ABSTRACT
Recent research has revealed that DAG signaling is activated in liver cells stimulated by thyroid hormone. L-thyroxine triggers a dual phospholipase pathway that is sequentially and synchronized with other pathways. PKC mediates the integration of both pKa and proline during the sustained phase of agonist stimulation, while lysine disrupts the signalling response.

INTRODUCTION
Thyroxine (T3) is a hormone that is released from the thyroid gland when there is a deficiency of the breakdown product of protein synthesis (Proteins). T3 is a major thyroid hormone in the body and plays an important role in the function of the thyroid gland. A deficiency of T3 can lead to the following symptoms:

Insomnia

Weight gain

Fatigue

Cervical, ovary, testicular and breast pain

A syndrome called Thyroiditis can make it difficult for the thyroid gland to produce T3 and this can cause symptoms such as:

Thyroid cancer

Cervical cancer

Skin cancer

Heart failure

Anemia Thyroid hormone exerts a wide range of effects on development, growth, and metabolism. Its actions are primarily influenced by its interaction with nuclear receptors that bind to regulatory regions of genes and modify their expression. The interaction between Ca+2 mobilization hormones and transmitters with cell surface receptors leads to phospholipid breakdown under PLC or -D action, accumulation of inosite phosphates (DAG), and generation of regulatory molecules through increase in intracellular free calcium concentration and PKC activation, which is widely demonstrated on various cell types. Rat hepatocytes and single heart cells experience a rapid increase in intracellular calcium concentrations when physiological doses of thyroid hormones are added to the cell suspension. However, there is no information available about the accumulation of other PKC modulator - DAG in cells upon administration of T4. Steroid hormones have a genomic independent effect on different cell types, which is comparable to the mechanism of action of thyroid hormone. We have investigated the impact of thyroid hormones on DAG formation and PKC activation in liver cells. The results demonstrated that L-T4 induces the biphasic accumulation of DGA in both liver slices and isolated hepatocytes, with sequential activations of PLC and -D leading to increased DAF formation during sustained agonist stimulation.

CONCLUSION
Remarkable conclusions According to the investigations conducted, L-T4

stimulates the hydrolysis of polyphosphates by PLC and activates DAG and PKC activation in liver cells. This study also revealed that PLD significantly enhances PC cleavage in hepatocytes, as well as in other cells operated by Ca2+-mobilizing receptors.