



KOL Webinar – BK Virus Nephropathy and the Promise of MAU868

March 29, 2022

Forward Looking Statements

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Agenda for Today's Meeting

Corporate Overview	Marshall Fordyce, MD	<i>Chief Executive Officer Vera Therapeutics</i>
Patient Caregiver Story	Claire Flores	<i>Wife of late Art Flores and Organ Donor</i>
Clinical Impact of BK Virus Reactivation	Stan Jordan, MD	<i>Director of Nephrology & Transplant Immunology and Medical Director of Kidney Transplant Program Cedars-Sinai Medical Center</i>
MAU868 Development	Celia Lin, MD	<i>Chief Medical Officer (CMO) Vera Therapeutics</i>
Q&A	Marshall Fordyce, MD	<i>Moderator</i>

Corporate Highlights



Vera's vision is to **improve standard of care for patients with immunologic diseases**. Our first three clinical programs focus on kidney disease.



Phase 2b “ORIGIN” study: atacicept for patients with IgA nephropathy – **primary endpoint readout expected Q4 2022**



Phase 3 study: atacicept for patients with lupus nephritis – **trial expected to initiate 2H 2022**



Phase 2 study: MAU868 for patients with post-renal transplant BK virus reactivation – plan to share interim results mid-2022



Strong financial profile sufficient to fund current operations to Q2 2024

Strategic Vision: Develop Transformative Therapeutics for Immunologic Diseases

- Lead indications with **large markets and validating clinical data**
- Vera has **worldwide, exclusive licenses to develop and commercialize** atacicept from Merck KGaA and MAU868 from Pfizer/Novartis
- Experienced corporate development team with a **strategic focus to develop and commercialize novel therapies for immunologic diseases**

New Asset MAU868

BK Virus Nephropathy

- Late-stage clinical asset for BK viral disease
- No currently FDA approved therapies
- Phase 2 **interim results expected mid-2022**

Lead Clinical-Stage Asset, Atacicept

IgA Nephropathy (IgAN)

- Conducting Phase 2b trial, **primary endpoint data expected Q4 2022**
- Tarpeyo approval sets precedent for UPCR endpoint

Lupus Nephritis (LN)

- Phase 2 results in SLE show **clinical efficacy in severe patients**
- Plan to initiate Phase 3 trial in 2H 2022

Additional Autoimmune Indications

- Dual inhibition of BLYS and APRIL **reduces disease-associated antibodies** with dose-dependence
- Potential for **best-in-class for B-cell targeting biologics**

Vera Is Well-Positioned to Develop and Commercialize MAU868

BK Virus Nephropathy

- **Leading cause of kidney transplant failure**, leading to high morbidity and healthcare cost
- **No available BK virus-specific treatments**
- MAU868: **late-stage clinical asset** with encouraging data
- We believe that MAU868 is poised to **change the standard of care** for transplant patients



Committed to the Development of MAU868

- ✓ Team with proven track record in **antiviral development and commercialization**
- ✓ **Development leadership maintained** – Ciara Kennedy (former CEO of Amplyx), brought MAU868 through Phase 2, now serves as Senior Advisor of Vera
- ✓ **Clinical and commercial synergy** with Vera's nephrology focus

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Stanley Jordan, MD, FASN, FAST

- Director of Nephrology & Transplant Immunology and Medical Director of the Kidney Transplant Program at Cedars-Sinai Medical Center
- **>30 years improving transplant care**, including first desensitization protocol for HLA incompatibility, and establishing the Transplant Immunotherapy Program at Cedars-Sinai in 2004
- **Published >400 peer reviewed manuscripts** and served on the editorial boards of numerous professional journals
- **Numerous awards**, including the Medawar Prize, The Transplantation Society's highest distinction for lifetime contributions in the transplantation field, the National Kidney Foundation "Gift of Life Award", and the inaugural Cedars-Sinai Prize for Research in Scientific Medicine (PRISM)



The Clinical Impact of BK Virus Reactivation

Stanley C. Jordan, MD, FASN, FAST

Director of Nephrology & Transplant
Immunology

Cedars-Sinai Medical Center

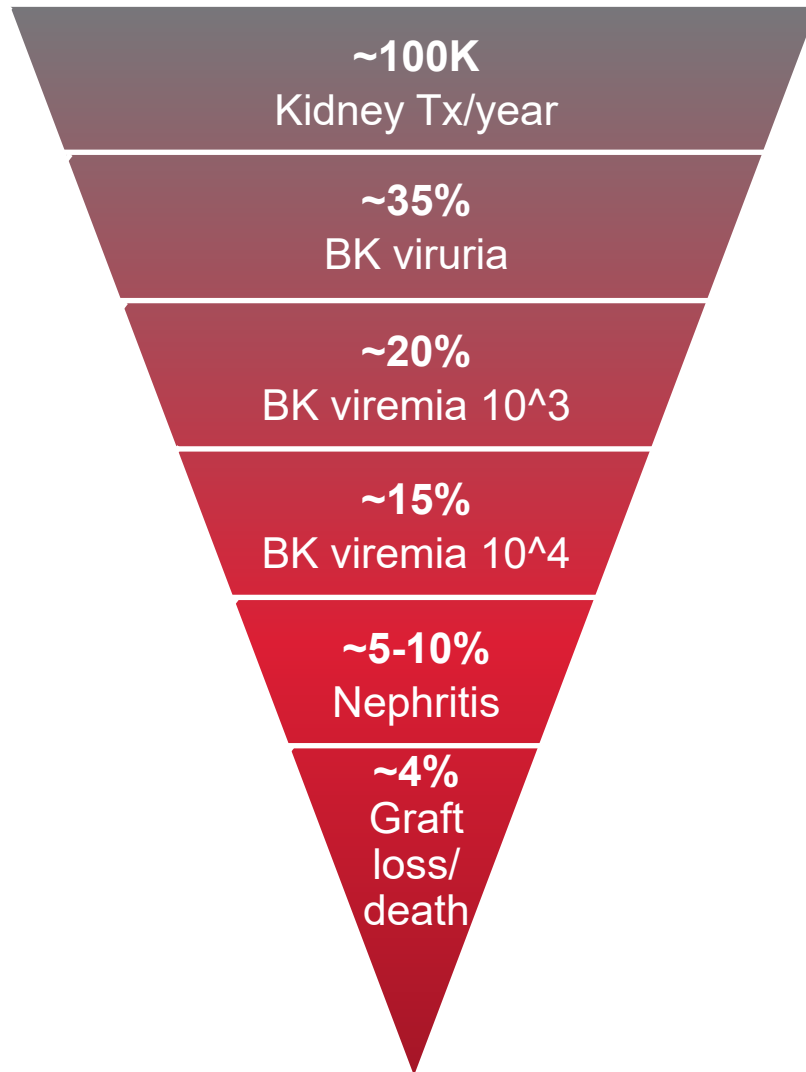
Comprehensive Transplant Center

BK Virus is an Opportunistic Infection Associated with Significant Morbidity and Mortality in Transplant Patients

- BK Virus is one of 13 known polyomaviruses (DNA viruses)
 - 80-90% of adults infected by BK virus (BKV) worldwide
 - Persistent infection established in renal and urinary tract epithelium
- Reactivation of persistent infection in immunosuppressed hosts leads to disease in two key patient populations
 - Nephropathy and allograft loss in kidney transplant recipients
 - Hemorrhagic cystitis in hematopoietic stem cell transplant (HSCT) recipients
- Main risk factor is the overall degree of immunosuppression
- Currently no approved or effective anti-BKV therapies, mainstay of management is reduction of immunosuppression which increases risk of allograft rejection



BK Virus is a Common and Important Threat to Renal Transplant Patients



- 100,000 Kidney transplants each year, with nearly 100,000 patients in the US alone on the waiting list
- BK reactivation will occur in up to 40% of patients post-transplant, virus is first detectable in the urine, with viremia developing several weeks later.
- There is a general consensus to intervene when a BK viremia of 10^3 copies/ml is detected
- BK viremia may indicate irreversible damage to allograft despite no apparent change in graft function
- Kidney function deteriorates rapidly with persistent BK viremia $\geq 10^4$ and can be associated with rapid graft loss
- Without effective treatments, the current standard of care is reduction of immunosuppression, but this can lead to acute rejection in up to 12% of patients
- 1-10% of patients will develop BKV nephropathy resulting in irreversible damage and significant risk of rejection

BKV Nephropathy is a Leading Cause of Allograft Loss In Kidney Transplant Recipients

BKV Nephropathy after Kidney Transplant: Risk Factors

Immunosuppression:

- **The intensity of immunosuppression is the main risk factor for BKV reactivation and disease**
- Replication rates are higher in the early posttransplant period and following treatment for allograft rejection when immunosuppression intensity is highest
- No specific immunosuppressive drug or regimen has been definitively associated with clinically significant BKV infection
- BKV replication and BKVAN have occurred in patients receiving nearly all immunosuppressive drugs and their combinations

Other risk factors include:

Donor risk factors:

BK virus seropositive donor
Degree of HLA mismatching

Recipient risk factors:

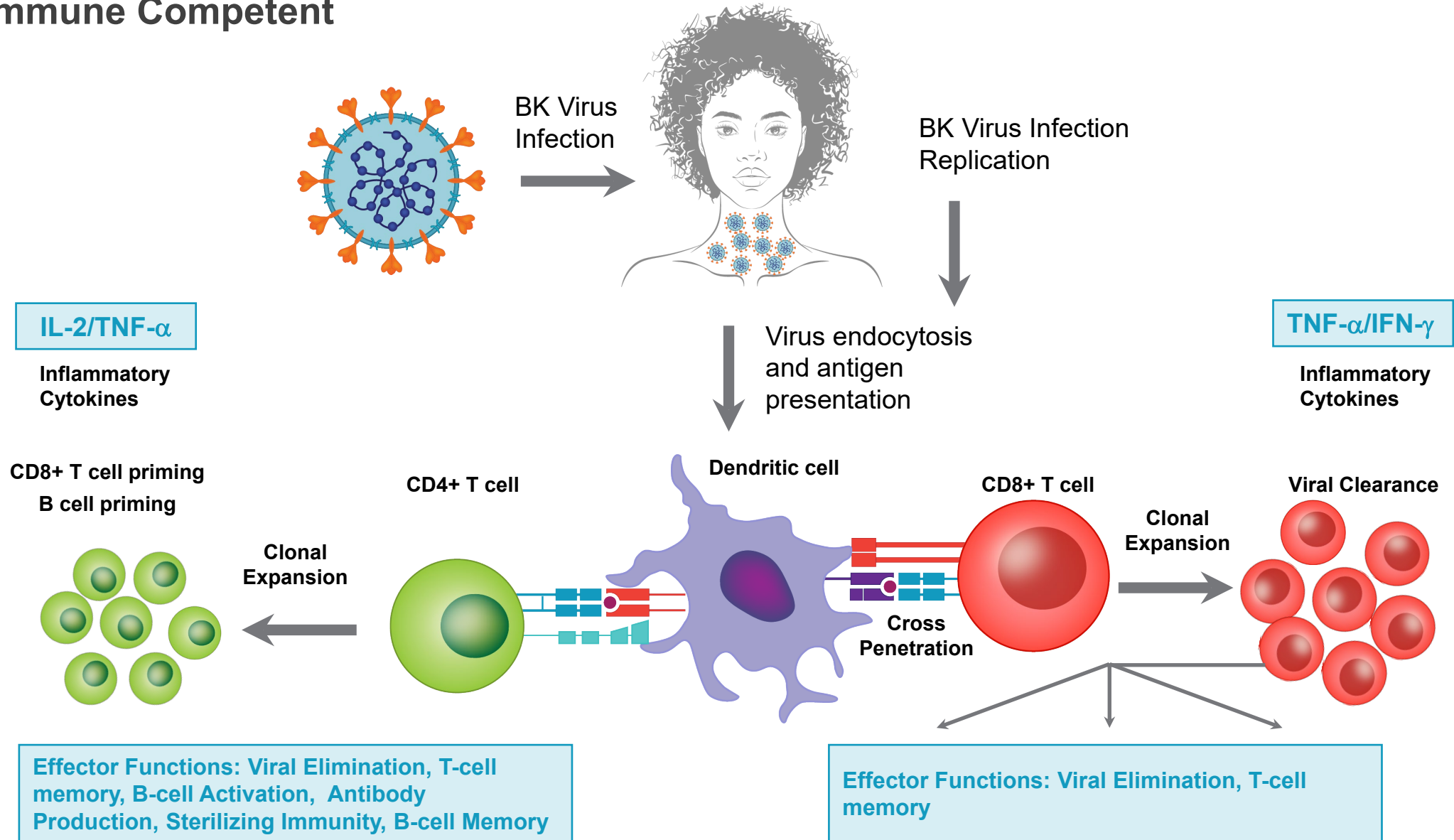
Older recipient age
Male recipient
Recipient race
(non-African American)
Diabetes

Transplant risk factors:

Acute rejection episodes
Cold ischemia time
Delayed graft function
Ureteral stent placement

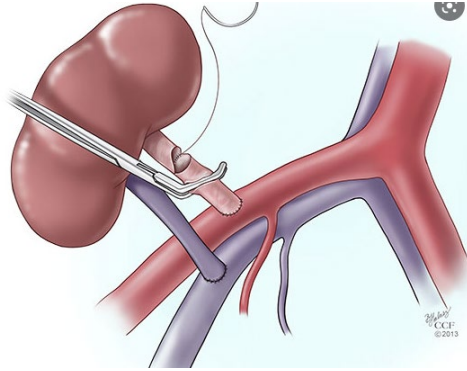
T-cell Activation After BK Virus Infection

Immune Competent



BKV Infection in Immunocompromised Kidney Transplant Patient

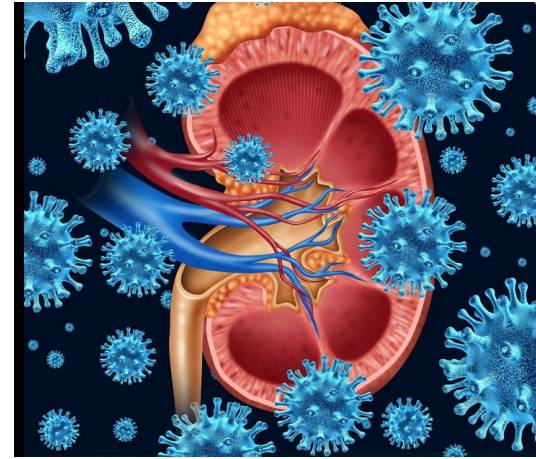
Dormant BK Virus in 80-90%



Immunosuppression



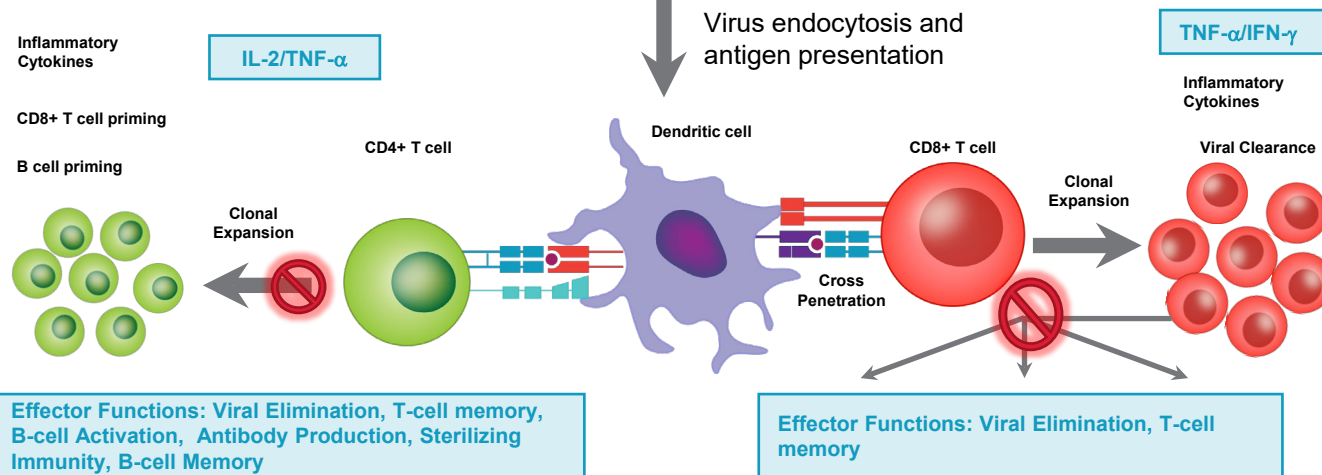
BK Virus Replication



Persistent BK Virus Replication Results in: BKV->BKAN->Graft Loss

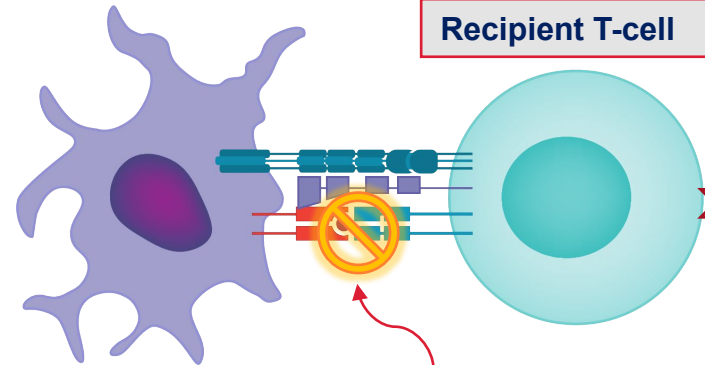
Kidney Transplantation

Immunosuppression



Donor Dendritic Cell

Recipient T-cell



Poor T-cell Immunity

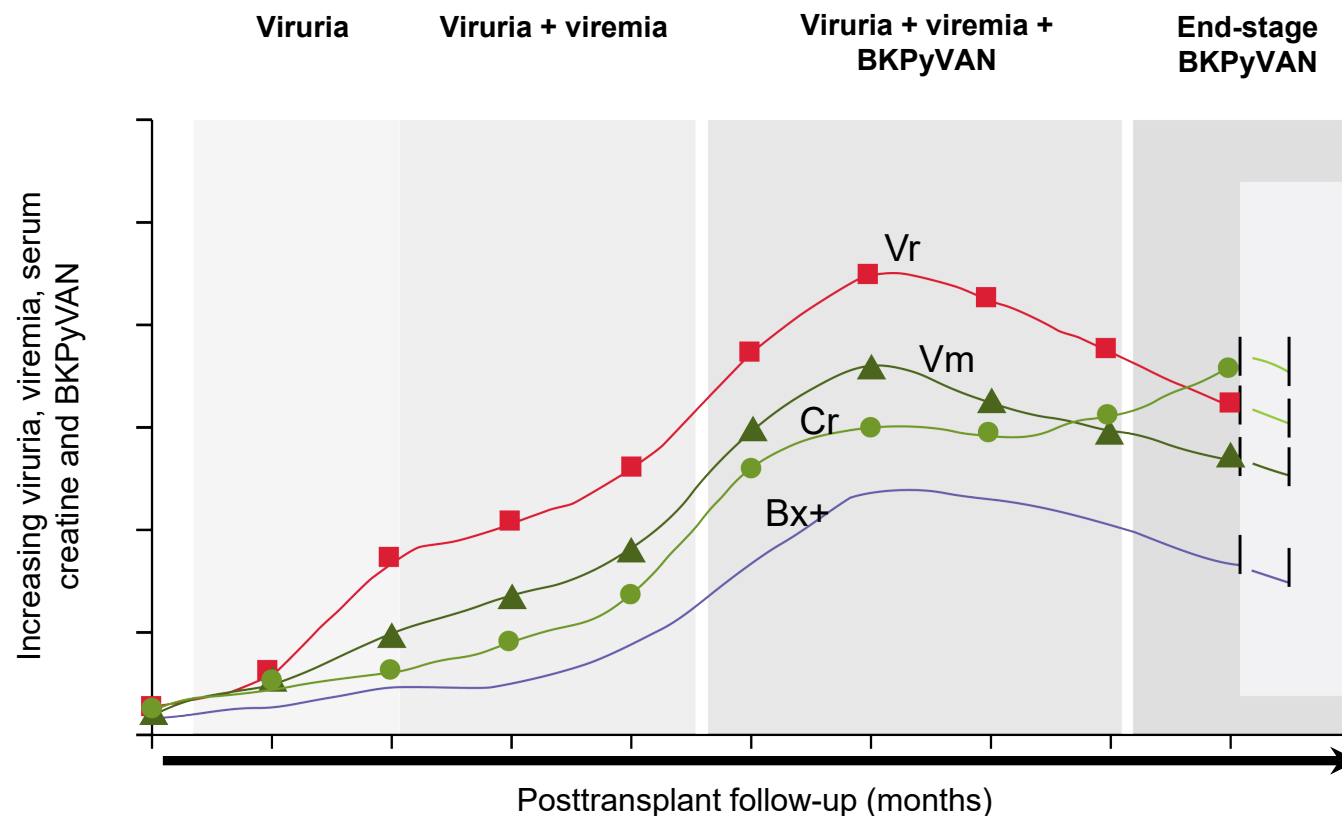
BKV Peptides Not seen when Presented by **Non-Self Dendritic Cells** Interacting with Self T-cells. Antigen Presentation is restricted to Self MHC

Screening and Diagnosis of BKV Infection

- All patients are screened for BKV reactivation after kidney transplantation.
- Screen for viremia with quantitative PCR of serum or plasma samples, by the time BKVAN is diagnosed the serum creatinine is significantly raised and subsequent graft survival is worse.
- The frequency of monitoring can vary between transplant centers but is typically:
 - Monthly for the first 3-6 months after transplantation
 - Every 3 months until the end of the first post-transplant year
 - Patients also undergo PRC-based screening for BKV every time an unexplained rise in serum creatinine occurs and after treatment for acute rejection
- Some transplant centers also screen urine for polyomavirus-bearing 'decoy cells' or urine DNA-PCR.
- The definitive diagnosis of BKVN requires a renal biopsy showing polyomavirus-induced cytopathic changes in tubular or glomerular epithelial cells.




Time to BKV Reactivation and BKVAN after Transplant

- BKV reactivation occurs within weeks to months of transplantation
- Can occur later in patients with prolonged immunosuppression
- Clearance of viremia after modifying immunosuppression can take months to years
- Viremia predicts progression of nephropathy
 - Clinicians accept detection of viremia as presumptive evidence
- Viruria represents a higher risk group for viremia but has lower predictive value of nephropathy



Diagnosis of BKVAN

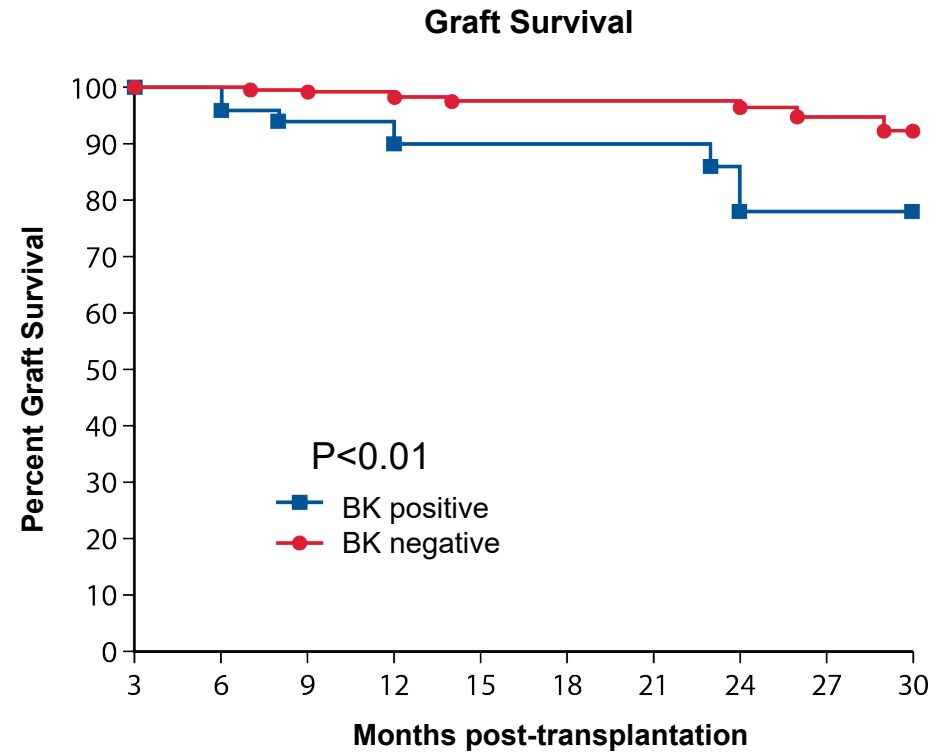
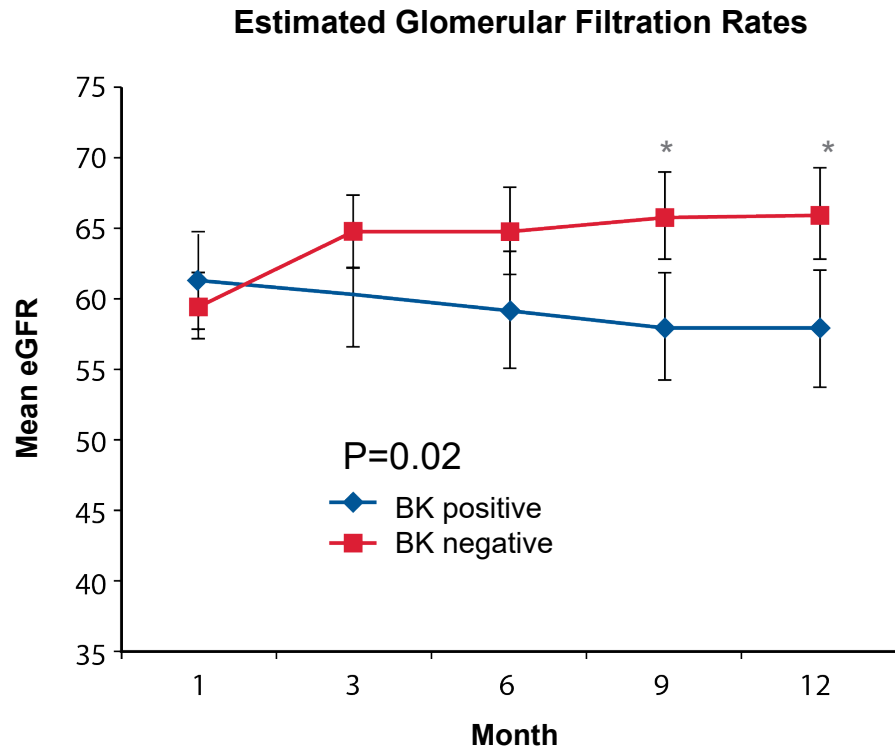
Renal biopsy is not always feasible or reliable, therefore, consensus groups have definitions for probable or presumptive BKVAN based on level of viremia

Consensus Group	Proven BKVAN	Probable/Presumptive BKVAN
	Biopsy confirmed cytopathic changes of tubular epithelia cells in the allograft tissue	Probable: viremia >1,000 copies/mL in two measurements within 3 weeks Presumptive: viremia >10,000 copies/mL in at least one of two measurements
	Biopsy confirmed viral cytopathic changes and antibodies against SV40 in kidney	Possible: high level viruria, marked by high level of DNAuria, high VP1 mRNA load, Haufen, or decoy cells.
	NA	Probable: Plasma BKV load \geq 10,000 copies/mL in at least one measurement

Viremia Associated with Reduction in Renal Function and Allograft Survival

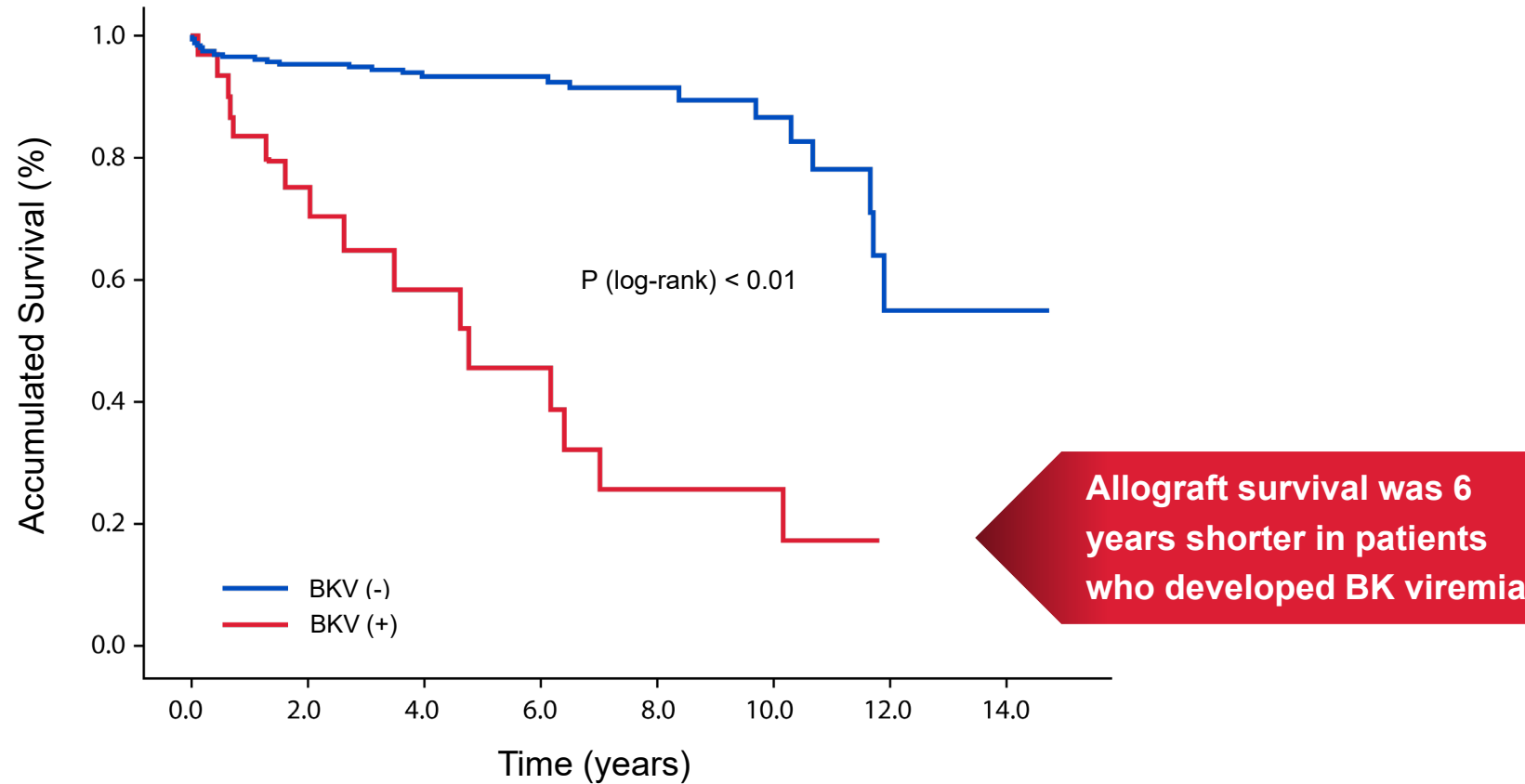
Retrospective analysis of 349 kidney transplant recipients, 57 (16%) of whom developed viremia

- Median time to BK viremia was 3 months (1-17)
- BKV-related allograft loss in 7% of patients



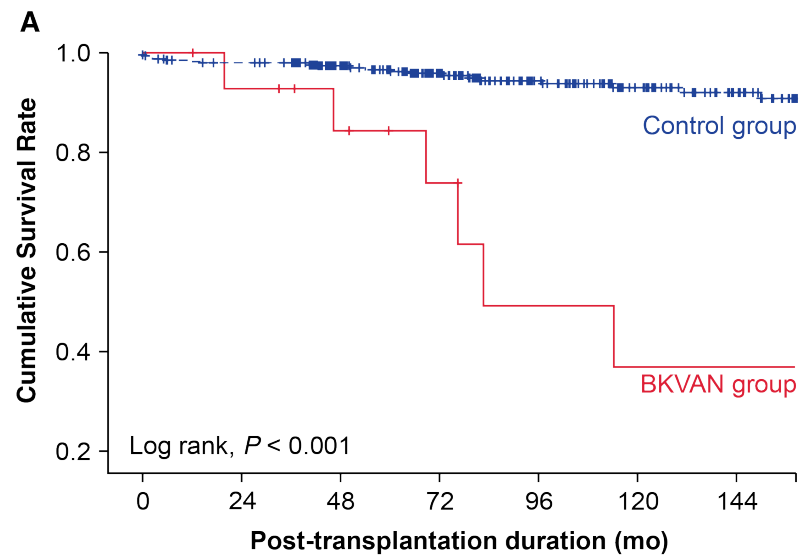
BK Viremia has profound effect on graft survival

Patients with BK viremia had significantly shorter allograft survival

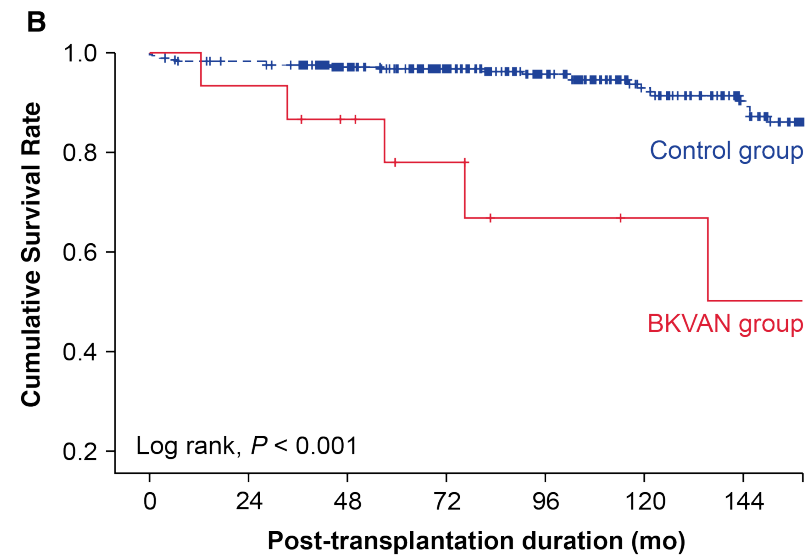


Poor long-term prognosis of patients with BKVAN

- Single center retrospective analysis of kidney transplants between 2001-2014
- BKVAN was associated with a significantly worse graft survival and patient survival rate



Control	356	349	347	343	340	338	337
BKVAN	15	14	13	12	10	9	9

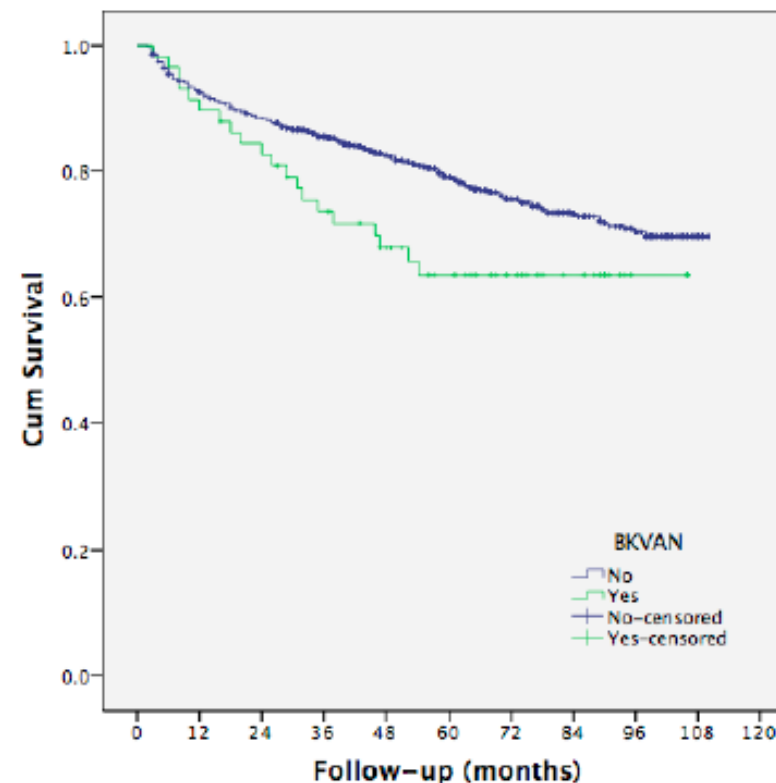


Control	356	352	348	347	345	341	338
BKVAN	15	14	13	12	11	11	10

Comparison of (A) death-censored graft survival rate, and (B) patient survival rate between kidney transplant recipients with BK virus associated nephropathy and those without BK virus-associated nephropathy (BKVAN).

Increased allograft loss in patients with BKV with immunosuppression reduction

- Single center retrospective evaluation of kidney transplants with biopsy-proven BKVAN compared with no-BKVAN over 2 years
 - 1404 kidney transplants analyzed during study period
 - 58 (4.1%) diagnosed with BKVAN
- Graft survival at year 5 was significantly lower in patients with BKV despite immunosuppression reduction



Patients at risk									
No-BKVAN	1346	1235	1166	979	758	562	378	251	128
BKVAN	58	52	47	40	33	27	19	12	3
KM (%)									
No-BKVAN	100	92.9	88.6	85.5	82.7	79.0	75.5	73.1	70.3
BKVAN	100	89.7	82.7	73.6	67.8	63.4	63.4	63.4	63.4

Figure 1B: Graft survival (uncensored by death) according to BK virus-associated neuropathy (BKVAN) after kidney transplantation

ARCIA VD, Raupp FV, MEINERZ G, Piegas K, KEITEL E. Epidemiology and Outcomes of BKV Nephropathy Management by Screening Policy in a Real-Life Setting [abstract]. *Am J Transplant.* 2019; 19 (suppl 3).

BKV Management Strategies

- In the absence of effective therapies, current management for BKV involves careful reduction of immunosuppression
- Critical balancing act: lower immunosuppression to control the BKV, with increased risk of allograft rejection
 - Acute rejection can occur in up to 12 percent of patients with BK viremia following a reduction in immunosuppression
- Antiviral agents have been tried with little to no efficacy including:
 - cidofovir, leflunomide, quinolone antibiotics and IVIG
- Although early diagnosis and prompt therapeutic intervention have reduced rates of overt graft loss to approximately 15%, surviving grafts frequently show progressive decline in graft function
- Acute rejection after immunosuppression reduction for BKVN showed worse allograft survival¹

Large unmet need for effective, anti-BKV therapies

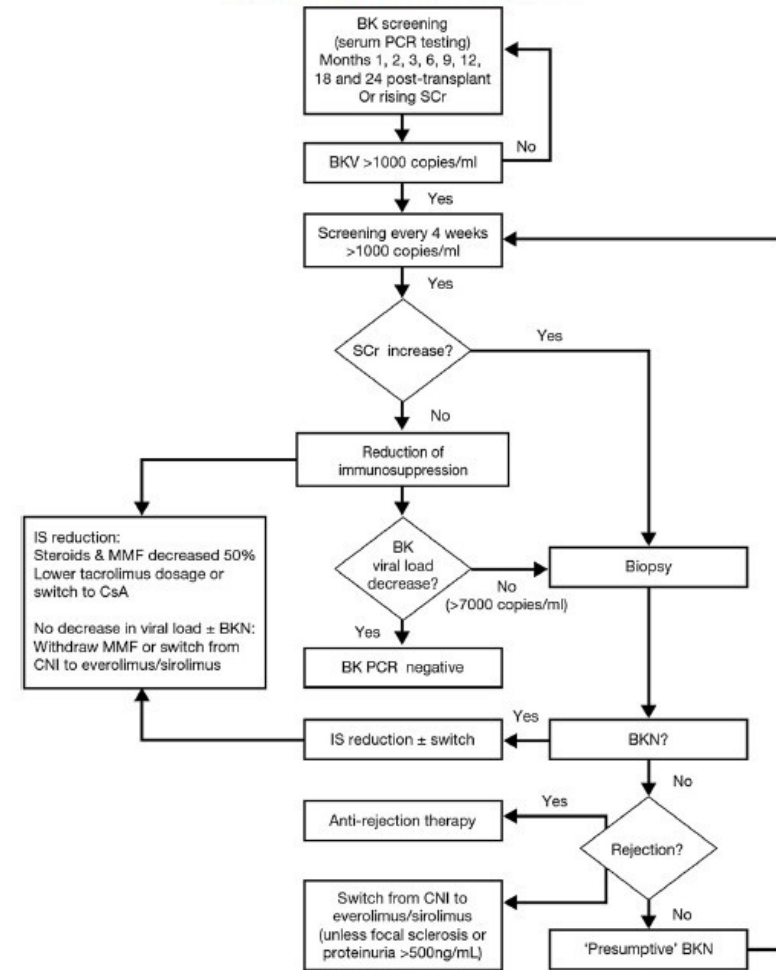


Fig. 1. Proposed BK screening and management protocol. BK screening is performed by PCR analysis of serum. SCr indicates serum creatinine; IS, immunosuppression.

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MAU868, A Novel Monoclonal Antibody Against BK Virus

Celia Lin, MD

BK Virus Infection: Unserved market in Renal Transplant and HSCT



BK Virus (BKV) leads to significant morbidity and mortality in transplant patients



80-90% of healthy adults have been infected with BKV and the virus remains latent in healthy adults



BKV can be reactivated when a patient is immunocompromised



BKV impacts two immunocompromised populations including **kidney transplant patients** and **hematopoietic stem cell transplant (HSCT) recipients**



No approved anti-BKV treatments in the United States

Unserved Market ~\$1B+ Commercial Opportunity WW in 2036¹

Kidney Transplants: ~80,000 RTx per year WW

Viruria (30-50%)	40,000 pts – measurable BKV
Viremia (10-20%)	15,000 pts – kidney at risk
Nephropathy (3-4%)	3,200 pts – irreversible damage
Rejection (1-2%)	1,500 pts – kidney loss

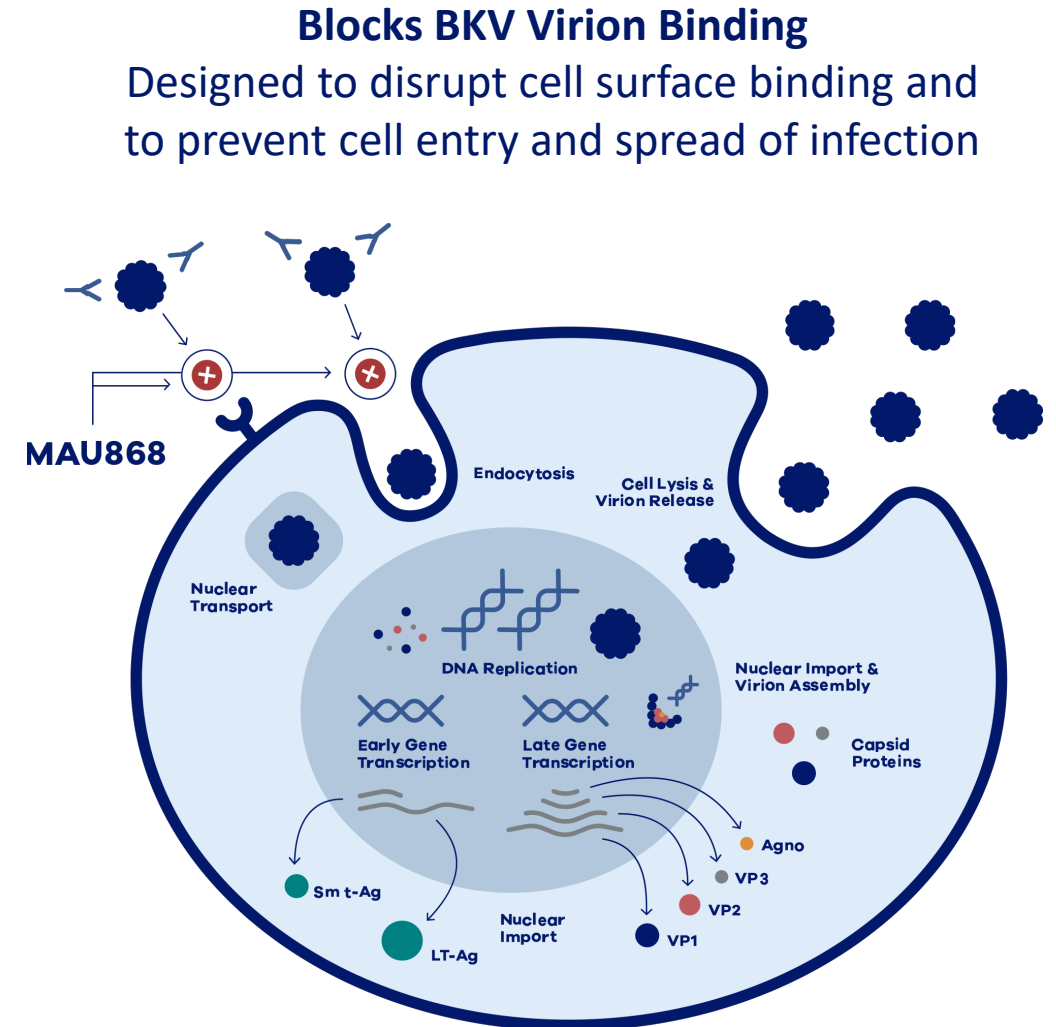
HSCT Procedures: ~100,000 HSCT per year WW

Allogeneic (50%)	50,000 pts – higher risk of BKV
Viremia (10-35%)	22,500 pts – risk of cystitis
Cystitis (6-16%)	10,500 pts – hemorrhagic cystitis

- BKV Nephropathy is the **leading cause of allograft loss**
- BKV in HSCT patients have **increased risk of severe hemorrhagic cystitis**

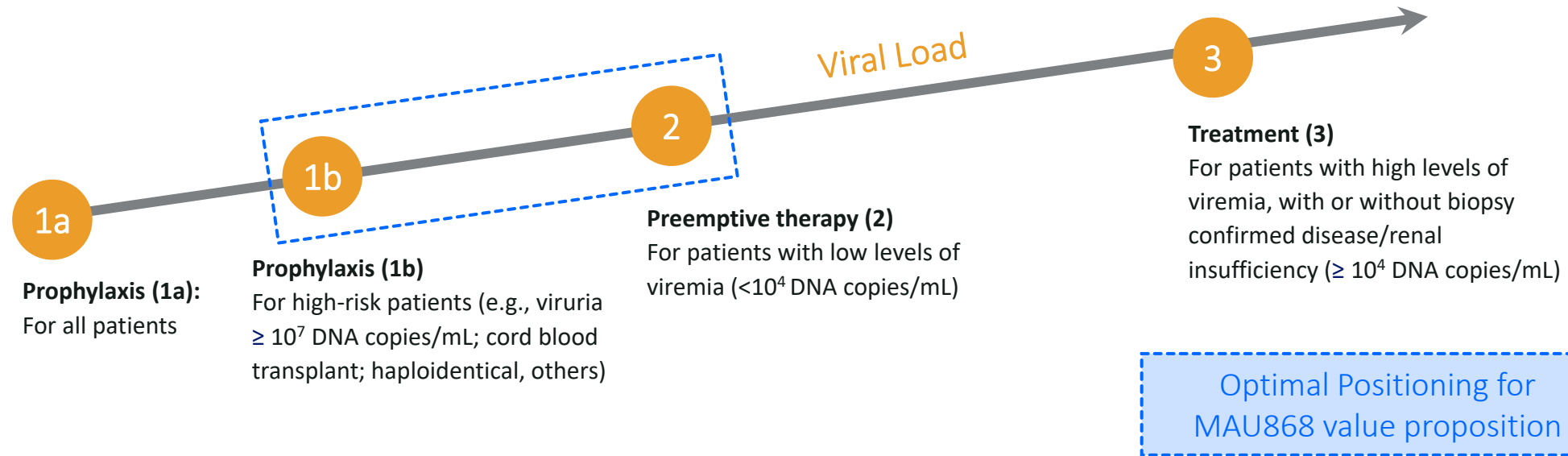
MAU868: First Known Neutralizing Antibody Targeting BK Virus

- **Novel Target:** mAB that neutralizes infection by blocking BKV virion binding to host cells
- **Active Against All Genotypes:** Sub-nanomolar potency against all major genotypes
- **Proven Mechanism:** Neutralization of virus infection effective in other approved mAB therapies
- **More Potent than IVIG:** ~10,000 fold more potent *in vitro*



BKV Treatment Paradigms

Goal of therapy: prevent clinically significant viremia/disease



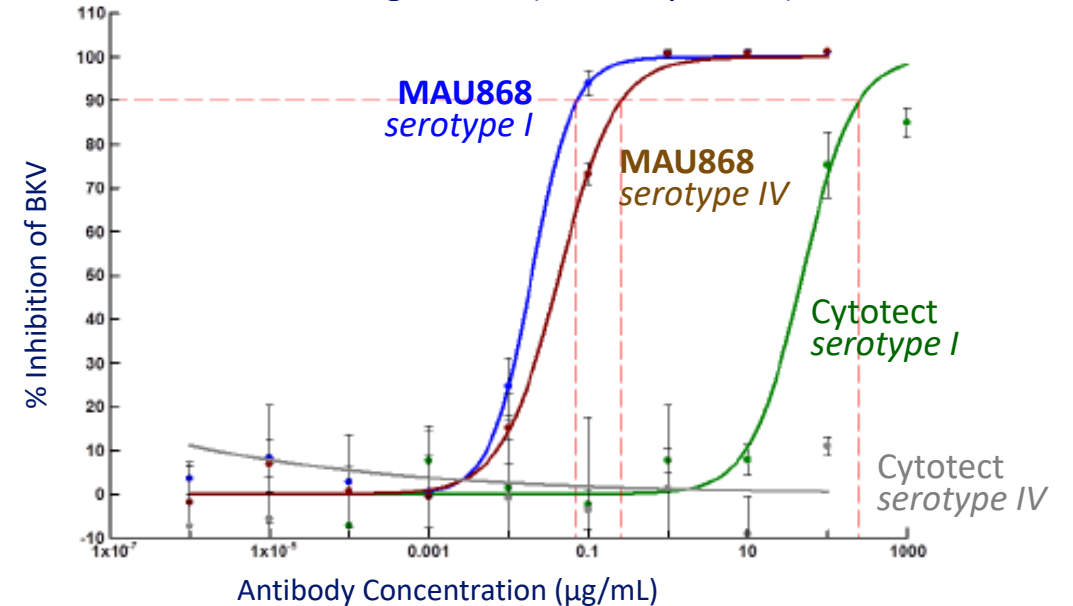
Intervention	Definition
Prophylaxis (a)	Administration of MAU868 to all patients <u>before</u> any evidence of BKV replication in plasma
Prophylaxis (b)	Administration of MAU868 to high-risk patients <u>before</u> any evidence of BKV replication in plasma
Preemptive	Administration of MAU868 at an <u>early stage</u> of BKV infection or replication (VL $<10^4$) in plasma
Treatment	Administration of MAU868 given at/after disease diagnosis (i.e. at VL $\geq 10^4$ in plasma)

DNA copies/mL $\geq 10^4$ has been correlated in two studies with BKV nephropathy (Hirsch et al; Limaye et al)
Clinically significant viremia is BKV mL $\geq 10^4$ DNA copies/mL

MAU868: Potently Neutralizes all BKV Serotypes

- MAU868 is a human IgG1- λ monoclonal antibody
- MAU868 potently binds to and neutralizes BKV
 - MAU868 neutralizes BKV infection of renal proximal tubular epithelial cells and prevents cell-to-cell spread
 - No resistance-associated serotype variants
- MAU868 is ~10,000-fold more potent than hyperimmune immunoglobulin (IVIg - Cytotect)
 - IVIg preparations like Cytotect are used to treat some patients with reactivated BKV
 - Clinical efficacy of IVIg preparations has not been consistently demonstrated
 - Observed IVIg activity has been limited to BKV serotype I

MAU868 Activity vs. Pooled Intravenous Immunoglobulin (IVIg - Cytotect)



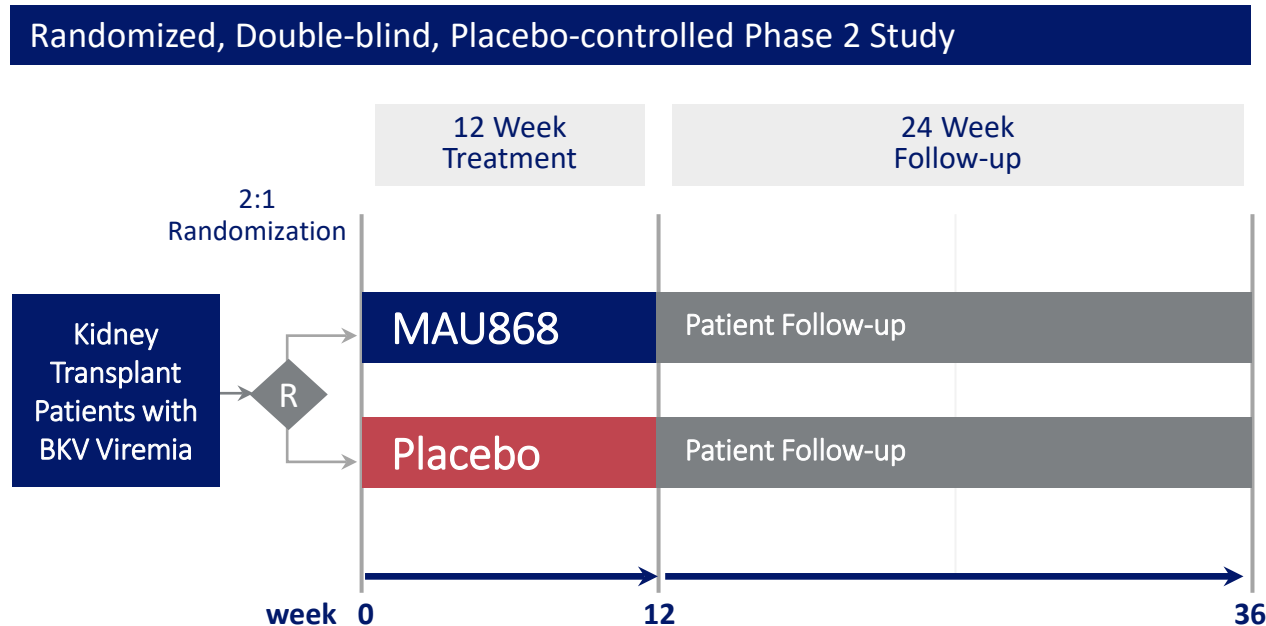
MAU868	K _D (pM)	EC ₅₀ (nM)	EC ₅₀ (µM/mL)
BKV Serotype I	5.8 ± 1.8	0.062 ± 0.068	0.009 ± 0.010
BKV Serotype II	2.8 ± 0.6	0.278 ± 0.175	0.040 ± 0.025
BKV Serotype III	8.4 ± 3.7	0.645 ± 0.397	0.093 ± 0.057
BKV Serotype IV	4.1 ± 1.3	0.143 ± 0.135	0.021 ± 0.020

Phase 2 Trial of MAU868 in Kidney Transplant Patients with Active BKV

MAU868-201 Trial Design

Study Population

- Kidney transplant within one year of enrollment in the trial
- Documented BKV viremia within 10 days prior to enrollment in the trial
- Viral load $\geq 10^4$ log₁₀ copies/ml, but no more than $\leq 10^7$ log₁₀ copies/ml, or consecutive positive VLs if most recent is $\geq 10^3$ log₁₀ copies/ml



Dose Cohorts (n=12 each) 4 IV doses over 12-wks

Cohort 1 1350 mg x 4

Cohort 2 6750 mg x 1 followed by 1350 mg x3

Study Endpoints

Primary

- Safety, tolerability

Secondary

- BKV-related outcomes including viremia, nephropathy, graft function and rejection, PK

Vera's Late-Stage Pipeline

Program	Indication	Phase 1	Phase 2	Phase 3	Next Milestone	Global Rights
Atacicept						
Fusion protein that blocks BLyS and APRIL	IgAN				 Ph 2b primary endpoint 4Q 2022	
	Lupus Nephritis				Ph 3 trial initiation	
MAU868						
Monoclonal antibody that neutralizes BK virus	BK Viremia in Renal Transplant				Ph 2 results mid-2022	
	BK Cystitis in Hematopoietic Stem Cell Transplant (HSCT)	<i>Under Exploration</i> 			Undisclosed	

Q&A

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