ShareVault®

Share documents. Simply, securely.

WHAT'S HOT & WHAT'S NOT IN

ANTIBODY-DRUG CONJUGATES (ADCs) LICENSING

Based on the web panel discussion of the same name that took place on January 24, 2019

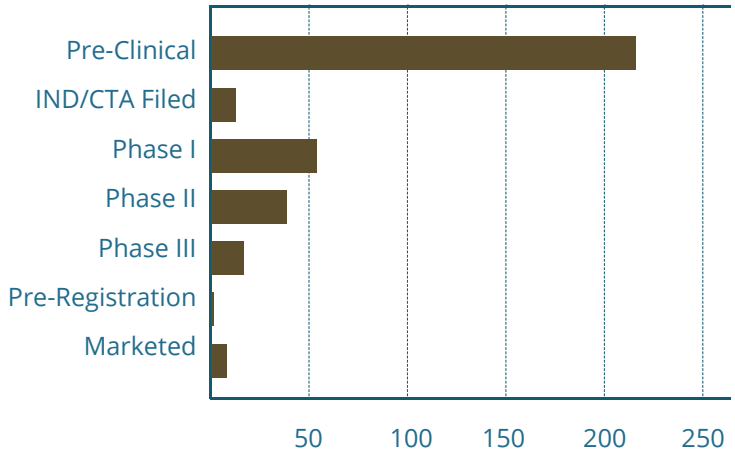
Antibody-drug conjugates, or ADCs, are a growing class of highly potent biopharmaceutical drugs that add the power of a potent drug, such as cancer killing cytotoxic small molecules, to the targeting specificity of antibody for greater efficacy while minimizing the damage to untargeted cells.

To date, six ADCs have received market approval and over 100 are in clinical development. Sales of ADCs were close to $2 billion in 2018 and expected to grow to $12 billion by 2024 (GlobalData). Many of the ADCs approved thus far have been antibodies conjugated with small molecule cytotoxics to kill the antibody targeted cancer cell after being internalized with the antibody on the receptor.

On January 24, 2019, ShareVault, in partnership with Pullan Consulting, Cello Health, BIO and LES, hosted a web panel discussion exploring the mushrooming field of ADCs and the challenges and potential for patient care, licensing deals, and the oncology marketplace. The web panel was made up of:

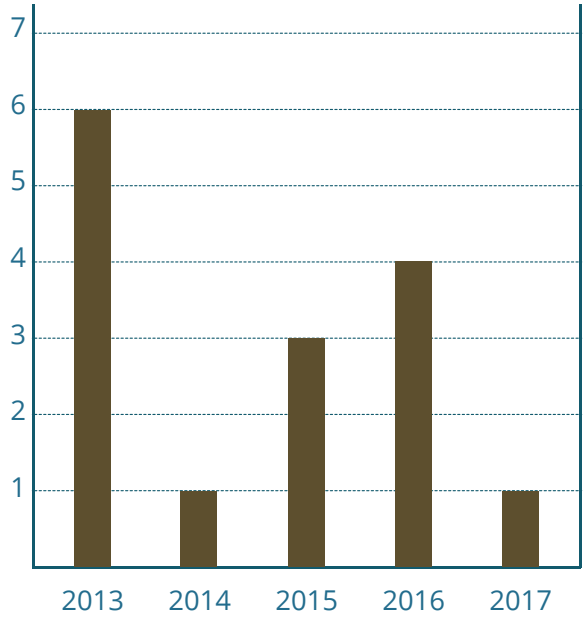
- **Linda Pullan of Pullan Consulting, Moderator**
- **Jeff Bockman of Cello Health BioConsulting (Previously Defined Health)**
- **Peter Dragovich of Genentech**
- **Jason Kim of Molecular Templates**
- **Neela Patel of Seattle Genetics**

Number of ADCs by Stage



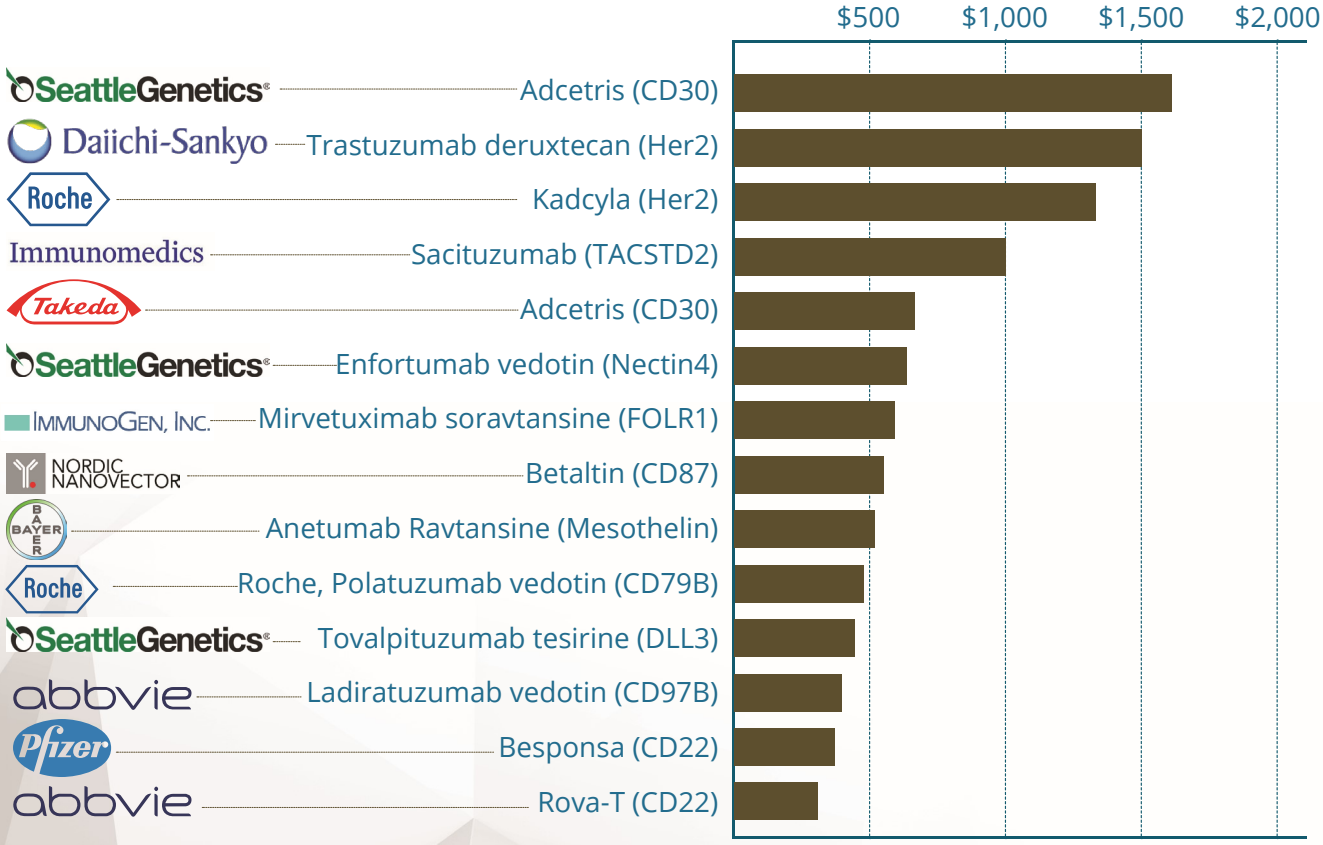
Source: GlobalData

Number of Deals for ADCs



Source: GlobalData

Top 2024 Sales Forecasts (Millions of $USD)



Source: GlobalData

TARGETS

Linda Pullan: The top targets for ADCs are well known in cancer biology, but let's talk more broadly about what's needed in a target.

Jason Kim: An ideal ADC target is one that's highly expressed with low heterogeneity on the tumor while having a low expression on normal cells. Ideally, it's also a target that's not secreted or shed at high levels in order to mitigate some of the issues that might come up with binding and circulation.

The target must be able to internalize efficiently via receptor-mediated endocytosis once the ADC is bound. Once inside the cells, the ADC needs to be trafficked to the lysosome for drug release. All of these factors are relevant for how amenable a target may be when being considered as an ADC target.

Of course, because the drug conjugate effect is a stoichiometric function of the drug amount delivered, the aim is obviously to get as much drug into the tumor as possible given the tolerability profile of the ADC.

Linda: How do ADC targets compare to other therapeutic modalities?

Jeff Bockman: One thing that's clear is that oncology is, and always has been, hungry for targets. That's become even more paramount as we've moved beyond antibodies and ADCs and now to bispecifics of various sorts, especially redirecting ones, and various types of adoptive cell therapies such as CARTs, TCRs, et cetera.

The chart below shows many of the usual suspects that are being pursued. That's not to say there isn't an unmet

Number of Agents in Development per Target, by Modality PC → MRKT	HER2	CD19	CD20	PSMA	BCMA	CEA/CEACAM5	CD33	CD123	EpCAM	ROR1	GPC3	5T4	B7H3	P-cadherin	A33	CEACAM6	CEACAM1	CLEC12A
CAR-T cells	6	55	6	5	12	2	6	7	1	3	6	1				2		1
Antibody-drug conjugate	45	10	7	12	2	2	9	4	8	2	1	2	4	3	2			
Bispecific/trispecific antibody	20	15	12	7	13	5	5	6	7	4	3	1	1	1	1			1
Naked monoclonal antibody	23	9	18			3	1	2		3	3		1		1	1	3	
Small molecule	33			8			1			3								
Cancer vaccine	32					4				1	1			1				
Fusion protein	12	4	6			3	1	2	1			1						
Other cell therapy	3	6	1	1	4	1					1							
Peptide	4																	
Oncolytic virus						3						1						
Undefined		1	1	1														
Recombinant product	1																	
Other						1												

Sources: Adis R&D Insight; Clarivate Analytics Cortellis, Cello Health BioConsulting/Defined Health analysis

Key (# of agents)				
1-5	6-10	11-15	16-20	>20

need for many of these targets, such as HER2, CD19 and CD20, which are defined by current antibodies. Or, in the case of CD19, first generation CARTs. Many of those targets to the right are known, but less validated, certainly not validated in the sense of being approved.

When any company is pursuing targets, whether it's an ADC company or a bispecific company, it behooves them to think broadly. As we saw from ASH (American Society of Hematology) with the data from the redirecting BCMA program, some of these can be directly competitive with CARTs.

If a company has an ADC, CART or bispecific directed at one of these targets, that company needs to be thinking broadly about other types of modalities that could be competitive. That broad hunger for targets means that there's a lot of potential for those companies that are in any sort of target discovery mode.

There are a lot of companies doing that. Some of them are dedicated to the discovery of surface antigens and novel targets. They come in all sorts of flavors, from the T-cell peptide targets from the discovery platform of an Immatics to the antibody and antigen target discovery approach that Atreca uses by mining elite responders to checkpoint inhibitors for antibodies.

Any company operating in a space where they need targets would be well advised to seek partnerships or collaborations with some of these other discovery companies.

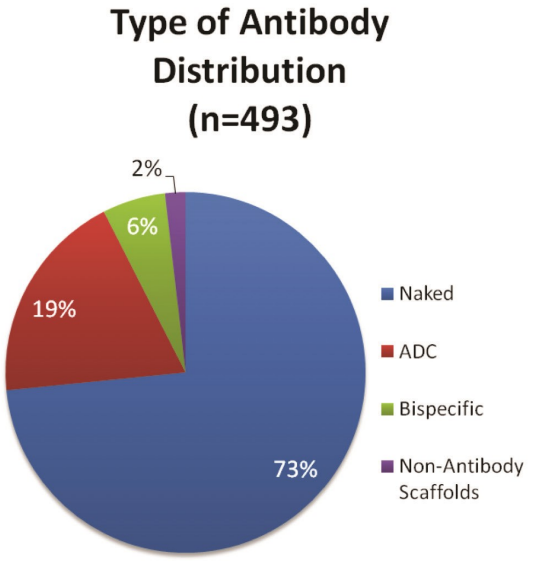
DRUGS

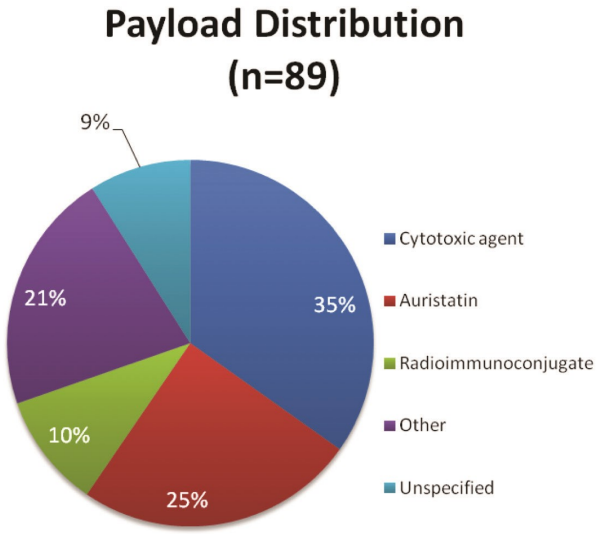
Linda: What kinds of drugs are suitable for ADCs and what is the consideration for payloads?

Pete Dragovich: Originally, the payloads that were used were very potent agents, typically targeting tubulin or DNA. We are all familiar with the auristatins MMAE and MMAF as payloads used on many ADCs, many of which are still in the clinic. Also in the tubulin binder payload class are the maytansines (DM1, DM4), which have also served quite well.

One of the original DNA damaging agents that was used was calicheamicin. It's an enediyne antibiotic and is also a very potent agent. That molecule functions by damaging DNA. It's worth pointing out that two of the approved ADCs use calicheamicin as a payload, so it is possible to have a DNA damaging agent included in an approved drug.

However, recently the field has shifted to a different class of DNA damaging agents, namely the PBD dimer (pyrrolobenzodiazepine) class of DNA damaging agents.





Sources: Adis R&D Insight, Clarivate Analytics Cortellis; , Cello Health BioConsulting/Defined Health analysis

These are alkylating compounds that form crosslinks with DNA and are significantly more potent than systemic chemotherapeutic drugs. As DNA minor groove binding agents, pyrrolobenzodiazepines bind and crosslink specific sites of DNA of the cancer cell. This blocks the cell's division without distorting its DNA helix, thus potentially avoiding the common phenomenon of emergent drug resistance.

It's important to note that there have been a number of setbacks with these types of payloads in the clinic from a variety of different companies. It remains to be seen how far this particular payload class is going to progress and exactly which indications or applications it will be most suitable for.

There are also examples of dimeric molecules made from duocarmycin-type compounds, which also crosslink DNA and tend to be very potent DNA damaging agents.

Some companies are currently attempting to attenuate what the PBD dimer class can accomplish. Instead of forming bisadducts (binding two sites simultaneous) with DNA, they are modifying these payloads so they only form a single alkylation event with the nucleic acid. An example is ImmunoGen's IGN class of payloads. There are also companies attempting to make monoalkylator PBD dimers or PBD analogs going forward.

Linda: Why is that a driving direction?

Pete: ImmunoGen has shown some very interesting data that suggests that by monoalkylating instead of crosslinking the DNA, there may be a better toxicity profile. They have a couple of molecules that contain the monoalkylator payload class in the clinic, both with themselves and with partners. It will be interesting to see how that payload class performs relative to traditional PBD dimer molecules.

Another example of a monoalkylator is a duocarmycin-class monomer that is present in the SYD985 HER2 conjugate that Synthon is progressing. That is now in Phase III in the clinic.

Neela: Traditionally, many of us in the field have thought that more potent is better. The PBD dimer class points out some of the shortcomings and faulty understanding of what is happening with antibody-drug conjugates. There was a time in the field when we thought about them fairly simplistically—here's the antibody, it targets an antigen and it's going to deliver a payload selectively to the tumor cells. I think what we're understanding now is that it's far more complicated with respect to internalization. Where is the payload released? How much payload is necessary? Is there a low level of that antigen being expressed on normal tissues and are some normal tissues more sensitive to that payload than others?

It turns into a multi-parameter optimization, very similar to small molecule drug discovery where you are simultaneously optimizing for potency, for ADME (absorption, distributions, metabolism, excretion) PK (pharmacokinetic) characteristics, for tox. We're finding that we need to optimize all the pieces simultaneously in order to get the therapeutic index that we want.

Linda: Historically, radioimmunoconjugates was an area that was explored and then seemed to disappear. Now there are few players.

Jason: Before small molecules, radioisotopes and protein-based toxins predated the current advent of ADCs. Today, we're seeing a little bit of resurgence around different types of radioisotopes, such as alpha emitters. There are also transcriptional inhibitors, such as toxic peptides that are being used for a payload. Bacterial toxins are also still being evaluated. Lumoxiti is a recent approval that was a PE (pseudomonas endotoxin) toxin targeting CD22.

Neela: The problem with radioimmunoconjugates is that we haven't

solved the basic CMC issue around shelf life, manufacturing location, and getting it to patients on time. A synchrotron is needed and you need to be in close proximity to a major facility that can do the conjugation. It's not trivial. It's true that there's been a small resurgence, but we don't see that this is going to become a major player in the future.

Jeff: There is some hope as the newer generation of radioconjugates are using different isotopes to address some of the manufacturing and safety issues that have bogged down other programs, but it remains to be determined. Certainly, even with an improved type of isotope and manufacturing and safety profile, there will still be perceptual baggage that there are other non-radiolabeled approaches available. I wouldn't be surprised if those would be defaulted to still.

Linda: Pete, you did do a marvelous job of laying out the scheme of things, but it makes me wonder if the stoichiometry requirements are just so onerous that we will never really get very far away from things that have to kill the cell as opposed to things that modulate the cell.

Pete: Historically, one of the reasons to go to those potent cytotoxics was the drug loading that was achievable at the time, especially in terms of maintaining decent antibody pharmacokinetic (PK) properties. That is now changing. The field is moving into delivering less potent cytotoxic compounds.

Great examples are the topoisomerase inhibitors from Immunomedics. Daiichi Sankyo also has one in the clinic. These are definitely less potent molecules. They tend to be more heavily loaded onto the antibodies and they're progressing relatively well in the clinic. The question the field is now facing and is beginning to explore is: How many more of these types of ADCs can be progressed in the clinic?

LINKERS

Linda: What are the most important trends in linkers, cleavable and non-cleavable? What are you aiming to achieve with new variations?

Pete: The purpose of a linker is to keep the payload in question attached to the antibody in an appropriate way so it's stable in circulation. Then, as the conjugate is internalized into the cell, the payload is liberated in a bioactive form. That can be accomplished via a cleavable linker, or simply a linker that remains attached to the payload once it's inside the cell. Both are possible.

With cleavable linkers, peptide-based triggers were historically used to affect the release of the payloads inside of cells. That technology works extremely well. Going forward, there's been an attempt to introduce more tumor specific release into some of the linkers by making the peptides, or peptide mimetics, more tumor selective.

This is an area that's still under development. Some companies have used glucuronide-based linkers instead of peptides to affect that release. Disulfides have been used as well. In some very creative applications, various companies have used pyrophosphates to affect this type of release.

The linker also plays a role in helping to mask the hydrophobicity of a payload that would otherwise be problematic to attach to an antibody. Yet another function of the linkers, especially moving forward, is to enable the higher loading of payloads, particularly weak or active payloads onto antibodies, which was otherwise unachievable just a few years ago.

CONJUGATION

Jeff: There's another aspect of linkers, which is where they get placed on the antibody. There are a number of companies, such as Ambrx, that have shown that where the linker is placed can affect the behavior of the antibody in either a positive or negative way.

Neela: The question around site specificity and conjugation is really an important one. There are two aspects of it. One is getting a defined drug antibody ratio. Instead of ending up with a heterogeneous drug product where some antibodies will have two linkers attached and the corresponding drug payloads, some will have four, some six, some eight linkers.

That question of heterogeneity is a question that the FDA is going to be sensitive to; they certainly prefer a homogeneous drug product. There's also the question of what happens when you actually have the higher loading. Some of the older linkers and toxins, when they got to eight loads or ten loads, often resulted in aggregation, which is something you don't want for ADCs.

One issue is getting a defined DAR (Drug Antibody Ratio) by using a specific site. The second issue has to do with the location. Different companies have

different thoughts about which locations for conjugation are best.

Seattle Genetics has a mutation that allows for site-specific conjugation. Genentech and other companies, such as Ambrx, also have their proprietary means for achieving it.

Pete: As a chemist, if I'm trying to optimize the performance of a molecular entity, I would like it to be a single molecular entity, not a mixture of four or five different things. It being as homogeneous as possible is definitely a plus.

I will acknowledge that the marketed ADCs are not necessarily completely homogeneous. If somebody had data showing that a non-site specific conjugate was performing well, I would pay attention. But if I were starting a new research program, I would want it to be as site specific as possible.

There's also the issue of how stable that attachment connection is. The field has improved quite a bit in the last five or ten years in terms of making those connections as stable as possible. The site does impact the stability and that's pretty well documented in a number of publications now. The chemistry that's used to affect the connection will also impact that stability. I don't think there's any reason that any company couldn't make a stable connection now using a variety of different approaches that are available in the literature.

CELL PROCESSING

Linda: How important is it to focus on cell processing of the target cell in order to increase efficiency?

Jason: This is probably the central, multifactorial optimization question we ask. This is where we look for opportunities to increase efficiency at the level of the antibody domain, the linker, or the payload itself. If we look at the level of the antibody when selecting an antibody, we can consider the ability of the antibody to induce crosslinking via capping (where the antibodies are clustered at 1 pole of the cell) for more efficient internalization, or perhaps there are differential epitopes on the target as well that could help improve in the internalization kinetics of the receptor for improved efficiency.

The biparatopic approaches targeting two epitopes on a target are interesting in that regard. Some have also looked at bispecific approaches where we're binding through primary and secondary co-expressed targets. In that case, there will be internalization, or lysosomal delivery of ADCs, as well. There are a lot of options to consider.

From a linker standpoint, depending on the target antibody or drug, there may be an optimal linker design. For instance, you might be aware of a known resistance mechanism that might favor a particular linkage strategy. There are reports of diminished

vascuolar-ATPase activity in tumors that may reduce the ability of Trastuzumab DM1 to be metabolized in the lysosomal compartment, but this seems to be overcome using protease cleavable linkers.

The same types of considerations apply for the drug. It has to be relevant for the mechanism of action for the tumor and the indication that's being targeted. However, at the end of day, all of this is still largely based on an empirical process of optimizing on these factors to really understand and determine how you may have increased the efficacy of the ADC.

BYSTANDER EFFECT

Linda: We've been talking about ways to increase the specificity to the cells that the drug is delivered to, yet there have been advantages of killing adjacent cells. Let's discuss the bystander effect.

Neela: In oncology, with solid tumors, the issue of heterogeneity of expression of the antigens is a very real one. In some cases, a bystander effect can be beneficial. What we're talking about is killing or targeting cells that don't necessarily have the antigen on their surface.

This kind of approach requires two things. First, it's necessary to have a cell permeable payload and a linker that is cleavable. If an antibody is bound to its antigen on a target cell and it's internalized, and there is cleavage of the linker, now there is a free drug, which is active in that cell. If that drug is also cell permeable, it can exit the target cell and then through diffusion enter nearby cells. That's one possibility.

The other possibility: Even for antigens that are not internalized, or internalized more slowly, if the linker being used is, for example, pH sensitive or even proteolytically cleaved by enzymes that are present at reasonable concentrations in the extracellular matrix, the drug can get released outside

of the target cell. Assuming that the payload is not charged, it has the opportunity to enter into nearby cells and be active there. There can be a benefit, but there is also a potential downside for normal tissues. It depends on the local concentration and what tissues express the target antigen.

Pete: Whether the bystander effect is generated or not, possibly including modification of the payload controlled through the chemical design of the linker, it's worth understanding how important the bystander effect is for the indication or application that one is going after, sooner rather than later. Ideally, that can be accomplished through *in vitro* experimentation, but sometimes that requires *in vivo* experimentation to fully get the answer.

DESIGN

Linda: Talk to us about design.

Pete: Design has become somewhat more predictable, but there is still an element of empiricism in the optimization process. Areas that have become more predictable are the areas where we can control whether or not we have a bystander effect through the modification of the linkers and/or the payloads and predicting the stability of a conjugate in terms of connection stability or biotransformation of a payload.

The technologies to assess ADC stability using *in vitro* methods have improved quite a bit. The likelihood of being surprised once a conjugate is put *in vivo* by unexpected *in vivo* instability is lower now than in the past. It's not completely zero, but there are things that we can do that will enable more prediction in that area.

Where it gets more tricky is predicting efficacy and then, ultimately, the safety of the conjugates. The field is still working to improve predictiveness and ultimately to make things as plug and play as possible.

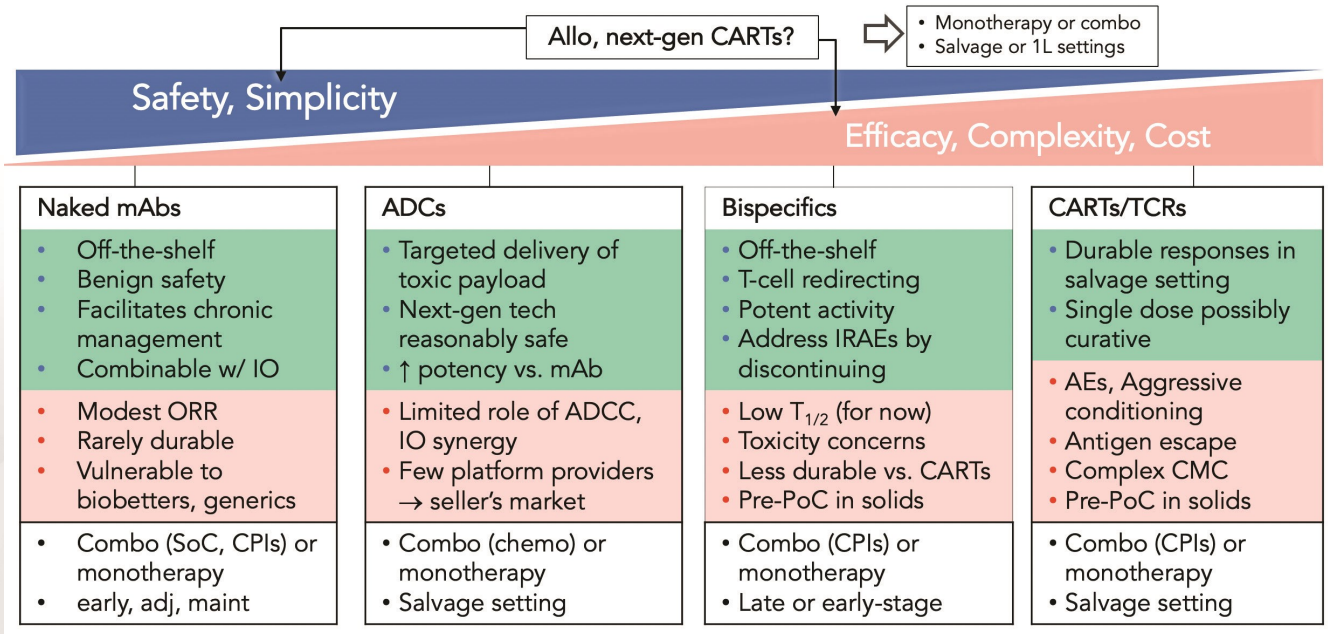
Jeff: We're in the age of engineering in biology. Presumably, it could be possible to mix and match masking and bi-specificity and ADCs. What are your thoughts on moving beyond single chain antibodies or fragments to more

complex types of biologics and targeting and dual targeting in the context of ADCs?

Neela: I'm really excited by the opportunities of the various formats. We've mentioned biparatopics, where there is a single antigen target, but two different epitopes are recognized. It's also exciting to have the potential to target two different antigens, which may help to achieve more specificity with respect to tumor cells and not hit normal tissues by requiring both targets to be expressed simultaneously. There are a variety of formats and the jury is still out with respect to what works best.

Is it a smaller format, or a single-chain or fab, or something even smaller? What provides the greatest tissue penetration, and therefore greater efficacy? Can we achieve good enough PK properties? We're always looking at that balance, because the very small formats have very short half-lives. We've seen that with Amgen's Blincyto where you end up with a delivery pump, which is not ideal for the patient. The developments are exciting; we just have to wait and see how they play out in the clinic.

Jason: From our perspective at Molecular Templates, we're also very excited about the development around engineering for the antibody portion of the molecule. Obviously, as those platforms and constructs become more validated and optimized, there are certainly approaches we would consider putting into our engineered toxin format. Because a lot of our biology is really driven by the payload, we've spent a tremendous amount of time engineering the payload itself.



We've tried to make it modular and plug-and-play, such as engineering our scaffold interactions with known innate receptors like TLR4 to reduce innate immunogenicity. We also reshape and modify the scaffold for reduced adaptive response. These are things that are fixed into our scaffold. As these new antibody binding or antibody-like binding domains become available, those are certainly technologies that we would look to integrate with our platform.

FIT IN THE MARKETPLACE

Linda: **ADCs are only one of a diverse array of biologics being deployed against cancer. How will these various platforms compete or play with each other?**

Jeff: When thinking about the value proposition for any of these agents, there's the historical evolution as viewed from left to right. That's somewhat simplistic, but certainly beginning with naked antibodies and then branching out into fragments and single chains and non-antibody scaffolds, et cetera.

ADCs have been a next step. Bispecifics are newer and, of course, the most complex in terms of engineering is cell therapies because we're dealing with live cells, especially autologous. But it's important not to just think about the targets, but also to balance the trade-offs—the time to produce, the ease of use, and the cost—all of the things that affect the settings of at least the current generation and deciding to use them versus an off-the-shelf ADC or antibody, or even a bispecific.

Then, it needs to be considered how these can be evolved and mixed and matched. When does the bispecific become an ADC? Is it a paratopic or two distinct targets, like HER2 and prolactin receptor? In any case, ADCs certainly have the potential to be competitive with other modalities.

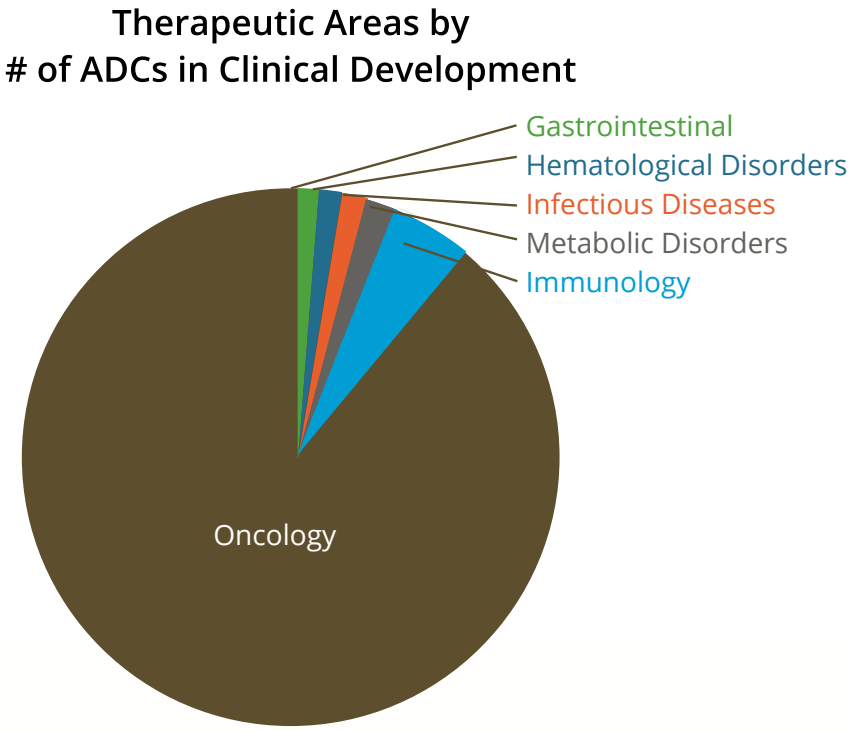
APPLICATIONS OUTSIDE OF ONCOLOGY

Linda: **What are some applications for ADCs outside of oncology?**

Pete: There are certainly initiatives for expanding the application of ADCs outside of oncology, or at least outside primarily cytotoxic therapy oncology. There are many examples of companies using antibodies to deliver immuno-modulatory agents. Examples include TLR7 agonist and STING agonists. Novartis recently initiated a Phase I trial with an unspecified immuno-targeting conjugate. The field is definitely moving into the area of trying to stimulate the immune system with antibody-drug conjugates.

There are also a few publications that would work in the other direction where immunosuppressive agents are being delivered using antibodies as well. That's a growing area of potential application for ADCs and we haven't yet seen how far it will go.

Concerning payloads, at Genentech we've actually used antibodies to deliver antibiotics to the surface of bacteria. That's a relatively unique application and just another example of how broad the applications could be for this type of technology. It remains to be seen exactly how far the technology can be applied in different settings, but companies are more open to exploring those applications today than they were five years ago.



Source: GlobalData

About Linda Pullan, PhD

Founder, Pullan Consulting

Linda and her team at Pullan Consulting offer biotech and pharmaceutical companies consulting in all aspects of partnering. Linda has a PhD in Biochemistry, a BS in Chemistry, and over twenty years of drug industry experience, including work on more than 75 deals.

About Jeff Bockman, PhD

EVP & Head of Oncology Practice,
Cello Health BioConsulting
(formerly Defined Health)

Jeff leads the Oncology and Virology Practices at Cello Health BioConsulting, formerly Defined Health. He received a BA from University of California at San Diego, a PhD in Medical Microbiology from the University of California at Berkeley, and an MA in English/Creative Writing from New York University.

About Peter Dragovich, PhD

Staff Scientist, Genentech

Peter Dragovich received a BS in chemistry from UC Berkeley and subsequently obtained a PhD in synthetic organic chemistry from Caltech under the direction of Professor Andrew Myers. He has worked in the pharmaceutical industry for more than 25 years in both large-pharma and biotech organizations and has performed a variety of research and management activities during that time. He contributed to the discovery and development of several antiviral agents that progressed to human clinical testing including the rhinovirus protease inhibitors Rupintrivir (AG-7088) and AG-7404 as well as the HCV polymerase inhibitors Filibuvir (PF-00868554) and Setrobuvir (ANA-598). He joined Genentech in 2010 and has since worked on multiple projects in both the immunology and oncology therapeutic areas. His Genentech activities include leading the company's efforts to identify novel payloads and linkers that can be utilized for the creation of new antibody-drug conjugates. His current research interests entail the targeted delivery of novel cargos via antibody-mediated technologies.

About Neela Patel, PhD

Executive Director of Business Development, Seattle Genetics

Neela Patel joined Seattle Genetics as Executive Director of Business Development in May 2016, with responsibility for identification, evaluation, and transaction of collaboration, licensing, and acquisition opportunities to diversify the pipeline. Previously, at AbbVie she identified, introduced, and led technical diligence, resulting in more than twenty-five executed deals including collaborations, licensing, and participation in consortia. Dr. Patel spent the first 16 years of her career in drug discovery management positions of increasing responsibility to advance small molecules and biologics from early stage discovery through IND filing at Poniard Pharmaceuticals, Genentech, SUGEN/Pharmacia, and Roche Bioscience.

About Jason Kim

President & COO, Molecular Templates

Jason Kim joined Molecular Templates in 2010 and serves as President and Chief Operating Officer. He has 16 years of experience in the biotechnology industry including operations, business development, and venture capital. He previously led corporate development and strategic planning initiatives at OSI Pharmaceuticals and ImClone Systems. He served as an investment professional at Domain Associates where he focused on venture and public investments in biotechnology. Mr. Kim holds an MBA from The Wharton School and a BA in Neuroscience from Wesleyan University.

About BIO

BIO is the world's largest trade association representing biotechnology companies, academic institutions, state biotechnology centers and related organizations across the United States and in more than 30 other nations. BIO members are involved in the research and development of innovative healthcare, agricultural, industrial and environmental biotechnology products. BIO also produces the BIO International Convention, the world's largest gathering of the biotechnology industry, along with industry-leading investor and partnering meetings held around the world.

www.bio.org

About LES

For more than 50 years, LES has been the leading association for intellectual property, technology, and business development professionals to achieve professional and personal success. Whether you are new to licensing or an experienced licensing executive, LES is your professional home.

LES is a welcoming business community that empowers, connects, and celebrates IP professionals through: Education, Best Practices, Networking, Participation and Mentoring.

www.lesusacanada.org

About ShareVault®

ShareVault offers secure, cloud-based document sharing solutions, also known as virtual data rooms, for organizations of all sizes looking to securely control and monitor highly confidential documents being shared with outside parties. Backed by the experience of billions of dollars in deal transactions, ShareVault's solutions are used in due diligence for:

- Licensing and Partnering
- Fundraising and M&A

ShareVault is also used in other sensitive applications such as:

- CROs and CMOs sharing and protecting their SOPs
- Document sharing with consultants, vendors, distributors, Scientific Advisory Board and Board of Director members
- Document archiving for sponsor and regulatory audits
- University tech transfer licensing processes

ShareVault's robust features together with its wide adoption and recognition in Life Sciences (44 out of the 45 largest pharma have used ShareVault) are just two of many reasons BIO and more than 50+ other regional associations have chosen ShareVault for their member savings programs. It is also why organizations in 48 countries have trusted ShareVault for their secure document sharing needs.

Pandesa Corporation,
dba ShareVault

Headquarters
16795 Lark Avenue, Suite 210
Los Gatos, CA 95032

Toll-free USA 800.380.7652 | Worldwide 1.408.717.4955
sharevault.com