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ASSOCIATION OF ONCOGENE RAC 1 WITH HER-2 TUMORS AND WITH AGGRESSIVITY OF TRIPLE NEGATIVE TUMORS

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Objective: To evaluate the expression of RAC 1 in patients with breast cancer and its molecular subtypes. **Method:** Samples were collected from 41 patients who underwent mastectomy at the Barao Lucena Hospital, 10 luminal A, 15 luminal B, 1 luminal (undifferentiated), 6 HER-2, 9 Triple- negative. RNA was purified by RNAeasy KIT (Qiagen) and quantified by Nano Drop 2000 (Thermo). The cDNA was synthesized with the QuantINova ReverseTranscription kit (Qiagen) and real-time PCR was performed in StepOnePlus (Applied Biosystems) with the Go Taq Qpcr Master Mix kit (Promega). The expression of b-actin was used as endogenous control and the ACT was calculated to analyze the reactive quantification of RAC 1 in each sample. Statistical analyzes were performed with R. **Results:** The relative expression of RAC 1 presented a metric behavior. The HER2 subtype had the highest RAC 1 expression compared to luminal ($p=0.0006899$), even when stratified in Luminal A ($p=0.003592$) and in Luminal B ($p=0.00762$) and with larger tumor size ($p=0.01441$). No association was observed between RAC 1 expression and KI-67 LOW (KI-67 lower 20%), with p values ranging from 0.2186 to 0.9472. **Conclusion:** The absence of the estrogen receptor seems to amplify the expression of RAC 1 in response to the metabolic pathway DE her 2. Patients who underwent hysterectomy showed a reduction in the expression of RAC 1. RAC 1 presents a great potential for new progression studies tumor in triple-negative tumors.