2-2021

The Role of Toll-Like Receptor 4 in the Progression of Keloids

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SI/CTR Abstract
Word Count: 248

The Role of Toll-Like Receptor 4 in the Progression of Keloids

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Keloids are highly prevalent in the population and their pathogenesis is largely unknown. T-cell lymphoma research shows that toll like receptor-4 (TLR-4) is highly expressed on CD206+ immunosuppressive macrophages and is implicated in maintaining the tumor microenvironment. Additionally, fibronectin, specifically extra domain A (EDA), is highly expressed and is associated with immunosuppression via the TLR-4 pathway. Assuming that keloids mimic the tumor microenvironment, we hypothesize that TLR-4 is implicated in the pathogenesis of keloids. Using keloid tissue samples and normal breast tissue as a control, immunohistochemistry was performed to observe the presence and co-localization of these markers. Three antibodies were used to examine co-localization of CD-206 with TLR-4 and CD-206 with EDA to determine if these three components were localized to the same region in the keloid tissue. The secondary antibodies were immunofluorescent, and the tissue was analyzed using electron microscopy. Upon analysis, it was found that in keloid tissue CD-206 and TLR-4 were co-localized. To further support the hypothesis, it was found that CD-206 and EDA were also co-localized. In the control group, there was no evidence of co-localization of CD-206 with TLR-4 or with EDA. These findings support the overall hypothesis that TLR-4 is involved in the pathogenesis of keloids. Discovering that CD-206 co-localizes with both TLR-4 and EDA indicates that these three molecules interact to help form the tumor microenvironment needed to support keloid formation. Knowing this information, studies can begin to look into possible immunological targets for keloid treatment that access this immunosuppressive pathway.