Supporting Information

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SI Experimental Procedures

Cells, Viruses, Virus Infections, and Virus Yield Determination. HeLa (ATCC # CCL2), MDCK, and TZM-bl cells were maintained in DMEM supplemented with 10% FBS and antibiotics. BHK-21 cells were maintained in MEM with 5% FBS. LCMVr3-GFP (1) and HPIV3-GFP (2) were grown in BHK-21 cells and titrated in HeLa cells by fluorescent focus forming assay. Clonal stock of VSV-eGFP was prepared by growing the virus in BHK-21 cells and titrating in HeLa cells by plaque assay. VSV-PeGFPΔG virus has been described (3). VSV yield was determined by plaque assay on HeLa cells.

Antibodies. Anti-M (23H12) monoclonal antibody was from Douglas Lyles (Wake Forest University, Winston-Salem, NC). Anti-actin, anti-eGFP, and anti-*Renilla reniformis*-GFP (GFPrr) antibodies were from Santa Cruz; anti-βCOP, anti-ζCOP, anti-ARF1, and MAT2A antibodies were from Sigma; anti-GBF1 antibody was from BD Science; and anti-SLC46A1 antibody was from Abcam.

SDS/PAGE, Immunoblotting, and Quantitation. These procedures are described (3). Quantitation of proteins detected in the blots was performed by using Versadoc QuantityOne software (BioRad Laboratories).

NC Preparation and Transfection. VSV-eGFP NCs were prepared, and transfection was performed by using Lipofectamine 2000 as described (3).

RNAi Screen. The primary screen was performed at the Duke University RNAi screening facility by using the Qiagen genomic siRNA library v 1.0 consisting of four distinct siRNAs (A, B, C, and D) targeting 22,909 known and putative human genes. The four siRNAs were grouped into two pools, with each pool containing two siRNAs (set AB and set CD). This format resulted in seventy-four 384-well plates per set and a total of 148 plates for the entire screen. This 2 × 2 pool design allowed each gene to be tested by two independent siRNA sets. AllStars nontargeting (NT) siRNA (Qiagen) was used as a negative control. Also, siRNAs targeting N and L genes (two per gene) of VSV were synthesized and used as additional positive control siRNAs. The sequences of the siRNAs targeting the VSV genes are as follows. N1: 5′-CUGCAAGGCCUAAGAGAGA-3′; N2: 5′-UGGAAUACCCGGCAGAUUA-3′; L1: 5′-GCAGUUAUCCAGCAAUCAU-3′; and L2: 5′-GAGAAACGUUGUAGAAUUA-3′.

Corning 384-well tissue culture plates were prearrayed with 1 pmol of siRNA per well by using the Velocity Bravo liquid handling system (Agilent Technologies). Each plate was also seeded with siRNAs targeting VSV N and L gene as well as the NT siRNA. Lipofectamine RNAiMAX (Invitrogen) was used in the amount of 0.05 μL per well in 10 μL of OptiMEM (Invitrogen). Reverse transfection of 3,000 HeLa cells was performed with 15.4 nM final concentration of siRNAs in a 65 μL of total volume. VSV-eGFP virus (grown in BHK-21 cells and titrated in HeLa cells) was used. Initial studies of siRNA transfections (48 and 72 hpt), virus infections at different MOIs (0.2, 0.4, 0.5, 0.6, 0.8, 1, 2, and 3 MOI), and for various lengths of time postinfection (8, 10, 12, 14, 16, and 18 h) were conducted to identify suitable conditions for primary high-throughput screen. To identify factors involved in all stages of the VSV life cycle, such as entry and uncoating, replication, and budding and release, the screen was optimized for MOI and time to allow multiple rounds of virus infection to occur. As a quality control metrics for the assay, Z′ factor (4) was calculated between NT siRNA treated wells and N and L siRNA treated cells. The multiplicity and time of infection was optimized to obtain a Z′ factor value of >0.5, which would indicate a robust assay. Based on these initial standardization experiments, the genome-wide screen was performed by using 0.5 MOI of VSV and 18 h of infection. In the genomic screen, the Z′ factor between NT and L was calculated to be 0.44 for AB set and 0.51 for CD set. These Z′ factor values indicated that we would be able to distinguish the positive control wells (L and N siRNA treated cells) from control NT siRNA treated wells in both AB pool and CD pool.

Automated Image Analysis. At the end of the infection, cells were fixed with 4% paraformaldehyde in PBS, permeabilized with 0.1% Triton X-100 in PBS, and stained with Hoechst 33342 in PBS for 30 min. Stained cells were imaged with a Cellomics ArrayScan VTI automated microscope. Images were analyzed with vHCS Scan Target Activation software v 5.1.2 to identify infected cells. Cells without VSV-eGFP infection served as reference population for background fluorescence. Four fields per well of a 384-well plate were imaged at 10× magnification. First, cells were identified by their nuclei staining in channel 1 of Cellomics. Cells that scored positive in channel 1 were analyzed for GFP expression in channel 2, and GFP intensity was calculated for the cells. Frequency distribution of GFP intensity was plotted and compared between NT siRNA transfected controls infected with VSV-eGFP or uninfected reference population. For selection of infected cells, threshold was set by manual inspection of representative images from VSV-eGFP infected and uninfected cells. Cells appearing at least 5 SDs away from the average intensity of uninfected control population in the same plate or same batch were considered positive for GFP expression. Once the threshold was determined, all of the plates were subjected to data analysis by vHCS Target activation software. Finally, data were obtained by using vHCS View software v 5.1.2, and the numbers of cells present in the well were identified as "Valid Object Count" (VOC), whereas the percentage of infection was determined as "% selected."

Statistical Analysis. Transfection of siRNAs and subsequent infection with VSV resulted in reduced cell number for some wells due to the combined effects of siRNA toxicity, loss of cell survival factors, and VSV cytopathogenicity. We used a cutoff value of 800 or more cell number in four fields per well for further analysis. This criterion requires that wells transfected with siRNA sets AB and CD for a particular gene should have a VOC of at least 800. Using this criterion, we discarded ~24% of the genes in the library. Because our screen was designed to identify factors whose depletion would reduce VSV infection, the genes discarded from further analysis due to reduced cell numbers may potentially represent restriction factors and/or factors required for cell survival. In other words, if a gene is required for VSV infection, its depletion would reduce VSV replication and thus would not promote VSV-mediated cell death. We would then expect to observe higher cell number in those wells. So, rationally, the remaining 76% of the genes in the library should include factors required for VSV replication.

In the screen, for both AB set and CD set, we did not observe a correlation between percent infection and cell number [r (correlation coefficient) = 0.308 (for AB set) and 0.295 (for CD set)]. It has been reported that cell density may impact clathrin-mediated endocytosis and may subsequently affect virus infection (5). However, throughout the primary screen, we used a cell density (3,000 plated cells per well) and MOI (0.5) as well as time (18 hpi)

conditions in which the rate of infection remained in the linear range. The distribution of percent infection for genomic population for the AB and CD pools did not follow a normal distribution, and thus parametric tests would not have given meaningful results. Therefore, we used a nonparametric analysis, the Sum rank statistics (6), to identify the hits from the primary screen. Because of day-to-day variation in percent infection of NT control population, we decided to use batch-wise analysis to get the primary hits. Percent infection values for set AB and set CD were ranked independently from lowest to highest and given ranks from 1 to n (n = the number of samples analyzed), respectively. Sum rank for a particular gene is the sum of rank in AB set and rank in CD set for that gene. The P value for each gene was then determined, and finally samples were aligned according to the P value. A total of 233 genes with $P < 0.01$ were identified as primary candidate hits.

Validation Screen. The validation screen was conducted at the Eppley Institute for Cancer Research at the University of Nebraska Medical College (UNMC) by using Dharmacon ON-TARGETplus pool of four siRNA. The sequences of Dharmacon siRNAs did not overlap with those of Qiagen siRNAs, allowing us to test multiple siRNA. The assay was conducted by using 96-well plates. Six microliters of pooled siRNA (500 nM) was prearrayed per well in 96-well plates by using the Biomek FX liquid handling system. Lipofectamine RNAiMAX and OptiMEM mix was prepared (0.25 μL of LipofectamineRNAiMAX in 20 μL of OptiMEM per well). For experiments using VSV, 15,000 cells in 75 μL of DMEM with 10% FBS and 1× PKS per well were added to each well to yield a final concentration of 30 nM siRNAs. For LCMV and HPIV infection, 10,000 cells per well were added in 75 μL of above medium. Cells were incubated for 50 h for knockdown of the genes. For VSV-eGFP infection, 0.05 MOI of virus was added in 100 μL of DMEM 2% FBS and 1× PKS per well by using a multichannel pipette. LCMVr3-GFP (0.05 MOI) and HPIV3-GFP (0.2 MOI) were used to infect HeLa cells. Virus inoculum containing required MOI of viruses were prepared in 40 μL of DMEM (with 5% FBS and PKS) per well. After 1 h of infection, 60 μL of DMEM with 5% FBS and 1× PKS was added to each well. Cells were infected with VSV for 14 h, with LCMV for 36 h, or with HPIV3 for 41 h. Infected cells were fixed as per the protocol described in the primary screen. Cells were stained with DAPI to stain the nuclei. Image analysis was performed in Cellomics ArrayScan VTI at UNMC to obtain the VOC and percent infection. Experiment was repeated four times for VSV and two times each for LCMV and HPIV3. Meaningful between-plate comparisons required normalization due to variations among plates. Therefore, we normalized the percent infection values by plate-wise medians, separately for siRNA-treated and untreated control samples. We applied the nonparametric Wilcoxon-Mann-Whitney (WMW) rank-sum test (7). Despite the lower statistical power of the nonparametric tests compared with the parametric tests, the WMW test indicated significant effects of the siRNA treatment in the majority of genes compared with untreated controls at the $P \leq 0.01$ level. Normalization was performed by in-house PERL and MATLAB programs. The WMW tests, heat map representations, and other graphics were carried out in MATLAB.

siRNA Transfection in 12-Well Plate. Reverse transfection of HeLa cell was performed in 12-well tissue culture plates. Final concentrations of siRNAs were 20 nM, except for COPI where 10 nM concentrations of siRNAs were used. Required amount of siRNAs were plated in the wells. Lipofectamine RNAiMAX in OptiMEM was prepared (2 μL of Lipofectamine in 300 μL of OptiMEM per well) and added to the wells. Plates were incubated for 30 min for complex formation. HeLa cells (200,000 cells per well in 500 μL of DMEM 10% FBS+ PKS) were added for COPI experiments. For other siRNA transfection experiments, 100,000 cells were added. Cells were further incubated for 42–44 h for COPI siRNAs and 66–68 h for other siRNA transfection. All of the experiments were conducted with Dharmacon ON-TARGET-plus siRNAs, except that where indicated, the Qiagen siRNAs were used.

Quantitative RT-PCR (qRT-PCR). qRT-PCR was used to measure the VSV P mRNA, genome, and anti-genome levels. Total RNA was extracted from cells by TRIzol according to manufacturer's protocol (Invitrogen). First-strand cDNA was synthesized from 200 ng of total RNA by using M-MLV reverse transcriptase according to manufacturer's protocol (Invitrogen). For quantification of P mRNA and the internal control β-actin mRNA, oligo-(dT) was used as primer in one RT reaction. For quantification of anti-genome and the internal control β-actin mRNA, VSV2955R and β-actin1 R (each 2 pmol) were used as primers in another RT reaction. These cDNAs were used as templates in qRT-PCR reactions that were carried out in Cepheid Smart Cycler. The sequences and optimized concentrations of the primers and probes are listed in Tables S1 and S2, respectively. Lightcycler 480 probe master mix (Roche Applied Sciences) was used in qRT-PCR reactions. The thermal setup conditions used included initial denaturation at 95 °C for 5 min, denaturation at 95 °C for 30 s, and annealing and extension at 60 °C for 30 s for a total of 40 cycles. Relative fold change in VSV P mRNA and anti-genome levels was calculated by ΔΔCt method. Briefly, the difference in threshold cycle (ΔCt) was calculated by dividing Ct value of VSV P mRNA or anti-genome with Ct value of the respective β-actin internal control. The relative fold change in VSV P mRNA or anti-genome levels was calculated by dividing the ΔCt value of siRNA treated sample with ΔCt value of NT siRNA-treated sample.

Semiquantitative RT-PCR. VSV N mRNA and anti-genome levels were examined as described in our previous publication (3).

Cell Viability Measurement. Cell viability was measured by CellTiter Glo assay (Promega). Briefly, 30,000 cells were plated per well in a clear bottom black wall 96-well plate. Cells were treated with the indicated concentrations of the drugs in 100 μL of volume and further incubated for the appropriate length of time. One hundred microliters of CellTiter Glo reagent was added to the wells and incubated at room temperature for 10 min. Readings were taken by using a luminometer.

HIV Pseudotype Experiment. For this experiment, pHIV NL4-3-based, GFP-expressing reporter construct (8) was used. To generate virus stock, 293T cells were cotransfected with an envelope (VSV-G or HIV NL4-3) expressing plasmid, along with an Env-deficient, pHIV NL4-3. HeLa or TZM-bl cells were reverse transfected with siRNAs against COPZ1, COPG, or ATP6V0B. For COPZ1 and COPG transfection, cells were incubated for 28–30 h before infection. For ATP6V0B, cells were incubated for 52 h before infection. The siRNA transfected cells were infected with 1.0×10^5 $TCID_{50}$ VSV-G pseudotyped (HeLa) or NL4-3 env packaged (TZM-bl) virus stock, and GFP-positive cells were measured by flow cytometry (BD FACScan) at 40 hpi. To enhance the infectivity with the NL4-3 env packaged HIV, TZM-bl cells were exposed to DEAE dextran (33.3 μg/mL) simultaneously with virus for 3.5 h. Cells were washed two times with PBS and supplemented with DMEM plus 10% FBS and incubated for 37 h more.

Protein–Protein Interaction Network. Protein–protein interaction network data were downloaded from the STRING Database (9) and manually validated for each interaction. Each network was represented by using the Cytoscape suite (10) as follows: the focal protein for each network (COPA, MAT2A, SCL46A1, and ADAL) and their respective subunits were color-coded and aligned hori-

zontally. Network visualizations were redrawn by centering to these focal proteins and their subunits and to preserve the topologies of most other highly interconnected subnetworks to the highest possible extent.

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2. Zhang L, et al. (2005) Infection of ciliated cells by human parainfluenza virus type 3 in an in vitro model of human airway epithelium. *J Virol* 79:1113–1124.
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9. Szklarczyk D, et al. (2011) The STRING database in 2011: Functional interaction networks of proteins, globally integrated and scored. *Nucleic Acids Res* 39(Database issue):D561–D568.
10. Smoot ME, Ono K, Ruscheinski J, Wang PL, Ideker T (2011) Cytoscape 2.8: New features for data integration and network visualization. *Bioinformatics* 27:431–432.

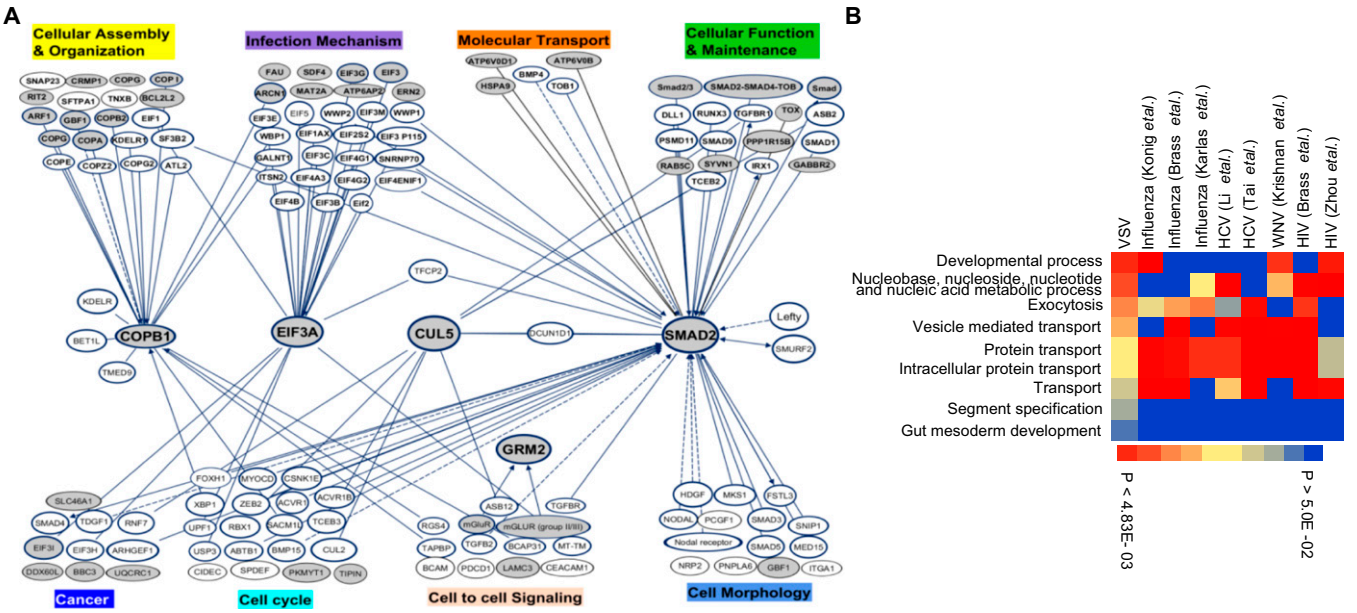

Fig. S1. Computational pathway analysis of identified factors. (*A*) Ingenuity pathway analysis (IPA; Ingenuity Systems) identified 8 top biological functional annotations associated with the 173 identified candidates. Integration of these functions is presented in the form of relationships as direct (solid lines) and indirect (discontinuous lines) interactions with arrows indicating the direction of the underlying relationship when appropriate. The nodes with gray background represent molecules identified in our screen, and those with white background are the molecules forecasted by IPA knowledge base. (*B*) Biological processes enriched among the 72 genes identified in the validation screen for VSV were compared with those identified for other viruses (1–8). Hit lists were accessed from these published articles and subjected to the *Panther* biological classification analysis. The color intensities represent degree of requirement by the indicated virus. Red and gray represent high degree of dependency. Blue represents little or no dependency on the indicated pathway or process.

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2. Brass AL, et al. (2009) The IFITM proteins mediate cellular resistance to influenza A H1N1 virus, West Nile virus, and dengue virus. *Cell* 139:1243–1254.
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4. Li Q, et al. (2009) A genome-wide genetic screen for host factors required for hepatitis C virus propagation. *Proc Natl Acad Sci USA* 106:16410–16415.
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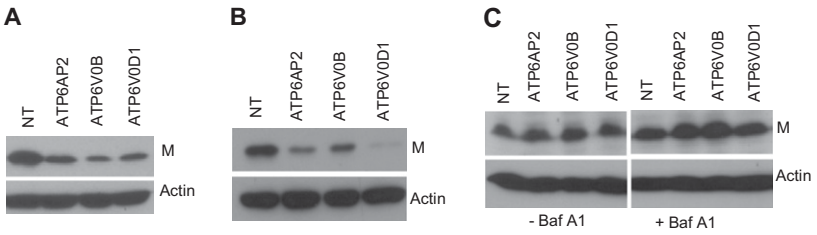

Fig. S2. vATPase is required for VSV infection. (*A*) Cells were transfected with pool of four siRNAs for indicated vATPase subunits for 72 h and infected with VSV (MOI = 0.01) for 14 h. Cell extracts were examined for VSV gene expression (viral M protein) by immunoblotting. Actin is shown for loading control. (*B*) HeLa cells were transfected with the indicated siRNAs and then infected with VSVΔG virus. M protein expression was examined at 5 hpi. (*C*) Cells transfected as above were treated with (+) or without (–) bafilomycin A1 (Baf A1) for 30 min and then transfected with VSV NC. VSV gene expression (M protein) was examined by immunoblotting at 6 h after NC transfection.

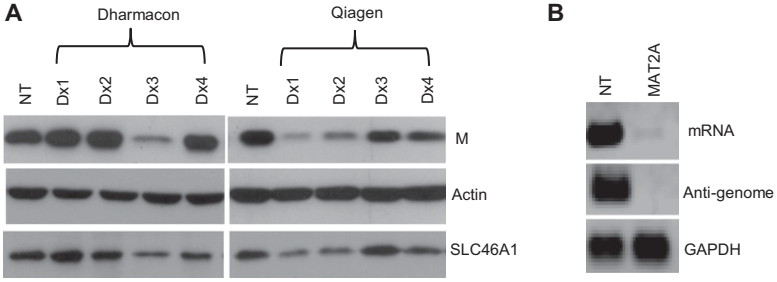

Fig. S3. SLC46A1 and MAT2A are required for VSV infection. (*A*) HeLa cells transfected with individual siRNA duplexes targeting SLC46A1 were subsequently infected with 0.005 MOI of VSV for 14 h. Cell lysates were collected and the viral M protein, actin, and SLC46A1 were examined by immunoblotting. (*B*) HeLa cells were transfected with pool of four siRNAs targeting MAT2A and at 48 hpt were infected with VSV. At 4 hpi, levels of mRNA and antigenomic RNAs were determined as described (1). Levels of glyceraldehyde-3-phosphate dehydrogenase (GAPDH) was shown as internal control.

1. Dinh PX, Beura LK, Panda D, Das A, Pattnaik AK (2011) Antagonistic effects of cellular poly(C) binding proteins on vesicular stomatitis virus gene expression. *J Virol* 85:9459–9471.

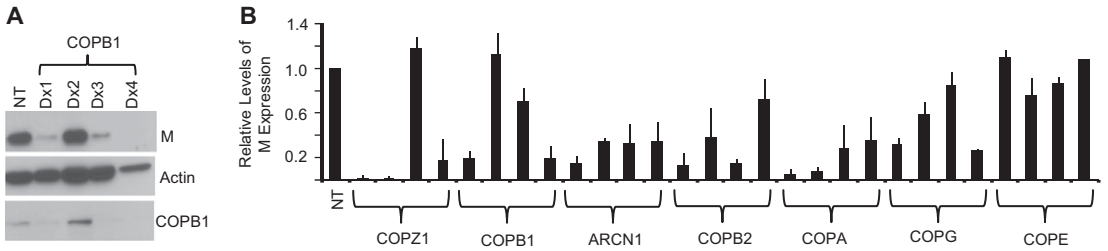

Fig. S4. Effect of depletion of COPI subunits on VSV gene expression. (*A*) HeLa cells transfected with individual siRNA duplexes targeting COPB1 subunit and subsequently were infected with VSV (MOI = 0.5) for 4 h. VSV gene expression was assessed by immunoblotting for M. Depletion of COPB1 was examined by Western blotting. (*B*) HeLa cells transfected with individual siRNA duplexes (four per gene) targeting each of the seven COPI subunits were infected with VSV (MOI = 0.5) for 4 h. VSV gene expression was assessed by immunoblotting for M. Relative levels of M (mean ± SD) from three experiments are presented.

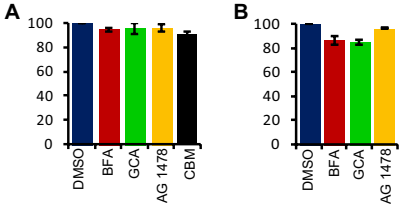

Fig. S5. Cell viability after drug treatment. (*A*) HeLa cells plated in 96-well plate were treated with Brefeldin A (BFA; 5 μg/mL), Golgicide A (10 μM), or AG-1478 (30 μM) for 6 h or CBM (2 mM) for 4 h. Cell viability was measured by CellTiter-Glo assay. (*B*) Cells plated as described in *A* were treated with BFA (200 ng/mL), Golgicide A (5 μM), or AG-1478 (20 μM) for 14 h. Cell viability was measured as described in *A*.

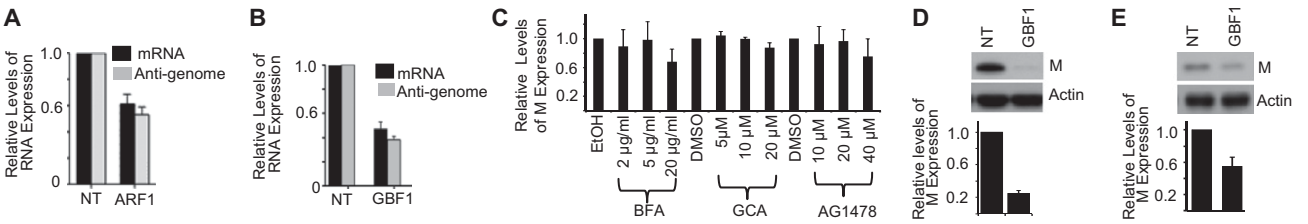

Fig. S6. Disruption of ARF1 and GBF1 functions affects VSV RNA synthesis. (*A* and *B*) HeLa cells were treated with pool of four siRNAs for ARF1 (*A*) or GBF1 (*B*) and then infected with VSV. VSV-P mRNA or anti-genome levels were examined by qRT-PCR. Values show mean ± SE of measurement (SEM) of duplicate reactions from two experiments after normalizing to NT control. (*C*) MDCK cells infected with 1 MOI of VSV were treated with indicated concentrations of the drugs at 1 hpi. Cell lysates were prepared at 4 hpi, and M and actin levels were assessed by immunoblotting. Relative levels of M (mean ± SD) compared with vehicle controls from three experiments are presented. (*D* and *E*) HeLa cells depleted of GBF1 protein by pool of four siRNAs were infected with VSVΔG virus (*D*) or transfected with NCs (*E*). M protein expression was examined at 5 hpi (*D*) or 6 hpt (*E*).

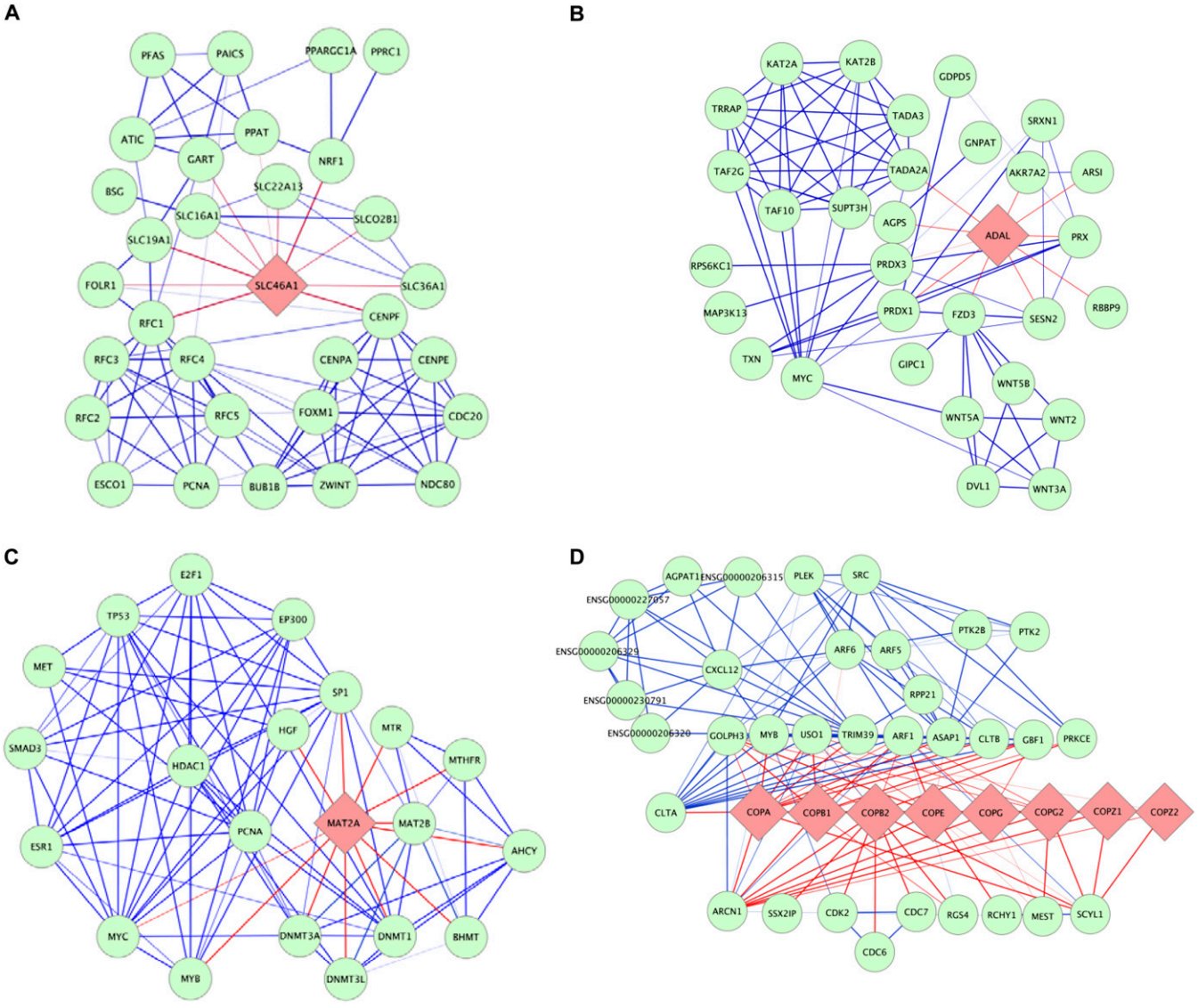

Fig. S7. Protein-protein interaction networks. The protein–protein interaction network for solute carrier family 46, member 1 protein (SLC46A1; proton-coupled folate transporter, PCFT) (*A*), ADAL (*B*), MAT2A (*C*), and COPI (*D*). Edges (interactions) are indicated by red colors. Only the proteins directly interacting with SLC46A1, ADAL, MAT2A, and COPI and their directly interacting proteins are shown. Interactions and annotations were obtained from the STRING Database (1) and from GeneCards (2), and network visualizations were created using Cytoscape (3).

1. Szklarczyk D, et al. (2011) The STRING database in 2011: Functional interaction networks of proteins, globally integrated and scored. *Nucleic Acids Res* 39(Database issue):D561–D568.
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3. Smoot ME, Ono K, Ruscheinski J, Wang PL, Ideker T (2011) Cytoscape 2.8: New features for data integration and network visualization. *Bioinformatics* 27:431–432.

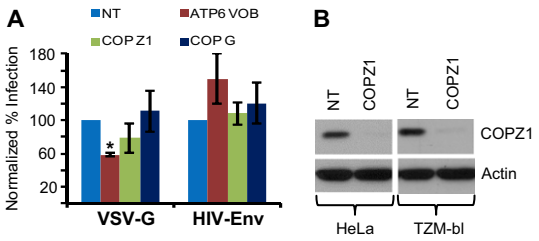

Fig. S8. Depletion of COPI does not affect VSV-G mediated HIV entry. (*A*) COPZ1, COPG, or ATP6V0B siRNAs transfected HeLa cells or TZM-bl cells were infected with VSV-G pseudotyped HIV or NL4-3 env packaged virus, respectively. GFP expressing cells were measured by flow cytometry at 40 hpi. Asterisk indicates statistically significant difference ($P = 0.0001$). (*B*) Depletion of COPZ1 protein was examined by immunoblotting after 24–26 h post siRNA transfection in HeLa and TZM-bl cells.

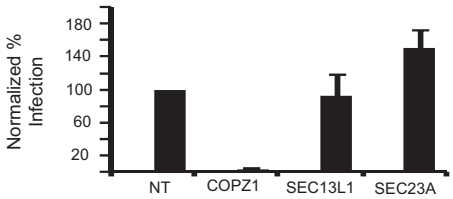

Fig. S9. Transfection of COPII siRNA does not affect VSV infection. HeLa cells were transfected with siRNAs for SEC13L1 and SEC23A (two of the COPII subunits) or against COPZ1. At 48 hpt, cells were infected with VSV (MOI = 0.01), and normalized percent infection relative to NT siRNA at 16 hpi was determined.

Table S1. Primer sequences

Primer	Sequence
VSV 2795F	GTGACGGACGAATGTCTCATAA
VSV2860R	TTTGACTCTCGCCTGATTGTAC
VSV2955R	TGATGAATGGATTGGGATAACA
β-Actin-F	CAAGTACTCCGTGTGTGGAT
β-Actin-R	CATACTCCTGCTTGCTGAT
VSV 2825 probe	6-FAM/CCATCCTGCTCGGCCTGAGATAC/TAMRA
β-Actin probe	Cy5/TCGCTGTCCACCTTCCAGCAGAT/BHQ

Table S2. Primer and probe concentrations

Primer	Primer concentrations, nM	Probe	Probe concentrations, nM
VSV P mRNA			
VSV 2795-F	500	VSV 2825	200
VSV 2860-R	500		
VSV anti-genome			
VSV 2795-F	1,000	VSV 2825	200
VSV 2955-R	1,000		
β-Actin			
β-Actin-F	500	βActin	1,000
β-Actin-R	500		

Dataset S1. List of genes identified in the primary screen for VSV

Dataset S1

Dataset S2. List of genes identified for VSV, VSV/LCMV/HPIV3, VSV/LCMV, or VSV/HPIV3

Dataset S2

Dataset S3. Known and putative biological functions and subcellular localization of genes required for VSV

Dataset S3