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CORRECTION

# Correction to: Abstracts

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K. Sokolova[1] · I. Gette[1] · I. Danilova[1] · M. Abidov[1]

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The authors of **PS-03-015** wish to clarify the Funding details related to their abstract. The abstract is included in full below – no changes have been made to the abstract beyond listing the Funding information.

**Background & Objective:** Damage and dysfunction of β-cells lead to hyperglycemia. That is why β-cells are the main target of regenerative therapy of diabetes. Insulin-producing cells (IPC), located solitary or in clusters, may occur in acinar part or in ducts of the pancreas. Proposed, that formation of such cells occurs due to the damage of pancreas, mediated by macrophage infiltration of organ. Strategy, aims to proliferation and maturation of these specific β-cells subpopulations, may represent a promising direction in the regulation of hyperglycemia. Objective: to characterize quantity, localization, functional activity of extra-islet IPC in diabetic rats and after modulation macrophage activity.
**Method:** 20 Wistar rats were divided into 4 groups: 1 – control; 2 and 3–30 and 60 days of streptozotocin-induced diabetes correspondingly, 4–30 days of diabetes + injection of 3-aminophtalhydrazine derivatives, which modulate macrophage activity and reduce inflammation. Insulin-positive cells were detected by immunohistochemistry.
**Results:** In 30 days of diabetes number of solitary IPC in acinar part increased more than 3 times with normal functional activity level and decreased in 60 days. Number of IPC in ducts at diabetes almost unchanged. Modulation of macrophages activity promotes increase the number of solitary IPC in acinar and ductal parts and ductal insulin-positive clusters as well; growth of functional activity of extra-islet IPC was detected.
**Conclusion:** Exposed increase of number and functional activity of endocrine extra-islet cell subpopulations proves heterogeneity and plasticity of pancreatic cells, due to which they can serve as an additional source of β-cells, while macrophage-centered therapy offer promising possibilities at correction of hyperglycemia.

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✉ K. Sokolova
kssokolova@bk.ru

[1] Dept. of Natural Science, Ural Federal University, Yekaterinburg, Russia