	N = 54	%
ADV alone	18	33
ADV + BK virus	4	8
ADV + CMV	6	11
ADV + BK virus + CMV	5	9
ADV + HHV6	2	4
BK virus alone	11	20
BK virus + CMV	1	2
CMV alone	6	11
CMV + HHV6	1	2

Table 1: Indications for cidofovir administration.
ADV: Adenovirus, CMV: Cytomegalovirus, HHV6: Human Herpes virus 6.

		AKI[1] at EOT N=13	%	No AKI[1] N=41	%
HCT type					
	Conventional	3	23	9	22
	T cell depleted	10	77	32	78
Cidofovir timing					
	≤30 days from HCT	4	31	3	7
	≤100 days from HCT	7	54	26	63
	>100 days from HCT	6	46	15	37
Cidofovir dose					
	Low (0.25 - 1mg/kg)	0		13	32
	High (3-5mg/kg)	13	100	28	68
Cidofovir number of doses					
	1-3	9	69	21	51
	>4	4	31	20	49
Active GvHD at cidofovir start		4	31	19	46
Abnormal baseline creatinine		4	31	11	27
EOD involving genitourinary tract		3	23	20	49
Concomitant nephrotoxic drugs[2]		12	92	32	78
Death within 4 weeks from last dose		8	62	11	27

Table 2: Characteristics of patients with acute kidney injury.
[1]Acute kidney injury defined as a rise of ≥1.5 times the baseline value.
[2]including vancomycin, aminoglycozides, cyclosporine, tacrolimus, amphotericin b and intravenous voriconazole/posaconazole.
AKI: Acute kidney injury, EOT: End of treatment, HCT: Hematopoietic cell transplantation, GvHD: Graft versus host disease, EOD: End organ disease.

Rates and Outcomes of End Organ Disease								
		N	Probable[1]	Proven[1]	Clinical response[2]	%	death within 4 weeks	%
Adenovirus		35			18	51	11	31
	All EOD	30	25	5	12	40	14	47
	EOD involving GIT[3]	15	11	4	8	53	8	53
	EOD involving GUT[4]	3	3	0	2	67	0	
	EOD involving more than one site	11	10	1	2	18	8	73
BK virus		21			12	57	6	29
	EOD (All involving GUT[4])	19	19	0	11	58	5	26
CMV		19			7	37	10	53
	All EOD	5	2	3	3	60	1	20
	EOD involving GIT	3	1	2	2	67	0	
	EOD involving lungs	1	0	1	0		1	100
	Retinitis	1	1	0	1	100	0	

Table 3: EOD: End organ disease, GIT: Gastrointestinal tract, GUT: Genitourinary tract, CMV: Cytomegalovirus.
[1]Definition of probable and proven end organ disease based on standard criteria
[2]Clinical response as implied by treating physician's clinical impression
[3]Duadenitis, colitis or both
[4]Mainly hemorrhagic cystitis

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1762. Genotype Prevalence and Molecular Characteristics of Human Adenovirus in Pediatric Hematopoietic Stem Cell Transplant Recipients

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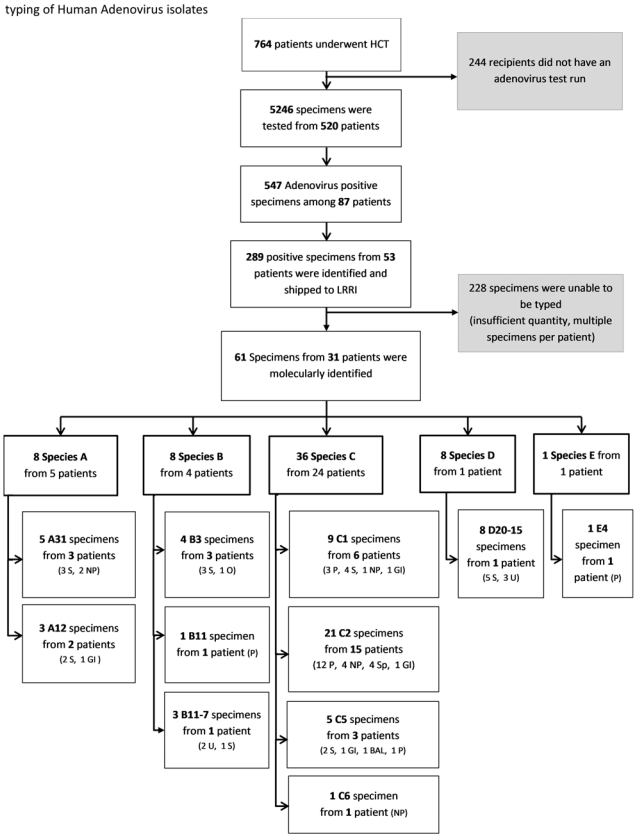
Background. Human adenovirus (HAdV) is a documented source of morbidity and mortality after hematopoietic cell transplant (HCT); however, there are limited data documenting HAdV species and type in this population. Understanding the molecular characteristics of HAdV could inform the development and assessment of interventions. The species and type of HAdV-positive specimens are detailed using an archived convenience sample of specimens obtained in pediatric HCT recipients.

Methods. The cohort included autologous and allogeneic HCT recipients between January 2000 and December 2013. An archived clinical repository of frozen specimens was interrogated to identify residual HAdV-positive specimens, which were sent to Lovelace Respiratory Research Institute (LRRI) to determine species and type. Medical chart review was performed to determine whether an isolate was related to HAdV disease or HAdV-attributable death.

Results. There were 547 HAdV PCR-positive clinical specimens from 87 HCT recipients. Of the 547 specimens, 289 were identified from an archived repository and sent to LRRI to determine species and type, and HAdV was successfully isolated and typed from 61 (Figure 1). Species C was the most common species (59.0%) with C2 being the most frequent type (34.4%). Of the 15 recipients with type C2, plasma was the most common specimen source (57.1%). Three recipients with C2 had this species and type detected from multiple sources (Tables 1 and 2). Among those with a typing result, type C2 also was responsible for 33.3% of all HAdV-attributed disease and 38.1% of all HAdV-attributed death.

Conclusion. Species C was the most common species to be isolated in a convenience sample of HAdV-positive clinical specimens from a single-center cohort of pediatric HCT recipients. Type C2 was most commonly associated with HAdV disease and attributable death. These results suggest HAdV species and type influence the impact of HAdV in this patient population. The findings need to be confirmed in prospective cohorts but suggest real-time molecular typing may be relevant and provide possible targets for the development of future interventions. These results must be interpreted with caution; not all clinical specimens were available for molecular typing, and it is possible C2 is easier to isolate from archived specimens.

Figure 1: Flow diagram of specimens collected from Pediatric Hematopoietic Cell Transplant (HCT) Recipients to typing of Human Adenovirus isolates



Abbreviations: HCT = Hematopoietic Cell Transplant P=Plasma/Blood; NP=Nasopharyngeal aspirate; S=Stool; U=Urine; BAL=Bronchoalveolar lavage; GI=Gastrointestinal tissue; Sp=Sputum; L=Liver; O=Other

Table 1. Species, type and clinical manifestations of Human Adenovirus isolates (patients under surveillance)

ID	Number of specimens retrieved from archive, by source									Species/Type (source)[£]	Transplant type	Disease	Days from detection to disease [¥]	Deceased (within 180 days post-transplant)
	BAL	GI	L	NP	P	S	Sp	U	O					
1					1					C2	Allo	Pneumonitis (prov)	0	N
												Hepatitis (pos)	5	
2					8					C2; E4	Auto	Hepatitis (pos)	5	N
3					8				1	C2 (P); B3 (O)	Allo	Hepatitis (pos)	6	N
4					6					C1	Allo	Hepatitis (prob)	7	N
6					2					C5	Allo	None	N/A	N
7				2	7	1				A31 (NP; S) C2 (P)	Allo	Pneumonitis (pos)	36	Y[†]
												Hepatitis (pos)	38	
8				1	9				0	C2 (P; NP)	Allo	Pneumonitis (prob)	25	N
												Colitis (pos)	43	
9				1	9					C2(P)	Allo	Pneumonitis (pos)	32	N
11					1	6		3	1	D20-15 (S; U)	Allo	Hepatitis (pos)	42	N
												Cystitis (pos)	63	
												Colitis (pos)	82	
12		1			4	2				C1 (GI; S)	Allo	Colitis (prob)	17	N
13		1			4		1	1		C2 (P)	Allo	Cystitis (pos)	0	Y[†]
												Hepatitis (pos)	0	
												Colitis (pos)	3	
												Pneumonitis (pos)	17	
14			1		9					C1 (P)	Allo	Hepatitis (pos)	23	N
15		1		4	7	1		1		C2 (NP; S)	Allo	Colitis (prob)	13	Y[†]
												Cystitis (pos)	36	
												Pneumonitis (prob)	90	
16					17	2		2		C1 (S)	Allo	Hepatitis (pos)	0	Y[†]
												Colitis (pos)	3	
												Cystitis (pos)	37	
												Pneumonitis (pos)	70	
17						2				B3	Allo	Colitis (pos)	0	N
18		1			2	2				C5 (GI; S)	Allo	Colitis (pos)	0	N
												Hepatitis (pos)	2	
19					5					C2	Allo	Hepatitis (prob)	0	N
21					2	1				A12 (S)	Allo	None	N/A	N
23					2	2		1		C2 (S) A31 (S)	Allo	Colitis (pos)	23	Y
												Hepatitis (pos)	23	
												Cystitis (prob)	38	
24		1				3				A12 (GI; S)	Allo	Colitis (prob)	2	N
25	1	2		1	3					C2 (GI; NP; BAL)	Allo	Colitis (pos)	100	Y[†]
												Pneumonitis (prov)	108	
26					1	2				A31 (S)	Allo	Colitis (prob)	2	N
27					19	2				C2 (S)	Allo	Colitis (pos)	0	N
												Hepatitis (pos)	1	
												Pneumonitis (pos)	8	
28					3					C2	Allo	None	N/A	N
29				2	14	1				C1 (P; S)	Allo	Pneumonitis (prob)	87	Y[†]
												Hepatitis (prob)	87	
												Colitis (prob)	87	
												CNS (prov)	87	
30						1				C2	Allo	None	N/A	N
31				1	7	1		2		B11-7 (S; U) B11 (P)	Allo	Hepatitis (prob)	0	Y[†]
												Cystitis (pos)	14	
												Colitis (pos)	49	
												Pneumonitis (prov)	55	

Abbreviations: P=Plasma/Blood; NP=Nasopharyngeal aspirate; S=Stool; U=Urine; BAL=Bronchoalveolar Lavage; GI=Gastrointestinal tissue; Sp=Sputum; L=Liver; O=Other; pos=possible; prob=probable; prov=proven

[£]The species and type were identified from at least one of the clinically positive specimens. When specimens from more than one source were sent, the source in which the molecular type was identified, is specified in the parentheses next to the type.

[¥]Time from first positive to first disease designation

[†]Death was attributable to Adenovirus

Table 2. Species, type and clinical manifestations of Human Adenovirus isolates (patients not under surveillance)

ID	Number of specimens retrieved from archive, by source									Species/Type (source)[£]	Transplant type	Disease	Days from detection to disease [¥]	Deceased (within 180 days post-transplant)
	BAL	GI	L	NP	P	S	Sp	U	O					
5				2						C6	Allo	Pneumonitis (pos)	0	N
10					5					C2	Allo	Cystitis (prob)	0	Y
												Hepatitis (pos)	0	
20				1						C1	Allo	Pneumonitis (pos)	0	Y[†]
22						4				B3	Allo	Cystitis (pos)	0	N
												Colitis (prob)	14	

Abbreviations: P=Plasma/Blood; NP=Nasopharyngeal aspirate; S=Stool; U=Urine; BAL=Bronchoalveolar lavage; GI=Gastrointestinal tissue; Sp=Sputum; L=Liver; O=Other; pos=possible; prob=probable; prov=proven

[£]The species and type were identified from at least one of the clinically positive specimens. When specimens from more than one source were sent, the source in which the molecular type was identified, is specified in the parenthesis next to the type.

[¥]Time from first positive to first disease designation

[†]Death was attributable to Adenovirus

Disclosures. **All authors:** No reported disclosures.

1763. The Use of Haploidentical Donors Compared with HLA-Matched Unrelated Donors is Associated with Increased Risk of BK Viruria and Hemorrhagic Cystitis

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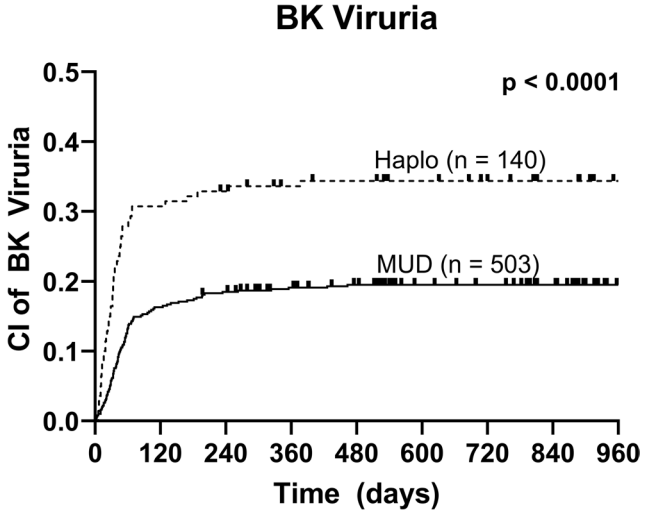
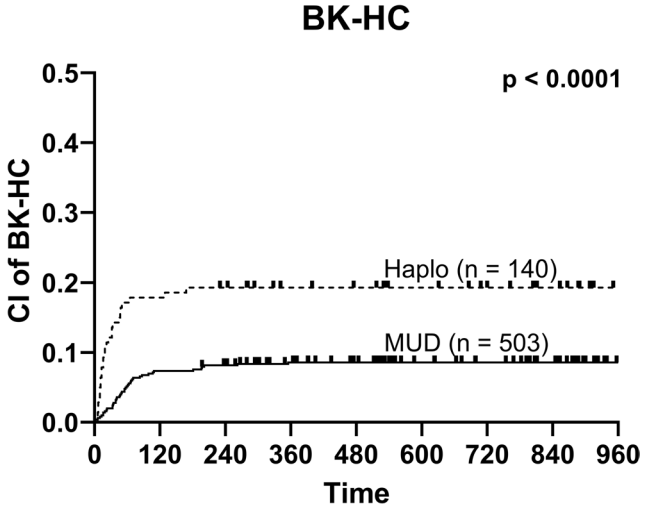
Background. BK virus-associated hemorrhagic cystitis (BK-HC) is a common and often serious complication of hematopoietic cell transplantation (HCT). Studies have suggested a higher incidence of BK-HC in patients receiving haploidentical (haplo) HCTs compared with those receiving matched unrelated donor (MUD) transplants.

Methods. We retrospectively identified all adult patients receiving HCT from MUD or haplo donors at Washington University School of Medicine between January 1, 2011 and January 1, 2016. Via informatics queries, we obtained the results of every urine BK test performed on these patients. Patients with BK viruria were then evaluated for BK-HC and graded according to established criteria. The last day of follow-up was April 31, 2017.

Results. 503 MUDs and 140 haplos were identified for inclusion in the study. Haplo patients were significantly more likely to be nonwhite (21% vs. 5%, $P < 0.001$) and were younger (median age: 51.5 vs. 55, $P = 0.01$). Conditioning regimens were also significantly different; haplos were less likely to receive myeloablative conditioning (44% vs. 57%, $P < 0.001$) and busulfan-based conditioning (13% vs. 39%, $P < 0.001$), but were more likely to receive total body irradiation-based conditioning (83% vs. 26%, $P < 0.001$). Haplos were also more likely to have undergone previous allogeneic HCT (26% vs. 6%, $P < 0.001$). The cumulative incidence of both BK viruria and BK-HC were significantly higher in haplos (both $P < 0.001$). This was observed at 100 days, 180 and 365 days (Table 1).

Conclusion. We found a significantly higher incidence of both BK viruria and BK-HC in patients receiving haplo HCT compared MUD HCT. Significant demographic and clinical imbalances exist between our two cohorts and attribution of increased risk for BK-HC to donor type vs. other factors should be further explored.





	MUD	Haplo
BK Viruria (%, 95% C.I)		
At 100 days	16 (13 - 19)	31 (23 - 38)
At 180 days	17 (14 - 21)	32 (24 - 40)
At 1 year	19 (16 - 23)	34 (26 - 41)
BK-HC (%, 95% C.I)		
At 100 days	7 (5 - 9)	18 (12 - 24)
At 180 days	7 (5 - 10)	19 (13 - 26)
At 1 year	9 (6 - 11)	19 (13 - 26)

Table 1: BK viruria and BK-HC by donor type

Disclosures. **All authors:** No reported disclosures.

1764. Use of Intravesical BCG for Treatment of Bladder Cancer in a Renal Transplant Recipient, with Subsequent Resolution of Chronic BK Viremia

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