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Titel des Beitrags: Expression of inhibin/activin subunits alpha (-alpha), betaA (-betaA), and betaB (-betaB) in placental tissue of normal, preeclamptic, and HELLP pregnancies.

Abstract: During human pregnancy the placenta produces a variety of proteins for the establishment of the fetoplacental unit, including inhibins and activins. Inhibins are dimeric glycoproteins, composed of an alpha-subunit and one of two possible beta-subunits (betaA or betaB). Aims of the present study were (a) the determination of the frequency and tissue distribution patterns of the inhibin/activin subunits in human placental tissue of normal pregnancies and pregnancies complicated with preeclampsia and HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) and (b) the assessment of a combined expression of inhibin-alpha- and both beta-subunits (betaA-and betaB-subunits) using double immunofluorescence technique. A significant lower expression of the inhibin-alpha subunit in preeclamptic and HELLP placental tissue compared to normal pregnancies was observed, while the inhibin-alpha immunostaining was significantly upregulated in syncytiotrophoblast. Additionally, we demonstrated a significant down-regulation of inhibin-betaB subunit in extravillous trophoblast cells between normal and preeclamptic compared to HELLP placental tissue, while inhibin-betaA-subunit was significantly higher in preeclamptic syncytiotrophoblast cells. A colocalization of inhibin-alpha and the
beta-subunits could be demonstrated, suggesting a production and secretion of intact inhibin A and inhibin B. Therefore, inhibin A and activin A might be useful markers in preeclampsia. Valuable parameters in HELLP syndrome could be inhibin A, rather than inhibin B, and activin B. Furthermore, the lower betaB-subunit production in extravillous trophoblast cells demonstrates that this subunit might have an important role in the pathogenesis of HELLP syndrome. Additionally, the higher production of the betaA-subunit in syncyotrophoblast cells suggest a higher production of activin A rather than inhibin A in preeclampsia that might be utilized as a marker of placental function.