

**3<sup>rd</sup> International Conference on**

# **Hematology & Blood Disorders**

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## **Immunotherapy targeting the FcγRIIB receptor**

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FcγRIIB has long been known to inhibit the activity of therapeutic mAb. In addition to inhibition of effector cells, we recently showed that FcγRIIB can also engage mAb at the tumor cell surface transmit inhibitory signals and elicit internalization, thereby reducing efficacy. Here we show that highly specific antagonistic hFcγRIIB mAb are able to overcome many of these resistance mechanisms and so warrant clinical investigation in combination with existing therapeutic mAb.

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## **Testosterone therapy interacts with previously undiagnosed familial thrombophilia, facilitating development of osteonecrosis**

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Idiopathic osteonecrosis (ON) (not secondary to high dose long term corticosteroids, alcoholism, fracture-dislocation, etc.) can be caused by osseous venous occlusion leading to increased osseous venous pressure, reduced arterial influx and ischemic necrosis. Although not widely recognized; familial thrombophilia is commonly pathoetiologic for the development of ON. In this study, we examined the interaction between testosterone treatment (TT) and previously undiagnosed thrombophilia facilitating the development of ON in 12 men and 4 women who developed idiopathic ON 6 months (median) after starting TT. When TT is given to patients with previously undiagnosed familial thrombophilia, ON commonly occurs, particularly in the presence of Factor V Leiden heterozygosity. Elevation of (E2) derived from the aromatization of testosterone interacts with thrombophilia to promote development of ON. Screening for common familial thrombophilias prior to starting TT is a tool to optimize estimation of the benefit/risk ratio of TT.

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## **Notes:**