multiple myeloma (MM) is an incurable disseminated cancer of antibody-secreting plasma cells that localize primarily to the bone marrow, which is characterized by the development of progressive and destructive osteolytic bone disease (1–7). MM is the second most prevalent blood cancer after non-Hodgkin’s lymphoma, representing 1% of all cancers and 2% of all cancer deaths (1, 8). The American Cancer Society estimates that in the United States about 50,000 patients have MM, approximately 20,000 new cases are being diagnosed every year, and MM is responsible for the death of about 10,000 annually (1, 4, 9). Significant progress in the understanding of disease pathogenesis and emerging therapies has been made; however, MM remains an incurable disease with a median survival of 5 to 6 years after conventional therapies (3–6, 8, 10). This underscores the urgent need for the development of alternative approaches to therapy.

Oncolytic viruses can selectively infect tumor cells and cause direct cell destruction and/or elicit antitumor innate and cellular immune responses (11–18). Oncolytic viruses have shown potential utility for the treatment of MM (19–23). We are particularly interested in utilizing vesicular stomatitis virus (VSV) for the treatment of MM. VSV is a *Vesiculovirus* of the family *Rhabdoviridae* with a negative-sense RNA genome that encodes a nucleocapsid protein (N), a phosphoprotein (P), a matrix protein (M), a glycoprotein (G), and an RNA-dependent RNA polymerase (L) (24). VSV infects a wide variety of animals and different cells. VSV has a rapid replication cycle (24) and the ability to reach high titers in infected tumor cells, while normal cells are effectively protected by the antiviral activity of the host interferon (IFN) response (25–29). These attributes, together with the presumed lack of a preexisting antibody response to VSV in humans, make VSV a preferred candidate platform for development of oncolytic virus against a variety of cancers (17, 21, 22, 27, 29–32). Studies from our laboratory and others show that VSV and its recombinant derivatives have some promise as antimyeloma agents (17, 19, 21, 22, 31, 33).

For cancers like MM that are disseminated from the onset, systemic delivery of oncolytic viruses that can target both primary and metastatic deposits at the same time is the preferred option for better efficacy (17, 34, 35). However, intravenous (i.v.) delivery presents its own challenges, such as potential failure of oncolytic virus extravasation from tumor blood vessels, mislocalization in liver and spleen, and neutralization by antiviral antibodies (17, 20, 36–39). These potential challenges to systemic virus delivery emphasize the difficulties of using free virus as an oncolytic agent in immunocompetent hosts. In an effort to circumvent the neutralization of oncolytic viruses and to increase the efficiency of virus delivery, different approaches have been employed, such as the use of cell carriers, serotype switching, and polymer shielding (17, 33, 40–45). We are testing covalent modification of VSV with polyethylene glycol (PEG) molecules and noncovalent membrane-inserting function-spacer-lipid (FSL)–PEG construct, termed PEGylation, to overcome the challenges for i.v. delivery of VSV. PEG is an uncharged, hydrophilic, linear polymer that is nonimmunogenic and that has low toxicity, leading to its approval by the Food and Drug Administration (FDA) for human use (40, 46). PEGylation can potentially protect oncolytic viruses from serum neutralization and from nonspecific localization in organs such as liver, leading to an increased virus circulation half-life (40, 42,
Accordingly, we hypothesized that PEGylation of VSV will increase its stability in blood circulation and that this will enhance virus delivery into tumor sites and improve its antitumor efficacy.

In this study, we characterized the interaction of VSV with nonimmune normonuclear serum from human and mouse. We found that clinical-grade VSV is rapidly neutralized by nonimmune human and mouse serum. Neutralization requires both IgM antibody and a heat-labile factor(s). In addition, we demonstrate that covalent modification of VSV with PEG molecules or with membrane-inserting FSL-PEG constructs (PEGylation) protects the virus from serum neutralization in vitro. Using passively immunized mice, we further show that PEGylation extends the persistence of VSV in the circulation and increases virus trafficking to tumor growth sites while reducing virus-induced hepatoxicity. We are currently investigating if this increase in VSV circulating half-life in blood can translate to better VSV efficacy in mouse models of multiple-myeloma tumors.

**MATERIALS AND METHODS**

**Cells.** Human 293T cells and African green monkey kidney Vero cells were cultured in Dulbecco modified Eagle medium (DMEM) supplemented with 10% heat-inactivated fetal calf serum (FCS) for 293T cells and mouse plasmacytoma (MPC11) cells (ATCC CCL-167; American Type Culture Collection) or 5% FCS for Vero cells, penicillin, and streptomycin (Gibco).

**Virus.** We obtained VSV coding for mouse beta IFN (mIFN-β) or green fluorescent protein (GFP; Indiana strain) from the laboratory of Glen N. Barber (University of Miami School of Medicine, Miami, FL) (27) that was constructed by insertion of the IFN-β or GFP gene at Xhol/Nhel restriction sites between the viral G and L genes. Clinical-grade oncolytic VSV was manufactured in the Mayo Clinic Vector Manufacturing Facility.

**Virus neutralization assays.** For neutralization assays, VSV-GFP (5-μl volume with 1 × 10⁵ 50% tissue culture infective doses [TCID₅₀/g]) was incubated with 100 μl undiluted nonimmune human serum (serum from a pool of 3 to 5 volunteer healthy donors) or naïve mouse serum at 37°C for 1 h or for the indicated times, followed by determination of the virus titer (number of TCID₅₀/ml) on Vero cells (1 × 10⁴ cells/well) that were plated overnight prior to infection with VSV treated with serum. In some cases, heat inactivation of serum was performed at 56°C for 30 min (32). The effect of serum IgM was analyzed by preincubation of serum with anti-IgM antibody (equal volumes for 30 min at room temperature). As a control, virus was incubated with medium only and plated on Vero cells. The neutralization assay result was read at 48 h after infection with virus that was treated with or without serum. All virus neutralization assay experiments were conducted in triplicate.

In vivo mice experiments, VSV-GFP (5 × 10⁴ TCID₅₀) was i.v. injected into immunocompetent BALB/c or C57BL/6 mice to which VSV-immune serum (1:100-diluted serum that was harvested from actively immunized mice) had been passively transferred by intraperitoneal (i.p.) injection 24 h prior to i.v. tail vein injection of VSV. Blood samples were drawn via the retroorbital and/or submandibular method at the indicated time points for determination of the number of virus TCID₅₀/ml on Vero cells and viral RNA analysis by quantitative PCR (qPCR).

For survival studies, BALB/c mice (4 to 6 weeks old) were immunized by i.v. challenge with VSV–mIFN-β (1 × 10⁸ TCID₅₀/mouse). Two weeks after immunization, the titer in serum was determined using neutralization assay. Mice with similar serum titers (see Fig. 2a) were selected, and MPC11 subcutaneous tumors were established by injecting 5 × 10⁶ MPC11 cells subcutaneously, including into control naïve mice. Once tumors were established (tumor volume, ~100 mm³), mice were randomly divided into three groups: group 1 was immunized and i.v. injected with no virus (phosphate-buffered saline [PBS] only; n = 8), group 2 was immunized and i.v. injected with VSV–mIFN-β (2.5 × 10⁷ TCID₅₀/mouse; n = 9), and group 3 was nonimmunized (naïve) and i.v. injected with VSV–mIFN-β (2.5 × 10⁷ TCID₅₀/mouse; n = 5). Tumor size and survival data were collected, as shown in the appropriate figure legend. All of the animal experimental procedures in this study were performed following the guidelines of the Mayo Clinic Institutional Animal Care and Use Committee (IACUC).

**VSV PEGylation.** VSV was covalently modified with activated monomethoxy-polyethylene glycol (PEG5000) succinimidyl succinate (Jenkem Technology, Beijing, China). A range of concentrations of PEG (0 to 20 mg) was added to 5 × 10⁷ TCID₅₀ of VSV with a final volume of 500 μl in 100 mM potassium PBS (pH 7.4) for each preparation. PEG conjugation reactions were performed as described previously (40, 45, 53). Briefly, all conjugation reactions were performed at 25°C with gentle agitation. The reactions were stopped by 15 min incubation at 4°C (45). Reaction products were purified by buffer exchange over a Micro-Bio Spin P-30 chromatography column (Bio-Rad) equilibrated with 100 mM potassium PBS (pH 7.4) following the supplier’s protocol. A similar approach was used to modify VSV with FSL-PEG5000 (KODE Biotech, Auckland, New Zealand), but it was noncovalently used to modify VSV by simple direct contact (53, 54). As a control, VSV was incubated with 100 mM potassium PBS (pH 7.4) without PEG or FSL-PEG under similar conditions and was processed in a manner similar to that for the PEG-conjugated virus. PEGylation status was assessed by in vitro infectivity assay on Vero cells. Serum neutralization assay was performed after exposing viruses to different concentrations of PEG-treated (0.31 mg to 20 mg) or FSL-PEG-treated (5 to 200 μg) VSV.

**Purification of RNA and cDNA synthesis for qPCR analysis.** Tumor tissue disruption and homogenization for RNA harvest were performed using a TissueLyser apparatus in combination with a Qiagen sample purification kit. Total blood RNA was harvested using a RNAeasy Protect animal blood kit (Qiagen). Total RNA (160 ng) was subjected to reverse transcription-PCR (RT-PCR) assay for cDNA synthesis using a SuperScript III first-strand synthesis Supermix kit (Invitrogen) with random hexamers following the manufacturer’s instructions.

**qPCR analysis.** To measure the number of VSV genome copies in total RNA samples harvested from blood or tumor, cDNA was subjected to qPCR analysis with Mx4000 multiplex quantitative PCR systems (Stratagene) with primers specific to VSV N (primers VSVN-F [5'-GGAAATGAGCTTGACCA-3'] and VSVN-R [5'-CTAACCA-3']) and as internal control specific primers for GAPDH (glyceraldehyde-3-phosphate dehydrogenase; primers GAPDH-F [5'-GCCACCTCTCCACCTTTG-3'] and GAPDH-R [5'-CTAACCA GGAATGCGTGGACCA-3']). Amplified sequence detection was performed by SYBR green-based real-time quantitative PCR assay (Roche Diagnostics, Indianapolis, IN). Data are presented as the VSV N genome copy number, determined using a standard curve of known quantities of VSV N plasmid.

**Statistical analysis.** Data analysis was carried out using GraphPad Prism software (GraphPad Software, CA). A two-tailed Student’s t test was used to compare mean values. A P value of <0.05 was considered statistically significant. Error bars represent standard deviations. For survival studies, comparison was performed using the log-rank test from Kaplan-Meier survival curves.

**RESULTS**

VSV is rapidly neutralized by nonimmune normal serum. Presumed advantages for using VSV as an oncolytic virus are that human infections are rare and that preexisting anti-VSV immunity is lacking, which is very important for clinical success (17). To test these assumptions, we performed neutralization assays by exposing clinical-grade VSV to normal human or mouse serum for 1 h or at earlier and later time points at 37°C, and the virus titer was determined on Vero cells. Our data show that normal human serum quickly neutralized VSV, resulting in up to a 4-log-unit
virus titer reduction compared to the titer for control virus incubated with medium only. Normal mouse serum similarly neutralized VSV, albeit to a lesser extent than that observed with human serum (Fig. 1a, b, and d). We also show that the anti-VSV activity of normal human serum can be largely abrogated by anti-IgM antibody pretreatment (Fig. 1b) or by heat inactivation of the serum (Fig. 1a) prior to incubation with virus, suggesting the involvement of the antibody and a heat-labile factor(s) of normal nonimmune serum in the anti-VSV activity, which is in agreement with findings from previous studies (52).

**VSV is rapidly cleared from blood circulation in vivo.** We assessed the stability of VSV after intravenous injection into immunocompetent C57BL/KaLwRij mice, using the 5TGM1 multiple-myeloma mouse model (55). Our in vivo VSV stability studies showed that VSV is rapidly cleared from the bloodstream, with up to a 3-log-unit reduction in VSV titer within 10 min after virus injection into nonimmunized mice (Fig. 1c). In addition, using actively immunized BALB/c mice bearing MPC11 subcutaneous tumors, we show that immune anti-VSV antibody completely negates the antitumor efficacy of VSV (Fig. 2b and c), underscoring a challenge for repeat VSV administration.

Our in vitro and in vivo data show that nonimmune serum can effectively neutralize VSV and the antitumor efficacy of VSV is negated by VSV-specific immune serum. These results, together with the potential failure of oncolytic VSV to extravasate from tumor blood vessels and its sequestration in liver and spleen (17, 20, 37–39), emphasize the difficulties in using free VSV as an oncolytic agent in an immunocompetent host. Because of this, we are focused on finding ways to circumvent serum neutralization of VSV in the blood circulation and to increase the efficiency of VSV delivery into tumor growth sites. One of the innovative approaches being tested is covalent and noncovalent modification of VSV with PEG, termed PEGylation.

**PEGylation protects VSV from neutralization with VSV-specific immune serum.** PEGylation, the covalent modification of virus with PEG molecules, is one of the ways to protect nonenveloped viruses from serum neutralization being tested and is showing some promise (40, 42, 47–50). We therefore tested if PEGylation can render VSV, an enveloped virus, resistant to serum neutralization. To our knowledge, this is the first report of modification of an enveloped animal virus with PEG for protection of virus from serum neutralization. We used two approaches to bind PEG molecules to the virus surface, either by directly PEGylating the VSV glycoprotein G (VSV G), which is required for virus attachment and membrane fusion during VSV entry into susceptible cells and is the target for host neutralizing antibody (24), or, alternatively, by using FSL-PEG conjugates that insert into the VSV lipid membrane rather than covalently bind to VSV G (53, 54). We proposed that PEGylation can protect VSV from serum neutralization and limit nonspecific hepatosplenic sequestration, extending virus persistence in blood and increasing its trafficking to sites of multiple-myeloma tumor growth. While the direct VSV G covalent modification with PEG may render VSV, an enveloped virus, resistant to serum neutralization. To our knowledge, this is the first report of modification of an enveloped animal virus with PEG for protection of virus from serum neutralization. We used two approaches to bind PEG molecules to the virus surface, either by directly PEGylating the VSV glycoprotein G (VSV G), which is required for virus attachment and membrane fusion during VSV entry into susceptible cells and is the target for host neutralizing antibody (24), or, alternatively, by using FSL-PEG conjugates that insert into the VSV lipid membrane rather than covalently bind to VSV G (53, 54). We proposed that PEGylation can protect VSV from serum neutralization and limit nonspecific hepatosplenic sequestration, extending virus persistence in blood and increasing its trafficking to sites of multiple-myeloma tumor growth. While the direct VSV G covalent modification with PEG may result in reduced virus infectivity, the use of FSL-PEG, which inserts into the virus lipid membrane, leaves VSV G available for receptor binding (53, 54) and may be less likely to block virus infectivity. To test our hypothesis, we first identified PEGylation conditions that can provide both a good level of virus infectivity and protection from serum neutralization.

We assessed the level of PEGylation using fluorescence-activated cell sorter analysis of FSL-fluorescein membrane incorporation (data not shown) (54) and inhibition of VSV infectivity due to increased concentrations of PEG or FSL-PEG (Fig. 3 and 4a). We show that while low levels of PEG or FSL-PEG modification maintain VSV infectivity, increasing the concentration of PEG molecules leads to loss of VSV infectivity in vitro (Fig. 3 and 4a). We found that in our hands, depending on the freshness of the

![FIG 1](http://jvi.asm.org/)
stock used, we needed a 10 to 200 times increased amount of PEG than FSL-PEG to inhibit the infectivity of VSV in vitro (Fig. 3 and Fig. 4a, medium). We speculate that the need for an increased amount of PEG compared to that of FSL-PEG to result in a loss of VSV infectivity is due to either an increased efficiency of binding of FSL-PEG as it inserts into the virus membrane envelope compared to that of PEG, which covalently binds to VSVG, or the presence of FSL, which increases the length of the polymer compared to that with PEG alone and which may lead to increased inhibition of VSV infectivity with fewer FSL-PEG molecules per virion. In addition, we determined the level of modification needed for protection of VSV from in vitro neutralization with VSV-specific immune serum, a very stringent condition compared to that achieved with nonimmune serum (Fig. 4a).

PEGylation of VSV extends virus RNA persistence in blood. Our in vitro data show that serum from naïve mice can inactivate VSV at a rate similar to that by nonimmune human serum (Fig. 1a,b, and d) and that the virus is rapidly cleared from the circulation in vivo (Fig. 1c). Interestingly, we show that VSV can be conjugated with PEG (Fig. 3a) or modified with FSL-PEG (Fig. 3b) and that PEGylation can protect VSV from FSL-PEG molecules per virion. In addition, we determined the level of modification needed for protection of VSV from in vitro neutralization with VSV-specific immune serum, a very stringent condition compared to that achieved with nonimmune serum (Fig. 4a).

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PEGylation increases virus genome accumulation in tumor and lowers virus-induced hepatotoxicity. Considering the improved persistence of PEGylated VSV in blood (Fig. 4c), we hy-
The fluorescent (top) and phase-contrast (bottom) images taken at 24 h after virus injection showed increased virus trafficking in tumor tissues. PEGylation status was assessed by similar conditions and processed in the same manner as the conjugated virus. PEGylation status was assessed by infectivity on Vero cells.

When liver, lung, kidney, and spleens were assayed for VSV deposition at day 4, PEGylation increased virus genome levels in these tissues (data not shown). The PEGylation-related increase in virus level in tissues could be due to initial virus protection from serum neutralization in the circulation followed by virus amplification in tissues.

In an independent experiment, we investigated virus-induced hepatotoxicity by performing blood chemistry to determine serum alanine aminotransferase (ALT) and alkaline phosphatase (ALP) levels at day 3 after virus injection in BALB/c mice. ALT and ALP levels were significantly increased in animals treated with unmodified VSV compared to control animals (P = 0.0003 and P = 0.00045, respectively) or those treated with PEGylated VSV (P = 0.0092 and P = 0.0003, respectively). Conversely, animals treated with PEGylated VSV had lower levels of ALT and ALP that did not differ significantly from the levels in control animals treated with PBS (P = 0.3449 and P = 0.4922, respectively) (Fig. 6a and b).

**DISCUSSION**

We are developing oncolytic VSV, a negative-stranded RNA virus, for treatment of multiple myeloma, an incurable cancer of antibody-secreting plasma cells primarily localized to bone marrow (1, 2, 4–6). VSV is a promising oncolytic virus that specifically infects and kills tumor cells and has shown promise in preclinical studies for treatment of various tumors (17, 21, 22, 25–32). In this work, we show that nonimmune normal human serum rapidly neutralizes clinical-grade VSV, a process that is dependent on a heat-labile factor(s) and IgM antibody and that reduces the viral titer by about 4 log units within 1 h. In contrast, VSV-immune serum very rapidly neutralizes VSV and negates its antitumor efficacy in the MPC11 multiple-myeloma BALB/c mouse model (Fig. 2b and c). Together, these data underscore the challenges of using VSV as an oncolytic agent in an immunocompetent subject.

We are investigating different approaches that can protect VSV from serum neutralization, increase virus trafficking to tumor growth sites, and lead to increased virus efficacy. One of the innovative ways to protect virus from serum neutralization is virus PEGylation, a covalent conjugation of PEG molecules to the virus surface (40, 42, 47–50). We have developed PEGylation conditions for VSV. Our in vitro data show that low concentrations of PEG did not compromise VSV infectivity. However, higher concentrations of PEG led to a loss of virus infectivity (Fig. 3a and b). Our in vitro studies further demonstrate that PEGylation of VSV with PEG or FSL-PEG protects VSV from neutralization with VSV-specific immune serum, a very stringent condition compared to that with nonimmune serum (Fig. 4a). Using BALB/c mice bearing subcutaneous MPC11 multiple-myeloma tumors, we show that virus RNA persisted for a longer time in the circulation of mice treated with PEGylated VSV than in mice treated with unmodified VSV in the presence or absence of passively transferred VSV-specific immune serum.  

More importantly, we show that VSV RNA was enriched in tumor tissues harvested on day 4 from mice treated with PEGylated VSV compared to the level in mice that received unmodified VSV (Fig. 5). FSL-PEG-VSV levels tend to be higher in blood (Fig. 4c), while they are lower than PEG-VSV levels in tumor tissues. We speculate that this may be related to differences in sequestration levels by other tissues, particularly lung and liver.

**FIG 3** PEGylation of VSV. Different amounts of PEG5000 activated by succinimidyl (Jenkem Technology, Beijing, China) (a) or FSL-PEG2000 (KODE Biotech, Auckland, New Zealand) (b) were added for 5 × 10^9 TCID50 of VSV. All conjugation reactions were performed at 25°C with gentle agitation. The reactions were stopped by 15 min incubation at 4°C as described by O’Riordan et al. (45). Unreacted PEG and reaction by-products were eliminated by buffer exchange over a Micro-Bio Spin P-30 chromatography column (Bio-Rad) equilibrated with 100 mM potassium PBS (pH 7.4). As a control, VSV with the same PBS buffer but without PEG or FSL-PEG was incubated under a similar condition and processed in the same manner as the conjugated virus. PEGylation status was assessed by infectivity on Vero cells. The fluorescent (top) and phase-contrast (bottom) images taken at 24 h postinfection are shown for each condition.

We hypothesized that this increase in the plasma half-life of PEGylated VSV may lead to an increase in virus trafficking to tumor sites. To test our hypothesis, we compared the accumulation of unmodified VSV to that of PEGylated VSV following i.v. injection of VSV into four subcutaneous MPC11 tumor-bearing BALB/c mice in the presence of passively transferred VSV-specific immune serum. Control mice were treated with 200 μl of potassium PBS. At 4 days after virus injection, mice were euthanized and total RNA was isolated from the tumor and then analyzed for VSV N genome copy number using qPCR. Animals that were treated with PEGylated VSV had lower levels of ALP and ALT that did not differ significantly from the levels in control animals treated with PBS (P = 0.3449 and P = 0.4922, respectively) (Fig. 6a and b).
and binding to and/or infection of blood cells. The larger molecular size of FSL-PEG may reduce VSV sequestration by liver compared with that of PEG-bound VSV, leading to an increased level of FSL-PEG-VSV in blood (Fig. 4c) (62). However, FSL-PEG that inserts into the VSV membrane envelope is likely to retain VSV G free to effect binding and infection of blood cells, which may reduce its level in tumor and other tissues (63, 64) compared to the level of PEG-VSV, whereas binding of PEG directly on the VSV G may reduce its infectivity of blood cells, leading to increased levels in tumors (Fig. 5).

Interestingly, virus-induced hepatotoxicity assessed using blood levels of ALP and ALT showed that mice treated with unmodified VSV had significantly elevated enzyme levels compared to animals treated with PEGylated VSV or PBS-treated mice (Fig. 6a and b).

Overall, our data identified barriers to systemic VSV delivery in immunocompetent mice and demonstrated the feasibility of using covalent modification of VSV with PEG molecules for virus protection from serum neutralization. We further established that PEGylation extended the persistence of VSV in the circulation in the presence of neutralizing anti-VSV antibodies and improved virus trafficking to tumor sites with no noticeable virus-induced
The results of our studies are in line with published results of preclinical studies on PEGylation of nonenveloped viruses that demonstrated that PEGylation provided virus protection from serum neutralization and reduced virus-induced toxicity \((40, 42, 47–51)\). To our knowledge, this is the first study to report the feasibility of PEGylation of an enveloped animal virus and to demonstrate that PEGylation protects VSV from serum neutralization. We are currently investigating if the increased stability of PEGylated VSV in blood will improve the efficacy of the virus as an oncolytic agent following intravenous delivery into immunocompetent multiple-myaloma mouse models in the presence of neutralizing antibodies.

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REFERENCES

29. Stojdl DF, Lichy BD, tenOever BR, Paterson JM, Power AT, Knowles


Erratum for Tesfay et al., PEGylation of Vesicular Stomatitis Virus Extends Virus Persistence in Blood Circulation of Passively Immunized Mice


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Volume 87, no. 7, p. 3752–3759, 2013. Page 3754, Fig. 1b: “P/H11005 0.004” should read “P/H11005 0.0004” and “P/H11005 0.012” should read “P/H11005 0.0174.”