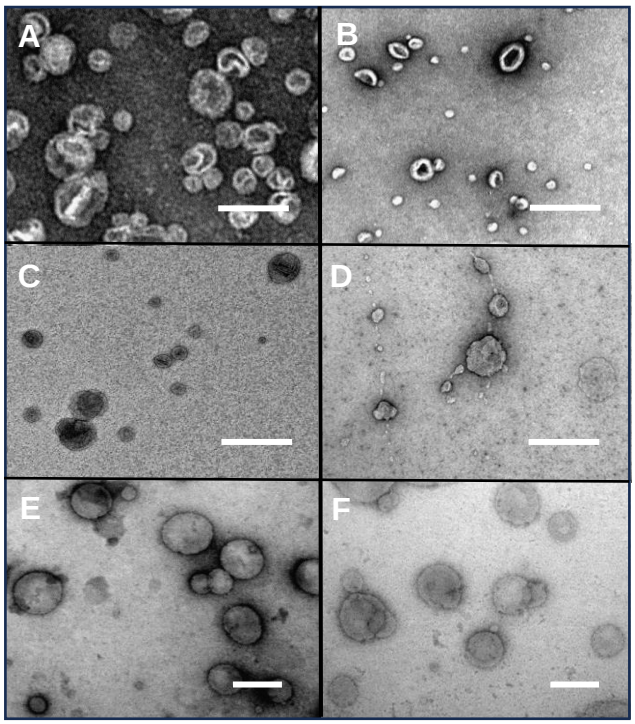


Figure S1. TEM images of Lipid vesicles. Small Unilamellar Vesicles (SUVs) POPC (A), POPS (B), POPA (C) and POPE (D). Large Unilamellar vesicles (LUVs) POPC (E) POPS (F). Scale bars: 100 nm. Negatively stained with Uranyl acetate (20 mg/mL). SUVs and LUVs are typically 50 and 100 nm in diameter, respectively.

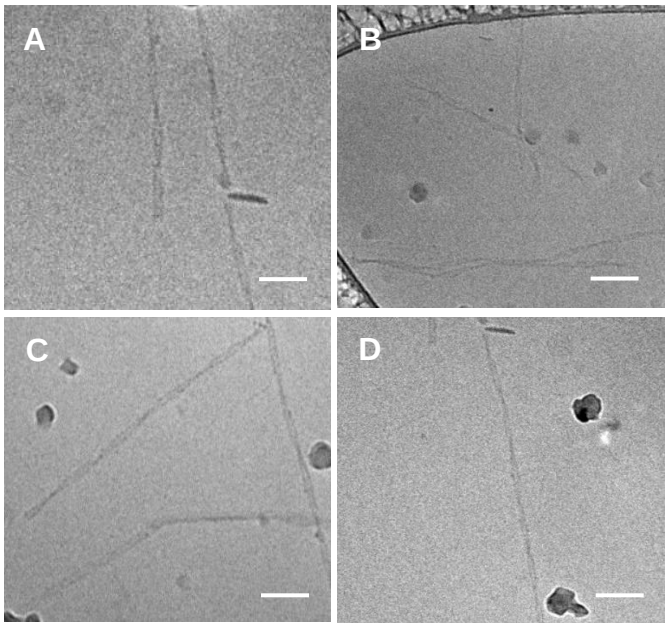


Figure S2. Cryo-EM images of Aβ40 (5 μM) fibrils formed in the absence (A,C) and presence (B,D) of POPS (0.2 mg/mL) SUVs vesicles in HEPES buffer (50 mM). Scale bars: 100 nm.

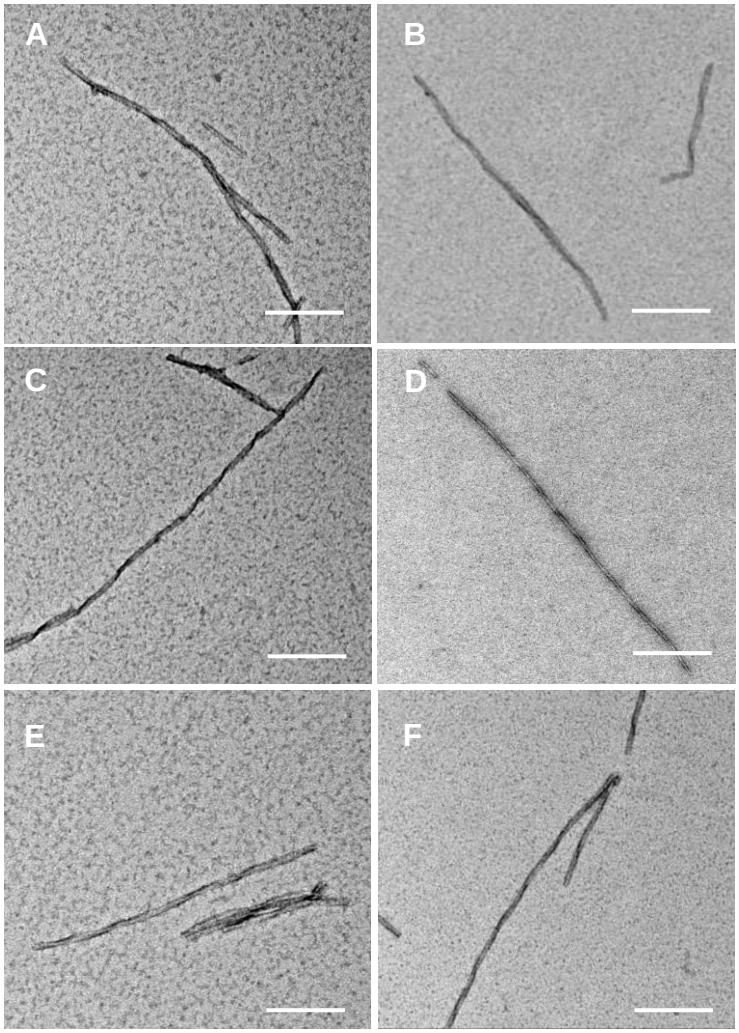


Figure S3. **Figure S3.** TEM images of Aβ40 (5 μM) fibril formed in the absence of lipid vesicles (A,C,E) or formed in the presence of POPC (B), POPS (D) and POPA (E) SUVs (0.2 mg/mL). Negatively stained with Uranyl acetate (2 mg mL-1, in HEPES buffer (50 mM). Scale bars: 200 nm.

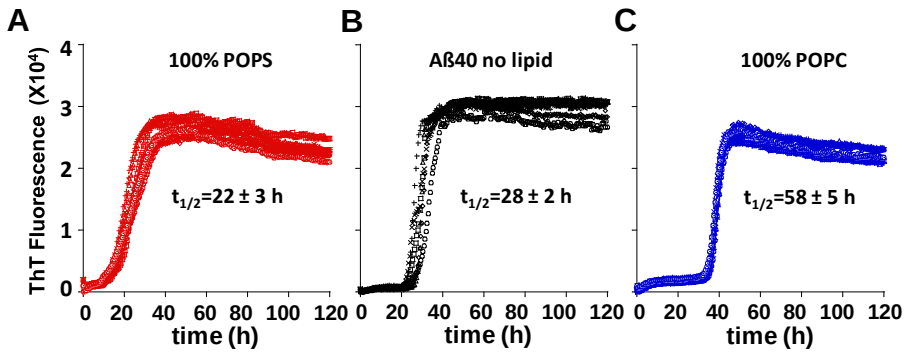


Figure S4. Impact of POPC and POPS vesicles (LUVs) on Aβ40 fibril formation. Kinetic traces of synthetic Aβ40 (5 μM) fibril growth in the presence of lipid vesicles (0.2 mg/mL) 100% POPS (red) and 100% POPC (blue) and without LUVs (black) in $NaHPO_4$ (50 mM), NaCl (50 mM) at 30°C pH 7.4, under quiescent conditions. N=6 traces for each condition. In the presence of NaCl, POPS accelerates lag-times and POPC slows lag-times.

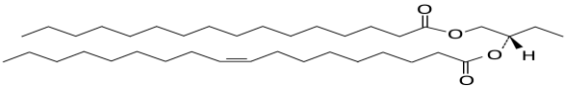
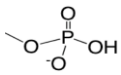
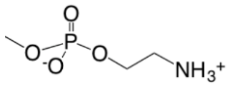
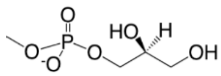
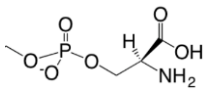
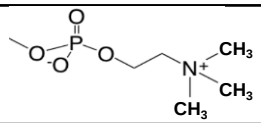
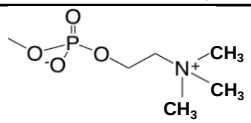
Acyl chain PO 16:0,18:1 				
Lipid	**Symbol**	**Headgroup Structure**	**Headgroup Mol. Wt.**	**Charge**
Phosphatidic Acid	PA		17	Anionic
Phosphatidyl Ethanolamine	PE		45	Neutral* (Anionic-like)
Phosphatidyl Glycerol	PG		75	Anionic
Phosphatidyl Serine	PS		87	Anionic
Phosphatidyl Choline	PC		87	Neutral
Sphingomyelin	SM		87	Neutral

Figure S5. Structure and properties of phospholipids. Structure of Phospholipid Acyl side chain PO 16:0, 18:1 (1-palmitoyl-2-oleoyl-sn-glycero-3-phospho-lipid). *PE has a neutral headgroup at pH 7.4 as an isolated phospholipid, but within the lipid bilayer it has more anionic properties.

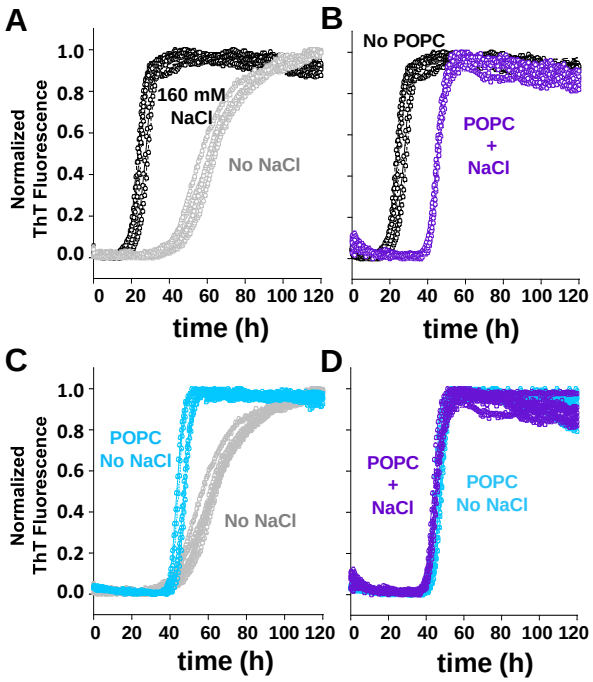


Figure S6. Impact of POPC lipid vesicles in the presence and absence of NaCl. Aβ40 (5 μM) with (black) and without (grey) 160 mM NaCl and with POPC LUVs (264 μM) in the absence (blue) and presence of NaCl (purple). In the presence of NaCl POPC slows fibril formation, relative to Aβ with NaCl only (B). While in the absence of NaCl, POPC accelerates fibril formation, relative to Aβ in the absence of NaCl (C). HEPEs buffer (50 mM) at 30˚C and pH 7.4, under quiescent conditions, n=6 kinetic traces.

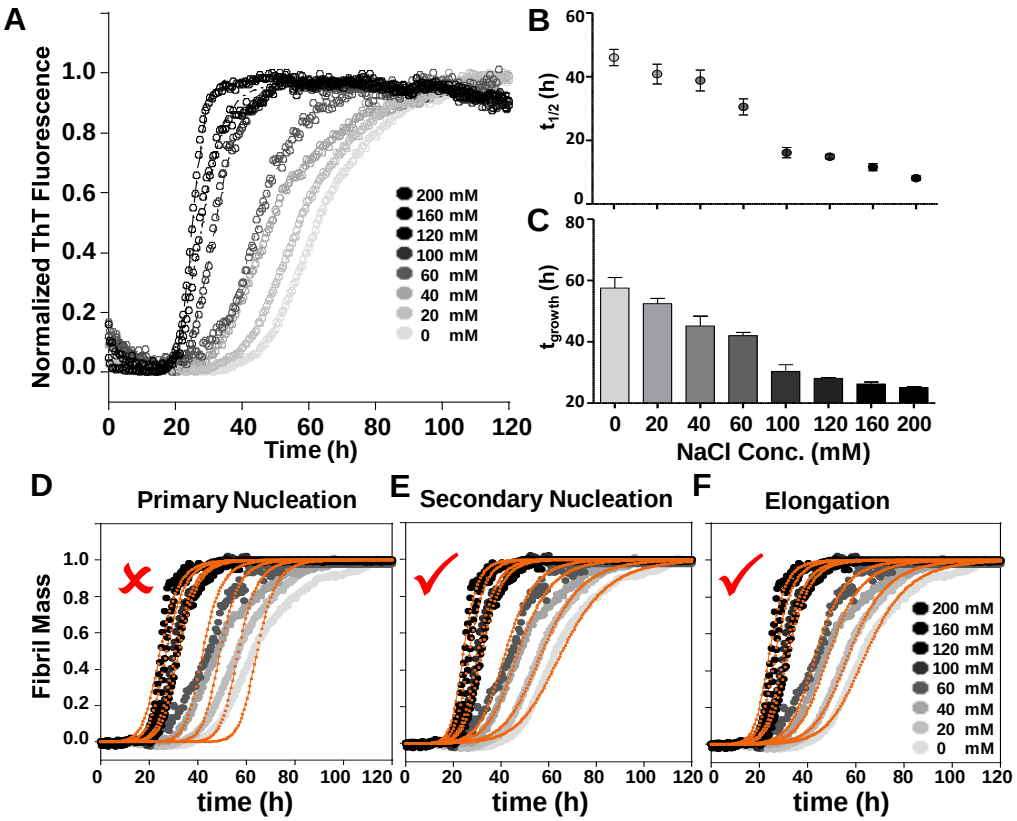


Figure S7. Impact of a range of NaCl concentration on Aβ fibril formation. Single representative kinetic profiles of Aβ40 (5 μM) in the presence of 200, 160, 100, 60, 40, 20, 0 mM NaCl, from black to pale grey, with no lipid present (A). Half-time $t_{1/2}$ (hrs) and growth time t_{growth} (hrs) for fibril formation. Error bars are standard deviation of the mean (B, C). Global fits of the kinetic traces when only primary nucleation (D), secondary nucleation (E) and fibril elongation (F) rate constants are altered to globally fit kinetic traces. In HEPEs buffer (50 mM) at 30°C and pH 7.4, under quiescent conditions.

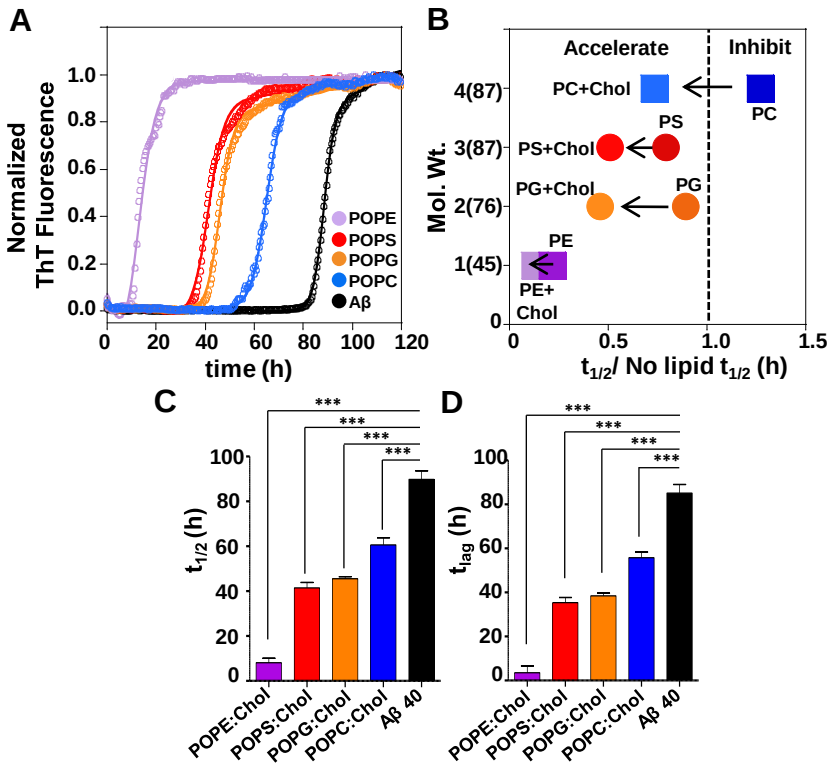


Figure S8. Cholesterol accelerates fibril growth by reducing lag-times in all phospholipids vesicles studied. Single representative kinetic profiles showing normalized ThT Fluorescence intensity from left POPE (purple), POPS (red), POPG (orange), POPC (blue) all with 30 % cholesterol, Aβ 40 (5 μM) with no lipid (black) (A). Relative $t_{1/2}$ *versus* Molecular Weight of Lipid headgroups. The dotted line represent $t_{1/2}$ for Aβ40 in the absence of lipid. Relative $t_{1/2}$ for the same four phospholipids but with no cholesterol in the vesicle is also indicated (in a darker shade) and highlights how the cholesterol accelerates lag-times in all lipid vesicles (B). Bar-graphs representing half time $t_{1/2}$ (C) and lag-time (D) Error bars represent standard deviation (SD) of the mean of seven replicates. One-way ANOVA test, ***P ≤ 0.001. HEPEs (50 mM), NaCl (160 mM) at 30˚C pH 7.4, under quiescent conditions.

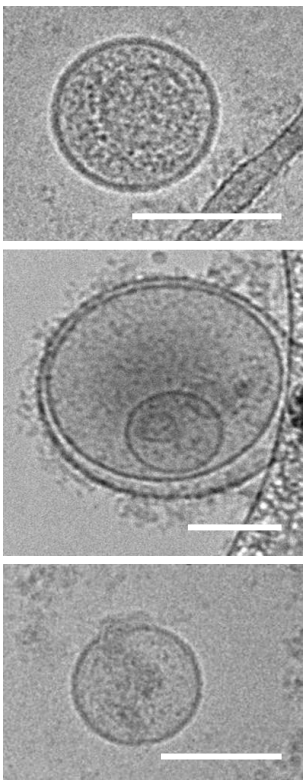


Figure S9. Cryo-EM images of Exosomes. Extracted from HEK-293 cells. Scale bars: 100 nm.

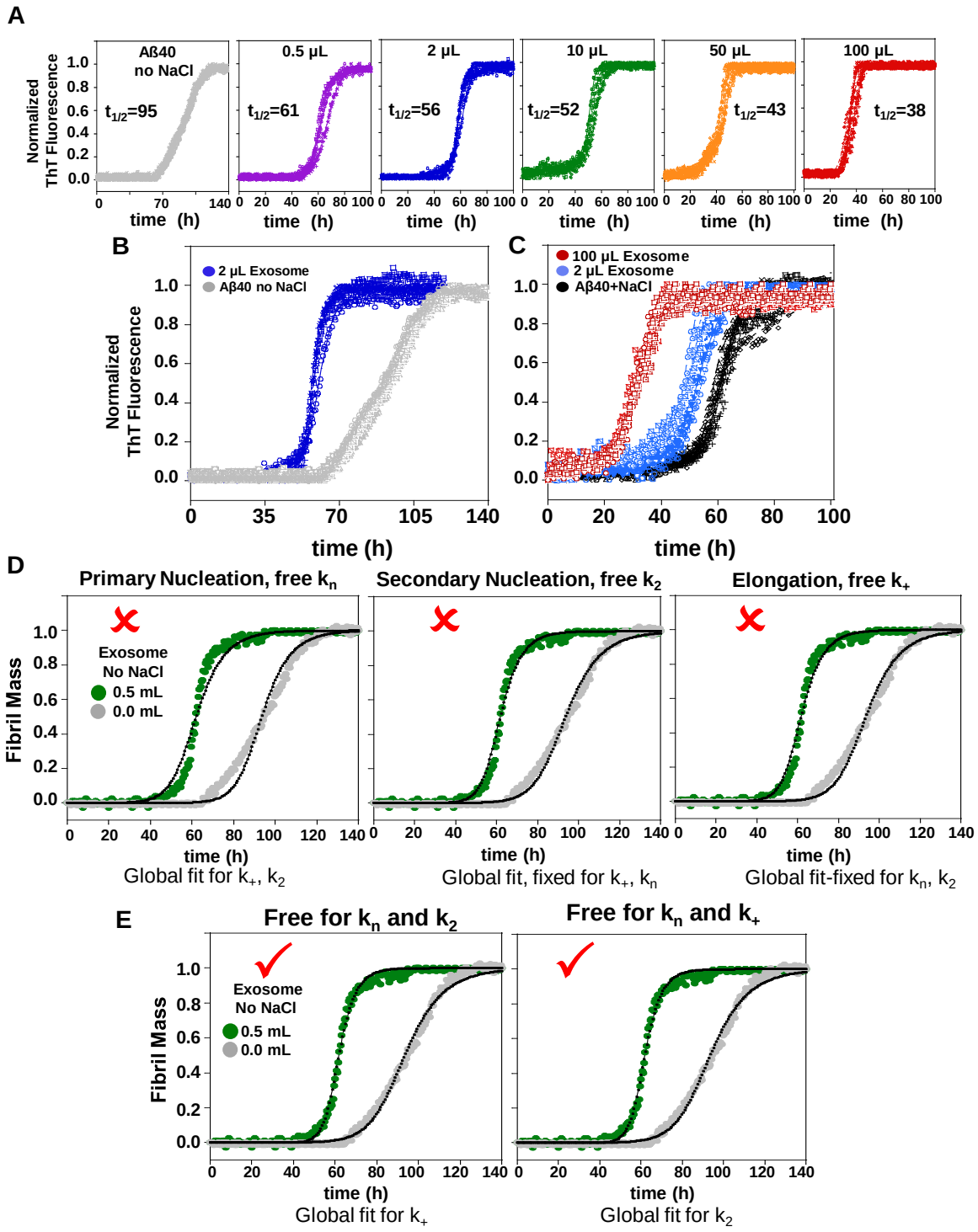


Figure S10. Aβ fibril formation in the presence of Exosomes. Kinetics traces of Aβ40 (5 µM) fibril growth in the presence increasing exosome levels: no lipid (grey); exosomes 0.5 µL (purple); 2 µL (blue); 10 µL (green); 50 µL (orange); 100 µL (red) with no NaCl, HEPEs buffer (50 mM) at 30°C, pH 7.4, under quiescent conditions. N=6 traces for each condition (A). Aβ40 (5 µM) kinetic traces showing normalized ThT fluorescence intensity in the presence of 2 µL exosome and 0 µL exosome concentration with no NaCl (B) and in the presence of 100 µL (red), 2 µL (blue) and 0 µL (black) exosome concentration with 160 mM NaCl, 50 mM HEPEs, at 30°C, pH 7.4, under quiescent conditions (C). Global fits to kinetic data for Aβ with and without exosomes, with only k_n, k_2 or k_+ free to vairy fits poorly to the data (D), exosomes impact both k_n and k_2 or k_+ (E).

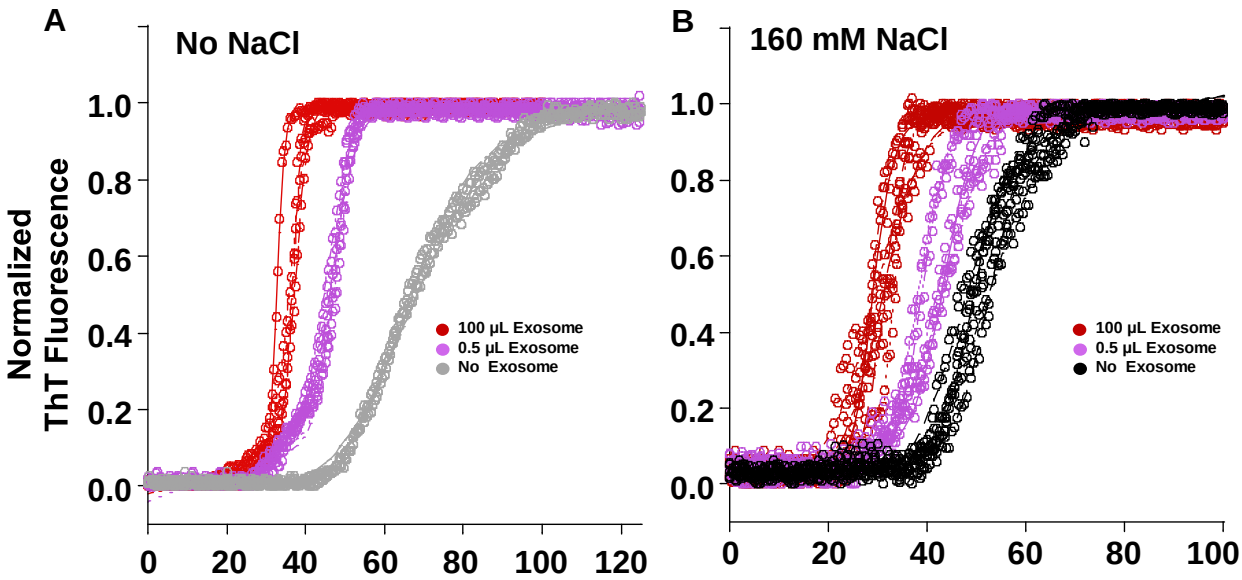


Figure S11. Aβ42 fibril formation in the presence of Exosomes. Aβ42 (5 µM) kinetic traces showing normalized ThT fluorescence intensity in the presence of 0.5 µL exosome (purple) and 100 µL exosome concentration (red). With no NaCl present (A) and with 160 mM NaCl (B). Exosomes will accelerate both the lag-time and the slope, while only a reduction in lag-times are apparent when NaCl is also present. N=5 traces for each condition. HEPES buffer (50 mM) at 30˚C, pH 7.4, under quiescent conditions.

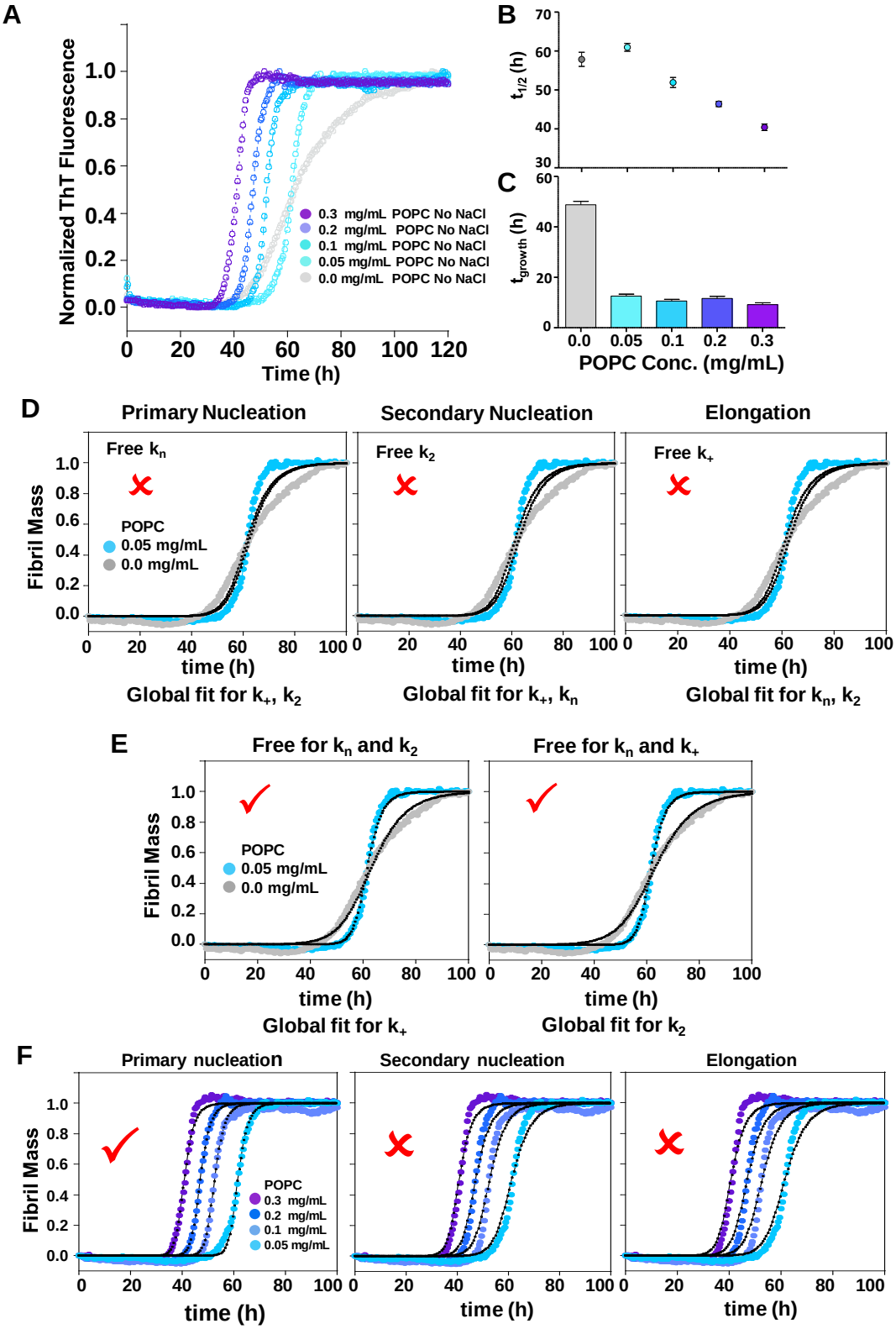


Figure S12. Impact of POPC on Aβ fibril. kinetics Single representative kinetic profiles showing normalized ThT Fluorescence, with a range of POPC LUV concentrations, all in the absence of NaCl (A). Half-time $t_{1/2}$ (hrs) and growth time t_{growth} (hrs) for fibril formation, error bars are standard deviation of the mean (B, C). Global fits for Aβ with and without POPC (0.05 mg/mL), when only primary nucleation (k_n), secondary nucleation (k_2), fibril elongation (k_+) rate constants are altered to globally fit traces (D). Global fits do not fit this kinetic behaviour and a second-rate constant is required (E) of the kinetic traces when primary and secondary nucleation rate constants and primary and elongation rate constants for POPC (0.05 mg/mL) and Aβ 40 (5 μM) traces are fitted together (E). Global fits of the Aβ 40 in the presence of increasing POPC only primary nucleation, secondary nucleation, fibril elongation rate constants are altered to globally fit the kinetic data, with the lipid free trace not present, the kinetic traces fit well when varying primary nucleation only (F).