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LPS retard chondrocyte hypertrophy in the growth plate through elevating Sox9 expression

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Accumulating data show that the cytotoxicity of bacterial lipopolysaccharides (LPS) from microbiota or infection is associated with many disorders in clinic. However, it is still obscure whether or not embryonic osteogenesis is affected by the LPS exposure during gestation. we could demonstrate that LPS exposure inhibited chondrogenesis of the 8-day chicken embryos and osteogenesis of 17-day. The further analysis of the growth plates showed that the proportion of proliferating zone (PZ) increased and hypertrophic zone (HZ) decreased following LPS exposure, while there was no significant change on cell proliferation in the growth plates. Sox9 and Col2a1 were highly expressed at the mRNA level and protein was also abundant. LPS exposure caused a down-regulation of Runx2 and Col10a1 expression in 8-day hindlimbs, and a suppression of Runx2, Col10a1 and Vegfa expression in 17-day phalanges. Knocking-down Sox9 in ATDC5 cells by siRNA transfection lead to the expression reduction of Col2a1, Runx2, Col10a1, implying the vital role of Sox9 in the process of LPS-induced delay from proliferating chondrocytes to hypertrophic chondrocytes in the growth plate. In presence of LPS, Nrf2 was highly expressed in 17-day phalanges and ADTC5 cells, so was intracellular ROS. When Nrf2 expression was knocked-down in ATDC5 cells, the expressions of Sox9, Col2a1, Runx2, Col10a1 and Vegfa were also going down as well. our current data suggest that LPS exposure during gestation could restrict the chondrocytes conversion from proliferating to hypertrophic in the growth plate, in which LPS-induced Sox9 plays a crucial role to trigger the cascade of downstream genes by excessive ROS production and Nrf2 elevation.