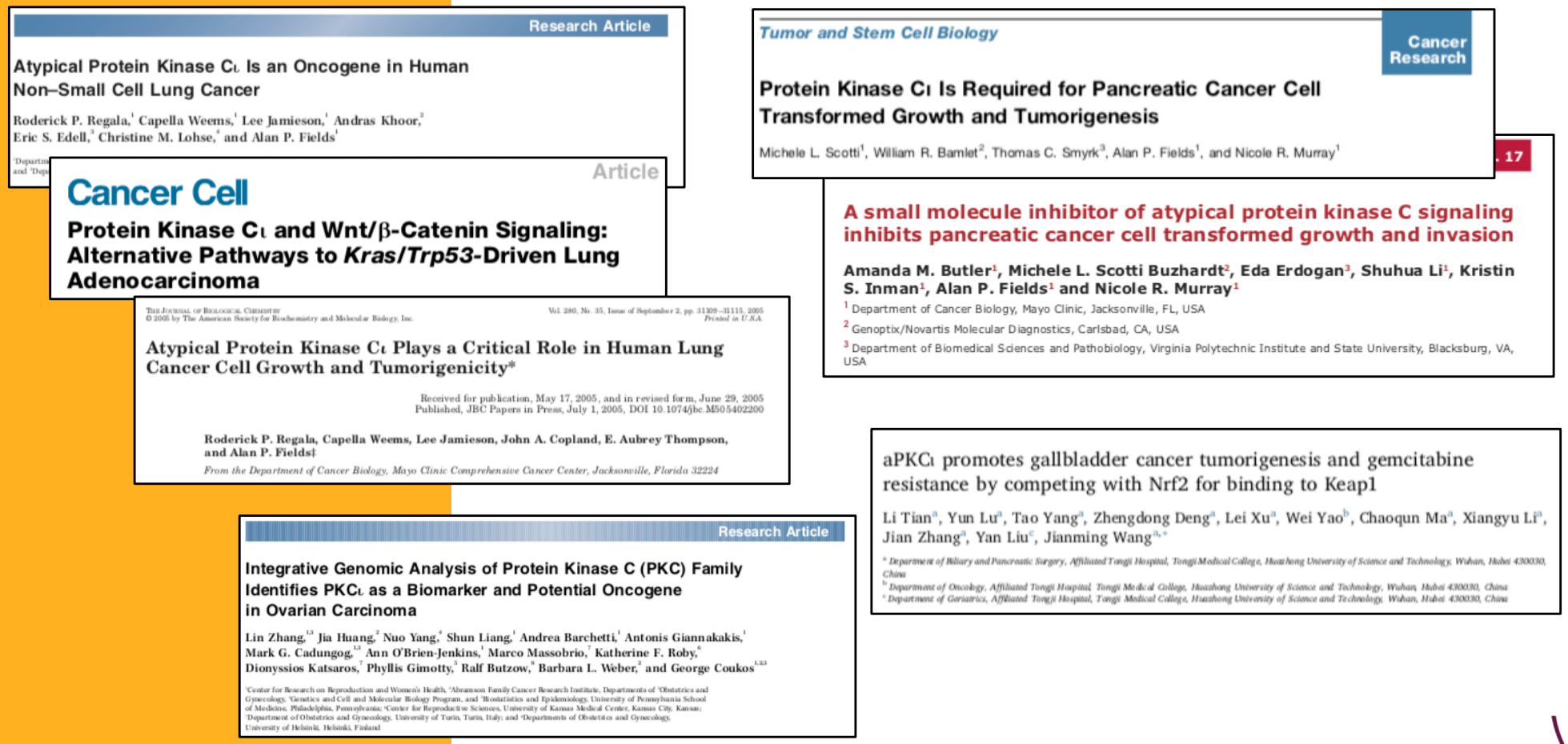
VAR-102 – *aPKCi in Solid Tumors*

Increasing Body of Scientific Evidence Supporting aPKCi / Kras Activation in Multiple Solid Malignancies



Research Article

Atypical Protein Kinase Cι Is an Oncogene in Human Non–Small Cell Lung Cancer

Roderick P. Regala,[1] Capella Weems,[1] Lee Jamieson,[1] Andras Khoor,[2] Eric S. Edell,[3] Christine M. Lohse,[4] and Alan P. Fields[1]

Article

Cancer Cell

Protein Kinase Cι and Wnt/β-Catenin Signaling: Alternative Pathways to *Kras/Trp53*-Driven Lung Adenocarcinoma

THE JOURNAL OF BIOLOGICAL CHEMISTRY © 2005 by The American Society for Biochemistry and Molecular Biology, Inc. Vol. 280, No. 35, Issue of September 2, pp. 31309–31315, 2005 *Printed in U.S.A.*

Atypical Protein Kinase Cι Plays a Critical Role in Human Lung Cancer Cell Growth and Tumorigenicity*

Received for publication, May 17, 2005, and in revised form, June 29, 2005
Published, JBC Papers in Press, July 1, 2005, DOI 10.1074/jbc.M505402200

Roderick P. Regala, Capella Weems, Lee Jamieson, John A. Copland, E. Aubrey Thompson, and Alan P. Fields‡

From the Department of Cancer Biology, Mayo Clinic Comprehensive Cancer Center, Jacksonville, Florida 32224

Tumor and Stem Cell Biology

Cancer Research

Protein Kinase Cι Is Required for Pancreatic Cancer Cell Transformed Growth and Tumorigenesis

Michele L. Scotti[1], William R. Bamlet[2], Thomas C. Smyrk[3], Alan P. Fields[1], and Nicole R. Murray[1]

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A small molecule inhibitor of atypical protein kinase C signaling inhibits pancreatic cancer cell transformed growth and invasion

Amanda M. Butler[1], Michele L. Scotti Buzhardt[2], Eda Erdogan[3], Shuhua Li[1], Kristin S. Inman[1], Alan P. Fields[1] and Nicole R. Murray[1]

[1] Department of Cancer Biology, Mayo Clinic, Jacksonville, FL, USA

[2] Genoptix/Novartis Molecular Diagnostics, Carlsbad, CA, USA

[3] Department of Biomedical Sciences and Pathobiology, Virginia Polytechnic Institute and State University, Blacksburg, VA, USA

aPKCι promotes gallbladder cancer tumorigenesis and gemcitabine resistance by competing with Nrf2 for binding to Keap1

Li Tian[a], Yun Lu[a], Tao Yang[a], Zhengdong Deng[a], Lei Xu[a], Wei Yao[b], Chaoqun Ma[a], Xiangyu Li[a], Jian Zhang[a], Yan Liu[c], Jianming Wang[a,*]

[a] *Department of Biliary and Pancreatic Surgery, Affiliated Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei 430030, China*

[b] *Department of Oncology, Affiliated Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei 430030, China*

[c] *Department of Geriatrics, Affiliated Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei 430030, China*

Research Article

Integrative Genomic Analysis of Protein Kinase C (PKC) Family Identifies PKCι as a Biomarker and Potential Oncogene in Ovarian Carcinoma

Lin Zhang,[1,3] Jia Huang,[2] Nuo Yang,[4] Shun Liang,[1] Andrea Barchetti,[1] Antonis Giannakakis,[1] Mark G. Cadungog,[1,3] Ann O'Brien-Jenkins,[1] Marco Massobrio,[7] Katherine F. Roby,[6] Dionyssios Katsaros,[7] Phyllis Gimotty,[5] Ralf Butzow,[8] Barbara L. Weber,[2] and George Coukos[1,2,3]

[1]Center for Research on Reproduction and Women's Health, [2]Abramson Family Cancer Research Institute, Departments of [3]Obstetrics and Gynecology, [4]Genetics and Cell and Molecular Biology Program, and [5]Biostatistics and Epidemiology, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania; [6]Center for Reproductive Sciences, University of Kansas Medical Center, Kansas City, Kansas; [7]Department of Obstetrics and Gynecology, University of Turin, Turin, Italy; and [8]Departments of Obstetrics and Gynecology, University of Helsinki, Helsinki, Finland

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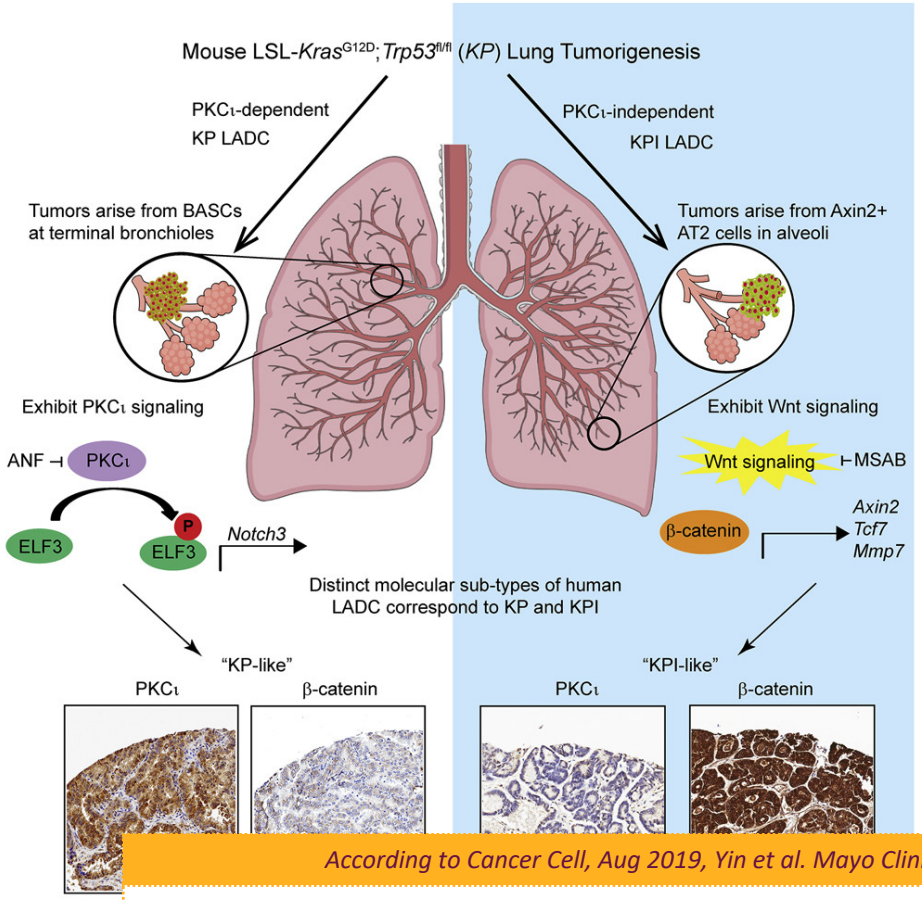
VAR-102 Systemic/Oral Opportunities



According to Cancer Cell, Aug 2019, Yin et al. Mayo Clinic

- *Lung cancer develops through both PKCi-dependent and PKCi-independent path-ways*
- *This results in tumors exhibiting distinct oncogenic signaling and pharmacologic vulnerabilities*

Multiple aPKCi / GLI-1 and KRAS Positive Opportunities

- **Varian plans to pursue systemic tumor indications in a "basket" solid tumor Phase 1 trial**
- **Biomarker-driven approach targeting aPCKi / Gli-1 / K-RAS positive tumors**
- **BCC appears to help form clinical "biomarker" training data set for solid tumors**
- **Target indications include NSCLC, CRC, Pancreatic, Sarcoma and other solid tumors (pre-clinical work to validate AML hypothesis)**
- **No potent, selective aPKCi inhibitors are currently known to being tested in the clinic**

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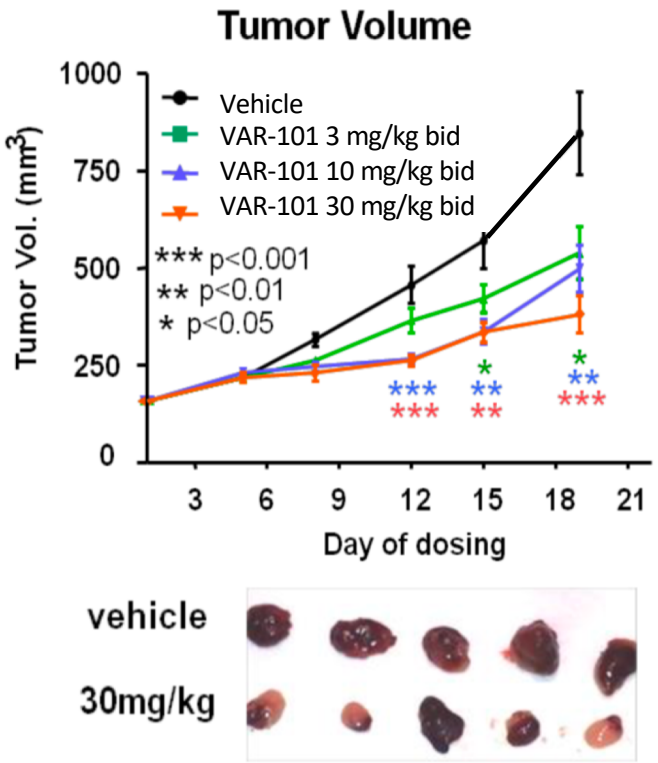
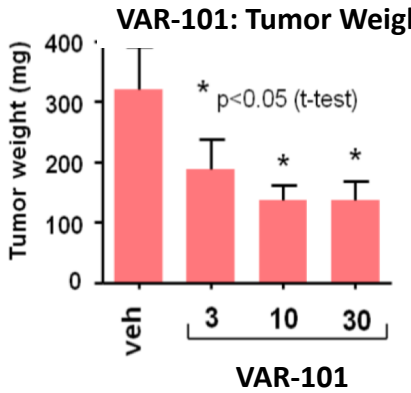
VAR-102 in NSCLC Models

Dose-related Anti-Tumor Activity Observed in H1703-ISO NSCL (SCC) Xenografts in Nude Mice



Compound and Dose	% TGI at d19	P value versus Vehicle
3mg/kg bid	**36**	**P<0.03**
10 mg/kg bid	**41**	**P<0.01**
30mg/kg bid	**55**	**P<0.009**



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VAR-102 in Small Animal Models

IV and PO Pharmacokinetic (PK) Profiles in Mice, Rats, Dogs and Monkeys

Species	Dose (iv/po, mg/kg)	CL (mL/min/kg)	Vd (L/kg)	Iv $t_{1/2}$ (h)	C_{max} at top dose (ng/mL)	F (%)
Mouse	1/10	205	11.2	0.6	62	37
Rat	5/55	12 ± 5	4.7 ± 2.9	3.7 ± 0.9	699 ± 76	13 ± 2
Dog	1/3 & 10	4.33	8.51 ± 3.1	23.0 ± 4.12	730 ± 172	92 ± 10.4
Monkey	1/3 & 10	18.2 ± 4.68	4.7 ± 1.18	3.93 ± 0.37	199 ± 9.29	38.9 ± 1.2

- ***In rat dose-related increases in exposure (Cmax and AUC) were observed to 100 mg/kg po, well in excess of the plasma activity levels observed in anti-tumor studies in mice***
- ***The plasma exposure in dog at 3 mg/kg po is sufficient to cover the exposures required for in vivo anti-tumor activity models in mice***
- ***No emesis observed in dog PK studies***

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Milestones / News Flow

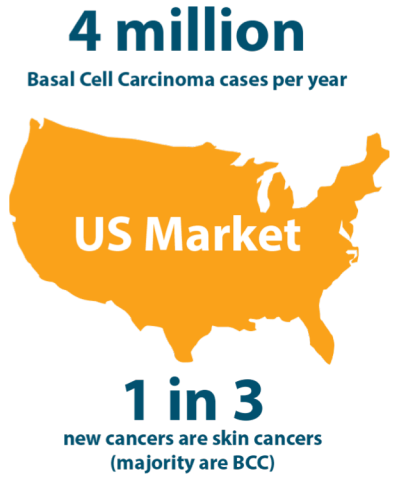
Significant News Flow Anticipated in 2022 and 2023

1H22

- CMC/Manufacturing – finalize optimization of API synthesis; produce initial quantities for permeability studies and initial formulations for VAR-101 (topical) and VAR-102 (oral)
- Plan IND-enabling pre-clinical studies with regulatory consultants; pre-pre-IND and/or pre-IND meetings with FDA (includes discussion of biomarker studies)
- Initiate discussions on next round financings and/or potential corporate partnerships by select geographic territories or by target indication

2H22/2023

- Complete pre-clinical IND-enabling studies
- GMP manufacturing for first-in-human studies of VAR-101
- Prepare IND (or equivalent) for submission in first indication (VAR-101 in Basel Cell Carcinoma)
- Initiate IND-enabling studies for VAR-102 (advanced malignancies to include pancreatic, NSCLC, colorectal, others)
- Discuss/plan scientific data presentations at leading cancer conferences



Valuation Comparable ***- Upon signals of activity in a Phase 1 study in Gorlin Syndrome with its topical SMO inhibitor, Pellepharm inked a $760 million deal with LEO Pharma ($70 million up front, and $690 million in milestones plus a double-digit royalty)***

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Intellectual Property Position

Composition of Matter and Method of Use IP through 2040

US patents include:

- **US 9,914,730:** Azaquinazoline inhibitors of atypical protein kinase C
- **US 9,896,446:** Azaquinazoline inhibitors of atypical protein kinase C
- **US 10,414,763:** Azaquinazoline inhibitors of atypical protein kinase C
- **US 17/598719 (PCT/US2020/025437**: Inhibitors of atypical protein kinase C and their use in treating hedgehog pathway dependent cancers

Over 60 patents (US and foreign counterparts) patents granted

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Transaction Overview

Transaction Highlights

Varian Bio to go public through a SPAC merger with SPK Acquisition Corp (SPK)

- SPK is a Special Purpose Acquisition Company ("SPAC") that raised approximately $50.9 million in its IPO in June 2021, with its common stock trading on the Nasdaq under the symbol SPK
- SPK to acquire Varian Bio for stock consideration valued at $45,000,000
- After the closing of the transaction, the combined company may receive up to $47.1[1] million in pro forma cash to fund operations (assuming no redemptions)
  - Net proceeds are primarily expected to fund clinical development of VAR-101/102
  - Expected to provide runway into 2024
- The combined company is to be led by the existing Varian Bio leadership, including experienced executives in the life sciences sector
- Varian Bio is expected to trade on the Nasdaq under the ticker "VBIO"[2] at closing
- The transaction is expected to close in Q2 2022

1. Pro forma cash balance calculated as sum of cash delivered from SPK trust, assuming no redemptions from the trust less assumed transaction expenses of $3.8M. Does not include Varian Bio's cash balance. 2. Subject to NASDAQ confirmation.

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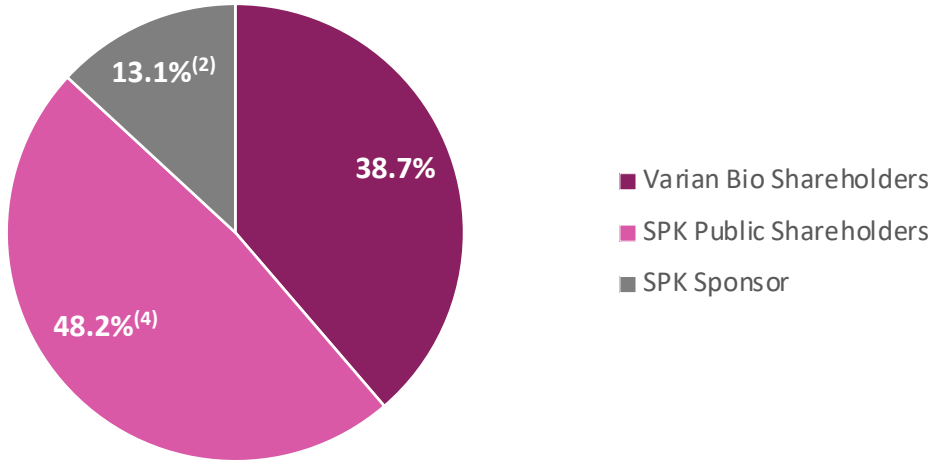
Transaction Overview

Sources and Uses	
Sources	**Amount ($M)**
Cash From SPK Trust[1]	$50.9
SPK Public Rights	$5.1
Varian Bio Equity Rollover	$45.0
SPK Sponsor Equity [2]	$15.3
Total Sources	**$116.3**

Uses	Amount ($M)
Varian Bio Equity Rollover	$45.0
SPK Sponsor Equity [2]	$15.3
Net Cash to Varian Bio BS[1]	$47.1
SPK Public Rights	$5.1
Assumed Transaction Expenses[3]	$3.8
Total Uses	**$116.3**

Pro Forma Valuation	
Particulars	**Amount**
Assumed Share Price	$10.00
Pro Forma Shares Outstanding[1][4]	11.6
Pro Forma Equity Value	**$116.3**
Less: Pro Forma Cash[5]	$47.1
Pro Forma Enterprise Value	**$69.2**

Pro Forma Ownership



(1) Assumes no redemptions from SPK trust. (2) Reflects sum of founder shares, private units, with private rights on an as converted basis, and 25,457 representative shares. (3) Excludes certain advisory fees that may be paid in equity in lieu of cash. (4) Includes 5.1M outstanding public rights eligible to be converted for 1/10th of a common share at the consummation of the business combination. (5) Pro forma cash balance calculated as sum of cash delivered from SPK trust, assuming no redemptions from the trust, less assumed transaction expenses of $3.8M. Does not include Varian Bio's cash balance.

VarianBio

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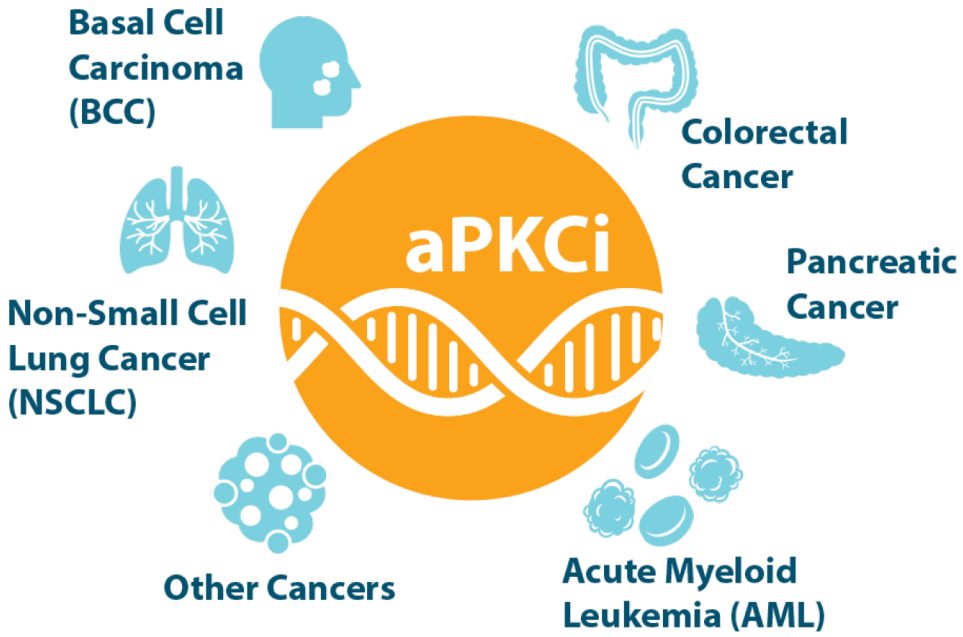

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