Introduction:
Activation of neutrophils is a mandatory stage and a sensitive marker of systemic inflammatory conditions that can lead to the development of multiorgan failure. The aim of the study was to investigate into the antiinflammatory effects of lithium chloride on human neutrophils in vitro.

Methods:
Study was carried out on neutrophils isolated from the blood of 5 healthy donors. 50% of neutrophils were activated by 100 mkM fMLP, 50% - by 100 ng/ml lipopolysaccharide (LPS); then their activity was evaluated by fluorescent antibodies to CD11b and CD66b degranulation markers. Intact and activated neutrophils were treated with a solution of lithium chloride (9 mmol). Immunoblotting was used to assess GSK3b activity in neutrophils. Mann-Whitney criterion and p<0.05 were used for statistics.

Results:
Lithium chloride 9 mmol decreased the level of expression of CD11b on intact neutrophils by 16% (p=0.07), CD66b by 15% (p=0.07). fMLP increased CD11b expression on neutrophils by 2.6 times (p=0.0007), CD66b by 2.5 times (p=0.0022). Addition of lithium chloride solution to fMLP activated neutrophils reduced the expression of CD11b (p=0.0317) and CD66b (p=0.0079). LPS increased CD11b and CD 66b expression by 2.1 times (p=0.0007, p=0.0022, respectively); addition of lithium chloride reduced the expression of CD11b (p=0.0317) and CD66b (p=0.0079) on neutrophils. fMLP led to a dephosphorylation of GSK-3b by 47% (p<0.05), lithium chloride increased its phosphorylation by 387% (p <0.05). Adding lithium chloride to activated fMLP neutrophils restored the level of GSK-3b phosphorylation by 277% compared to controls (p<0.05).

Conclusion:
Lithium chloride modulates the inflammatory activation of neutrophils by bacterial components through the phosphorylation of GSK3b in neutrophils.