Purpose: To model the ultrasound effects on cellular calcium ion (Ca2+) influx for exploring its potential impact on Ca2+ regulated cellular responses to ionizing radiation.
Methods: Based on Silva's membrane electrophysiology model and for the ultrasound (30 ~ 1000mW/cm2) considered, we adopted a linear relation between ultrasound induced cellular membrane strain deltaA/A0 and ultrasound wave amplitude p as deltaA/A0 ~ p/((Rho*C)*(Rho*C)) (A0 - undisturbed membrane area, deltaA - stress induced area increase, Rho - liquid density, C - sound velocity). The energy density W associated with membrane strain is proportional (deltaA)*(deltaA) while the ultrasound intensity I is proportional to p*p to yield W(I) = kw*I with a linearity coefficient kw. Open channel fraction f0 and rate of exogenous calcium influx qin can be expressed as the following.

f0 = 1/(1+a*exp(-fe*kw*I/(kTN)))

qin = 4f0*Pmax*VmF*F/(RT)(Caex-Cac*exp(2FVm/(RT)))/(1-exp(2FVm /(RT)))

(a - probability that a channel is in open state without load, fe - fraction of strain energy used to gate the channel, k - Boltzmann constant, T - temperature, N - area channel density, Pmax – membrane's ionic permeability when all channels are open, Vm - membrane potential, F - Faraday's constant, R - gas constant, Caex - extracellular calcium concentration, Cac - cytosolic free calcium concentration)
Results: A sigmoid relationship between qin and I is obtained, which is due to the Boltzmann character of the mechanosensitive channels. It's shown that a transient rise of qin can be induced at intensities higher than 40 mW/cm2 and stimulation over 1200 mW/cm2 would lead to unphysiological level of intracellular Ca2+.
Conclusions: The calcium transient induced by low-intensity ultrasound has been shown to be comparable to that induced by ionizing radiation reported in literatures. Further investigation is thus needed to examine the potential impact of ultrasound on cellular responses to ionizing radiation, such as bystander effect.