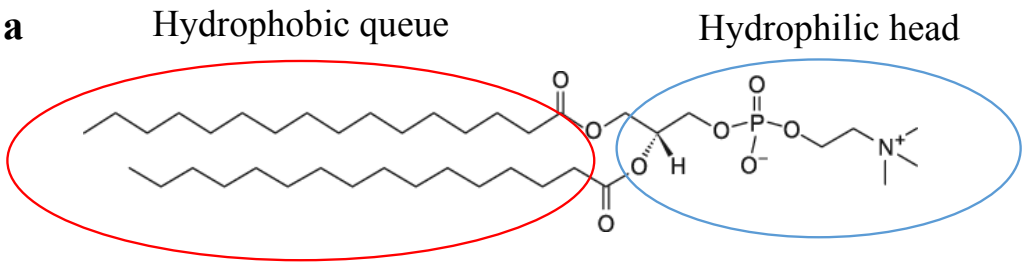
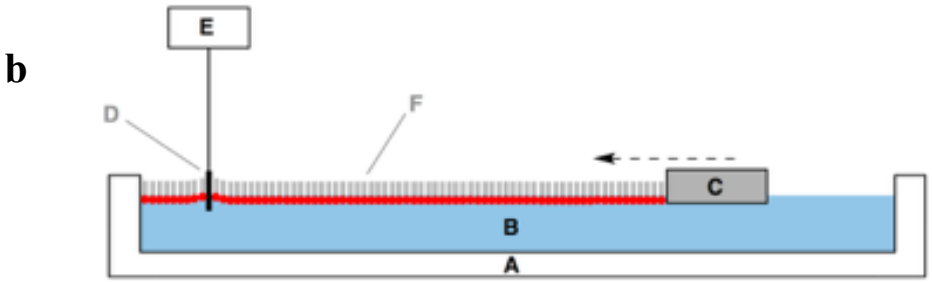
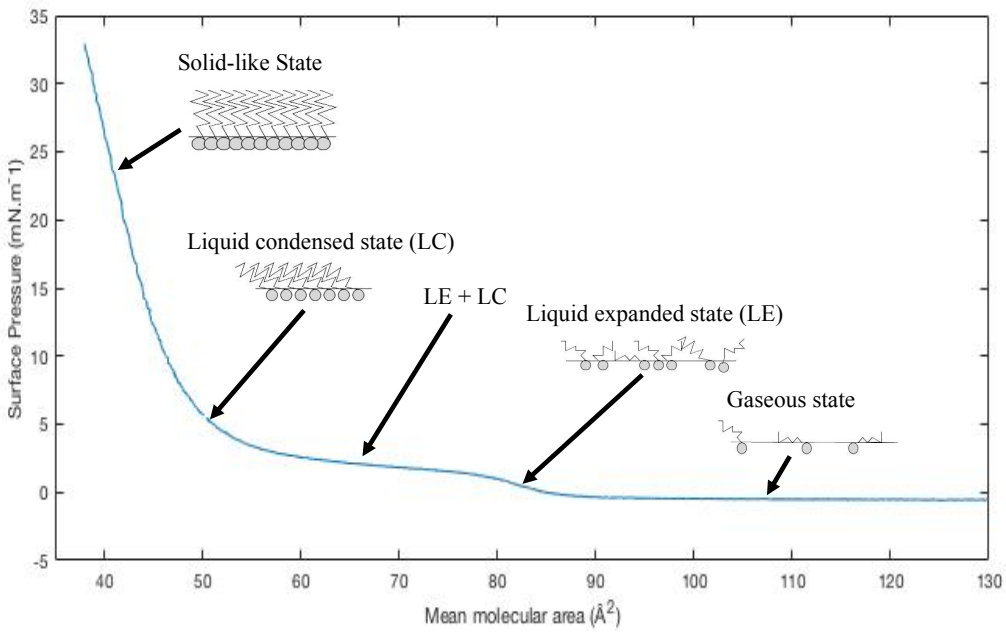
## Figure 1: Formation of a Langmuir film

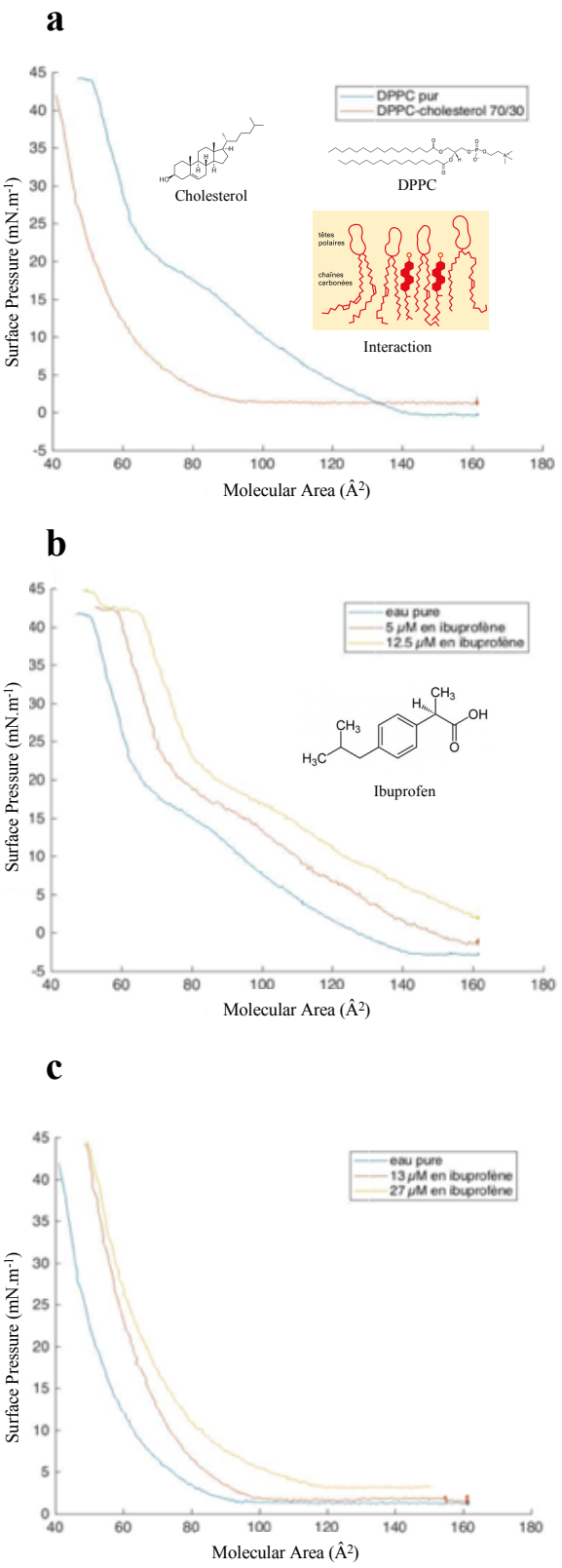


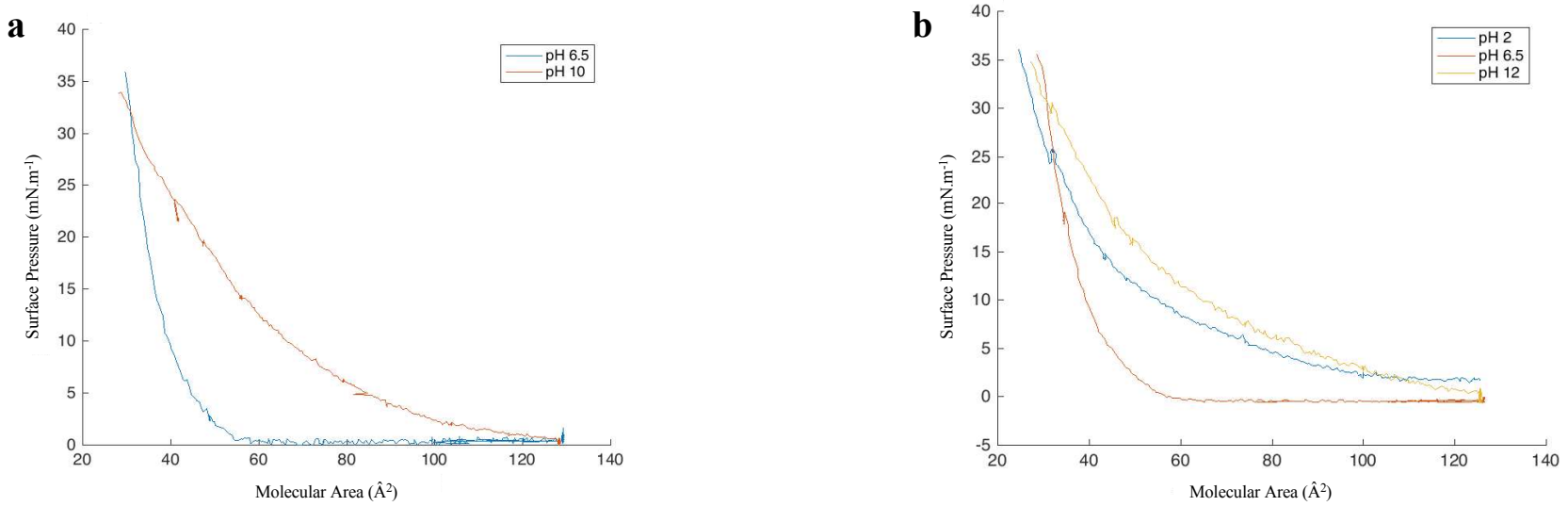

**a**, Chemical formula of 1,2-Dipalmitoylphosphatidylcholine (DPPC) phospholipid. DPPC is made of a hydrophobic queue and of a hydrophilic head. DPPC is the major constituent of many pulmonary surfactants. It is also used for research purposes in studying liposomes, lipid bilayers, and model biological membranes. **b**, Schema of a Langmuir balance: A: Teflon tank, B: aqueous sub-phase (milli-Q water, Millipore Corporation), C: movable barrier, D: Wilhelmy plate, E: Electrobalance, F: phospholipid monolayer. **c**, Experimental $\pi$(Surface pressure)-A(Molecular area) DPPC isotherm using a Wilhelmy plate on a Langmuir film at a constant temperature of 18°C. As the two movable barriers are compressing the Langmuir film at a constant speed, phospholipids go through different physical states: starting with a gaseous state, then a expanded liquid state, then a condensed liquid state and a solid state. At the end of the curve, we can observe a collapse which corresponds to the destruction of the monolayer.

# Figure 2: Influence of cholesterol and Ibuprofen on DPPC isotherms




**a**, Influence of cholesterol on a DPPC isotherm at 25°C. In red, DPPC isotherm (25 $\mu L$ , 1 mM) and in blue, DPPC/Cholesterol mix isotherm (25 $\mu L$, 1 mM, 70% of DPPC, 30 % of cholesterol). We can observe that intercalation of cholesterol in the membrane induce a stabilisation and a condensation of the membrane thanks to interactions between cholesterol nucleuses and DPPC carbon chains. Thereby, cholesterol promotes the formation of condensed monolayer domains. The LE to LC transition is affected by the presence of cholesterol. We can also observe that the Molecular Area at the collapse decreases with the presence of cholesterol (from 61 A°-2 to 40 A°-2), which is also coherent with a stiffening of the membrane: the membrane resists to a higher compression before collapsing. **b**, Influence of ibuprofen on a DPPC isotherm at 25 °C. We have here plotted three DPPC-isotherms for different concentrations of ibuprofen in the subphase (0 µM, 5 µM and 12.5 µM). We first observe an expansion of the monolayer: for the same Surface Pressure, the Molecular Area rises with the concentration of ibuprofen. This can be explained by the intercalation of ibuprofen into the hydrophobic region of the monolayer. We also note that the Molecular Area at the collapse increases with the concentration of ibuprofen, which is another proof of the enhancement of monolayer stability due to ibuprofen incorporation in DPPC monolayers. **c**, Influence of ibuprofen on a DPPC/cholesterol isotherm at 25 °C. We have here plotted three DPPC/cholesterol (70/30) isotherms for different concentrations of ibuprofen in the subphase (0 µM, 13 µM and 27 µM). We observed the same ibuprofen impact on isotherms than in **b**, but for higher concentrations of ibuprofen. This is due to the stabilizing effect of cholesterol which counters the effect of ibuprofen.

## Figure 3: pH Influence on DPPC/Cholesterol (70/30) and DPPC/Cholesterol (70/30)/Ibuprofen isotherms




**a**, pH Influence on a DPPC/Cholesterol (70/30) isotherm. It can be seen that the increase of the pH has a comparable effect to that of ibuprofen: the isotherm extension. This can be explained by the increase in the solvation sphere size in the hydrophilic head region due to the acid pH. Indeed, more counter ions are needed around the head to neutralize the charge. Around pH 7, the ammonium group and the phosphate group in the DPPC molecule have opposite charges, and so the hydrophilic region is approximately neutral, it needs less counter ions and it takes less place. **b**, pH influence on a DPPC/Cholesterol (70/30)/Ibuprofen isotherm. Same observation than in **a** can be made in presence of ibuprofen, for acid and basic pH.