

FIG E1. Knockdown of *Mrc1* polarizes macrophages toward M1 phenotypes. **A**, Real-time PCR analysis of *Mrc1* expression in macrophages with *Mrc1* knockdown by small interfering (*si*) RNA1 and siRNA2, respectively. **B**, Real-time PCR analysis of expression of M1 markers in untreated (control), LPS-treated (M1 macrophage), and IL-4-treated (M2 macrophage) BMDMs from WT (filled with black) or *Mrc1* (filled with gray) knockdown mice. **C**, Real-time PCR analysis of the expression of M2 markers in untreated (control), LPS-treated (M1 macrophage), and IL-4-treated (M2 macrophage) mice ($n = 2$ samples per condition). Data represent means. * $P < .05$.

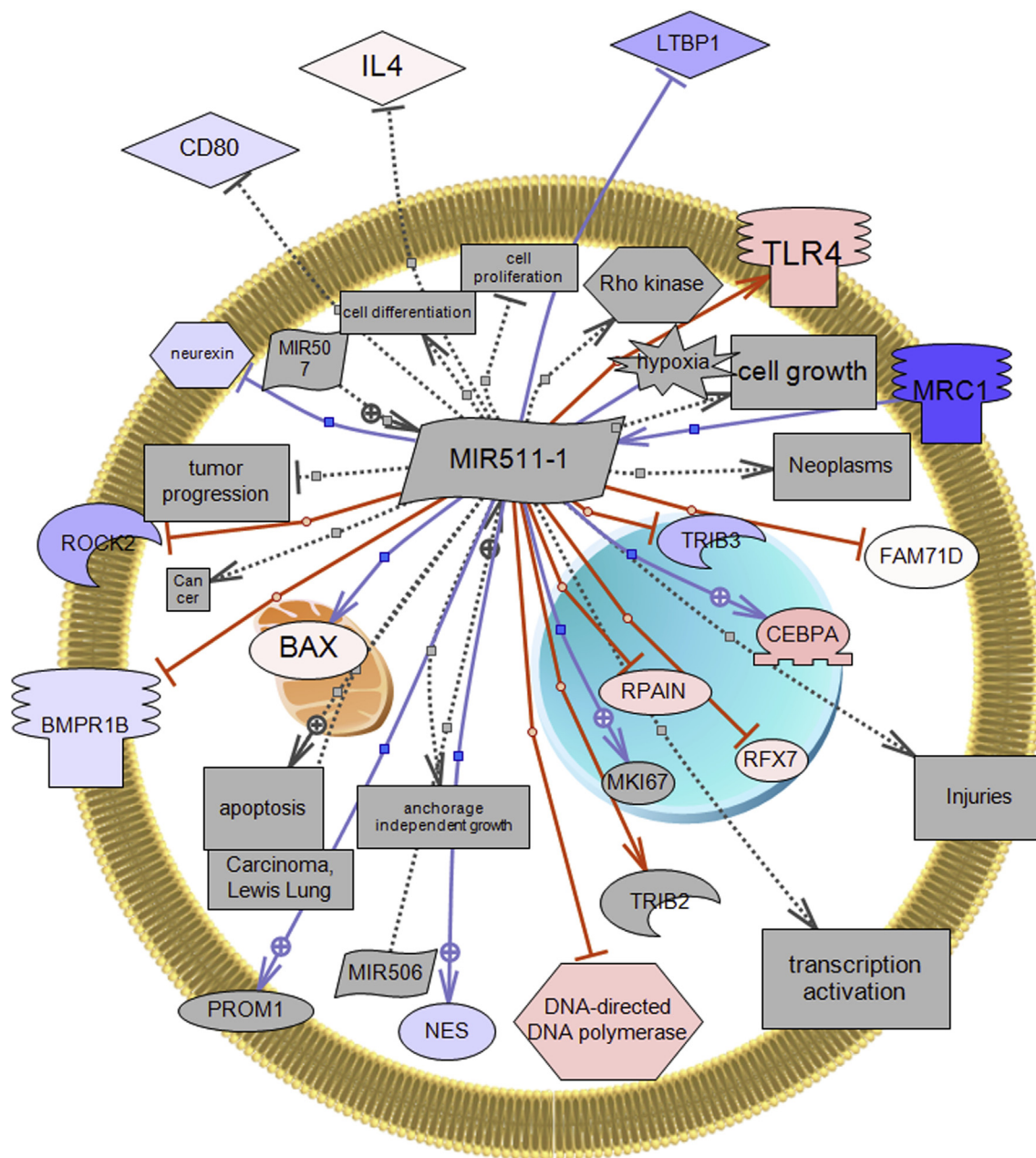


FIG E2. Interaction targets of miR-511-3p on Mrc1 networks. Interaction targets of miR-511-3p were identified by searching PubMed for all literature within the last 10 years and building up their connections by using the pathway studio 10 and rnen11 database. Blue, Downregulated by miR-511-3p; orange, upregulated by miR-511-3p.

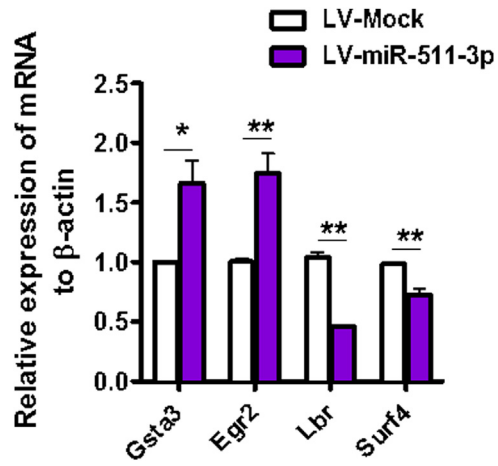
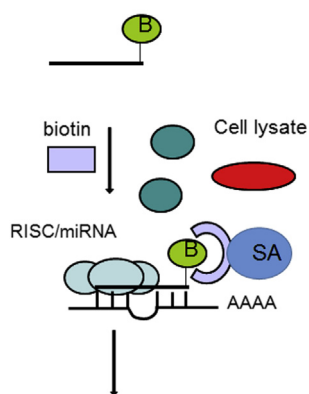


FIG E3. Validation in LV-miR-511-3-transfected BMDMs for the most upregulated or downregulated genes from gene array analysis ($n = 3$ samples per condition). Data represent means \pm SEMs. * $P < .05$ and ** $P < .01$.

Biotinylated miRNA: miR-511 or neg control



qPCR to identify bound mRNA

(compare mRNA bound by miR-511-3p vs neg control miRNA)

FIG E4. Affinity-based assay of miRNA to the *ptgds2* mRNA. A synthetic miR-511-3p containing a biotin at the 3' end (or a negative control biotinylated miRNA) was incubated with cell lysates of RAW cells stimulated with IL-4, and miRNA-mRNA complexes were isolated by using streptavidin beads. qPCR was performed to identify bound mRNA, and enrichment was analyzed by using miR-511-3p versus negative control miRNA. AAAAA, Polyadenylation; B, biotin; RISC, RNA-induced silencing complex; SA, streptavidin.

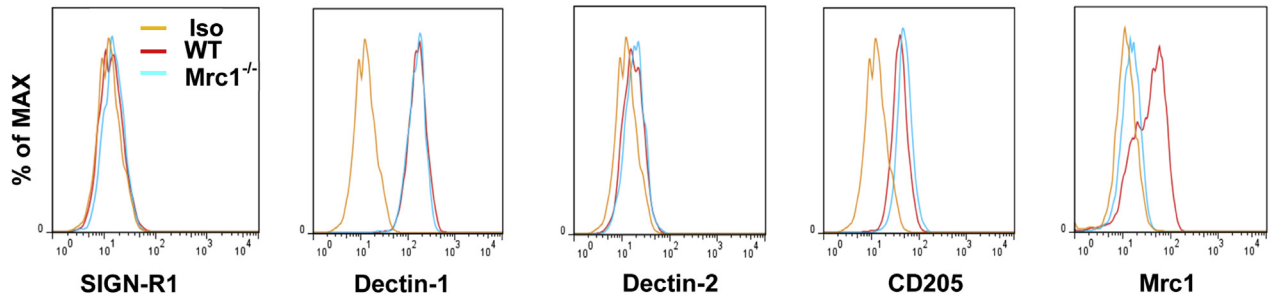


FIG E5. Expression of CLRs in lung macrophages from WT and *Mrc1*^{-/-} mice. Lung macrophages sorted from WT (red lines) and *Mrc1*^{-/-} (green line) mice by means of flow cytometry were stained with antibodies against isotype control (orange line) or CLRs (red lines; including *SIGN-R1*, *Dectin-1*, *Dectin-2*, *CD205*, and *Mrc1*).

TABLE E1. Interaction targets of the miR-511-3p identified by MedScan Reader 6

Name	z Ratio	Description
<i>Ltbp1</i>	-1.51	Latent transforming growth factor β binding protein 1
<i>Rock2</i>	-1.40	Rho-associated, coiled-coil containing protein kinase 2
<i>Tlr4</i>	2.20	Toll-like receptor 4
<i>Cebpa</i>	2.85	CCAAT/enhancer binding protein (C/EBP), α

TABLE E2. Clinical characteristics of the study subjects

Characteristic	Nonallergic nonasthmatic subjects (n = 12)	Allergic nonasthmatic subjects (n = 17)	Allergic asthmatic patients (n = 22)
Age (y)	38.5 ± 11.5	44.7 ± 17.6	42.1 ± 15.3
Sex, no. (M/F)	5/7	7/10	8/14
BMI (kg/m ²)	25.1 ± 5.0	27.8 ± 5.4	28.1 ± 5.9
Smoker, no. (%)	2 (16.7)	2 (11.7)	3 (13.6)
Allergic status, no. (%)	0 (0)	17 (100)	22 (100)
ACQ score	—	—	1.01 ± 0.59
Total IgE	—	—	334.5 ± 243.3
Peripheral blood eosinophils (cells/μL)	—	—	247.8 ± 163.4

Data for age, BMI, ACQ score, total IgE level, and eosinophil count are expressed as means ± SEMs.

ACQ, Asthma Control Questionnaire; BMI, body mass index.