UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 4, 2015

PIERIS PHARMACEUTICALS, INC.
(Exact Name of Registrant as Specified in its Charter)

Nevada	**333-190728**	**EIN 30-0784346**
(State of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

Lise-Meitner-Strasse 30
85354 Freising-Weihenstephan, Germany
(Address of principal executive offices, including zip code)

Registrant's telephone number, including area code: +49 81 6114 11400

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01 Regulation FD Disclosure.

Attached hereto as Exhibits 99.1 and 99.2 and incorporated by reference herein are industry conference presentations of Pieris Pharmaceuticals, Inc.

The information set forth under this "Item 7.01. Regulation FD Disclosure," including the exhibit attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, nor shall it be deemed incorporated by reference into any filing under the Securities Act of 1933, as amended, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits

(d) *Exhibits.*

99.1 Industry Conference Presentation of Pieris Pharmaceuticals, Inc. at BIO-Europe 2015, dated November 4, 2015.

99.2 Industry Conference Presentation of Pieris Pharmaceuticals, Inc. at PEGs Europe Protein & Antibody Engineering Summit, dated November 4, 2015.

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Dated: November 4, 2015

PIERIS PHARMACEUTICALS, INC.

By: /s/ Darlene Deptula-Hicks
Name: Darlene Deptula-Hicks
Title: Chief Financial Officer

EXHIBIT INDEX

Exhibit No.	Description
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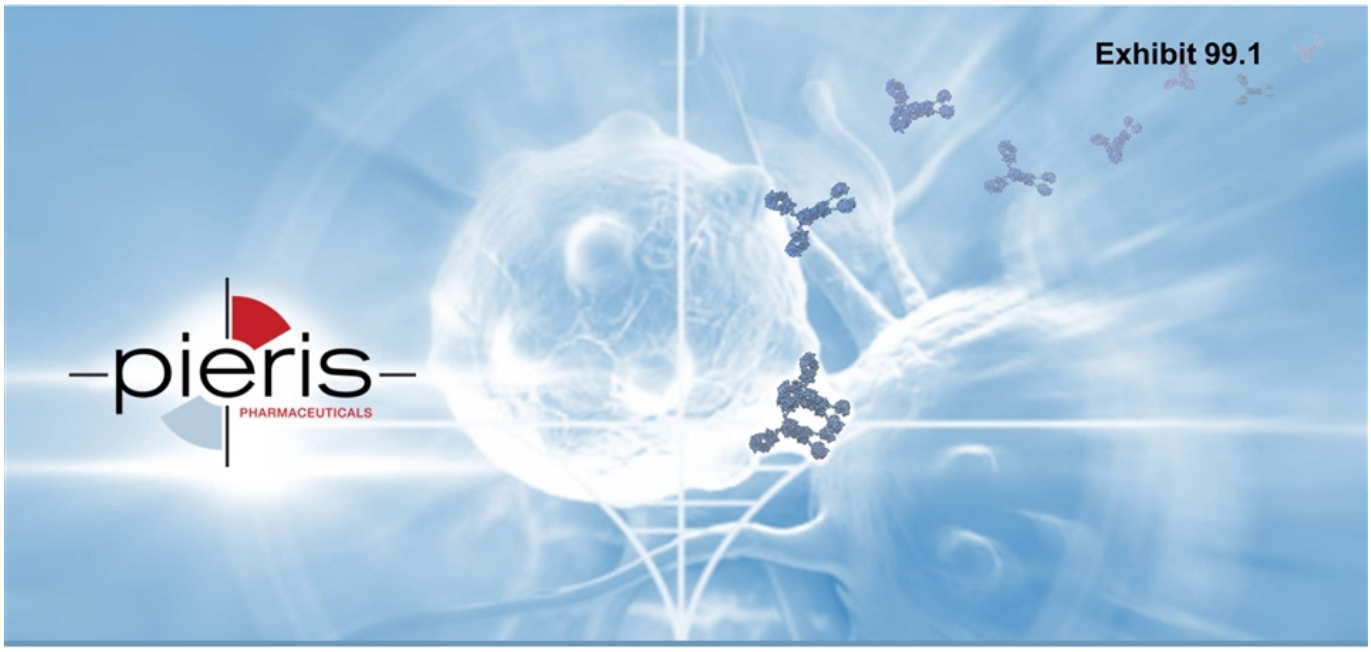


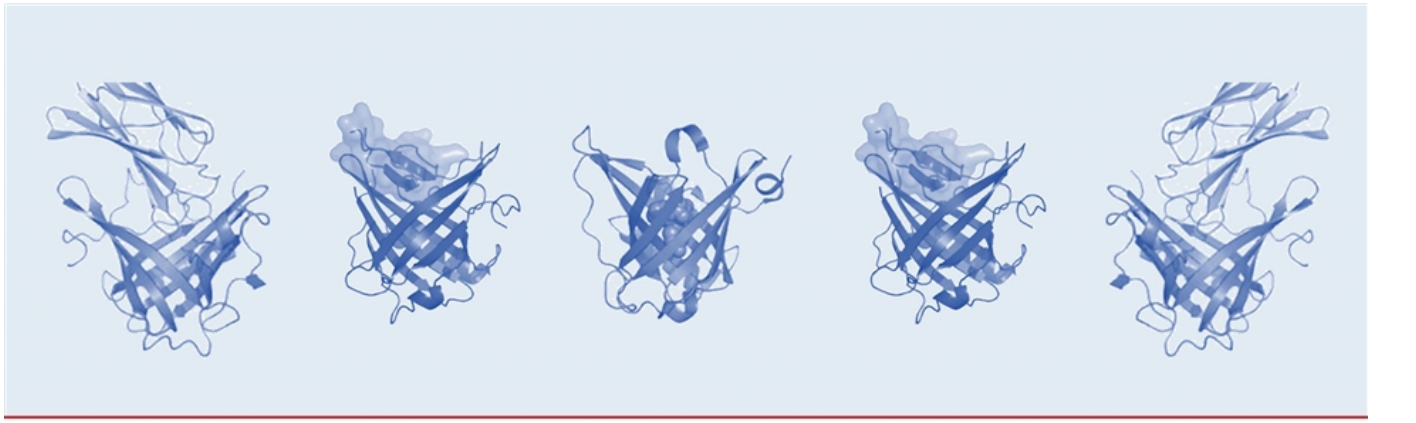
PRS-300 Series – Multispecific Anticalin® Fusions in Immuno-Oncology

BioEurope – Munich

November 04, 2015

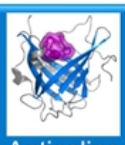
Forward Looking Statements

Statements in this presentation that are not descriptions of historical facts are forward-looking statements that are based on management's current expectations and assumptions and are subject to risks and uncertainties. In some cases, you can identify forward-looking statements by terminology including "anticipates," "believes," "can," "continue," "could," "estimates," "expects," "intends," "may," "plans," "potential," "predicts," "projects," "should," "will," "would" or the negative of these terms or other comparable terminology. Factors that could cause actual results to differ materially from those currently anticipated include, without limitation, risks relating to the results of our research and development activities, including uncertainties relating to the discovery of potential drug candidates and the preclinical and clinical testing of our drug candidates; the early stage of our drug candidates presently under development; our ability to obtain and, if obtained, maintain regulatory approval of our current drug candidates and any of our other future drug candidates; our need for substantial additional funds in order to continue our operations and the uncertainty of whether we will be able to obtain the funding we need; our ability to retain or hire key scientific or management personnel; our ability to protect our intellectual property rights that are valuable to our business, including patent and other intellectual property rights; our dependence on third-party manufacturers, suppliers, research organizations, testing laboratories and other potential collaborators; competition in our industry; regulatory developments in the U.S. and foreign countries; as well as those risks more fully discussed in the "Risk Factors" section of our Current Report on Form 8-K filed with the SEC on December 18, 2014, the Company's annual report on Form 10-K for the fiscal year ended December 31, 2014, the Company's quarterly reports on Form 10-Q, and the other reports we file with the SEC. In light of these risks, uncertainties and assumptions, the forward-looking statements regarding future events and circumstances discussed in this report may not occur and actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. The forward-looking statements included in this presentation speak only as of the date hereof, and except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in our expectations.



Company & Anticalin® Technology

Pieris Pharmaceuticals, Inc.



Anticalins

Proprietary Next-generation Therapeutic Proteins With Several Degrees of Validation

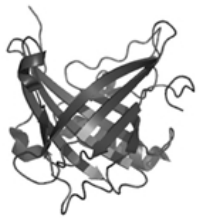
- **Human data demonstrating desired drug-like properties**
  - 26 solid tumor patients with VEGF-A antagonist
  - 36 healthy volunteers with hepcidin antagonist
- **Several R&D partnerships generating $40+ M in revenue**
  - Potential for future milestone and royalties
  - Retained commercial rights in major markets

Daiichi-Sankyo SANOFI ALLERGAN Zydus dedicated to life Stelis Biopharma

- **High Caliber Investors**
  - OrbiMed Advisors (~19%), Tekla Capital Management (~10%), Lombard Odier (~6.5%); Ally Bridge Group, Auriga, Emerald Mutual Fund, Forbion, Gilde, GLSV, Novo Nordisk, Sphera Funds, Zydus
  - $110M equity capital raised

Human Lipocalins – Scaffold for Novel Anticalin® Therapeutics

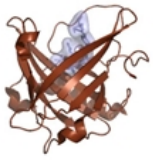
Human lipocalin "template"



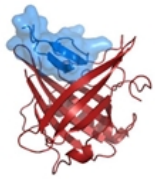
- Human, natural binding proteins
- Low molecular weight (~1/8 of mAb size)
- Extracellular
- Non-immunogenic
- Very stable "cup-like" structure

- Highly diverse libraries (>10^{11}) of potential drug candidates
- Highly automated selection and screening technology (phage display)
- Deep protein engineering know-how to yield ideal drug candidates

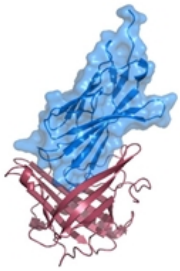
High-affinity (pM) Anticalin bound to



Small target

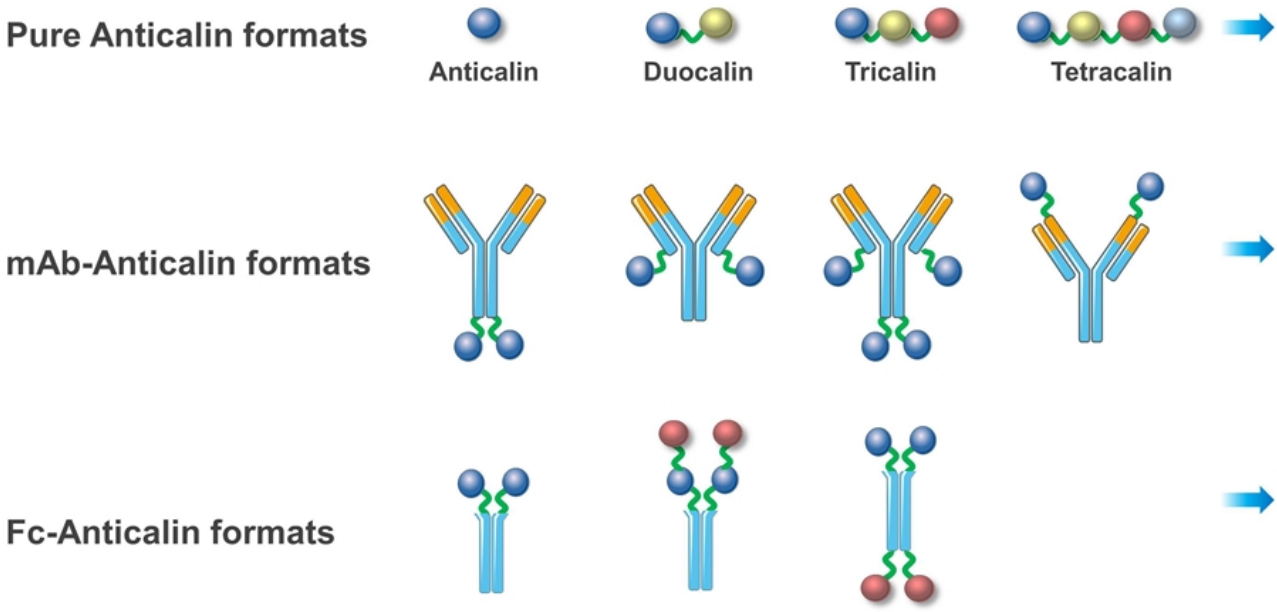


Medium target



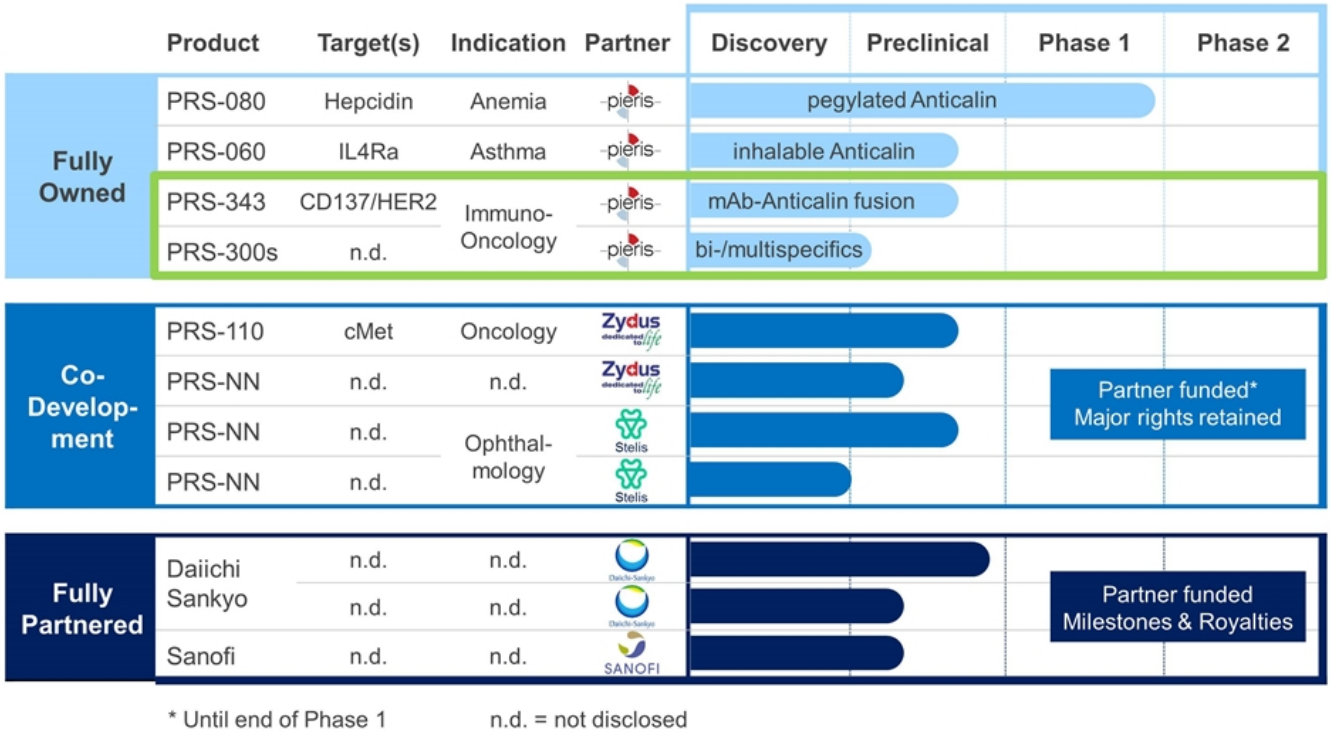
Large target

Going Beyond Anticalin Proteins – Multispecific Drug Candidate Formats

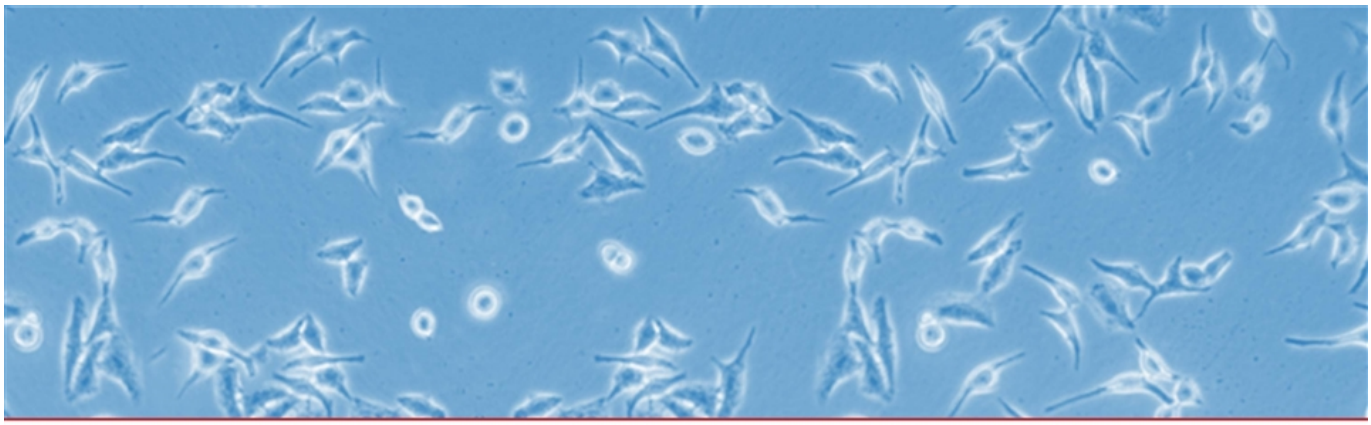


- Molecules designed for optimal target engagement and drug like properties
- Binding site geometry can be adjusted to biological need

Anticalin® Product Pipeline



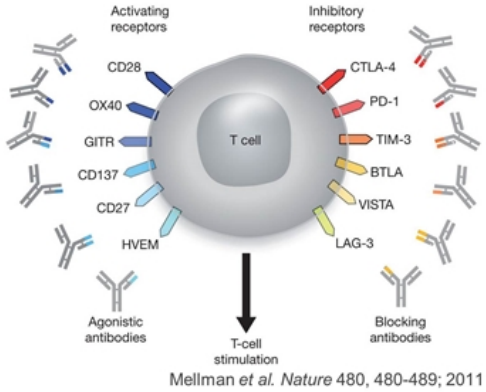
* Until end of Phase 1 n.d. = not disclosed



PRS-300 Series: Multispecifics for Immuno-Oncology

Pieris' Immuno-Oncology Approach – Localized Immune Activation

Monospecific Targeting

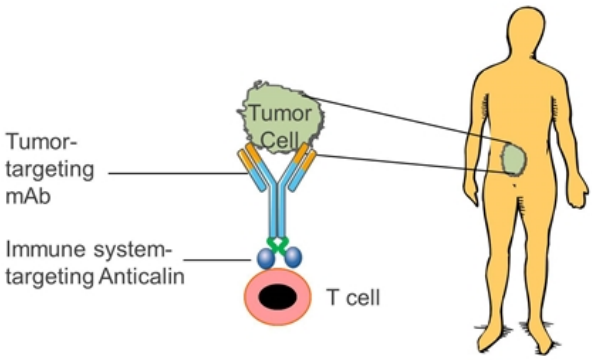


Mellman *et al. Nature* 480, 480-489; 2011

Challenges

- Systemic mAbs often show narrow therapeutic window
- mAbs are poor agonists for certain activating receptors and depend on Fc receptor clustering

Pieris' bispecific approach

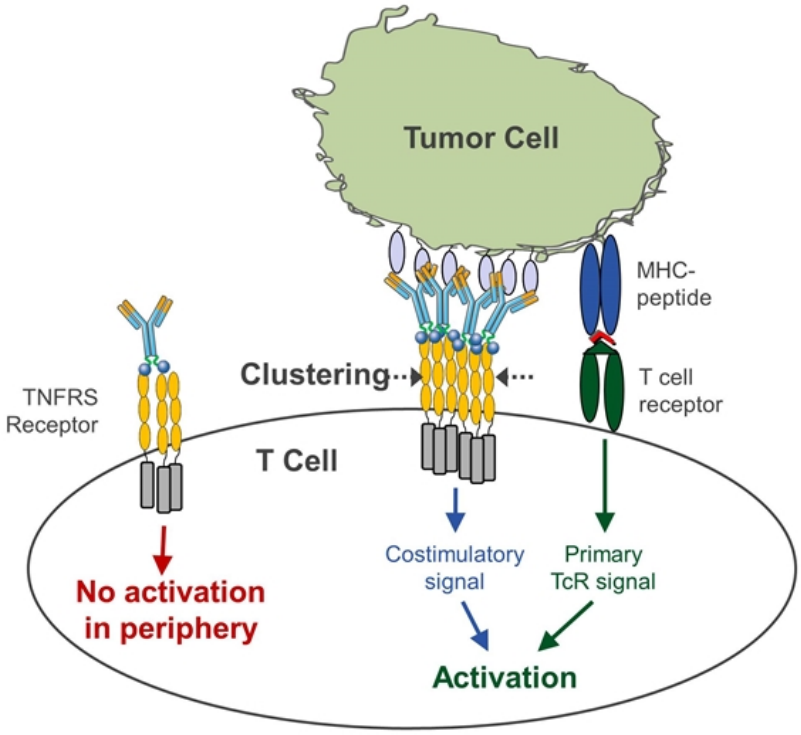


Potential benefits

- Enhanced tolerability with reduced "off-tumor" effects
- Tumor-mediated clustering drives signaling by activating receptors
- Increased efficacy in patients unresponsive to tumor-targeted therapies

Pieris is pursuing both activating and inhibitory IO targets

Costimulatory T cell Engagement in Tumor Microenvironment



Targeted Mode of Action

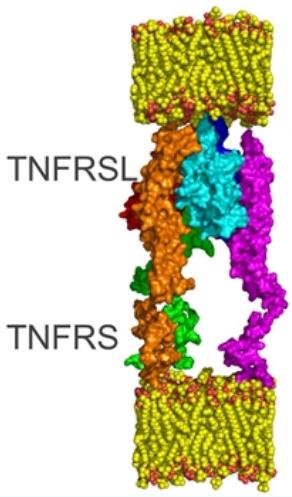
- Clustering of bispecific molecules in tumor microenvironment to drive costimulatory T cell engagement
- Maintaining T cell receptor-mediated tumor antigen specificity on activated T cells

PRS-300 Series
Differentiates from Current IO Approaches

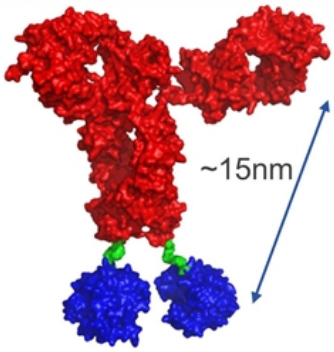
Approach	Tumor-targeted activation	TcR-mediated specificity	Toxicity	Delivery
PRS-300	Yes	Yes	Expected low	Injection
Agonistic mAbs	No	Yes	Low to significant	Injection
BiTE	Yes	No	Observed	Slow infusion
CAR-T	Yes	No	Observed	Individualized adoptive therapy

Bispecific Geometry May Create Different Pharmacodynamic Effects

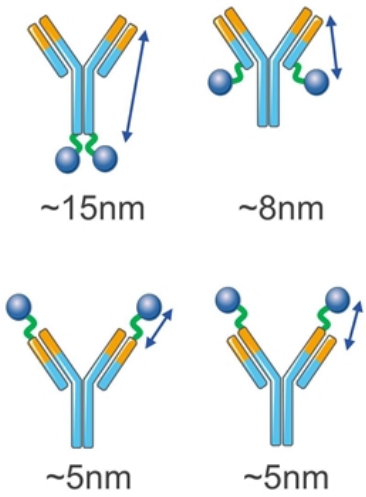
Membrane-to-membrane distances play a role in immunoreceptor interactions



Visualization of Antibody-Anticalin fusion

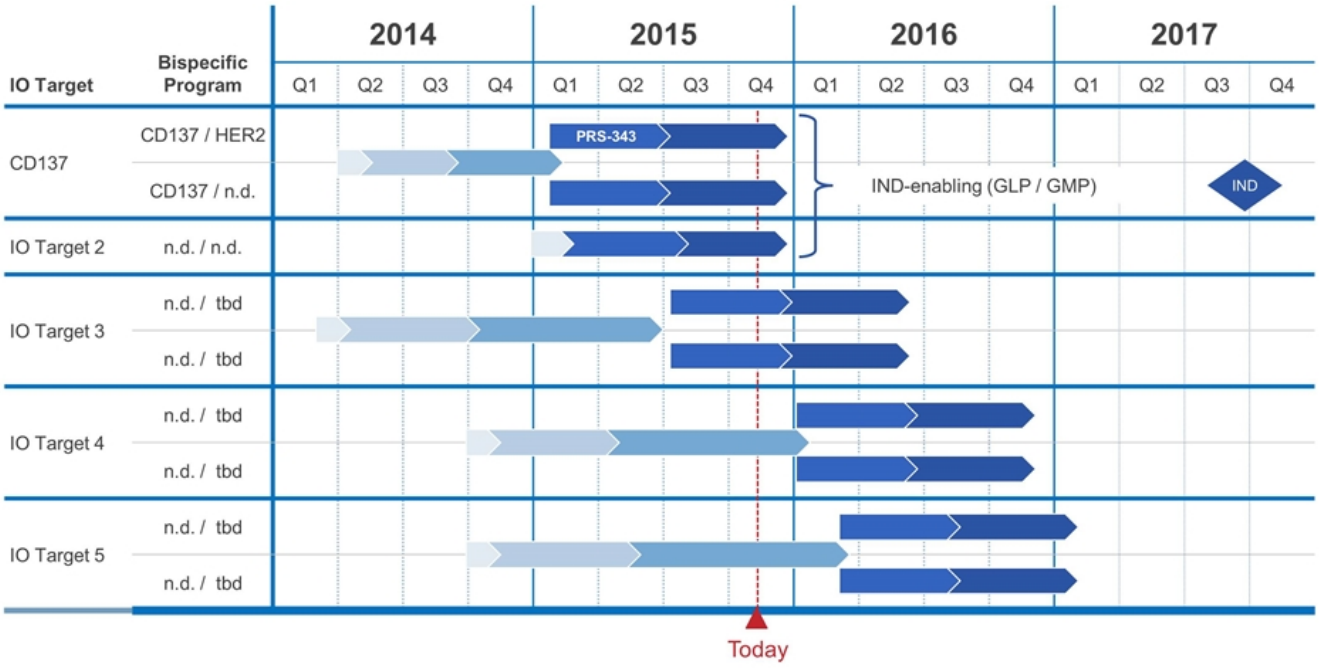


Expected distances between binding sites in different formats



- **Straightforward access to a range of distances between target binding sites**
- **Several formats to interrogate optimal target synapse for tumor cell killing**

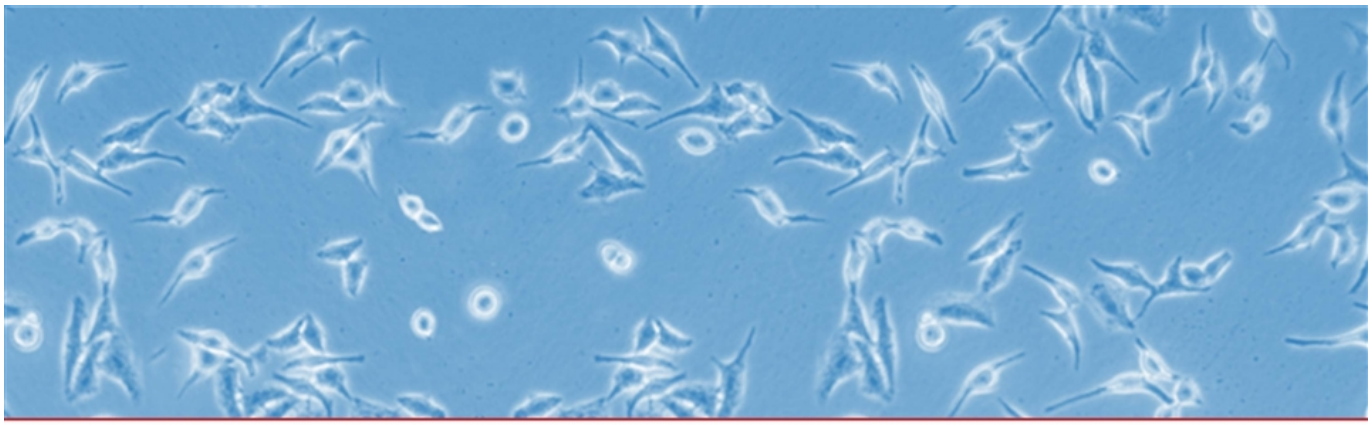
Pieris IO Pipeline Progressing
Multiple Shots on Goal



n.d.: not disclosed

tbd: 2nd target to be defined

Strategy & Target Idea → Discovery / Lead ID → Lead Optimization → Multispecifics & In vitro PoC → In vivo PoC



PRS-343: First-in-class HER2-CD137 Bispecific

PRS-343: HER2-CD137 Bispecific
Multiple Formats Under Preclinical Evaluation

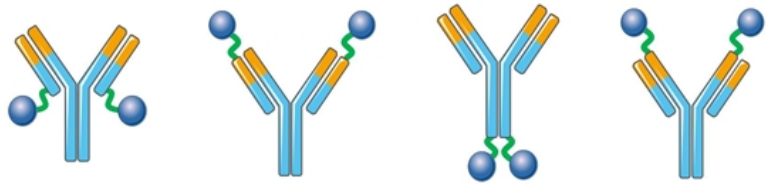
CD137 – a TNFR Costimulatory Target

- **Preclinically and clinically validated**
  - Marker for tumor-reactive T cells
  - Activation leads to tumor elimination in vivo
  - Signaling included in clinical CAR-T cells
- **mAbs struggle to find therapeutic window**
  - Activity depends on Fc receptor interaction
  - Doses required for T cell activation have led to toxicity
  - Current approaches focus on NK activation

HER 2 – Validated but not fully exploited

- **Upregulated on several solid tumors** with significant unmet medical need
  - Bladder, gastric, ovarian, breast cancer
- **Restricted expression on normal tissue** favors immunotherapy approach
- Bispecific immunotherapy approach may **expand responding population**
  - HER2+ tumors with lower expression levels not adequately addressed with current therapy

PRS-343



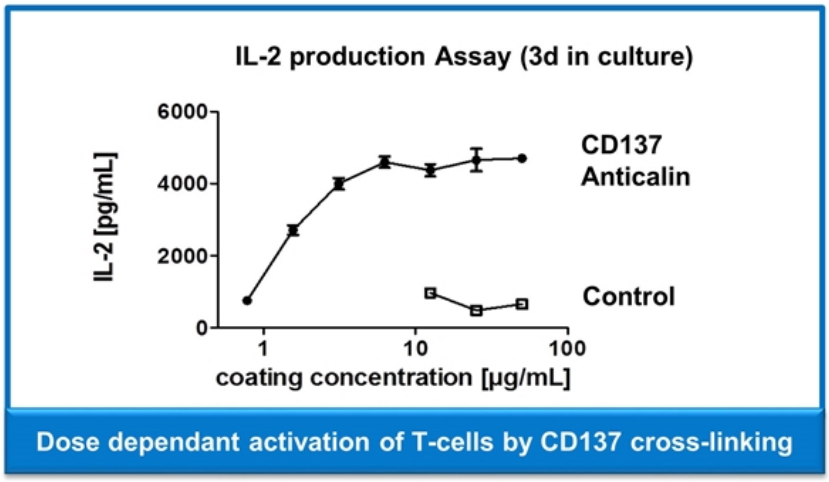
CD137-targeting Anticalin

HER2-targeting mAb (Trastuzumab derived)

CD137-Targeting Anticalin® Has Demonstrated Agonistic Properties

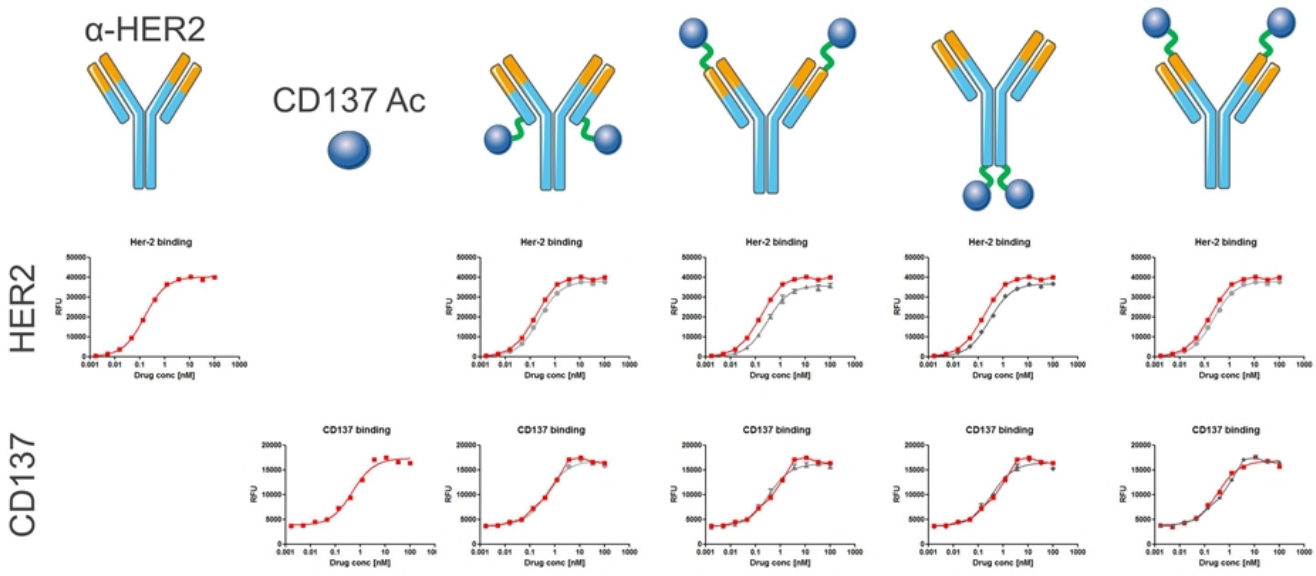
Lead CD137-targeting Anticalin identified (several backups available)

- Affinity: KDhCD137 = 2nM
- "Non-competitive" CD137 engagement preserves ligand-binding capability to CD137L
- Leads to T-cell activation in ex vivo human donor cell assay
- Good biophysical properties: 100% monomeric, high melting temperature (74°C), fully stable at 37°C in PBS or plasma



Dose dependant activation of T-cells by CD137 cross-linking

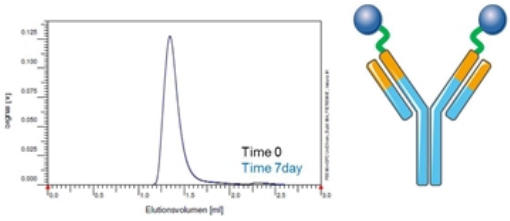
HER2-CD137 Bispecific Formats Retain Target Binding Capacity



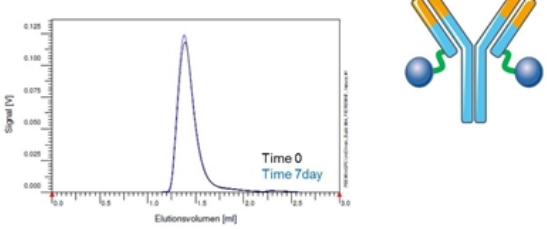
- Bispecific formats behave similarly to CD137 and HER2 building blocks
- Simultaneous target engagement confirmed for bispecific formats

HER2-CD137 Bispecific Formats Exhibit Favorable Biophysical Properties

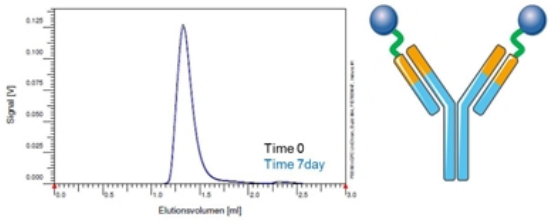
CD137-HER2-IgG4 (HC, N-term)



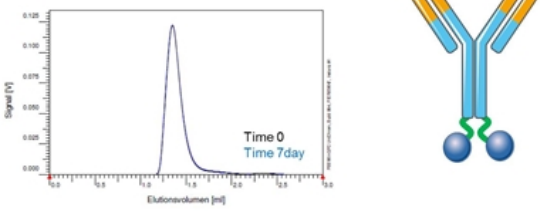
CD137-HER2-IgG4 (LC, C-term)



CD137-HER2-IgG4 (LC, N-term)

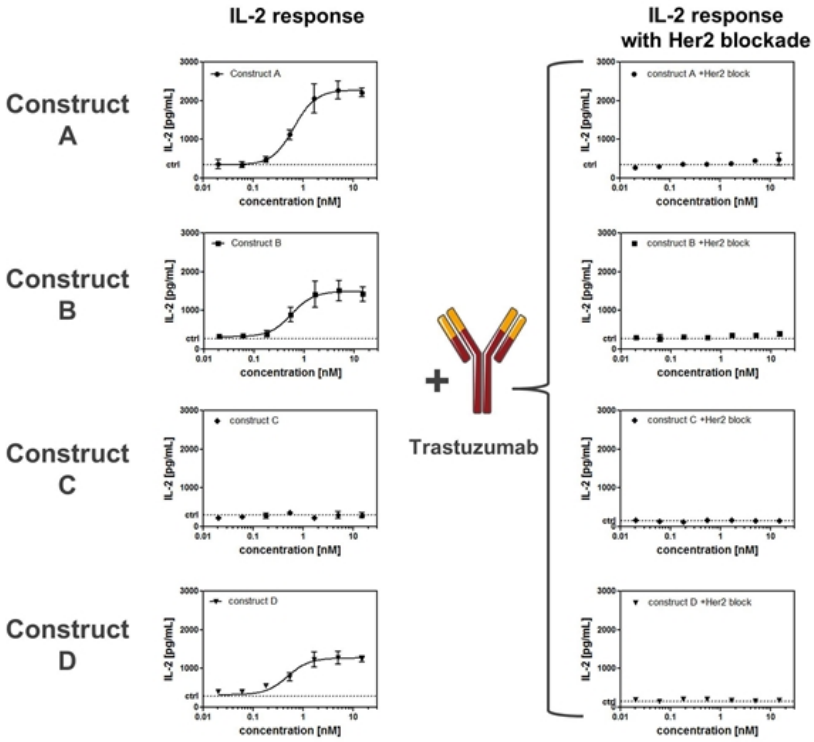


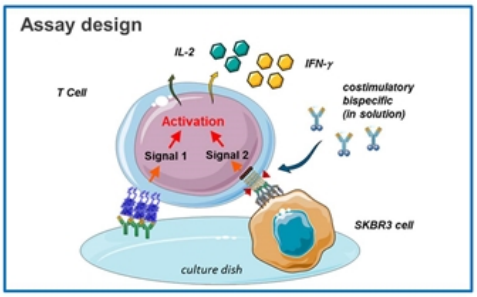
CD137-HER2-IgG4 (HC, C-term)



- **Constructs stable after one week in PBS at 37°C - no change in SEC profile observed**
- **Stability in human plasma also confirmed using a dual binding ELISA**

PoC: T Cell Activation by HER2-CD137 Bispecifics is HER2 Target-Dependent





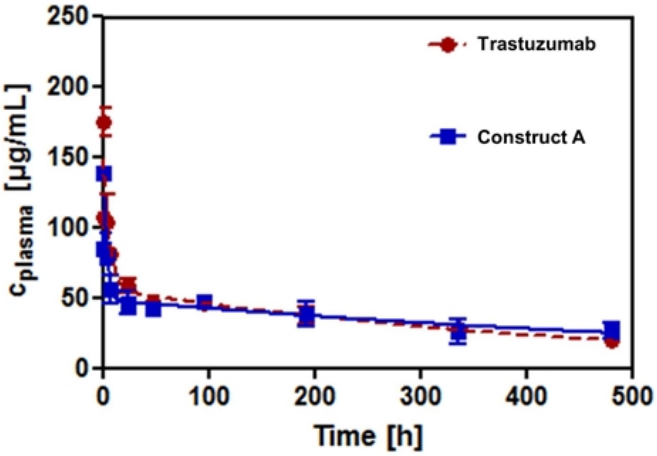
Geometry impacts activity of HER2-CD137 Bispecifics

- Three constructs are capable of activating T cells

Activity is HER2 target-dependent

- Addition of excess Trastuzumab prevents bispecific binding to HER2-positive cells and results in a loss of activity

Pharmacokinetics of HER2-CD137 Bispecifics in Mice Comparable to Trastuzumab

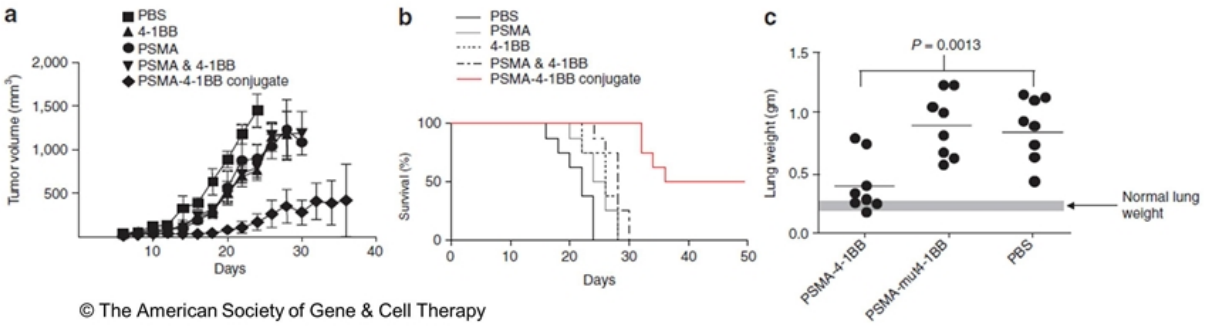


- 10mg/kg of bispecifics or Trastuzumab were injected i.v. in male CD-1 mice (3 mice per timepoint)
- Terminal half-lives of bispecifics range from 15-21 days compared to 13 days for Trastuzumab

➢ Beneficial half-life of parental antibody is preserved for all bispecifics or even exceeded

Preclinical Validation of Tumor-Localized Activation of CD137 (4-1BB)

Tumor Targeted Costimulation With Bi-specific aptamers



- Tumor-targeting CD137 bispecific aptamer leads to tumor growth inhibition and survival advantage *in vivo* compared to combination therapy
- Supports Pieris' bispecifics Mode of Action:
  - Tumor-specific activation of CD137 positive T cells

Pastor *et al,* Molecular Therapy: 2011, 10: 1878-1886

PRS-343 – Status & Path Forward

- **PRS-343: HER2-CD137 Bispecifics**
  - Exhibit excellent binding and drug-like properties with long half lives in mice
  - Induce strong T cell activation via tumor target-dependent costimulatory T cell engagement
  - Are expected to drive potent local activation of tumor-specific T cells with low systemic toxicity

- **PRS-343: Path to Clinic**
  - Cell line development initiated
  - Drug candidate nomination planned for YE 2015
    - Further *ex vivo* profiling:
      - Impact of clustering / receptor density on T cell / NK cell activation
      - Killing of target positive tumor cells
      - Testing of different target-positive tumor cells, different T cell subtypes, etc.
    - *In vivo* evaluation
      - Various animal models including patient derived xenograft (PDX) models
  - Initiate IND enabling studies in 2016
  - Aim to perform clinical trial in HER2 positive cancer in 2017

Summary of Pieris' Immuno-Oncology Efforts

- **Multispecifics to address non-responding patients and broaden therapeutic window**
  - Trafficking immunomodulation to tumor microenvironment
  - Ability to test for optimal synapse through varied geometry
- **Various formats**
  - mAb-Anticalin fusions (e.g., PRS-343)
  - Anticalin-Anticalin fusions (undisclosed)
- **Multiple targets**
  - Prioritization of costimulatory targets
  - Multiple checkpoint inhibitors also being investigated
  - Each immunomodulatory target combinable with different tumor targets
- **External collaborations complementing internal expertise and resources to advance drug candidates**

Pieris Pharmaceuticals, Inc.

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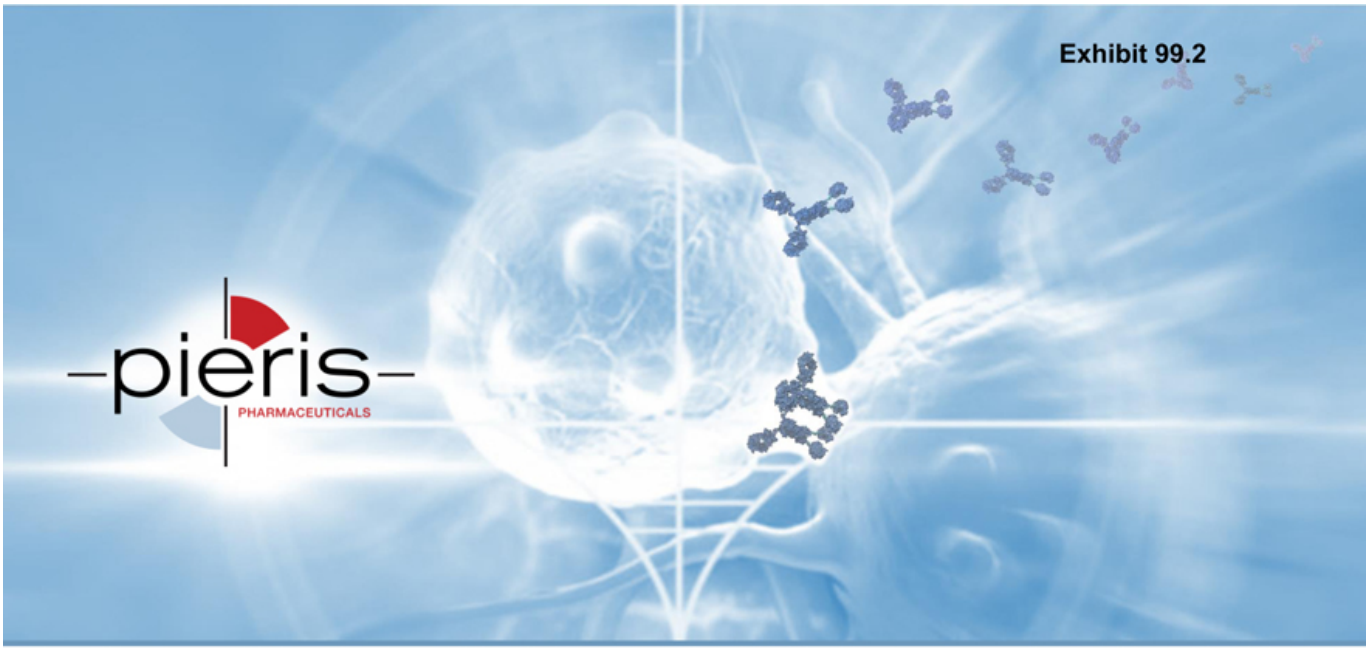
Boston, MA 02109
USA
info@pieris.com



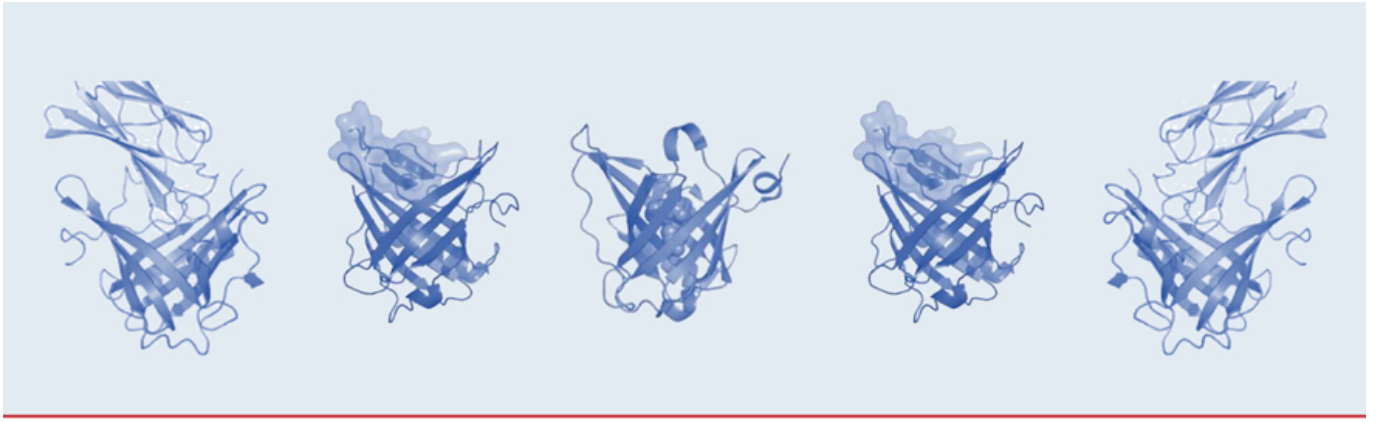
Bispecific Anticalin Fusion Proteins for Localized Targeting of Immune Cells for Application in Immuno-Oncology

Christine Rothe, Ph.D.

PEGS Europe Summit, Nov 4, 2016

Forward Looking Statements

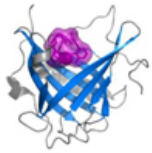
Statements in this presentation that are not descriptions of historical facts are forward-looking statements that are based on management's current expectations and assumptions and are subject to risks and uncertainties. In some cases, you can identify forward-looking statements by terminology including "anticipates," "believes," "can," "continue," "could," "estimates," "expects," "intends," "may," "plans," "potential," "predicts," "projects," "should," "will," "would" or the negative of these terms or other comparable terminology. Factors that could cause actual results to differ materially from those currently anticipated include, without limitation, risks relating to the results of our research and development activities, including uncertainties relating to the discovery of potential drug candidates and the preclinical and clinical testing of our drug candidates; the early stage of our drug candidates presently under development; our ability to obtain and, if obtained, maintain regulatory approval of our current drug candidates and any of our other future drug candidates; our need for substantial additional funds in order to continue our operations and the uncertainty of whether we will be able to obtain the funding we need; our ability to retain or hire key scientific or management personnel; our ability to protect our intellectual property rights that are valuable to our business, including patent and other intellectual property rights; our dependence on third-party manufacturers, suppliers, research organizations, testing laboratories and other potential collaborators; competition in our industry; regulatory developments in the U.S. and foreign countries; as well as those risks more fully discussed in the "Risk Factors" section of our Current Report on Form 8-K filed with the SEC on December 18, 2014, the Company's annual report on Form 10-K for the fiscal year ended December 31, 2014, the Company's quarterly reports on Form 10-Q, and the other reports we file with the SEC. In light of these risks, uncertainties and assumptions, the forward-looking statements regarding future events and circumstances discussed in this report may not occur and actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. The forward-looking statements included in this presentation speak only as of the date hereof, and except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation to conform these statements to actual results or to changes in our expectations.



Company and Technology Overview

Pieris Pharmaceuticals, Inc.

- **Anticalins** are a novel class of protein therapeutics, proprietary to Pieris, with several degrees of validation



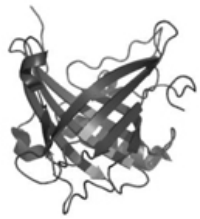
- Human data demonstrating desired drug-like properties
  - 26 solid tumor patients with VEGF-A antagonist
  - 36 healthy volunteers with hepcidin antagonist

- Proven track record for successful collaborations with Pharma

Daiichi-Sankyo SANOFI ALLERGAN Zydus dedicated to life Stelis Biopharma

Human Lipocalins – Scaffold for Novel Anticalin® Therapeutics

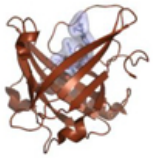
Human lipocalin "template"



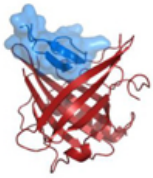
- Human, natural binding proteins
- Low molecular weight (~1/8 of mAb size)
- Extracellular
- Non-immunogenic
- Very stable "cup-like" structure

- Highly diverse phage display libraries (>10^{11}) of potential drug candidates
- Automated selection and screening technology
- Deep protein engineering know-how to yield ideal drug candidates

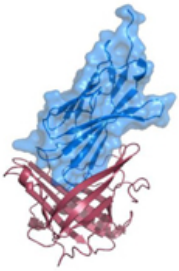
High-affinity (pM) Anticalin bound to



Small target

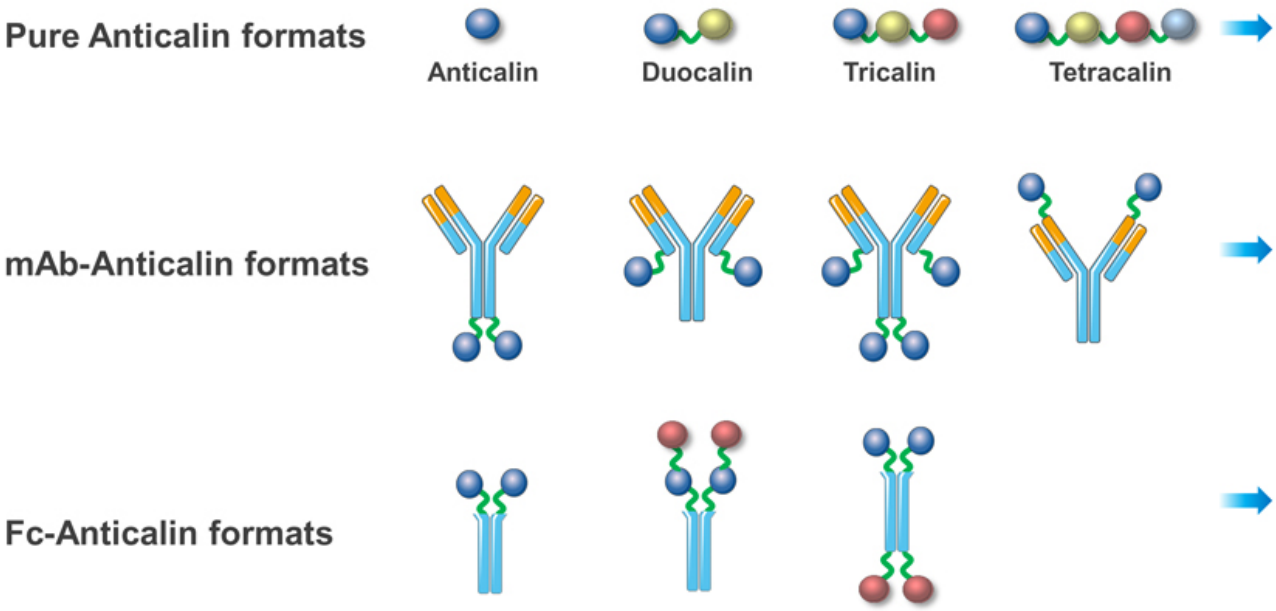


Medium target



Large target

Going Beyond Anticalin Proteins – Multispecific Drug Candidate Formats



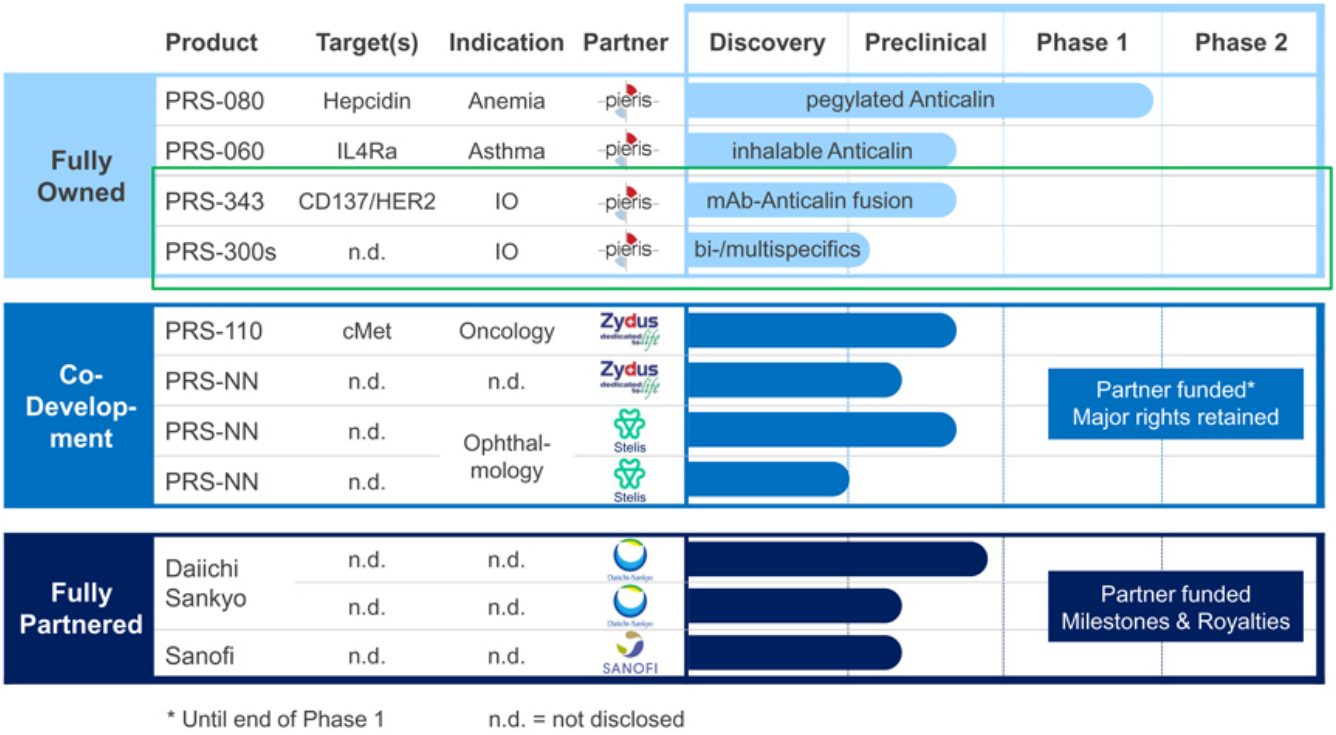
- Molecules designed for optimal target engagement and drug-like properties
- Binding site geometry can be adjusted to biological need

Anticalin® Product Pipeline

	Product	Target(s)	Indication	Partner	Discovery	Preclinical	Phase 1	Phase 2
Fully Owned	PRS-080	Hepcidin	Anemia	pieris	pegylated Anticalin			
	PRS-060	IL4Ra	Asthma	pieris	inhalable Anticalin			
	PRS-343	CD137/HER2	IO	pieris	mAb-Anticalin fusion			
	PRS-300s	n.d.	IO	pieris	bi-/multispecifics			
Co-Develop-ment	PRS-110	cMet	Oncology	Zydus			Partner funded* Major rights retained	
	PRS-NN	n.d.	n.d.	Zydus				
	PRS-NN	n.d.	Ophthal-mology	Stelis				
	PRS-NN	n.d.		Stelis				
Fully Partnered	Daiichi Sankyo	n.d.	n.d.	Daiichi-Sankyo			Partner funded Milestones & Royalties	
		n.d.	n.d.	Daiichi-Sankyo				
	Sanofi	n.d.	n.d.	SANOFI				

* Until end of Phase 1 n.d. = not disclosed

Anticalin® Product Pipeline



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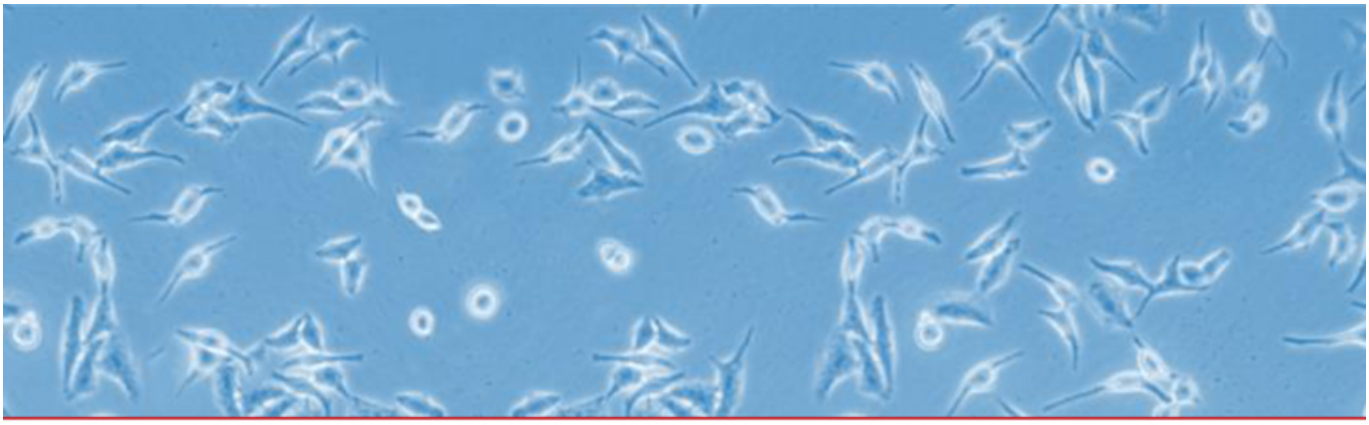
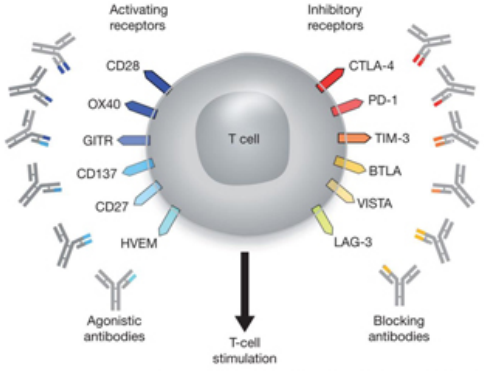


PRS-300 Series: Multispecifics for Immuno-Oncology

Pieris' Immuno-Oncology Approach – Localized Immune Activation

Monospecific Targeting

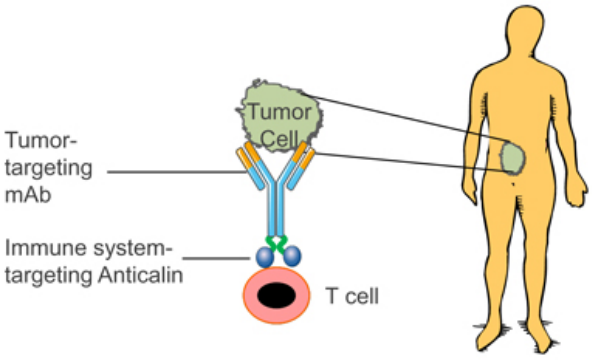


Mellman *et al. Nature* 480, 480-489; 2011

Challenges

- Systemic mAbs often show narrow therapeutic window
- mAbs are poor agonists for certain activating receptors and depend on Fc receptor clustering

Pieris' Bispecific Approach



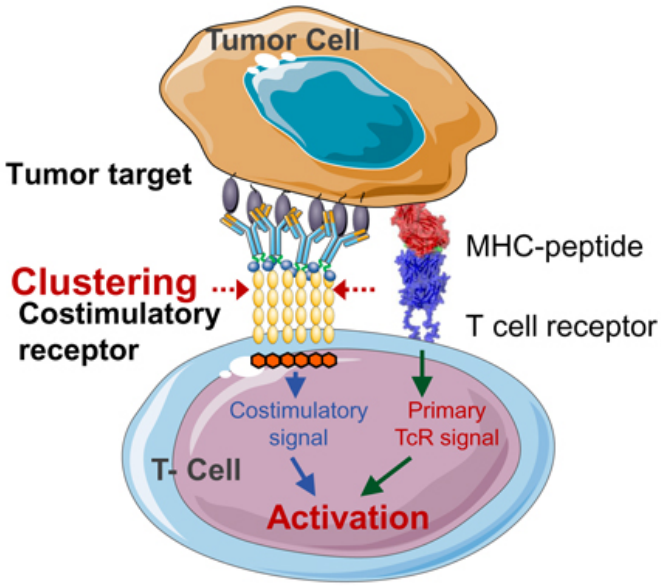
Potential benefits

- Enhanced tolerability with reduced "off-tumor" effects
- Tumor-mediated clustering drives signaling by activating receptors
- Increased efficacy in patients unresponsive to tumor-targeted therapies

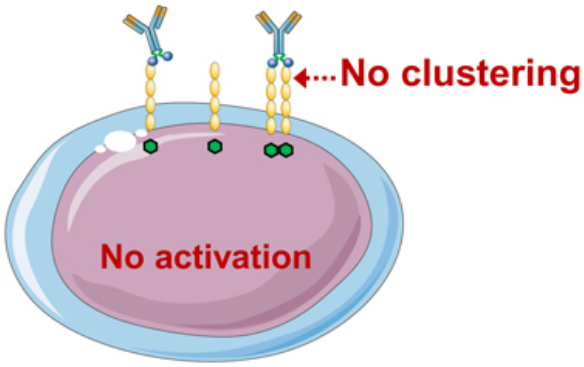
Pieris is pursuing both activating and inhibitory IO targets

Costimulatory T cell Engagement in Tumor Microenvironment

T cell costimulation in tumor

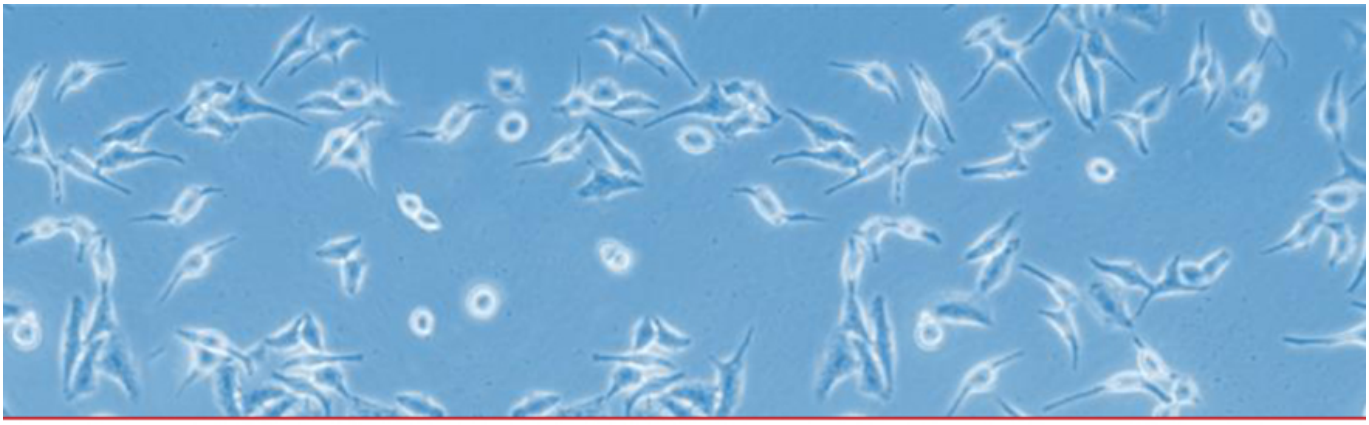


No T cell costimulation in periphery



Targeted Mode of Action

- Clustering of bispecific molecules in tumor microenvironment drives costimulatory T cell engagement
- Maintaining T cell receptor-mediated tumor antigen specificity on activated T cells



PRS-343

HER2-CD137 Bispecific Anticalin Fusion Proteins

PRS-343: HER2-CD137 Bispecifics

Multiple Formats Under Preclinical Evaluation

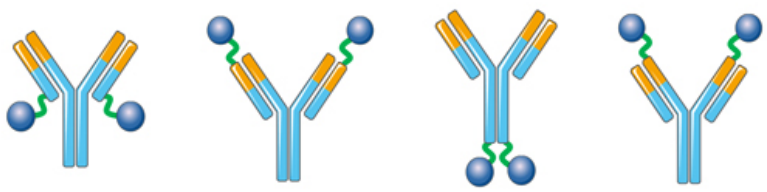
CD137 – a TNFR Costimulatory Target

- **Preclinically and clinically validated**
  - Marker for tumor-reactive T cells
  - Activation leads to tumor elimination in vivo
  - Signaling included in clinical CAR-T cells
- **mAbs struggle to find therapeutic window**
  - Activity depends on Fc receptor interaction
  - Doses required for T cell activation have led to toxicity

HER 2 – Validated but not fully exploited

- **Upregulated on several solid tumors** with significant unmet medical need
  - Bladder, gastric, ovarian, breast cancer
- **Restricted expression on normal tissue** favors immunotherapy approach
- Bispecific immunotherapy approach may **expand responding population**
  - HER2+ tumors with lower expression levels not adequately addressed with current therapy

PRS-343



CD137-targeting Anticalin

HER2-targeting mAb (trastuzumab derived)

CD137 Targeting Lead Anticalin® Has Demonstrated Agonistic Properties

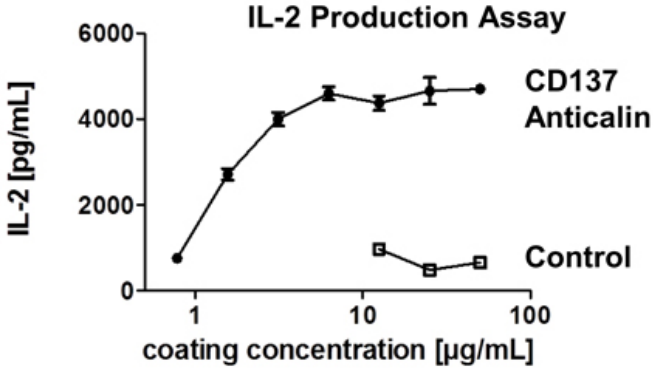
Binding to CD137

- Affinity: KD_{hCD137} = 2nM
- "Non-competitive" CD137 engagement preserves ligand-binding capability to CD137L

Biophysical properties

- 100% monomeric expression
- TM = 74°C (DSC)
- Fully stable after 1 week at 37°C in PBS, hu or mu plasma

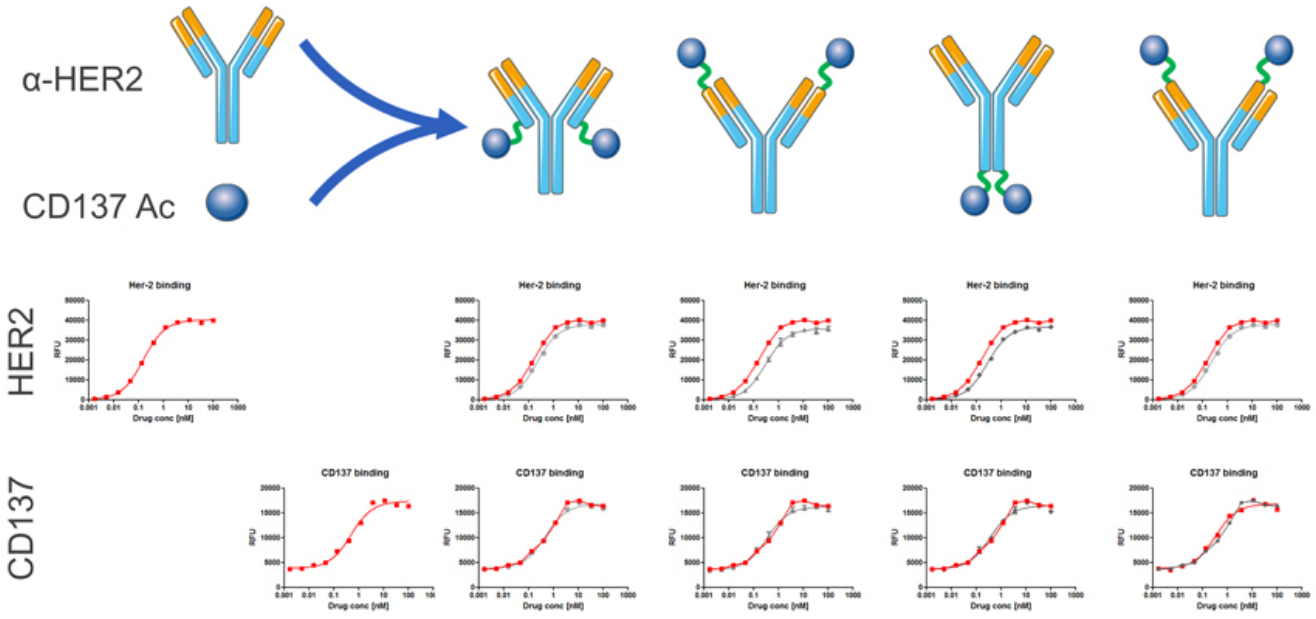
In vitro functional testing



Dose dependent T-cell activation in ex vivo human donor cell assay by CD137 clustering

- CD137-specific Anticalin coated together with subthreshold concentration of aCD3 antibody on ELISA plate

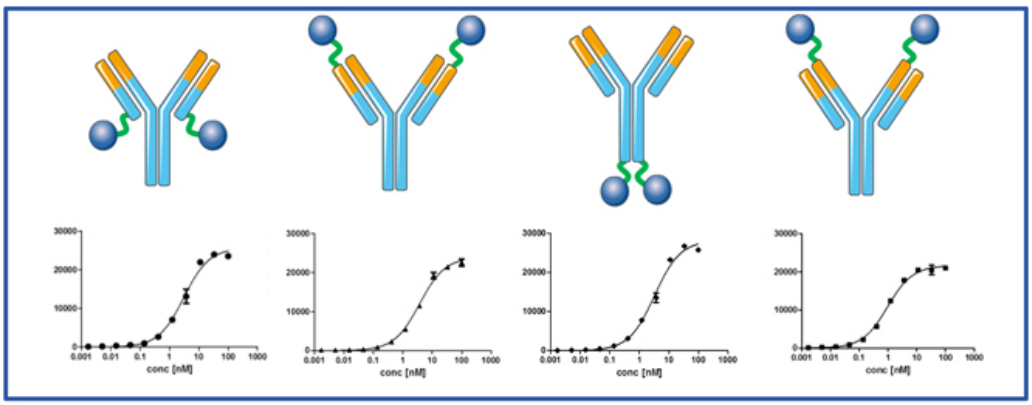
HER2-CD137 Bispecific Formats Retain Target Binding Capacity



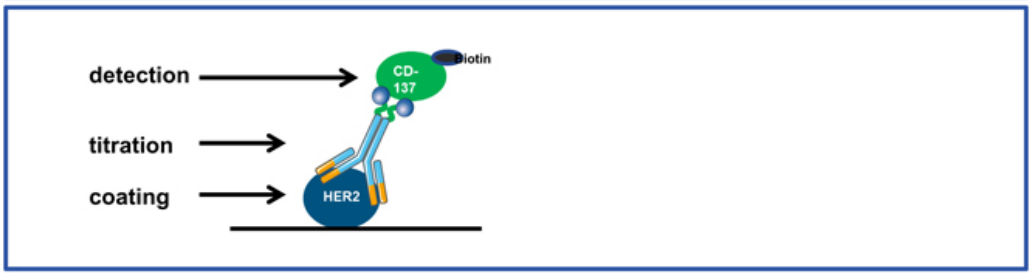
Bispecific formats show similar binding to CD137 and HER2 as building blocks

HER2-CD137 Bispecific Formats Bind Both Targets at the Same Time

Dual binding ELISA data

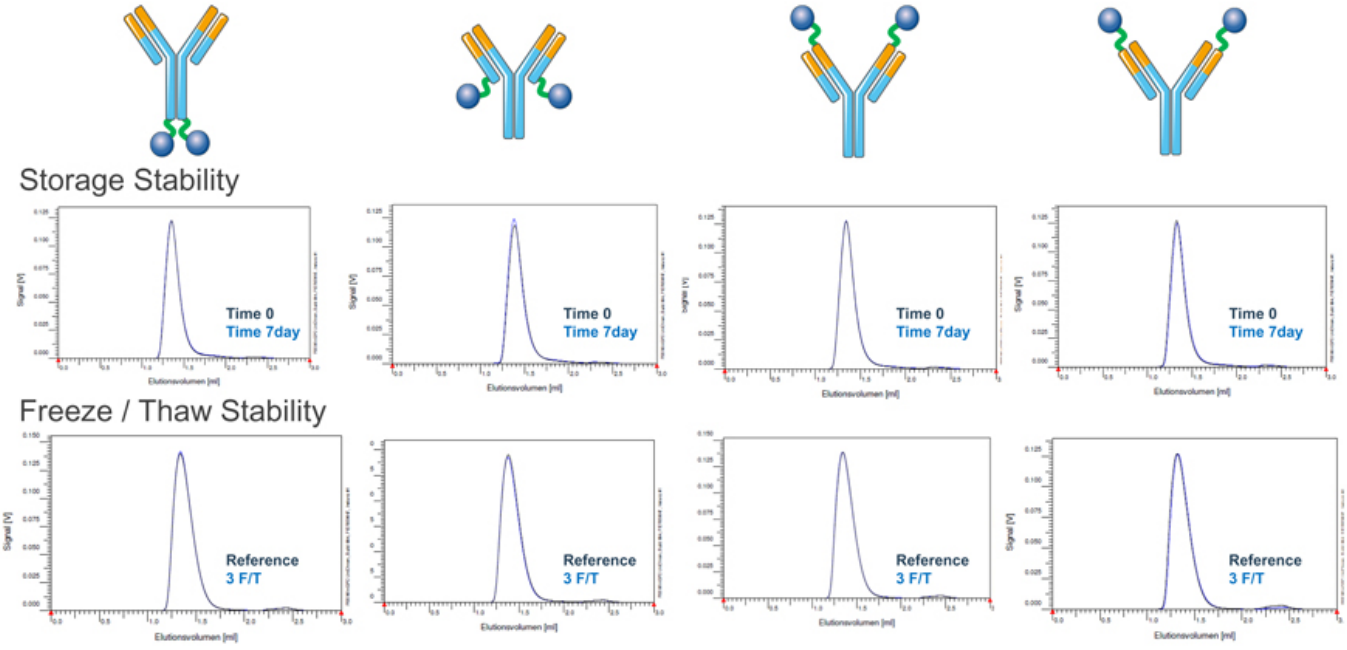


Dual binding assay format



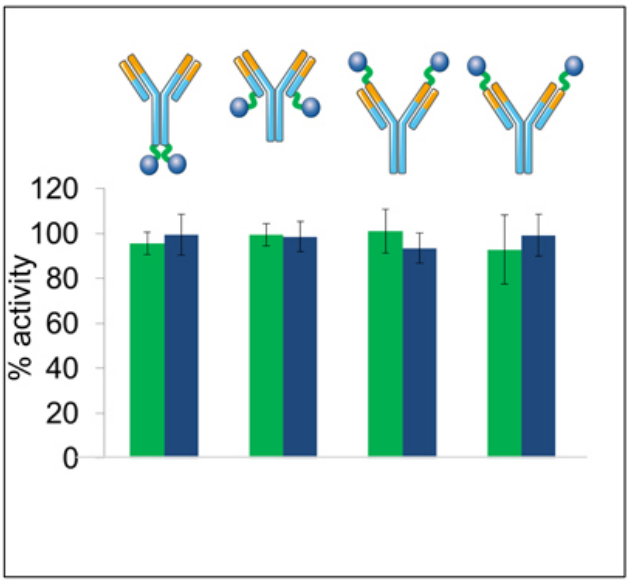
Simultaneous target engagement confirmed for all bispecific formats

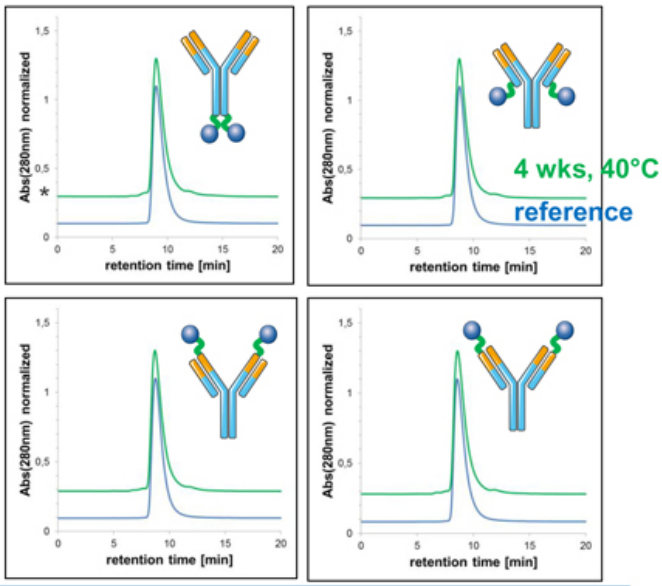
PRS-343 Bispecifics Exhibit Favorable Biophysical Properties



- Constructs are stable after freeze / thaw cycles and one-week storage in PBS at 37°C
- No change in SEC profile and full recovery of activity in qELISA

Plasma and Storage Stability Confirmed





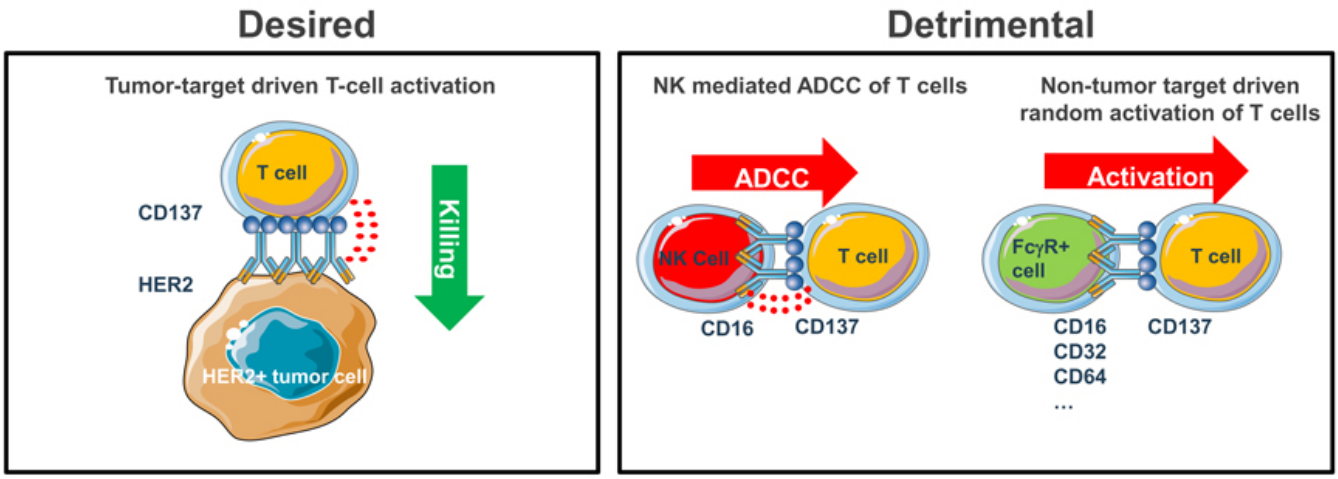
Plasma stability confirmed

- Fully active after 1 week in human (green) and mouse plasma (blue) at 37°C (0.5mg/ml; dual binding qELISA)

Storage stability confirmed

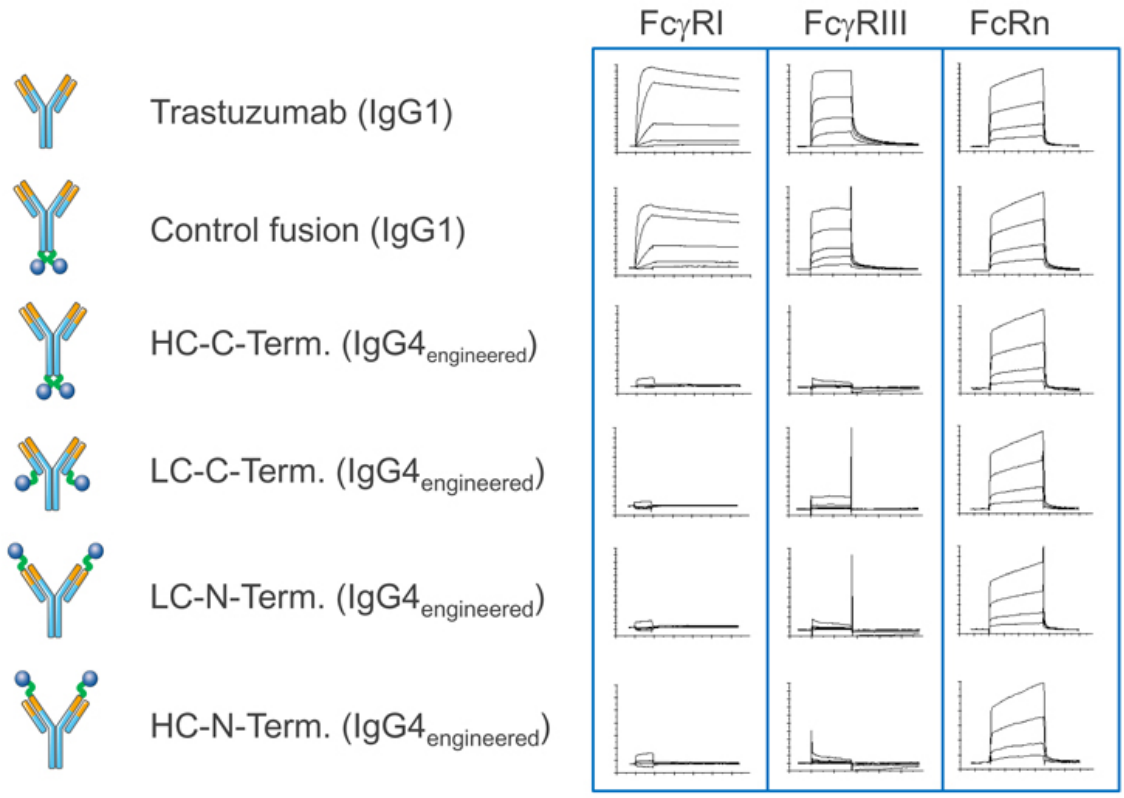
- Fully stable and active after 4 weeks at 40°C in PBS (20mg/mL, aSEC and dual binding qELISA); *blotted with an off-set on the y-axis for better visualization

HER2-CD137 Bispecifics Mode of Action – Relevance of Fc-γ Receptor Interaction



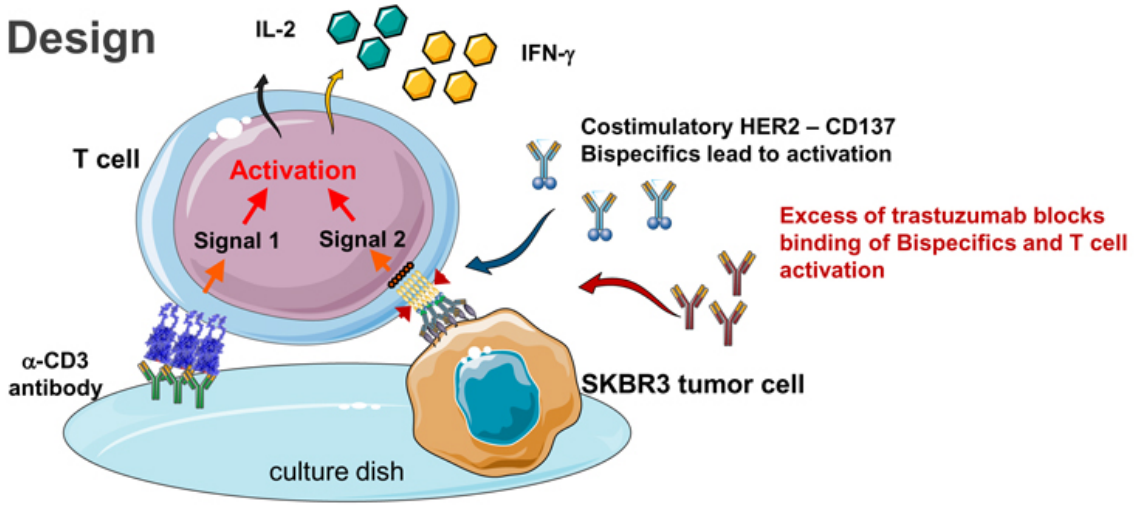
- Desired mode of action is HER2-dependent CD137 clustering and activation on T cells
- Trastuzumab IgG1 backbone could induce undesired side effects of ADCC directed against T cells and non-tumor localized activation of T-cells via FcγR positive cells in the periphery
- ➢ PRS-343 bispecifics contain trastuzumab with an engineered IgG4 backbone to minimize FcγR binding

Engineered IgG4 Backbone Ensures Reduced FcγRI & FcγRIII Interaction – FcRn Interaction Retained



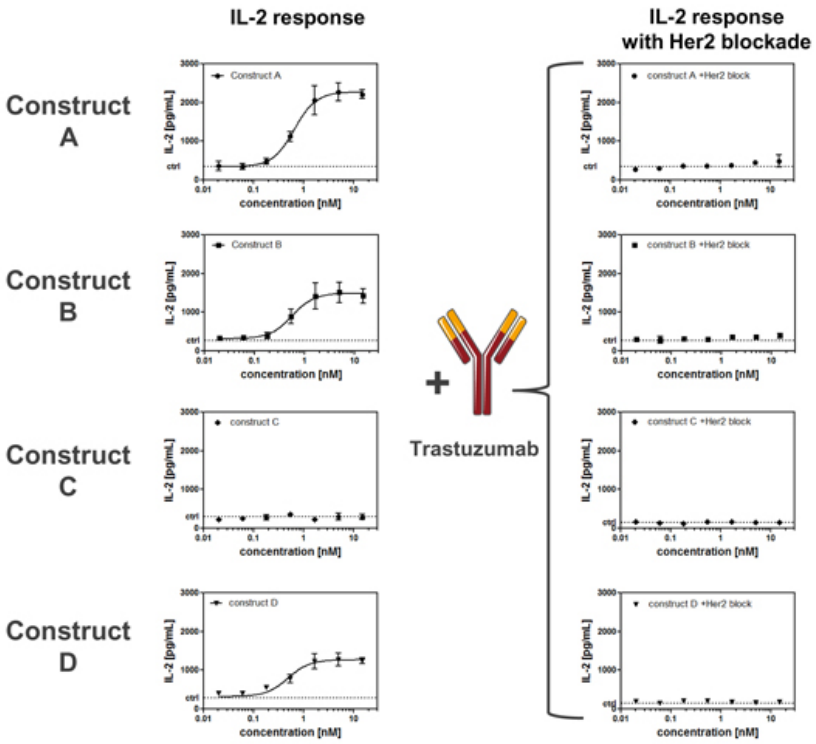
PoC: T Cell Activation by HER2-CD137 Bispecifics is HER2 Target-Dependent

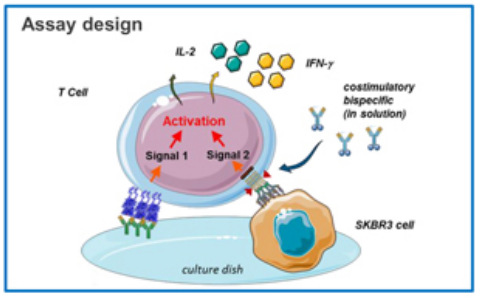
Assay Design



- Her-2 positive SKBR3 cells were grown on 96-well culture dishes, precoated with aCD3 antibody
- T cells from healthy donor PBMCs were added together with HER2 - CD137 bispecifics to activate T cells
- Excess of trastuzumab inhibits binding of HER2 – CD137 bispecifics and activation of T cells

PoC: T Cell Activation by HER2-CD137 Bispecifics is HER2 Target-Dependent





Activity is HER2 target-dependent

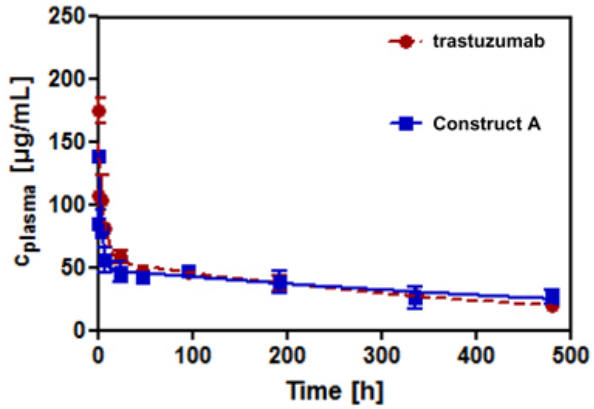
- Addition of excess trastuzumab prevents bispecific binding to HER2-positive cells and results in a loss of activity

Geometry impacts activity of HER2-CD137 Bispecifics

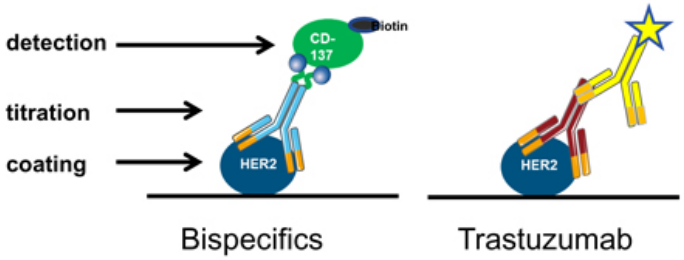
- Three constructs are capable of activating T cells

Pharmacokinetics of HER2-CD137 Bispecifics in Mice are Comparable to Trastuzumab

Pharmacokinetics in mice



PK assay set up



- 10mg/kg of bispecifics or trastuzumab were injected i.v. in male CD-1 mice (3 mice per timepoint)
- Terminal half-lives of bispecifics range from 15-21 days compared to 13 days for trastuzumab
- ➢ Beneficial half-life of parental antibody is preserved for all bispecifics or even exceeded

Summary and Path Forward

- **PRS-343: HER2-CD137 Bispecifics**
  - Exhibit excellent binding and drug-like properties with long half lives in mice
  - Induce strong T cell activation via tumor target-dependent costimulatory T cell engagement
  - Expected to allow potent local activation of tumor-specific T cells with low toxicity

- **PRS-343 Path to Clinic**
  - Drug candidate nomination planned for YE 2015
  - Initiate IND enabling studies in 2016
  - Aim to perform clinical trial in HER2-positive cancer in 2017

- **Pieris' IO pipeline focusing on multiple targets**
  - Pieris is pursuing both activating and inhibitory IO targets
  - Each immunomodulatory target combinable with different tumor targets
- **Bispecifics approach for tumor localized immune activation**
  - Variable bispecific geometry facilitates optimal engagement for all receptors

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Thanks to the Pieris Team!