

## ORIGINAL ARTICLE

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**Pathogenetic relevance of the pregnancy hormone relaxin to inborn hip instability**

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**Abstract** The etiology of inborn hip dysplasia is unknown. In general, a multifactorial genesis is assumed. The influence of hormones on the development of the fetal hip joint and its stability is discussed as well as mechanical influences. This study was carried out with the intention to examine the correlation between the concentration of the pregnancy hormone relaxin and the stability of the hip joint in newborns. Both hips of 90 newborn children were examined clinically and sonographically. In 25 hips (13.9%), pathological sonograms according to the classification of Graf were found. The relaxin concentration was measured in cord blood using a heterologous radioimmunoassay. Statistical evaluation revealed an insignificant decrease of relaxin concentration with increasing sonographic hip instability. The results indicate that hip instability frequently occurs with decreasing relaxin concentration. These facts contradict the earlier assumption that hip instability coincides with increased relaxin concentrations in newborns. We assume that there is a worse preparation of the pelvis and the birth canal during pregnancy due to the lower relaxin concentration and thus that there could be a higher pressure on the fetus in the perinatal phase. A decreased relaxin concentration seems to have no direct effect on the hip joint tissue, but indirectly there is consequent rigidity of the tissue in mother and child, which can further promote the development of hip instability.

**Introduction**

Inborn hip dysplasia occurs worldwide, with an incidence of 2.7–17 cases in 1000 live-born infants [24]. Without treatment, almost every 10th hip dysplasia develops into a hip luxation within the first months of life [1]. The hip dysplasia is with 48.6% the main cause of osteoarthritis of the hip in young adults [8]. The etiology of inborn hip

dysplasia has not been clarified up to now although a multifactorial genesis is commonly accepted [23]. In addition to genetic factors, an inborn laxity of the ligaments, external mechanical influences, and hormonal influences are also considered as causing the development of hip dysplasia [2, 23, 28].

The pregnancy hormone relaxin is supposed to lead to a laxity of fetal ligaments by influencing the connective tissue metabolism, as do oestrogens and progesterone [23, 28]. The action mechanism of relaxin was discovered in 1926 by Hisaw et al. [12]. The influence of this hormone on the connective tissue metabolism could be proved beyond doubt in animal experiments [26, 27]. A final explanation of its physiological role, however, is still to come. It is assumed that one of its main functions is the preparation of the birth canal by relaxing the mother's pelvic ligaments. This effect is caused by an influence on collagen synthesis [12, 26]. Relaxin increases the secretion of collagenase and plasminogen activator, two key enzymes of collagenolysis [14]. By neoformation and degradation of collagen due to a stimulation of fibroblast replication, the cellular tissue changes its formation [22]. This permits an extension and flexibility of the cells during growth in pregnancy [27].

The relaxin concentration is determined in the serum. Studies by Entenmann et al. [6] and Entenmann [5] prove that there was neither a significant difference in the concentration of relaxin in arterial or venous cord blood nor different concentrations in maternal and infant blood.

**Materials and methods**

In 90 newborn children (40 male, 50 female), 10 ml of cord blood were taken directly postpartum to determine the concentration of relaxin. The cord blood was mixed in a centrifugal tubule with one spatula tip each of sodium azide (0.02%) and phenylmethylsulfonylfluoride. This suspension was centrifuged in a laboratory centrifuge at 3000 U/min for 10 min. The extracted serum was stored at  $-20^{\circ}\text{C}$ . Relaxin has a short half-time in serum and can be protected against degradation by freezing and adding protease inhibitors [18]. Freezing does not change the relaxin concentration of serum specimens [27].