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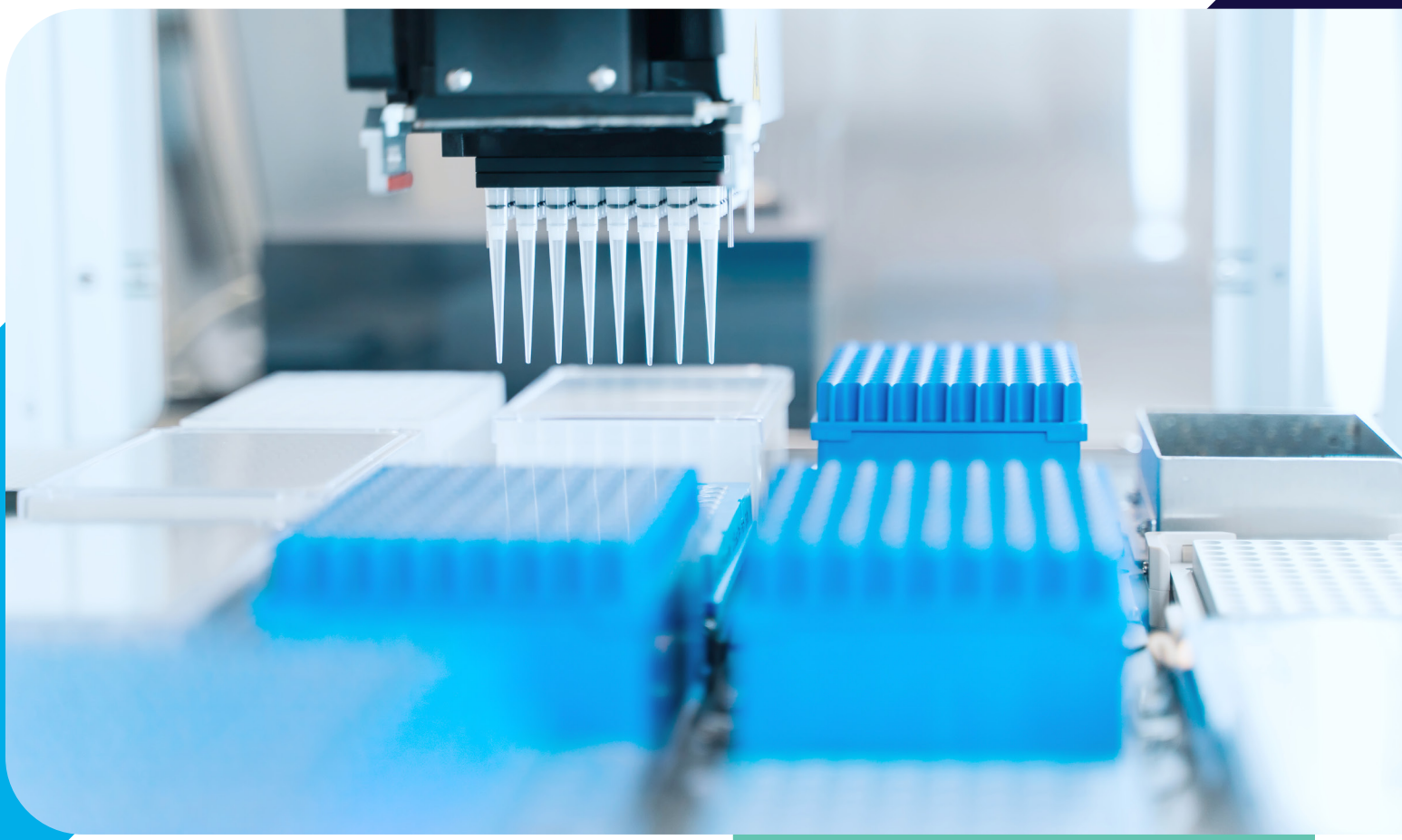
White Paper

Immunogenicity Testing: From Standardized Paradigms to Fit-for-Purpose Bioanalytical Strategies



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Introduction

Immunoassay-based immunogenicity testing has undergone substantial evolution over the past three decades, reflecting both advances in bioanalytical technologies and a maturing understanding of the clinical relevance of humoral immune responses to therapeutic proteins.[1-4] Approaches in the early 2000s relied primarily on relatively straightforward ligand binding assays which enabled quantitative or semi-quantitative measurement of Anti-Drug Antibodies (ADA) or Neutralizing Antibodies (NAb). These methods were frequently complemented by functional inhibitor assays in certain therapeutic areas (e.g., coagulation factors and cytokines), which provided some of the earliest evidence linking antibody magnitude to clinical outcomes such as reduced efficacy or altered pharmacokinetics.[5-6]

As the number and diversity of biotherapeutics expanded in the early-to-mid 2000s, concerns regarding assay sensitivity, drug interference, and inter-study variability prompted the emergence of the now widely adopted multi-tier immunogenicity testing paradigm, incorporating screening, confirmatory, and titer assays governed by statistically derived cut points.[7-11] This framework, while effective in standardizing detection and reducing false positives, emphasized conservative, highly sensitive identification of ADA in the absence of robust clinical context.

Over the past decade, accumulation of clinical data across therapeutic modalities has demonstrated that the presence of ADA alone is not uniformly predictive of clinical impact, and that immunogenicity risk is highly dependent on drug mechanism, patient population, and exposure–response relationships.[12-14] Consequently, there is a growing shift toward integrated, risk-based assessment strategies that contextualize ADA magnitude, whether expressed as titer or continuous signal (e.g., signal-to-noise ratios), alongside pharmacokinetic (PK), pharmacodynamic (PD), and clinical outcome data to more accurately define clinically meaningful immunogenicity.

This realization is driving a shift away from prescriptive, one-size-fits-all approaches toward strategies that prioritize context, clinical relevance, and fit-for-purpose assay design. The challenge, therefore, is not simply recognizing that context matters, but understanding which aspects of a program meaningfully influence immunogenicity risk and how those factors should shape testing strategy. Identifying these drivers is essential to moving from conceptual agreement to practical application.

What drives immunogenicity testing strategy?

The context of immunogenicity ADA testing extends beyond assay performance characteristics encompassing several interrelated factors which directly inform testing strategy, tier selection, and data interpretation. These include:

- **Molecular risk assessment and therapeutic attributes impact testing strategy:** Therapeutic modality, molecular size and structure, degree of molecular modification, and homology to endogenous proteins all influence the likelihood and the potential consequence of ADA formation.[15-16] For lower-risk modalities (e.g. Fully Human Monoclonal Antibodies with no Endogenous Analog), reduced sequence divergence and improved molecular design have been associated with lower immunogenicity risk and limited clinical consequences.[17] In such contexts, a streamlined approach such as reporting ADA screening tier signal to noise with omission of confirmatory, titer, and Neutralizing Antibody (NAb) testing is likely adequate to inform and address immunogenicity risk. Conversely, higher-risk programs (e.g. AAV vectors, enzyme replacement therapies, non-human or partially human proteins, fusion proteins with novel or repeated domains, cell therapies, etc.) introduce multiple immune activation pathways and often demonstrate direct clinical consequences of ADA and may warrant more comprehensive tiered evaluation.[18]
- **Drug target and mechanism of action have strong influence:** The clinical relevance of binding antibodies and neutralizing antibodies is highly dependent on mechanism of action and target engagement. In settings where pharmacokinetics, pharmacodynamic biomarkers, or downstream functional endpoints provide clear insight into the biological impact of ADA (e.g. IL-6, IL-17, TNF, cytokine-axis inhibitors), the incremental value of traditional ADA or NAb testing may be limited. In such cases, ADA screening-tier signal to noise may sufficiently inform interpretation of patient immunogenicity and functional outcomes, reducing reliance on standalone neutralization assays.[19]
- **Patient biology plays a role:** Disease-associated immune activation, immune suppression, or background inflammation can significantly influence ADA incidence, persistence, and assay behavior. These factors should be considered when determining whether traditional testing strategy adds clarity or introduces unnecessary complexity. In certain populations, ADA detection may reflect underlying disease biology rather than clinically meaningful immunogenicity, in such cases simplified testing strategies again may improve interpretability and operational efficiency.
- **Re-thinking Immunogenicity in the non-clinical space:** In the absence of formal regulatory guidance specific to non-clinical immunogenicity, industry lacks harmonization of approach.[20] Many sponsors prefer to apply three tier clinical assay paradigms in early development. In practice, non-clinical ADA data are primarily used to support interpretation of toxicology, pharmacokinetics, and exposure response relationships rather than to predict clinical immunogenicity risk.[21] The presence of ADA in non-clinical studies is in most cases anticipated, and generally manifests as altered exposure, accelerated clearance, or loss of pharmacologic activity. When such effects are not observed, ADA data often provides limited value. Accordingly, the intended use of non-clinical ADA data should guide a fit-for-purpose assay testing strategy tailored to what is necessary to inform study outcomes.

In many patient populations and therapeutic contexts, the presence of ADA does not equate to clinical relevance. When disease biology, immune competence, mechanism of action, and available PK/PD data collectively indicate low consequence, traditional ADA and NAb testing paradigms often provide limited value. In these settings, streamlined strategies can

generate data that are both scientifically sufficient and clinically meaningful. Conversely, clinically meaningful immunogenicity risk is not uniformly distributed across modalities. Platforms such as gene therapies, enzyme replacements, and non-human proteins carry inherent biological and clinical risk that potentially justify more comprehensive immunogenicity characterization. Aligning immunogenicity strategy with modality-specific risk is therefore essential to ensuring both patient safety and data utility.

When sensitivity becomes noise: Rethinking drug tolerance, cut points, and testing strategy

Across recent publications and scientific forums, several themes are emerging that challenge long-standing conventions. These include application of strategic and scientifically relevant approach to non-clinical immunogenicity testing, investigation into the value of cut points, the relationship between screening-tier signal-to-noise and titer, the value (or lack thereof) and necessity of confirmatory tiers, the utility of NAb assays when other data sources may sufficiently inform interpretation, and a trend away from pursuing ultra-high sensitivity, when clinically unnecessary, to minimize detection of low-level clinically irrelevant responses.[22]

Re-examining the historical role of cut points

Cut points have long served as a foundational element of immunogenicity assessment, providing a statistically derived threshold to distinguish baseline assay variability from putative ADA responses. Historically, their implementation was rooted in the need for objective, reproducible classification of samples in an environment where clinical experience with biologics was limited and the consequences of undetected immunogenicity were uncertain. By anchoring positivity to the distribution of responses observed in treatment-naïve populations, screening and confirmatory cut points enabled a standardized and conservative framework for identifying potential immune responses across studies and programs.

As immunogenicity testing paradigms matured, cut point methodologies became increasingly formalized, incorporating larger sample populations, tiered statistical approaches, and predefined false positive rate targets to balance sensitivity and specificity.[23] This evolution supported regulatory consistency and cross-program comparability, reinforcing the role of cut points as a central decision tool within multi-tier assay strategies. In this context, maximizing sensitivity, often accompanied by increased drug tolerance, was viewed as a necessary safeguard to ensure that clinically relevant immune responses were not overlooked.

However, the assumptions underlying this framework warrant reconsideration considering accumulated clinical and analytical experience. Cut points are inherently statistical constructs, reflecting the upper bounds of assay variability within a defined dataset rather than a biological threshold of clinical relevance. As assay sensitivity increases, signal distributions near the cut point become more densely populated, amplifying the influence of analytical variability and increasing the likelihood of transient or low-magnitude responses being classified as positive. These responses frequently lack persistence, magnitude, or correlation with pharmacokinetics, pharmacodynamics, safety, or efficacy outcomes.

Furthermore, the application of fixed or validation-derived cut points across diverse study populations and evolving assay conditions introduces additional complexity. Differences in matrix composition, disease state, concomitant medications, and immunological background can shift baseline signal distributions, leading to apparent changes in false positive rates or increased discordance between screening and confirmatory tiers. In many cases, these effects are concentrated near the assay baseline, where

classification is most sensitive to small variations in signal rather than meaningful biological differences.

Collectively, these observations highlight a central limitation: while cut points provide a structured and statistically defensible means of classification, they do not inherently confer clinical interpretability. As a result, increasing assay sensitivity without corresponding consideration of clinical context can lead to the disproportionate detection of low-level responses that add complexity without improving understanding. This has prompted a reevaluation of how cut points are defined, applied, and interpreted within modern immunogenicity testing strategies.

Screening tier Signal to Noise (S/N) as an indicator of intensity

In a properly developed and well controlled method (large dynamic range, absence of hook effect, minimal drug/target interference, precise performance of positive control S/N), screening tier S/N and titer responses typically demonstrate strong correlation.[24]

To proceed with removal of the titer tier, these factors should be accounted for within method development and established during the validation phase to provide confidence of screening S/N as a suitable substitute. Modality will influence the nature of these variables primarily through the dosing regimen, molecular half-life, presence of soluble target, presence of pre-existing antibodies, and the overall immunogenic nature of the molecule. In addition, the alternation of the therapeutic from its intended delivery state into that of an assay critical reagent may alter the structural form and stability of more sensitive molecules. Dynamic range and elimination of hook effect are largely based on platform selection and capture/detection optimization, drug and target interferences are mitigated through assay strategy, while precision relies on stability of critical reagents and the repeatable execution of the method procedure by the staff of the testing facility. Titer tiers are not without merit, they may maintain value in certain scenarios such as determining enrollment decisions for gene therapy programs or for enzyme replacement therapies with sustained high ADA response.

Reframing the role of the confirmatory tier

The confirmatory tier has long been viewed as an essential component of ADA testing yet growing evidence suggests its value is highly context-dependent. Confirmatory assays fundamentally assess inhibition by excess drug and therefore confirm drug related signals rather than immunological specificity. In the presence of soluble targets, drug–target complexes, multimeric targets, or aggregated species, non-ADA interactions may still bridge and inhibit, appearing indistinguishable from true drug specific ADA within the confirmatory format. Confirmatory assays are most useful when screening specificity is uncertain, matrix interference is present, or when distinguishing drug-specific antibodies from cross-reactive responses is clinically important. They may also remain appropriate for high-risk modalities or situations where antibody specificity directly influences patient safety or eligibility decisions. In contrast, for many modern biologics with well-behaved assays, strong correlation between screening signal magnitude and confirmatory outcomes means confirmation rarely alters interpretation.

Discordance between the screening and confirmatory results typically occurs near the cut points/assay baseline, which for most assays, falls well below the clinically relevant sensitivity of 100 ng/mL. When discordance is great, this is generally addressed through evaluation of pre-dose patient sample screening and confirmatory results, calculation of false positive rates, followed by adjustment of screening tier cut point in-study to ensure the data achieves statistical targets. In nearly all cases impact is observed within low-level responses near the cut point, of which there is rarely association to meaningful PK, safety, or efficacy effects. The value of minor shifts of false positive rates when applying an in-study cut point should be directly weighed against the improvement in clinical interpretation. In these settings, confirmatory testing often reinforces classification rather than insight. A risk, and context-driven approach, focused on whether confirmation changes clinical conclusions offers a more scientifically defensible alternative to universal three-tier testing.

Redefining the role of neutralizing antibody assays

Neutralizing antibody assays have historically been viewed as the most clinically consequential component of immunogenicity testing, but accumulated evidence now challenges their routine, universal application. Across multiple analyses, NAb positivity closely tracks with ADA magnitude and persistence, and in many cases functional impact is already captured through integrated evaluation of pharmacokinetics, pharmacodynamics, and efficacy. For low-risk biologics, particularly fully human monoclonal antibodies, stand-alone NAb assays rarely add interpretive value and often confirm what PK and PD data already reveal, making them largely a checkbox exercise. In contrast, NAb testing remains important for high-risk modalities, such as enzyme replacement therapies, gene therapies, and products with non-redundant endogenous counterparts, where neutralization can meaningfully impact safety or efficacy. Potentially even in moderate-risk programs, surrogate assessments of neutralizing activity through PK or PD endpoints may be more informative than highly sensitive NAb assays that detect clinically irrelevant responses.[25] The emerging consensus is that the question is not whether NAb activity should be understood, but whether a dedicated NAb assay is the most appropriate tool to do so. A context and mechanism-driven strategy that deploys NAb methodologies only when they meaningfully influence clinical interpretation represents a more rigorous and patient-focused approach than default inclusion.

The hidden cost of “check-the-box” Immunogenicity

As the understanding of immunogenicity’s role in drug development has matured, and as therapeutic modalities and mechanisms of action have become increasingly complex, it has become evident that a single, standardized testing paradigm cannot adequately address the needs of all programs. Nevertheless, for more than a decade, immunogenicity assessment has largely been guided by the implicit assumption that a uniform, multi-tier testing framework should be applied universally. While this approach provided consistency and regulatory confidence during earlier stages of biologic development, its continued application across diverse modalities has revealed important limitations.

Traditional assay strategies often require substantial laboratory investment, including development of critical reagents, multi-tier assay validation, and extensive sample analysis. However, in many programs, particularly those involving lower-risk biologics, these efforts yield limited incremental insight when evaluated alongside pharmacokinetic (PK), pharmacodynamic (PD), and clinical safety data. Increased assay sensitivity and drug tolerance, while analytically advantageous, frequently lead to the detection of low-magnitude responses that lack persistence, functional consequence, or correlation with clinical outcomes. As a result, the emphasis on maximizing detection can, in some cases, add complexity without improving interpretability.

This pattern is particularly evident in non-clinical development. In the absence of regulatory requirements specific to non-clinical immunogenicity, clinical-grade validation standards have often been applied by default. However, there is growing emphasis across the pharmaceutical industry and regulatory landscape on reducing reliance on animal studies and eliminating nonvalue added testing through the adoption of modern, science driven approaches. Consistent with this trend, non-clinical immunogenicity testing should be conducted only when there is a clear scientific rationale that ADA responses could meaningfully influence the interpretation of safety, pharmacology, exposure, or dose selection. When existing evidence demonstrates minimal impact on these endpoints, extensive ADA characterization provides limited incremental value relative to the resources required.

A similar principle applies to Neutralizing Antibody (NAb) assessments. Development and validation of complex cell based NAb assays often require substantial investment in specialized reagents, assay optimization, and long-term maintenance. Therefore, NAb testing should be guided by a risk-based evaluation of its potential to affect study interpretation, rather than being included as a routine procedural expectation when the resulting data are unlikely to influence development decisions.

Confirmatory testing presents an additional example of diminishing returns under certain conditions. In well-characterized assays with low variability and strong correlation between screening signal magnitude and inhibition, confirmatory testing frequently reinforces screening classifications rather than providing new insight. Furthermore, confirmatory workflows can introduce additional sample handling, including repeated freeze-thaw cycles and volume depletion, which may constrain the ability to perform subsequent analyses, particularly in studies with limited sample availability.

Collectively, these practices illustrate a broader challenge: when testing strategies are driven by convention rather than context, the incremental cost (in terms of time, resources, and sample utilization) may outweigh the improvement in scientific understanding. While each individual requirement may appear justified in isolation, their cumulative impact can extend development timelines and divert resources from areas of greater clinical relevance. At scale, these inefficiencies have the potential to influence portfolio prioritization and the pace at which new therapies advance through development.

Encouragingly, recent scientific discourse reflects a growing recognition that immunogenicity risk is not uniformly distributed across programs and that testing strategies should be aligned with modality-specific risk and intended data use. This shift toward fit-for-purpose design represents a critical inflection point, but it also introduces new challenges. Moving away from prescriptive frameworks reduces procedural certainty and places greater emphasis on scientific judgment, documentation, and the ability to provide a defensible rationale for decision-making.

In this evolving landscape, responsibility for appropriate strategy selection is inherently shared. Sponsors must clearly define development priorities and acceptable levels of risk, while Contract Research Organizations (CROs) must apply technical expertise to ensure that testing approaches are scientifically justified and proportionate to program needs. Progress therefore depends on early alignment, transparent decision-making, and a collective commitment to balancing analytical rigor with clinical relevance.

Fit-for-purpose doesn't mean risky: Staying regulatory-ready while evolving

As immunogenicity strategies evolve, clinical research organizations are increasingly accommodating sponsor requests for testing and data reporting approaches informed by contemporary scientific evidence. While this evolution is appropriate, deviation from the standard paradigm should never go undocumented. Modification requires deliberate implementation, rigorous oversight, and proactive discussion with sponsors and health authorities around the intended immunogenicity testing strategy to ensure success. Despite strong supporting data, testing laboratories remain accountable for maintaining robust methodologies and defensible results which assist clinical interpretation. While broader regulatory acceptance of these paradigm shifts will likely evolve over time, today these approaches must be carefully articulated, scientifically justified, and proactively aligned with health authorities to prevent downstream delays. The responsibility therefore falls on the scientific community to collectively share experience and evidence, ensuring that innovation and regulatory confidence advance together. The advancement of immunogenicity testing must balance innovation with diligence to ensure that data generated are clinically relevant, regulatory-ready, and aligned with the overarching goals of patient safety and therapeutic development.[28]

Translating strategy into practice

If you're evaluating how to align your immunogenicity strategy with your molecule, mechanism, and patient population, our expert scientific team would welcome the opportunity to exchange perspectives and explore the most appropriate path forward for your program.

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