CONCLUSION
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In the present study effects of concurrent and prior repeated treatment for a week with certain anti-inflammatory and beta-adrenergic blocking agents on telbuthamide-induced hypoglycemia in normal healthy as well as alloxan-induced diabetic rabbits were investigated. Among the anti-inflammatory drugs examined the lowest and tolbutamide and trametil the comparatively study introduced steroidal anti-inflammatory agents and among the beta-adrenergic blockers propranolol, the non-selective and atenolol, metoprolol and acebutolol the selective cardiac \( \beta_2 \) receptor blockers were chosen for the interaction study with telbuthamide. In order to determine the mechanism of interaction overm telbuthamide concentration was also measured along with blood sugar estimations.

From the results obtained the following conclusions can be drawn:

1. Our experiments show that telbuthamide produces a dose-dependent hypoglycemic action with a peak response at 3 hours in normal rabbits and 5 hours in diabetic rabbits and the effect remains persistent over 9 hours. The corresponding hyper
tolbutamide concentration has a significant
association with blood sugar changes (Fig. 19).

2. Aspirin, tolmetin, prasmanirol and atenacol
seem to have intrinsic hypoglycemic effect
whereas tronaril, metoprolol and acebutolol did
did not produce any significant change on blood
sugar level.

3. Out of three anti-inflammatory agents under study
aspirin and tolmetin on concurrent administration
and prior 7 days treatment were found to potentiate
the tolbutamide-induced hypoglycemia in normal
as well as diabetic rabbits with corresponding
decrease in serum tolbutamide level. However,
tronaril neither potentiated hypoglycemia nor
changed serum tolbutamide level pattern.

4. Since aspirin and tolmetin decreased serum
tolbutamide levels the potentiation of tolbutamide-
hypoglycemia is probably due to their intrinsic
hypoglycemic action and not due to pharmacokinetic
alterations.

5. Prasmanirol and atenacol when administered along
with tolbutamide increased tolbutamide hypogly-
cemic response without any effect on serum
tolbutamide concentration and tolbutamide half-life.
in normal rabbits. But metoprolol and acebutolol did not influence tolbutamide hypoglycemia and its serum level to any extent. But in diabetic rabbits all beta-blockers somehow potentiated tolbutamide hypoglycemia.

6. In normal rabbits pre-treated with beta-blockers for 7 days only propranolol and atenolol potentiated tolbutamide hypoglycemia. However atenolol showed a delayed response but metoprolol and acebutolol had no effect. The serum tolbutamide concentration remained unchanged.

7. It can be concluded that use of aspirin, tolmetin, propranolol, atenolol, metoprolol and acebutolol in diabetic individuals kept on tolbutamide treatment can increase chances of tolbutamide hypoglycemia episodes. Therefore due precautions should be taken to prevent such episodes by suitable dose adjustments or selecting alternative drugs for simultaneous treatment of cardiovascular or inflammatory conditions. However, tramadol is preferable than other anti-inflammatory agents for simultaneous use with tolbutamide. All the beta-blockers are potentially dangerous although cardiospecific drugs preferably metoprolol and acebutolol can be used carefully if use of a beta-blocker is needed.