**Supplementary Figure legends**

**Additional file 5: Figure S1. (A)** The number of PCa tissues andBPH in different staining index groups of ISH. **(B)** miR-154-5p expression levels in PCa tissues and the adjacent normal tissues (ANT) by analyzing the PCa miRNA sequencing dataset from TCGA (ANT, n = 52; PCa, n = 498). **(C)** miR-154-5p expression levels in 52 paired PCa tissues and the matching ANT by analyzing the PCa miRNA sequencing dataset from TCGA. **(D)** miR-154-5p expression levels in bone metastatic PCa tissues (PCa/BM) compared with that in non-bone metastatic PCa tissues (PCa/nBM) by analyzing the PCa miRNA sequencing dataset from TCGA. (PCa/nBM, n = 11; PCa/BM, n = 9). **(E)** Percentages and number of samples showed high or low miR-154-5p expression in PCa/nBM and PCa/BM in our PCa samples. **(F)** Percentages and number of samples showed high or low miR-154-5p expression in PCa/nBM and PCa/BM in PCa dataset from TCGA. **(G)** Percentages and number of samples showed high or low miR-154-5p expression in BPH and PCa tissues in our PCa samples. **(H)** Real-time PCR analysis of miR-154-5p expression levels in normal prostate epithelial cell (RWPE-1), primary PCa cell 22RV1, bone metastatic PCa cell lines (PC-3, C4-2B and VCaP) and brain metastatic cell line DU145 and lymph node metastatic cell line LNCaP. Transcript levels were normalized to *U6* expression. \**P* < 0.05.

**Additional file 6: Figure S2. Clinical correlation of miR-154-5p expression levels with clinicopathological characteristics in PCa patients.** **(A)** miR-154-5p expression levels in PCa tissues with different tumor volume as assessed by TCGA. **(B)** miR-154-5p expression levels in PCa tissues with different lymph node metastasis status as assessed by TCGA. **(C)** miR-154-5p expression levels in PCa tissues with different distant metastasis status as assessed by TCGA. **(D)** miR-154-5p expression levels in PCa tissues with different ISUP grade as assessed by TCGA.

**Additional file 7: Figure S3. (A)** Kaplan–Meier analysis of overall survival curves of PCa patients with high miR-154-5p expression (n = 72) versus low miR-154-5p expression (n = 73). **(B)** Kaplan–Meier analysis of overall survival curves of PCa patients with high miR-154-5p expression (n = 242) versus low miR-154-5p expression (n = 243) as assessed by TCGA. **(C)** Kaplan–Meier analysis of progression-free survival curves of PCa patients with high miR-154-5p expression (n = 221) versus low miR-154-5p expression (n = 222) as assessed by TCGA.

**Additional file 8: Figure S4. (A)** Real-time PCR analysis of miR-154-5p expression in the indicated PC-3, C4-2B and VCaP cells. Transcript levels were normalized by U6 expression. Error bars represent the mean ± s.d. of three independent experiments. \*P < 0.05. **(B)** Real-time PCR analysis of miR-154-3p expression in the indicated PC-3, C4-2B and VCaP cells. Transcript levels were normalized by U6 expression. Error bars represent the mean ± s.d. of three independent experiments. \*P < 0.05. **(C)** The effect of miR-154 overexpression on invasionand migration abilities in PC-3 and VCaP cells. Error bars represent the mean ± S.D. of three independent experiments. \**P* < 0.05. **(D** The effect of miR-154 overexpression or downexpression on invasionand migration ability in C4-2B cells. Error bars represent the mean ± S.D. of three independent experiments. \**P* < 0.05. **(E)** The effect of agomir-154-3p on invasionand migration abilities in PC-3 and VCaP cells. Error bars represent the mean ± S.D. of three independent experiments. \**P* < 0.05. **(F)** The effect of agomir-154-3p or antagomir-154-3p on invasionand migration ability in C4-2B cells. Error bars represent the mean ± S.D. of three independent experiments. \**P* < 0.05.

**Additional file 9: Figure S5. (A-H)** Gene set enrichment analysis (GSEA) revealed that miR-154-5p expression levels correlated with metastatic propensity.

**Additional file 10: Figure S6.** **(A and B)** GSEA revealed that miR-154-5p expression levels correlated with proliferation ability of cancer cells.

**Additional file 11: Figure S7. (A and B)** GSEA revealed that miR-154-5p expression levels correlated with AKT signaling activity.

**Additional file 12: Figure S8. (A)** AKT signaling inhibitors MK2206 inhibited the activity of AKT signaling in a dose-dependent manner in the indicated cells. Error bars represent the mean ± S.D. of three independent experiments. \**P* < 0.05, \*\**P* < 0.01 and \*\*\**P* < 0.001. **(B)** AKT inhibitors MK2206 (1 μM) attenuated the activity of AKT signaling in antagomir-154-5p-treated C4-2B cells.\*P < 0.05.

**Additional file 13: Figure S9. (A)** Predicted miR-154-5p targeting sequence and mutant sequences in 3′UTRs of EGFR, FGFR1, FGFR2, IGF1R, ERBB4 and INSR. **(B and C)** Real-time PCR analysis of EGFR, FGFR1, FGFR2, IGF1R, ERBB4 and INSR expression in the indicated VCaP and C4-2B cells. Transcript levels were normalized by GAPDH expression. Error bars represent the mean ± s.d. of three independent experiments. \*P < 0.05. **(D and E)** Luciferase assay of cells transfected with pmirGLO-3′UTR reporter of EGFR, IGF1R and FGFR1 in the indicated VCaP and C4-2B cells, respectively. \*P < 0.05.

**Additional file 14: Figure S10. (A-C)** ELISA analysis of EGF, bFGF, IGF1 and IGF2 concentration in the supernatant of the indicated cells.