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OPG / *total* sRANKL

in vascular calcification

ELISAs for research and routine

Vascular calcification often occurs with advancing age or with metabolilc disorders such as diabetes mellitus, leading to serious clinical consequences. Many key factors and cell types involved have been defined while searching strategies for clinical intervention in calcified vasculopathies. One emerging area in vascular biology involves the RANKL/RANK/OPG system.

Regulation of vascular calcification by osteoclast regulatory factors

OPG (Osteoprotegerin), RANKL (Receptor activator of nuclear factor NF-kB ligand) and RANK act as key regulators of bone metabolism and the immune system. **RANKL** is the essential factor for osteoclast formation, fusion, activation, and survival, resulting in bone resorption and bone loss. Moreover, RANKL is known to have several immunological effects, including T cell activation and regulation of various functions of antigen-presenting dendritic cells, as well as inhibition of apoptosis, induction of cluster formation, and stimulation of cytokine production. The biological effects of RANKL are counterbalanced by the endogenous antagonist osteoprotegerin (OPG) which binds to and neutralizes RANKL.
Recent findings suggest that OPG and RANKL may play an important role in the development of vascular disease. Jono et al. (2002) and Schoppet et al. (2003) showed that **serum OPG levels are associated with the presence and severity of coronary artery disease**. They conclude that alterations of the OPG system may contribute to the pathogenesis of human vascular disease. The mechanism by which serum OPG levels were increased in advanced coronary artery disease is still unknown. Another study showed that OPG is upregulated in symptomatic human carotid atherosclerosis with possible implications for plaque stability (Golledge et al. 2004).
Modulation of the RANKL/RANK/OPG system in animals results in a skeletal and vascular phenotype (Sattler et al. 2004).

OPG	
Matrix	Serum, EDTA plasma, Heparin plasma, Citrate plasma
Sample volume	20 µL
Test principle	ELISA
Cat. No.	K 1011

also available for your research: OPG Mouse/Rat (KR1020)

Why to measure „*total* sRANKL“?

The concentration of free sRANKL in serum/plasma is quiet low, that is why the determination is very difficult. For this reason we developed the ***total*** sRANKL ELISA. This ELISA determines the sum of the free sRANKL and the OPG bound sRANKL in serum or in cell culture supernatants. The test is based on the reaction with an excess of OPG and the simultaneous capture of the formed RANKL-Osteoprotegerin-complex to the solid phase by specific mouse monoclonal antibodies against a chosen adequate RANKL epitope.

total sRANKL – the advantages:

- **Detects free and OPG bound sRANKL: higher sensitivity than sRANKL-ELISA (differentiates clearly within the normal range)**
- **Free sRANKL can be determined mathematically by carrying out the assay two times: once with a prior addition of OPG (free sRANKL and OPG-sRANKL-complex are detected), once without an addition of OPG (only OPG-sRANKL-complexes are detected)**
- **Only 50 µl sample volume is needed**
- **High stability of the analyte (can be kept frozen for years until analysed)**

***total* sRANKL**	
Matrix	Serum, plasma
Sample volume	50 µL
Test principle	ELISA
Cat. No.	K 1016

References

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