аутореактивными специфическими рецепторами, либо *с* их анергией, либо же со снижением экспрессии провоспалительных цитокинов и хемокинов, включая IFNγ и CCL1 (Kim et al., 2020), которые ранее были определены как прямые мишени BOB1 (Shakya et al., 2015). Пептидный ингибитор белка BOB1 нормализовал уровень глюкозы, уменьшал инфильтрацию Т-клеток и экспрессию провоспалительных цитокинов у мышей NOD с ново-приобретенным диабетом (Kim et al., 2020), подтверждая тем самым идею о том, что BOB1 является мощным регулятором аутоиммунных процессов и перспективной мишенью для фармакологического ингибирования.

Таким образом, основываясь на многочисленных свидетельствах, можно предположить, что постоянное воздействие аутоантигена(ов) в контексте хронического воспаления повышает экспрессию регулятора транскрипции BOB1, который, взаимодействуя с факторами транскрипции OCT1 и OCT2 через еще не полностью определенные механизмы, индуцирует аутоиммунный ответ, способствуя дальнейшему усилению хронических воспалительных процессов в аутоиммунных очагах. Исследование механизмов, лежащих в основе работы BOB1/OCT1(2) в контексте развития аутоиммунных заболеваний, может стать в будущем основой для поиска потенциальных терапевтических мишеней при разработке методов лечения этих заболеваний.

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При подготовке работы не проводились какие-либо исследования с использованием животных или людей в качестве объектов исследований.

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Авторы заявляют об отсутствии конфликта интересов.

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Transcriptional Coactivator BOB1 (OBF1, OCA-B) in Autoimmune Diseases

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Despite significant efforts in biomedicine for several decades, autoimmune diseases continue to remain largely incurable and, moreover, poorly understood in terms of the molecular mechanisms underlying their onset and progression. It is generally accepted that autoimmune pathologies result from a malfunction of the adaptive immune system in genetically susceptible individuals leading to the appearance of autoreactive B- and T-lymphocytes. However, the exact molecular pathways that drive the activation of autoreactive lymphocytes, leading to the amplification

and perpetuation of self-directed immune responses are largely unknown. A number of experimental data accumulated over the past few years indicate a key role of BOB1, namely its imbalanced expression, in the onset of autoreactive lymphocytes. It has been postulated that the coactivator BOB1 affects transcription and local chromatin state indirectly, via selective interaction with DNA-binding POU-domain transcription factors – ubiquitous OCT1 and B-cell-specific OCT2, stabilises the binding of the OCT factors to DNA. The review lists the latest evidences of an important role of BOB1 in pathogenesis of autoimmune diseases and positions this protein as a promising target in the treatment of these diseases.

Keywords: BOB1, OCA-B, OBF1, POU2AF1, OCT1, OCT2, autoimmune diseases, autoreactive T- и B-cells