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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 eighth 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. *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 eighth 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.

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