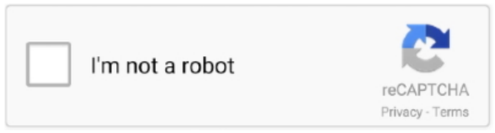

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This study adhered to the ARVO Statement for the Use of Animals in Ophthalmic and Vision Research and was performed in accordance with the guidelines provided by the Animal Care and Use Committee of the Affiliated Hospital of Inner Mongolia University for the Nationalities.. Neonatal mice (C57BL/6J) and adult mice (C57BL/6J, 20~25 g) were purchased from the animal center of Inner Mongolia University and were raised in the animal room of the Affiliated Hospital of Inner Mongolia University for the Nationalities.. Accumulating studies have shown that miR-155 is upregulated in several diseases, such as breast cancer, colon cancers, Down syndrome, Alzheimer disease, multiple sclerosis, and even AMD [-].. miRNAs play a critical role in the regulation of diverse biologic processes, such as cell proliferation, differentiation, apoptosis, tissue development, and homeostasis [-].. Abnormal miRNA expression is associated with various human diseases, such as cancer and metabolic disorders [].

Published online 2015 Oct 13 MiR-155 and the miR-17-92 cluster The first miRNAs to be found overexpressed in cancers were miR-155 and the miR-17-92 cluster of microRNAs.. Furthermore, several targets of miR-155, such as WEE1, SHIP1, and SOCS1, were identified in proteomic studies [,].. 2005; 65:6029–6033 Interestingly, it was found that miR-155, overexpressed in different lymphomas, including the activated B-cell–like type of diffuse large B-cell lymphoma, is also upregulated in aggressive CLLs, whereas members of the miR-29 family and miR-181 were found to be underexpressed and later demonstrated to directly regulate the TCL1 oncogene, which.. A laser-induced choroidal neovascularization (CNV) model was induced in adult C57BL/6J mice.. IntroductionRetinal neovascularization is a major cause of vision loss in various diseases, such as retinopathy of prematurity (ROP), diabetic retinopathy (DR), and age-related macular degeneration (AMD), and the incidence rates of these diseases have recently increased [].. Anti-miR-155 lentivirus reduced the VEGF-induced proliferation, migration, and tube formation abilities of HRMECs.. ResultsThe expression of miR-155 was elevated in HRMECs after treatment with vascular endothelial growth factor (VEGF) and in neovascularized mouse model retinas.. Others also reported that inflammatory cytokines increase miR-155 expression in human retinal pigment epithelial cells (RPEs) [], and in AMD miR-155 may be associated with inflammatory neurodegeneration [].

chanel

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In VEGF-treated HRMECs and retina neovascularization models, p-Akt (Ser473) was significantly upregulated, while SHIP1 was downregulated.. Anti-miR-155 was intravitreally injected on postnatal day 12, and the retinal non-perfused areas and extent of neovascularization were measured on postnatal day 18 using transcardiovascular fluorescein isothiocyanate (FITC)-dextran perfusion and retina sections.. Chan JA, Kritchevsky AM, Kasic KS MicroRNA-21 is an anti-apoptotic factor in human glioblastoma cells.. The efficiency of the lentiviral particles that produced anti-miR-155 in the transduced cells was confirmed via fluorescence microscopy.. The animals were housed with free access to laboratory food and water under a 12:12 h light:dark cycle.

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To evaluate the leakage areas, fundus fluorescein angiography was performed on day 14 after anti-miR-155 intravitreal injection.. Briefly, anti-miR-155-treated HRMECs were seeded at a density of 1×104 cells per well in 96-well plates and incubated for an additional 24 h and 48 h in a medium that contained VEGF165.. The results revealed that the downregulation of miR-155 attenuated retinal neovascularization via the PI3K/Akt pathway.. Lentivirus transduction in HRMECsHRMECs were transduced with anti-miR-155 (Thermo Scientific, MH12601, 5′-UUAAUGCUAAUCGUGAUAGGGGU-3′) or anti-control miRNA (Thermo Scientific, Anti-miR™ miRNA Inhibitor Negative Control #1, AM17010) in special endothelial growth medium (Endothelial Growth Medium, Cat# cAP-02, Angio-Proteomie, Boston, MA) for 24 h and cultured for another 24 h and 48 h in a medium that contained vascular endothelial growth factor (VEGF165, 25 ng/ml, BD Biosciences, Cat#293-VE) as a stimulating factor.. However, the role of miR-155 in retinal angiogenesis remains unknown In this study, we investigated the in vitro and in vivo anti-angiogenic effects of miR-155 using human primary retinal microvascular endothelial cells (HRMECs) and two kinds of mouse retinal neovascularization models.. MethodsmiR-155 was knocked down using lentivirus-mediated RNA interference The proliferation, migration, and tube formation of human retinal microvascular endothelial cells (HRMECs) were measured using BrdU, Transwell, and Matrigel assays, respectively.. MicroRNAs (miRNAs, miR) are small (~20–22 nucleotides), non-coding RNAs that post-transcriptionally regulate gene expression by binding to the 3′-untranslated regions of target mRNAs, leading to mRNA degradation or the inhibition of translation [].. Endothelial cell proliferation assaysThe effects of anti-miR-155 on cell proliferation were studied using a BrdU Cell Proliferation Assay Kit (#6813, Cell Signaling Technology) as previously described [].. Recently, miR-155 was reported to play an anti-angiogenic role in the regulation of neovascularization via the suppression of divergent cell-specific target genes [].

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PMID: 26539029This article has been cited by other articles in PMC AbstractPurposeWe aimed to investigate the anti-angiogenic properties of miR-155 via in vitro and in vivo studies.. Complications that result from uncontrolled retina neovascularization are the major causes of severe vision loss worldwide.. The neovascularization area of the CNV model was also examined in confocal and retina section studies.. An oxygen-induced retinopathy (OIR) model was induced using neonatal C57BL/6J pups.. MethodsCells and animalsHRMECs (Angio-Proteomie, Boston, MA) are primary cells that were used for in vitro studies and were cultured as previously described [].. Moreover, functional studies have indicated that the phosphatidylinositol 3-kinase (PI3K)/Akt, c-JUN, and JAK/STAT pathways are constitutively activated by miR-155 overexpression [,].. At the indicated time points, the proliferation assays were performed according to the manufacturer's instructions.. ConclusionsThe results revealed that the downregulation of miR-155 attenuated retinal neovascularization via the phosphatidylinositol 3-kinase (PI3K)/Akt pathway.. Anti-miR-155 attenuated retinal neovascularization in in vivo CNV and OIR models.. The expression levels of SHIP1 and p-Akt (Thr308, Ser473, and Thr450) were evaluated both in vitro and in vivo.. Conversely, the inhibition of miR-155 restored the expression of SHIP1 and reduced the phosphorylation of effectors in the Akt (Ser473) signaling pathway.. Because HRMECs are primary endothelial cells, they are sensitive to nutritional deficiencies; thus, all of the culture media in our experiments contained 10% FBS (Life Science Technology, as suggested by the manufacturer's culture protocol), including media for the proliferation assay, migration assay, tube formation assay, and western blot studies.. Each experiment was performed in five wells and repeated at least three times. miR-155 is a newly identified miRNA that has been associated with a large number of biologic activities, including lymphocyte activation, immune cell regulation, and microglia stimulation [,].. Anti-miR-155 or anti-miR GFP was applied with a lentivirus transduction agent (Thermo Scientific) following the guidelines provided by the manufacturer. d70b09c2d4