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## Summary & conclusions and recommendations

This study aims to detect the role of both endogenous and exogenous corticosteroids released in response to stress on some gastric changes. This study was done on adult male albino rats. The rats were classified into 4 groups:

### **Group 1 (control group):**

This group neither received any medications nor exposed to any forms of stress

### **Group II (stress group) :**

This group was exposed to acute cold stress for 3 hours by putting the rats' cage in a refrigerator at 4°C.

### **Group III (corticosteroid receptor antagonist) :**

This group was exposed to the same type of stress and given glucocorticoid receptor antagonist. This group we subdivided according to the dose into 3 subgroups.

**\*\* Subgroup III a:** This subgroup was given glucocorticoid receptor antagonist in a dose of 5 mg / kg.

**\*\* Subgroup III b:** This subgroup was given glucocorticoid receptor antagonist in a dose of 10 mg / kg.

**\*\* Subgroup III c:** This subgroup was given glucocorticoid receptor antagonist in a dose of 20 mg / kg.

### **Group IV:**

This group was subdivided into 4 subgroups

**\*\*Subgroup IV a:**

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This subgroup was given exogenous corticosteroid dexamethasone in a dose of 0.25 mg / kg.

**\*\*Subgroup IV b:**

This subgroup was given dexamethasone in a dose of 0.25 mg / kg. and exposed to acute cold stress.

**\*\*Subgroup IV c:**

This subgroup was given dexamethasone in a dose of 0.5 mg / kg

**\*\*Subgroup IV d:**

This subgroup was given dexamethasone in a dose of 0.5 mg / kg and exposed to acute cold stress.

In this study, exposure of rats in group II to cold stress increased all gastric mucosal parameters proving the ulcerogenic effect of stress. Meanwhile, the effect of endogenous corticosteroids on parameters of gastric mucosal injury was studied in group III by blocking of corticosteroid receptors by the use of its antagonist (RU-486). which caused an increase in parameters of gastric mucosal injury. This effect was dose dependant in the group received 20 mg/kg more than in groups received 5 mg/kg and 10 mg/kg. These results support the gastro-protective effect of endogenous corticosteroids. When the effect of exogenous corticosteroids (dexamethasone) on gastric mucosa was studied it was found that exogenous corticosteroids increased all the parameters of gastric injury and this effect was dose dependant as it increased in groups received dexamethasone in a dose of 0.5 mg/kg than received 0.25 mg/kg. The use of dexamethasone in stressed rats caused more gastric mucosal changes. This supports the ulcerogenic effect of exogenous corticosteroids. The controversy between the effects of endogenous and exogenous corticosteroids may be due to difference in the dose of endogenous released corticosteroid and the administrated dose of exogenous corticosteroids. Also, Chemical differences between

endogenous and exogenous corticosteroids may have a role in the different effects of corticosteroids on gastric mucosa as corticosteroids have different potency and their plasma biological half life is different. Different strain of rats may also cause different effects of corticosteroids on gastric mucosa.

## **Conclusion**

This study has proved that stress is an ulcerogenic factor to gastric mucosa and that endogenous corticosteroids released in response to stress may act as gastroprotective agents, while exogenous corticosteroids are suggested to have an ulcerogenic effect specially if accompanied with stress.

## **Recommendations**

Other studies are recommended to detect:

- The role of endogenous corticosteroids in the healing of pre-existing ulcer
- The effect of different exogenous corticosteroids in different doses on gastric mucosa
- The effect of different stress types as acute, chronic, water restraint stress, noise or immobilization stress on gastric mucosa.