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# Correction: Experimental models for developing oncolytic virotherapy for metastatic prostate cancer

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## KEYWORDS

oncolytic virus, metastatic prostate cancer, immunotherapy, virotherapy, experimental models

## A Correction on

### Experimental models for developing oncolytic virotherapy for metastatic prostate cancer

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There was a mistake in **Table 1** as published. There should be a separation slash between “Models and Hosts” and “Viruses”. The corrected **Table 1** appears below.

The virus particle unit number was incorrect.

A correction has been made to the section **Platforms to determine oncolytic virus cytotoxicity in prostate cancer**, *Focus on five-year clinical trials of OV in prostate cancer*, paragraph 3:

“The modified oncolytic adenovirus Ad5-yCD/mutTK<sub>SR39</sub>rep-hIL-12 was evaluated for its dosage tolerance and safety in a phase I clinical trial involving 15 patients with localized recurrent prostate cancer. A single intraprostatic dose of the virus, ranging from  $1 \times 10^{10}$  to  $1 \times 10^{12}$  viral particles, was administered on the first day of the trial, then followed by seven days of 5-fluorocytosine (5-FC) and valganciclovir (vGCV) chemotherapy (51). The Ad was designed to express cytosine deaminase (CD) and HSV thymidine kinase (TK), which convert the pro-drugs 5-FC and GCV into toxic agents that eliminate the cancer cells by interfering with DNA synthesis. The safety of this treatment was confirmed, with no reported DLTs, and 92% of side effects were classified as either grade 1 (mild) or grade 2 (moderate). Also, elevated levels of CD3<sup>+</sup>CD56<sup>+</sup> NK cells, CD3<sup>+</sup>CD4<sup>+</sup> T helper cells, and CD3<sup>+</sup>CD8<sup>+</sup> cytotoxic T cells were observed in patients, suggesting immune modulation in peripheral blood due to IL-12 expression from the Ad (104).”

The virus particle unit number was missing.

A correction has been made to the section **Trends of oncolytic viral therapy in cancer**, *Neutralization and clearance of OV within the system*, paragraph 1:

“Throughout history, humans have co-evolved with viruses, and it may come as a surprise that over 50% of the human genome originates from viruses and transposable elements. These genetic materials, acquired through horizontal gene transfer, crossover and recombination, and transformation, have significantly shaped who we are today (123), including our immunity. Over time, the diversity of the major histocompatibility complex

(MHC) has further impacted T cell and B cell specificity, as well as antibody production, thus strengthening our immune response to pathogens (124, 125). In a study by Alemany et al. in 2000 (10), it was reported that  $10^{10}$  transducing units (t.u.) of adenovirus serotype 5 particles have a half-life of less than 2 minutes following vena cava injection, with viral sequences being cleared by Kupffer cells in the liver within 24 hours (126). To prolong circulation time, scientists have genetically engineered viruses, equipping them with inhibitors of CD8+ T cells (127) or NK cell activation (128). Similarly, disguising the virus using polyethylene glycol (PEG) (for Ad and

VSV) or using mesenchymal stromal/stem cells (MSCs) as viral carriers has been shown also to prevent rapid clearance, thereby extending circulation time (126, 129–131). Protected virus not only extend their effective duration in the system but also enhance their migration toward target sites by reducing accumulation in the liver (129, 131). While rapid neutralization of oncolytic viruses might seem counter-intuitive as a therapeutic strategy, it could act to enhance immune cell infiltration into tumors, thereby improving therapeutic outcomes (132, 133)."

The original version of this article has been updated.

TABLE 1 Organized oncolytic virus publication on prostate cancer within 5 years.

Viruses	Models and Hosts	<i>in vitro</i> (2D culture)	<i>in vitro</i> (3D culture)	<i>in vivo</i>	Patients/ samples	Host reservoir
	Measles	PC3 (20)	–	–	–	Human
	Vaccinia Virus	PC3 (21)	–	PC3 (21)	–	Human and mammals
	Alphavirus	RM1 (22)	–	RM1 (22)	–	Human, Mammals, Marsupials, Birds, and Mosquitos
	Newcastle Disease Virus	DU145/PC3 (23) RM9 (24)	- -	DU145 (23) RM9 (24)	- -	Birds (Avians), can infect Human
	Epizootic Hemorrhagic Disease Virus	LNCaP/PC3 (25)	–	–	Patient samples (25)	Ruminants (Reoviridae)
	Orthoreovirus	PC3 (26)		–	–	Vertebrates
	Zika Virus	PC3 (27) PC3 (28)	- -	- -	- -	Monkey, Aedes mosquitos, and Human
	Parainfluenza Virus	22Rv1 (29)	22Rv1 (29)	–	–	Human
	Chimpanzee Adenovirus 6	RM1 (30)	–	RM1 (30)	–	Chimpanzee
	Sendai Virus	–	–	–	Patients (31)	Mice, Rats, Hamsters, and Guinea pigs
	Reovirus	TRAMP-C2/PC3/ DU145 (32) 22Rv1/DU145/ PC3 (33)	- MSK-PCa1/PDX from bone and liver metastasis (33)	TrampC2 (32) PC3 (33)	- Patient samples (33)	Vertebrates
	Herpes Simplex Virus (HSV)	TRAMP-C2/ DU145 (34) DU145 (35) PC3/LNCaP/ 22Rv1 (36)	Spheroid (34) - -	TrampC2/DU145 (34) - -	- - -	Human
	Vesicular Stomatitis Virus (VSV)	LNCaP/PC3 (37) PC3 (38) DU145/LNCaP/ PC3 (39) TRAMP-C2 (40)	- - -	LNCaP/PC3 (37) PC3 (38) - TrampC2 (40)	- - - Patient samples (40)	Indiana Vesicular Virus: Horse, Cattle, Pig, Sandflies, and Human
	Adenovirus	DU145/PC3 (41) LNCaP/C4-2 (42) DU145 (43) DU145/LNCaP/ PC3 (44) DU145/PC3 (45) PC3 (46) - DU145/LNCaP/ PC3 (47) 22Rv1/PC3 (48) LNCaP/PC3 (49) PC3 (50)	TRAMP-C2(42) - - - - - - - - - - -	PC3 (41) 224B1/Ki-ras (42) DU145 (43) PC3 (44) PC3 (45) PC3 (46) - PC3 (47) PC3 (48) LNCaP (49) PC3 (50)	- - Patients (43) - - - - Patients (51) - - - -	Human

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