International Society of Exercise and Immunology
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Exercise Immunology: Prescriptions for Health

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Authors (Underline the presenting author)	H Yano, M Uchida, E Oyanagi, A Yamauchi & MJ Kremenik
Department, Institution, Country	Department of Health and Sports Science, Kawasaki University of Medical Welfare, Kurashiki, Japan
Address Corresponding Author	288 Matsushima, Kurashiki, 701-0193, Japan, and E-mail address: yanohiro@mw.kawasaki-m.ac.jp
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Relationship between macrophage differentiation and the chemotactic activity toward damaged muscle cells

YANO H1, UCHIDA M1, OYANOGI E2, YAMAUCHI A3, and KREMENIK MJ1.

1Department of Health and Sports Science, Kawasaki University of Medical Welfare, Kurashiki, Japan
2Department of Health Promotion and Exercise, National Institute of Health and Nutrition, Tokyo, Japan
3Department of Biochemistry, Kawasaki Medical School, Kurashiki, Japan.

ABSTRACT

Aim: We investigated the effect of macrophage differentiation on the chemotactic activity to invade local damaged muscle using in vitro models of muscle injury. Methods: C2C12 cell myoblasts, and J774 cell macrophages were used. The "killed-C2C12" cells were combined with live C2C12 cells (live:killed C2C12 = 1:0.5) as a partially damaged muscle model. The J774 cells were stimulated with LPS and DEX. The chemotactic activity of J774 cells was examined using TAXIScan device. Results: Although the velocity of J774 cells was little affected by each type of C2C12 cells (live, killed and combination), the directionality of the J774 cells was increased. The highest directionality of J774 cells was observed when the ratio of live-:killed-C2C12 cells was 1:0.5.The TLR4 and CD11c expressions of LPS cells were higher than those in both Ctrl and DEX cells. The LPS cells were strongly stained around the cell membrane by phalloidin, but the F-actin expression in DEX cells was in an orderly line along the long axis of cells. DEX cells showed stretching toward C2C12 cells, and their length/width ratio was higher than that in both Ctrl and LPS cells. Although the chemotactic activity of LPS cells disappeared completely, DEX cells exhibited accelerated chemotactic activity toward damaged muscle cells. The MCP-1 production in live-:killed-C2C12 cells was higher than that in the live-C2C12 cells. The CCR2 expression in DEX cells was higher than that in both Ctrl and LPS cells. Conclusion: Our conclusion is that: 1) the chemotactic activity of macrophages toward areas of damaged muscle induces more live myoblasts than damaged cells, 2) the chemotactic activity of macrophages is not due to velocity, but depends on the directionality toward damaged muscle cells, and 3) macrophage differentiation influences their chemotactic activity toward damaged muscle cells through the expression of CCR2 and/or F-actin.