Supporting Information

Reiling et al. 10.1073/pnas.1018098108

SI Materials and Methods

Molecular Biology and Plasmids. The construction of gene trap (GT) vectors, mapping of GT integration sites, RT-PCR protocol to determine major facilitator domain containing 2A (MFSD2A) expression in the GT lines, and general screening approach are described in detail by Carette et al. (1).

MFSD2A cDNA was obtained by PCR amplification using primers with overhanging restriction sites (oJR261/SalI and oJR262/NotI) and a HEK293T cDNA pool as a DNA source. A PCR band of the correct length was TOPO TA (Invitrogen) subcloned, sequence-verified, and inserted as SalI/NotI fragment into SalI/NotI-digested lentiviral plasmids pLJM60 (puromycin resistant) or pLJM61 (neomycin resistant). This led to the generation of an N-terminally *Flag*-tagged *MFSD2A* cDNA construct used for lentivirus production. Flag-MFSD2AΔC and Flag-MFSD2AKKRQ [endoplasmic reticulum (ER) retention signal] mutant constructs were obtained with Flag-MFSD2A in pLJM61 as the DNA template and the following primers: Flag-MFSD2AΔC3: oJR261/SalI and oJR278/NotI; Flag-MFSD2AΔC10: oJR261/SalI and oJR323/NotI; Flag-MFSD2AΔC23: oJR261/SalI and oJR322/NotI; Flag-MFSD2AΔC40: oJR261/SalI and oJR321/NotI; Flag-MFSD2AKKRQ: oJR261/SalI and oJR314/NotI. For generation of the *N*-glycosylation site-deficient MFSD2A form (Flag-MFSD2AΔ*N*-glyco), the QuikChange site-directed mutagenesis kit (Agilent) was used according to the supplier's instructions using primers oJR352 and oJR353 and Flag-MFSD2A as the template. The R90A, D93A, D97A, and K436A MFSD2A mutant constructs were generated through site-directed mutagenesis using Flag-MFSD2A (WT) as the DNA template for the PCR reaction and the following primers: oJR374 and oJR375 (R90A), oJR368 and oJR369 (D93A), oJR370 and oJR371 (D97A), and oJR3776 and oJR377 (K436A). An EGFP-MFSD2A plasmid was made by (*i*) PCR-amplifying MFSD2A using Flag-MFSD2A in pLJM61 as the DNA template with primers oJR279/SalI and oJR280/BamHI, TOPO TA subcloning, digestion with SalI/BamHI, and insertion into pEGFP-C1 (Clontech); (*ii*) another PCR with oJR300/AgeI and oJR301/XbaI primers using *EGFP-MFSD2A* in pEGFP-C1 as the DNA template yielded the *EGFP-MFSD2A* cDNA that was cloned as an AgeI/XbaI fragment into digested lentiviral plasmid pLJM13. The entire EGFP-MFSD2A coding region was sequence-verified. γ-Tubulin and Rap2a in prk5 have been previously described (2). The cDNAs were excised as SalI/NotI fragments and cloned into pLJM60 or pLJM61. All constructs were checked for mutations by sequencing. An *MFSD1* cDNA clone was ordered from Open Biosystems and PCR-amplified with primers oJR350/SalI and oJR351/NotI before integration into pLJM60 and pLJM61 plasmids. *UDP-GlcNAc:dolichol phosphate GlcNAc-1-phosphate transferase* (*DPAGT1*) was cloned out of a HEK293T cDNA pool using oJR276/SalI and oJR277/NotI primers. After TOPO subcloning and sequence verification, a SalI/NotI-*DPAGT1* fragment was ligated into pLJM60/61.

Cell Lines and Tissue Culture. KBM7 were grown in Iscove's modified Dulbecco's medium (IMDM) with 10% heat-inactivated fetal serum (IFS) and antibiotics. All other cell lines with the exception of Jurkat T cells, which were grown in RPMI with 10% FBS, were cultured in DMEM supplemented with 10% IFS, 250 units/mL penicillin, and 250 μg/mL streptomycin.

Antibodies and Reagents. The following antibodies were used in this study: rabbit anti-GRP78 and goat anti-GRP94 (Santa Cruz); mouse anti–α tubulin (Sigma); rabbit anti-Flag, rabbit anti–inositol-requiring enzyme-1α (IRE1α), mouse anti-CHOP, rabbit anti-Raptor, rabbit anti-phospho eIF2α, rabbit anti-PDI, and rabbit anti-Ero1-Lα (Cell Signal Technologies). Rabbit anti-class I MHC serum (HC70) and mouse anti-W6/32 were described previously (3). Tunicamycin, thapsigargin, brefeldin A, etoposide, DTT, 2-deoxyglucose, Phloretin, and Chlorpromazine hydrochloride were purchased from Sigma-Aldrich; 16% paraformaldehyde solution (PFA) was obtained from Electron Microscopy.

Virus Production. HEK293T cells (800,000) were seeded into 6-cm culture dishes the day before transfection; 0.1 μg VSV-G (envelope plasmid), 0.9 μg Δvpr (packaging plasmid), and 1 μg lentiviral plasmid were transfected into cells using TransIT-LT1 reagent (Mirus). The transfection mix was removed ∼12 h later and replaced with 6 mL harvesting media (DMEM plus 30% IFS, 250 units/mL penicillin, 250 μg/mL streptomycin). Culture supernatants containing lentiviral particles were harvested 48 h after transfection. Lentiviral infections of target cells were done in the presence of 8 μg/mL polybrene, and cells were selected with the appropriate antibiotic 24 h after infection.

PNGAse F Treatment. A 40-μL slurry of Flag/Agarose beads was washed three times in lysis buffer. For immunopurification (IP), 500 μg protein lysate were combined with Flag beads and immunoprecipitated for 2.5 h at 4 °C. After three washing steps in lysis buffer, liquid was completely aspirated, and 35 μL denaturating buffer (New England Biolabs) were added to the bead pellet. The samples were scratched on a tube rack several times and boiled for 5 min before the eluate was loaded onto a microspin chromatogram column and briefly centrifuged to remove the beads; 5 μL PNGase F (New England Biolabs) were added to a 15-μL sample and incubated for 1 h at 37 °C. The reaction was stopped by adding 5 μL 5× sample buffer to the sample mix before subjecting it to SDS/PAGE.

Quantitative Real-Time PCR. RNA was extracted using TRIzol (Invitrogen) or RNeasy kit (Qiagen) followed by on-column DNase I (Qiagen) digest; 1 μg total RNA and SuperScript first-strand synthesis system (Invitrogen) were used for the reverse-transcription reaction using Oligo dT primers. SYBR Green PCR Master Mix (Applied Biosystems) was used according to manufacturer's instructions. Real-time PCR reactions were run on an ABI 7900HT machine. PCR volume was 10 μL (384-well plate), and data values were derived from three biological replicates using the comparative Ct method.

shRNA/cDNA Cotransfection. Two million HEK293T cells were seeded into 10-cm tissue culture dishes 24 h before transfection of 400 ng shRNA plasmids and 250 ng tagged cDNA construct. The total amount of plasmid DNA in each transfection was brought to 2 μg with empty vector. Cells were lysed 48 h after transfection, and Flag immunopurification was performed as described.

Bright-Field Images. Pictures of live cells were taken with a Canon Power Shot 5 digital camera mounted on a Zeiss Axiovert 40 CFL microscope.

Liquid Chromatography Tandem MS. This liquid chromatography tandem MS (LC-MS) procedure was used for tunicamycin (TM) detection as described in Fig. S8. KBM7 (Fig. S8*A*), stable PANC1 (Fig. S8*B*), or A549 (Fig. S8*C*) cells expressing the

protein of interest were washed three times with ice-cold PBS before lysis in 200–300 μL 80% or 100% methanol. Samples were pipetted up and down several times, briefly vortexed, and left standing at −20° for 1 h before further storage at −80 °C. Measurement of TM for Fig. S8 was carried out using a QSTAR XL hybrid quardupole/time of flight mass spectrometer equipped with a TurboSpray source (AB SCIEX) that was coupled to an Agilent 1100 HPLC system (Agilent Technologies). Chromatographic separations were achieved using a 150 × 2.1 mm Atlantis T3 column (Waters) that was initially eluted isocratically at 200 μL/min with 70% mobile phase A (0.1% formic acid in aqueous 10 mM ammonium formate) and 30% mobile phase B (0.1% formic acid in acetonitrile) for 3 min followed by a 17-min linear gradient to 90% mobile phase B. MS data were acquired in the positive ion mode using time of flight scanning over *m/z* 800–900 with an accumulation time of 0.5 s. MS electrospray ionization source settings were as follows: gas1 was 70, gas2 was 60, curtain gas was 30, spray voltage was 5.0 kV, and source temperature was 450 °C. Before LC-MS analyses, cell extracts were concentrated 10-fold by drying 300 μL extract using a Turbovap LV nitrogen evaporation system (Caliper) and then resuspending the sample in 30 μL methanol; 4 μL sample were injected for each LC-MS analysis. MultiQuant software (version 1.1; AB SCIEX) was used for peak integration, and data were manually reviewed for quality of integration. TM signal intensities were normalized to cell number and averaged from three biological replicates.

Primer Sequences.

RTMFSD2A-1_F Ctgtgctgtcatcctgatcc
RTMFSD2A-1_R Caagacaaagttcccctcca
RTMFSD2A-2_F Gcctccatcatcctgtttgt
RTMFSD2A-2_R Atggaaacacgtgaccattg
RTACTB-1_F gctcgtcgtcgacaacggctc
RTACTB-1_R Caaacatgatctgggtcatcttctc
h36B4_forward: CAGCAAGTGGGAAGGTGTAATCC
h36B4_reverse CCCATTCTATCATCAACGGGTACAA
oMFSD2A_Esnault1_forward: CTCCTGGCCATCATGCTCTC
oMFSD2A_Esnault1_reverse: GGCCACCAAGATGAGAAA
DPAGT1_forward1: CTCTGTGGTCAGGACCTCAAC
DPAGT1_reverse1GGGGAATGCCTTACACTGCT
oJR261/SalI: gtcgaccATGGCCAAAGGAGAAGGCGCCGAGAGC
oJR262/NotI: gcggccgcCTAGAGGATGCTAGCCAGCTCTGTGG
oJR276/SalI: gtcgaccATGTGGGCCTTCTCGGAATTGC
oJR277/NotI: gcggccgcGACTCAGACATCATAGAAGAGTCG
oJR278/NotI: gcggccgcctaAGCCAGCTCTGTGGAGTCTG
oJR279/SalI: GTCGACATGGCCAAAGGAGAAGGCGCCGAGAGC
oJR281/BamHI: ggatccCTAGAGGATGCTAGCCAGCTCTGTGG
oJR300/AgeI: accggtATGGTGAGCAAGGGCGAGGAGC
oJR301/XbaI: tctagaCTAGAGGATGCTAGCCAGCTCTGTGG
oJR314/NotI: gcggccgcTCAttgacgcttcttGAGGATGCTAGCCAGCTCTGTGG
oJR321/NotI: gcggccgcctaTTTGAAGAGCAGCAGGCCCAGC
oJR322/NotI: gcggccgcctaCTGCAGGGCCTTCTTATTCTGC
oJR323/NotI: gcggccgcctaTTCTGAGCAGCCAGAGCTGC
oJR350/SalI: gtcgacaATGGAGGAGGAGGATGAGG
oJR351/NotI: gcggccgcTCATTCAGTATGGGAAAATTTTATTTCTTCC
oJR352: CCTTGTTTCCAGGACCTCcAgAGCTCTACAGTAGCTTCACAAAGTGCCcAaCATACACATGGCACCACC
oJR353: GGTGGTGCCATGTGTATGtTgGGCACTTTGTGAAGCTACTGTAGAGCTcTgGAGGTCCTGGAAACAAGG
oJR368: CCTGTTTGTGGGCCGAGCCTGGGcTGCCATCACAGACC
oJR369: GGTCTGTGATGGCAgCCCAGGCTCGGCCCACAAACAGG
oJR370: CCTGGGATGCCATCACAGcCCCCCTGGTGGGCCTCTGC
oJR371: GCAGAGGCCCACCAGGGGGgCTGTGATGGCATCCCAGG
oJR374: CCTGTTTGTGGGCGCAGCCTGGGATGCC
oJR375: GGCATCCCAGGCTGCGCCCACAAACAGG
oJR376: CCTTCTATGTCTTCTTCACCGCGTTTGCCTCTGGAGTGTCACTGG
oJR377: CCAGTGACACTCCAGAGGCAAACGCGGTGAAGAAGACATAGAAGG

1. Carette JE, et al. (2009) Haploid genetic screens in human cells identify host factors used by pathogens. *Science* 326:1231–1235.
2. Sancak Y, et al. (2008) The Rag GTPases bind raptor and mediate amino acid signaling to mTORC1. *Science* 320:1496–1501.
3. Lorenzo ME, Jung JU, Ploegh HL (2002) Kaposi's sarcoma-associated herpesvirus K3 utilizes the ubiquitin-proteasome system in routing class major histocompatibility complexes to late endocytic compartments. *J Virol* 76:5522–5531.

Fig. S1. Multiple-sequence alignment of MFSD2A and bacterial MFS transporters. Protein sequence alignment of human MFSD2A, MFSD2B, and bacterial sugar transporters. Melibiose permease (MelB; melibiose sodium symporter [*Escherichia coli* ATCC 8739]); NP_414804 or YagG [predicted galactose-pentose-hexuronide (GPH) transporter from *E. coli* str. K-12 substr. MG1655]; YP_324084 galactoside symporter from Anabaena variabilis ATCC 29413; YP_001865367 (also known as Npun_F1762) sugar (GPH) transporter from Nostoc punctiforme PCC 73102; YP_002462749 (also known as Cagg_1406) sugar (GPH) transporter from Chloroflexus aggregans DSM 9485; YP_002483054 (also known as Cyan7425_2335) sugar (GPH) transporter of Cyanothece sp. PCC 7425.

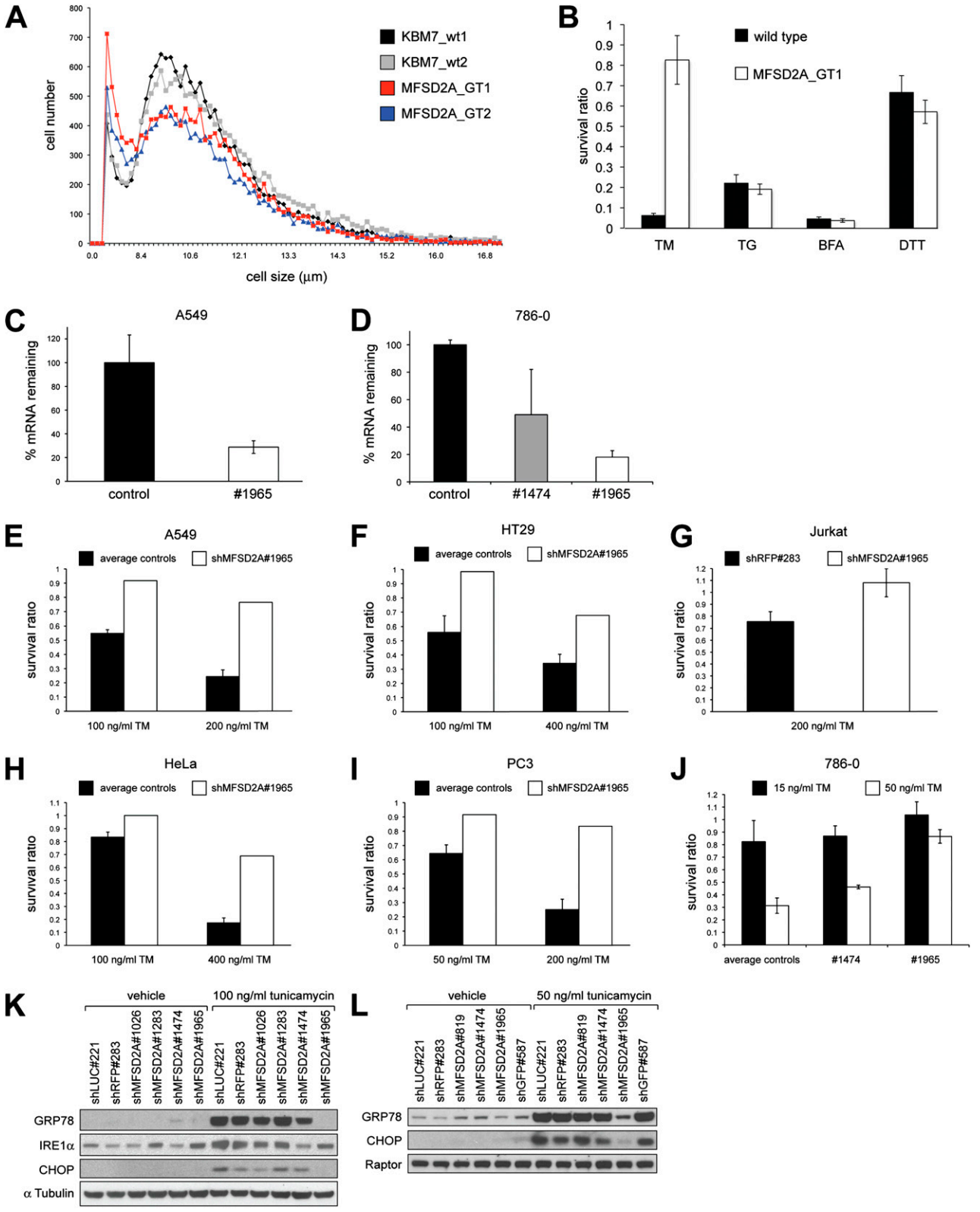

Fig. S2. MFSD2A knockdown (KD) in different cell types. (*A*) Cell size distribution of WT KBM7 or MFSD2A GT cells as determined with an automated Coulter Counter. (*B*) Treatment of WT KBM7 or MFSD2A GT cells for 3 d with various ER stress-inducing drugs. TM, 500 ng/mL tunicamycin; TG, 1.25 nM thapsigargin; BFA, 6.25 ng/mL brefeldin A; DTT, 2 mM DTT. MFSD2A KO cells are more resistant specifically to TM but not to other ER stressors compared with control cells. Viability was determined by the CellTiter Glo (CTG) assay (11 wells per genotype and conditions were analyzed). (*C* and *D*) Quantitative real-time PCR analysis of *MFSD2A* mRNA levels in A549 (*C*) or 786–0 (*D*) cells infected with shMFSD2A #1965 (denoted as #1965 in *C*, *D*, and *J*), shMFSD2A #1474 (denoted as #1474 in *D* and *J*), or control hairpins. shMFSD2A #1965-mediated MFSD2A KD in A549 and 786–0 cells showed a greater than 70% and 80% reduction of *MFSD2A* mRNA levels, respectively, whereas MFSD2A KD using shMFSD2A #1474 decreased transcript levels on average by 50% in 786–0 cells. *36B4* mRNA levels were used for normalization. (*E*–*J*) A number of cancer cell lines were infected with shMFSD2A #1965 that led to the strongest KD in A549 and 786–0 cells. MFSD2A

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KD and control cells (infected with lentiviruses carrying hairpins against luciferase, GFP, or RFP) were exposed to various TM concentrations for 3–6 d, and cell viability was assessed by either the CTG assay (*G*; Jurkat T cells) or direct cell number count using a particle counter device (all other cell lines). Jurkat cells (*G*) were seeded in 96-well plates, and bars represent the mean survival ratio ± SD of 11 wells for each condition. Representative results of multiple experiments are shown for A549, HT29, HeLa, and PC3 cells yielding qualitatively similar results. Two independent MFSD2A shRNAs (#1474 and #1965) were used for KD in 786–0 cells. Three wells per genotype and condition were counted for the 786–0 cells, and mean values ± SD are shown; $P < 0.05$ for both genotypes (50 ng/mL TM) using Student two-tailed *t* test (*J*). Survival ratio was determined by comparing TM-treated to untreated cells. Average controls refers to the mean ± SE of three different control hairpin survival ratios (duplicate samples for each hairpin). (*K*–*L*) Lentiviral shRNA-mediated MFSD2A KD in A549 (*K*) and 786–0 (*L*) cells and its effect on ER stress induction in response to TM. Cells infected with shMFSD2A #1965 and to a lesser extent, shMFSD2A #1474 mitigated ER stress marker induction in all cell lines tested. Cells were incubated with TM for 22 (A549) or 24 h (786-0).

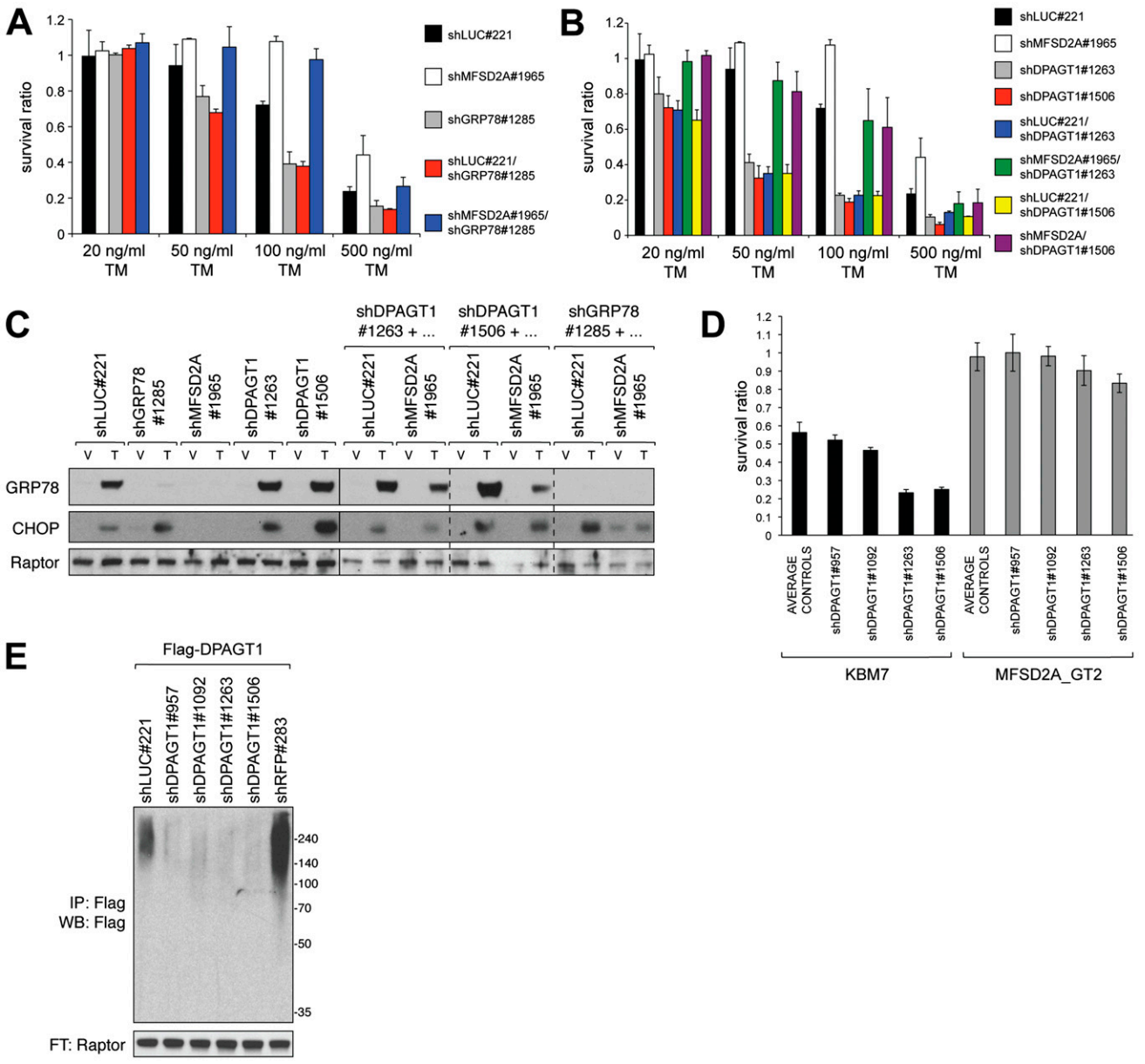

Fig. S3. Co-KD of MFSD2A/GRP78 and MFSD2A/DPAGT1 uncovers the single *MFSD2A* mutant phenotype. (*A* and *B*) A549 cells were transduced in a first round with lentiviruses bearing validated shRNAs against GRP78 (*A*) or DPAGT1 (*B*), and stable KD lines were obtained by antibiotic selection (see also *C* and *E* for KD validation). In a second round, shGRP78- and shDPAGT1-infected cells were reinfected with lentiviruses carrying either a control hairpin (shLUC#221) or shMFSD2A #1965. Double KD A549 cells were grown for 3 d without or with various TM concentrations, and cell survival was measured by scoring surviving cells as described previously. Average survival ratios ± SD of two independent experiments are presented. Infection efficiency for the second round of infection with shMFSD2A #1965 was ∼90%. Because the resistance marker for the second round of infection was the same as for the initial infection, the partial epistatic TM-resistance phenotype in the double KD lines might represent an underestimation of the strength of the MFSD2A single KD phenotype. (*C*) Western blot analysis of lysates of vehicle- or TM-treated A549 single and double KD lines described in *A* and *B*. Treatment duration was 24 h. V, vehicle; T, 100 ng/mL TM. (*D*) WT or MFSD2A_GT KBM7 cells were infected with four different DPAGT1 hairpins, and stable cell clones were established by puromycin selection. These DPAGT1 KD cells were grown for 88 h in 96-well plates in media containing 75 ng/mL TM. The CTG assay was used to determine the survival ratio calculated by dividing the mean luminescence values of 11 TM-treated wells by the corresponding untreated values for each genotype. (*E*) KD effectiveness of shDPAGT1 hairpins used in *B*–*D*. Flag-DPAGT1 and individual shDPAGT1 or control hairpin constructs were cotransfected into HEK293T cells and grown for 48 h before cell lysis and Flag immunopurification. Immunoblotting for Flag peptide was performed to assess DPAGT1 KD levels.

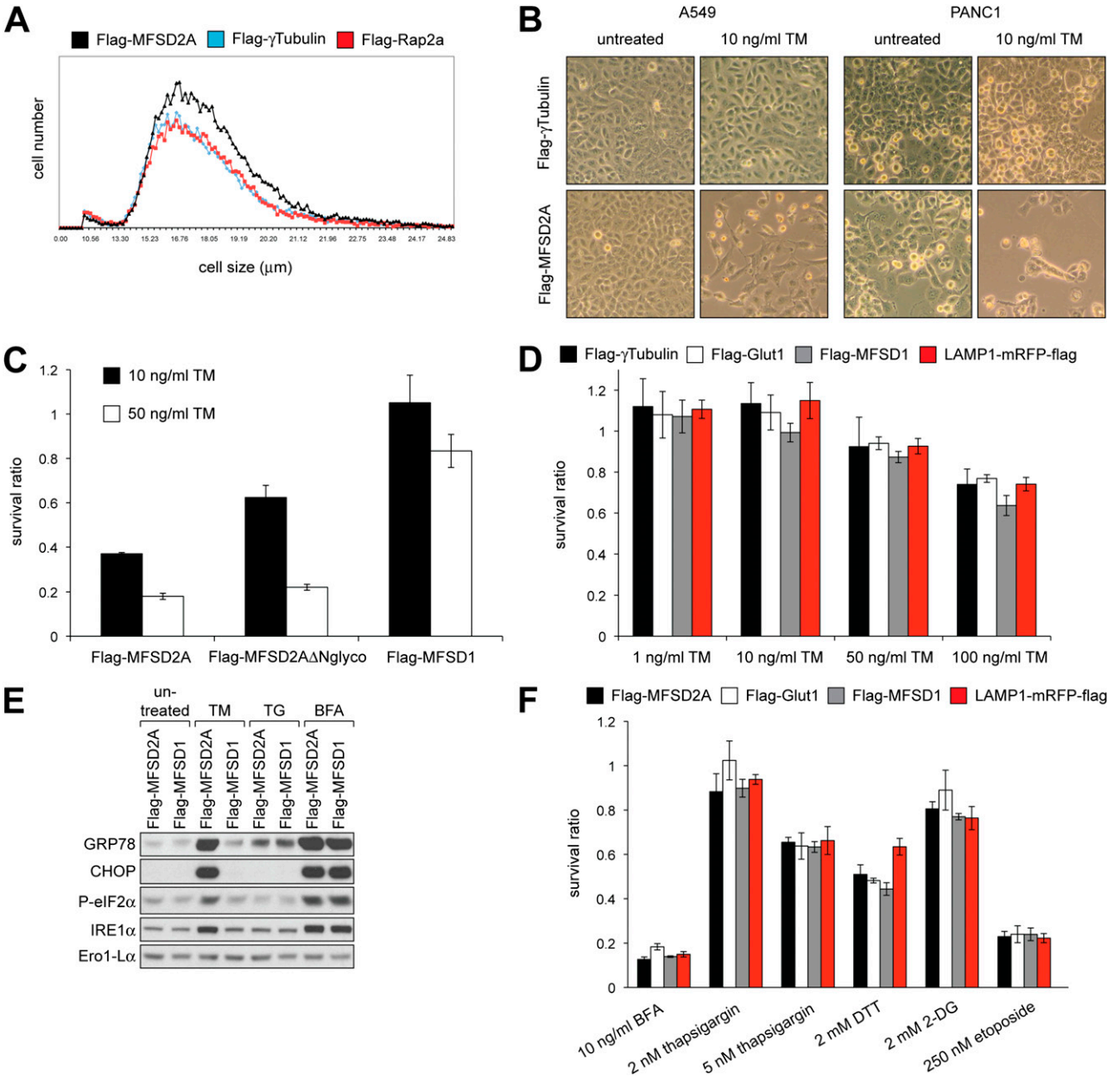

Fig. S4. Effects of MFSD2A overexpression on cell size and diverse drug treatments. (*A*) Stable A549 cells expressing the indicated proteins were seeded into 6-cm tissue culture dishes 24 h before cell size analysis. Cell size was measured using a Z2 Coulter Counter analyzer. Average size of Flag-MFSD2A–expressing cells is slightly bigger than control cells expressing γ-Tubulin or Rap2a, which is also indicated by the right shift of the curve ($P < 0.05$ using Student two-tailed *t* test). Three different plates for each genotype were seeded, and cell size data were analyzed in a pooled fashion for each genotype. Two independent experiments with similar results were performed. (*B*) Bright-field images of stable A549 or PANC1 cells overexpressing Flag-γ-Tubulin or Flag-MFSD2A and treated with TM for 3 (A549) or 5 d (PANC1). (*C*) Stable A549 cells expressing the indicated proteins were treated for 3 d with TM, and cell survival was determined with a Coulter Counter. Bars represent the mean ± SD of three replicates per genotype and condition. (*D*) A549 cells stably overexpressing various control proteins and treated with TM for 3 d have similar survival ratios. Three biological replicates of each genotype were counted with a Z2 Coulter Counter, and the mean ± SD is shown. (*E*) Comparison of ER stress marker induction between MFSD2A and MFSD1 overexpression in A549 cells treated with the indicated drugs. Treatment duration was 24 h, and the following concentrations were used: 10 ng/mL TM, 10 nM thapsigargin (TG), and 50 ng/mL BFA. (*F*) Stable A549 cells were exposed to various ER stress-inducing chemicals as well as the DNA damage-inducing drug etoposide for 3 d, and survival ratios were derived from Coulter Counter measurements. No differences in viability were observed between MFSD2A-overexpressing cells and control cells. Bars and error bars represent ratios of cell numbers of treated to untreated samples ± SD in triplicate studies.

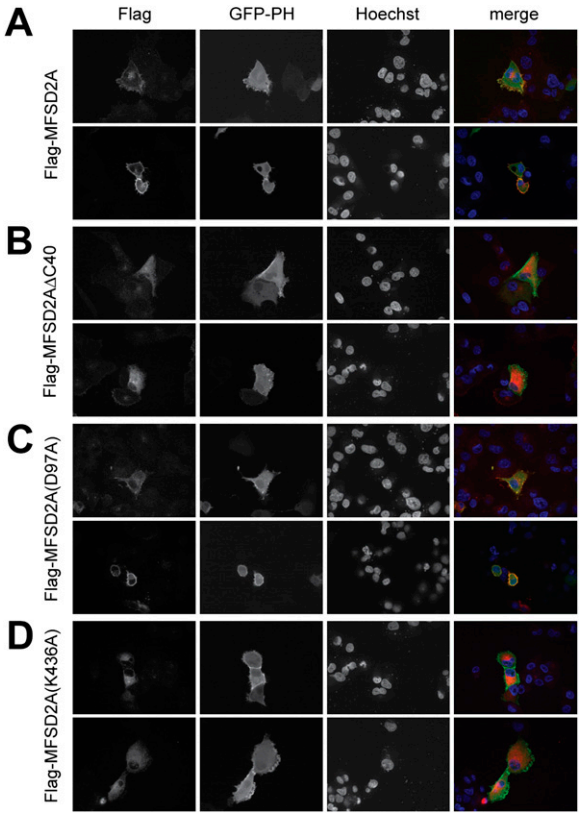

Fig. S5. MFSD2A localization in PANC1 cells. The indicated epitope-tagged MFSD2A constructs and the plasma membrane reporter GFP-PH were co-transfected into PANC1 cells. Cells were fixed 24 h after transfection and processed for immunofluorescence. Two independent examples of single optical sections of images obtained by confocal microscopy are presented. Additional details are in the text. (*A*) Flag-MFSD2A (WT), (*B*) Flag-MFSD2AΔC40, (*C*) Flag-MFSD2A(D97A), and (*D*) Flag-MFSD2A(K436A).

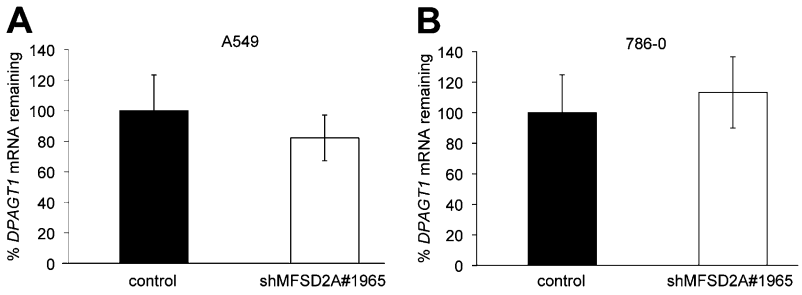

Fig. S6. No increase in *DPAGT1* mRNA levels in MFSD2A KD cells. Total mRNA isolated from A549 (*A*) or 786–0 (*B*) cells that were infected with control or shMFSD2A #1965 hairpins showed no statistically significant differences in *DPAGT1* transcript levels ($P = 0.34$ for *A* and $P = 0.53$ for *B* using Student two-tailed *t* test; experimental data derived from three biological replicates).

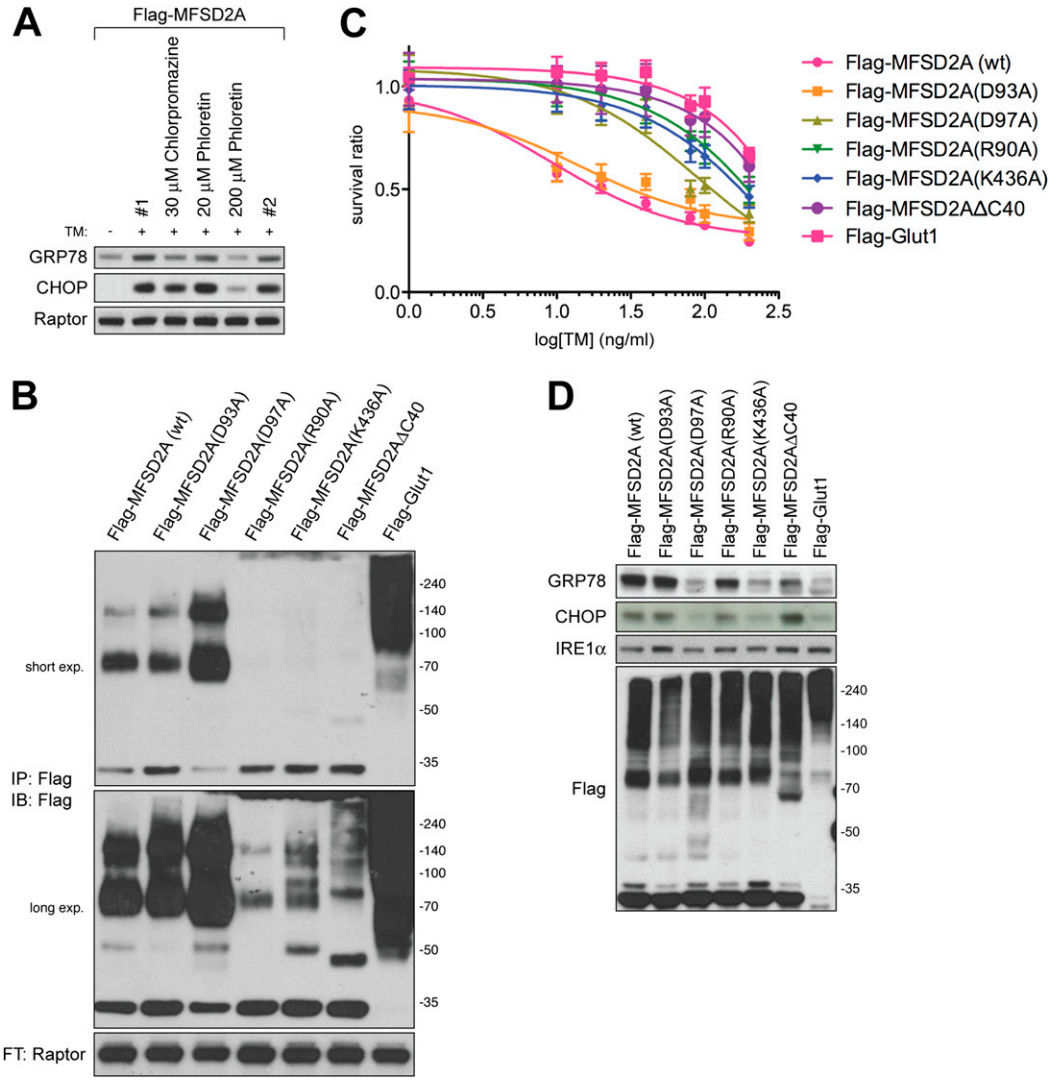

Fig. S7. Effects of alterations of cellular ion homeostasis and MFSD2A point mutations on TM sensitivity. (*A*) Flag-MFSD2A–overexpressing A549 cells were either left untreated (–) or cotreated for 6 h with 10 ng/mL TM (+) and Chlorpromazine (30 μM) or Phloretin (20 μM and 200 μM). #1 and #2 refer to TM-only treatments. (*B*) Expression levels of indicated MFSD2A mutants and control proteins (short and long blot exposure). The Flag-tagged proteins were immunopurified before SDS/PAGE. FT, flow through of lysate used for IP. (*C*) TM dose-response curves of individual A549 lines overexpressing different MFSD2A mutants or control proteins. Cells were treated with 1, 10, 20, 40, 80, 100, or 200 ng/mL TM for 3 d. Note that the R90A and K436A mutant lines showed reduced MFSD2A expression levels (*B*). (*D*) The indicated constructs were transfected into HEK293T cells and treated with 10 ng/mL TM for the last 22 h before lysis. MFSD2A(D97A)- and MFSD2A(K436)-expressing HEK293T cells do not up-regulate ER stress markers when treated with TM.

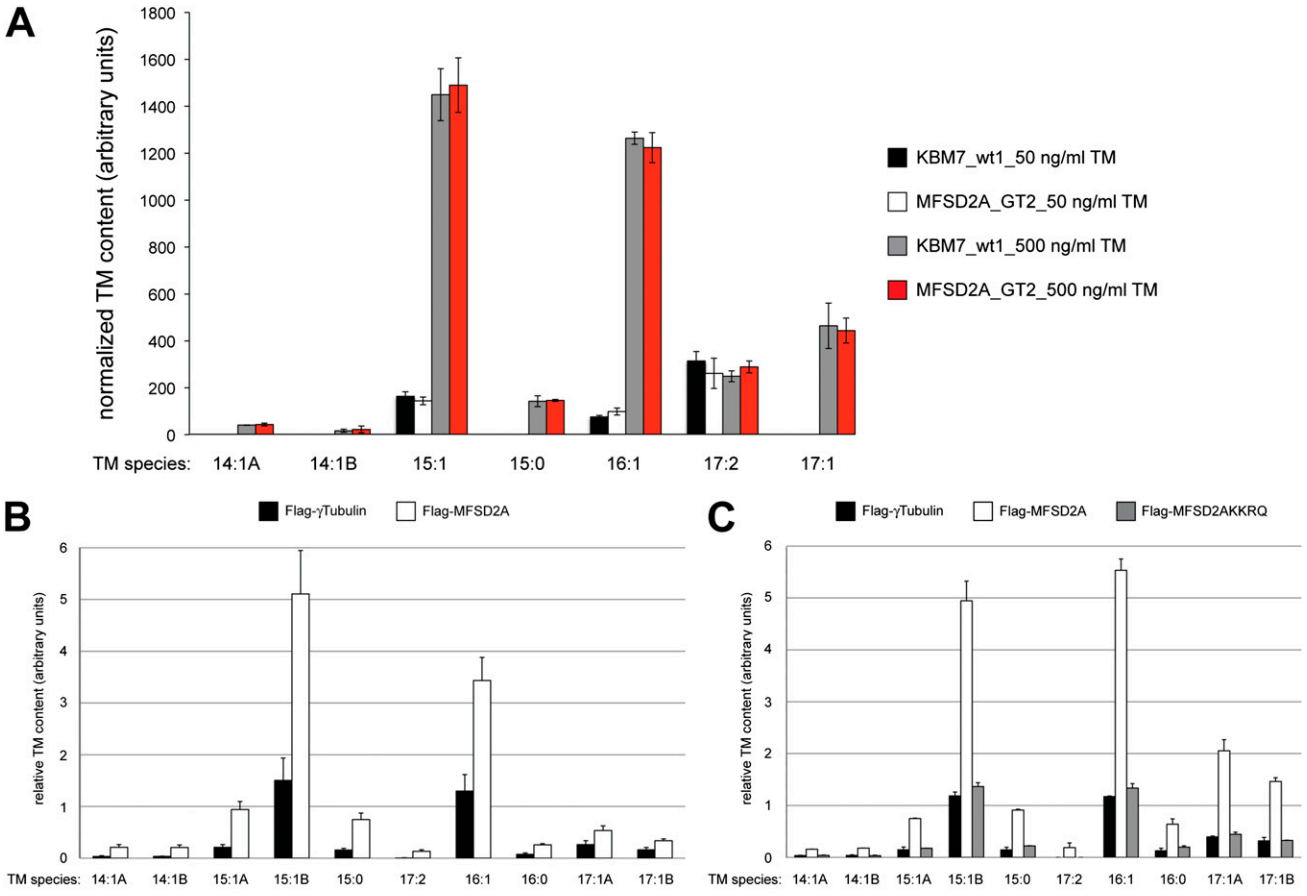

Fig. S8. Detection of TM by MS in MFSD2A GT or MFSD2A overexpression cells. (*A*) KBM7 WT or MFSD2A GT cells were incubated with 50 or 500 ng/mL TM for 22 h before lysis. No statistically significant difference in TM content could be detected. (*B* and *C*) Cells that overexpress MFSD2A show significantly enhanced TM uptake compared with control cells ($P < 0.05$ for stable A549 and $P < 0.03$ for PANC1 cell lines). Note that cells overexpressing ER-targeted MFSD2A (MFSD2AKKRQ) do not have increased TM content relative to control cells (*C*). (*B*) Stable PANC1 cells overexpressing MFSD2A or γ-Tubulin were treated for 7 h with 500 ng/mL TM before cell lysis and TM MS analysis. Sample processing and TM detection were done as described in *SI Materials and Methods*. (*C*) Stable A549 cell lines overexpressing WT MFSD2A, ER-directed MFSD2A, or γ-Tubulin were treated with 500 ng/mL TM for 24 h.

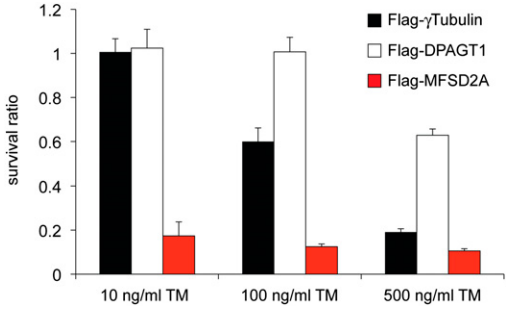

Fig. S9. DPAGT1 overexpression provides TM resistance. Stable A549 cell lines expressing Flag-DPAGT1, Flag-γ-Tubulin, or Flag-MFSD2A were treated for 3 d with TM, and surviving cells were scored with an automated Coulter Counter. Shown for illustration are the means ± SD of three replicates per genotype and condition.

Table S1. Hairpins

shRNA	NM number	TRC clone ID	Target sequence
shGFP#587	—	TRCN0000072186	TGCCCGACAACCACTACCTGA
shLUC#221	—	TRCN0000072246	CAAATCACAGAATCGTCGTAT
shRFP#283	—	TRCN0000072203	CGCGTGATGAACTTCGAGGAC
shMFSD2A#819	NM_032793.2–819s1c1	TRCN0000122696	GCTTCACAAAGTGCCAACCAT
shMFSD2A#1026	NM_032793.2–1026s1c1	TRCN0000139788	CACGGCCCATACATCAAACTT
shMFSD2A#1283	NM_032793.2–1283s1c1	TRCN0000143913	CATGGAGAGTAACCTCATCAT
shMFSD2A#1474	NM_032793.2–1474s1c1	TRCN0000140801	GAGTGTCACTGGGCATTTCTA
shMFSD2A#1965	NM_032793.2–1965s1c1	TRCN0000122777	CCACTGTGAATATGCCAAGGA
shDPAGT1#957	NM_001382.2–957s1c1	TRCN0000036023	CTGTACCAATGCCATCAATAT
shDPAGT1#1092	NM_001382.2–1092s1c1	TRCN0000036022	CCTCTACTTCATGATACCCTT
shDPAGT1#1263	NM_001382.2–1263s1c1	TRCN0000036019	CCAGGTGTTCAACTTCCTCTA
shDPAGT1#1506	NM_001382.2–1506s1c1	TRCN0000300800	CCTCATCAACTTGCTACTTAA
shGRP78#1285	NM_005347.x-1285s1c1	TRCN0000001024	CTTGTTGGTGGCTCGACTCGA

TRC, The RNAi Consortium.