Background Giving Brusein D or Gemcitabine to pancreatic carcinoma cells will re-inf ect genes that act as tumor suppressor genes. In Brusein D administration there will be activation of p38-MAPK, caspase 9 and caspase 3 as well as inhibition of NF-kB which will stop the synthesis of pancreatic carcinoma DNA so that the cell will carry out cell degradation by apoptosis. In the administration of Gemcitabine, there will be tumor suppressor gene activation, p38-MAPK, which in turn will trigger cell degradation with the mechanism of apoptosis.

Methods PANC-1 pancreatic carcinoma cell cultures were divided into three groups. The first group was in control, the second group was treated with three repetitions of the dose of gemcitabine, the third group was treated with three repetitions of the Brusein D dose, then the number of mutant p53 was calculated in each group.

Results Mutant p53 expression levels in cultures of pancreatic carcinoma PANC-1 cells in vitro were the least in the Brusein D treatment 2 ug/ml (B 2), which average 12.7%, then Gemcitabine 2 ug/ml (G 2) which is an average of 16.7%, followed by Brusein D 1 ug/ml (B 1) which is an average of 22.0%, then followed by Gemcitabine 1 ug/ml (G 1) which is an average of 22.2%, followed by Brusein D 0.5 ug/ml (B 0.5) which is an average of 28.8%, then followed by Gemcitabine 0.5 ug/ml (G 0.5), which is mean average of 29.0%. And the highest level of mutant p53 expression was control, with an average of 51.3%.

Conclusions Brusein D ligand from an ethanolic fraction of Makassar Fruit (Brueca javanica (L.) Merr) has the inhibitory potential for mutant p53 expression so that it can be a candidate for the chemotherapeutic agent in cancer with mutations through the p53 pathway.