Supporting Information

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Fig. S1. Immunoblot shows that male benign prostate hyperplasia (BPH-1) and prostate cancer (PC3) cells express the macrophage migration inhibitory factor receptor CD74. Whole-cell extracts of the cell lines were subjected to SDS/PAGE, blotted, and reacted with an anti-CD74 antibody.



Fig. S2. (A) Serum-starved BPH-1 cells were exposed to 1 ng/mL (80 pM) endotoxin-free *Trichomonas vaginalis* macrophage migration inhibitory factor (TvMIF) or human MIF (HuMIF) for 5, 10, or 15 min and probed to detect phosphorylated-ERK Thr202/Tyr204 (p-ERK1/2), total ERK1/2 (ERK1/2), and β -tubulin (Tubulin) loading control. (*B*) Serum-starved BPH-1 cells were exposed to 1 ng/mL endotoxin-free TvMIF or HuMIF for 15, 30, or 60 min and probed to detect phosphorylated Akt (p-Akt), total Akt (Akt), Bcl-2–associated death promoter (BAD) phosphorylated at ser136 (p-BAD), total BAD (BAD), and β -tubulin loading control. (*C*) Serum-starved PC3 cells were exposed to 1 ng/mL endotoxin-free TvMIF or HuMIF for 1, 2, or 4 h and probed to detect p-ERK1/2, ERK1/2, and β -tubulin loading control. (*Right*) Quantifications of mean fold increase of p-ERK1/2 levels (*D*) p-Akt (*E*), or p-BAD induction (*F*) in BPH-1 cells exposed to 1 ng/mL two exposures per experiment) ± SEM. (G) Mean fold increase from quantification of p-ERK1/2 levels from PC3 cells exposed to 1 ng/mL TvMIF or three independent experiments (two exposures per experiment) ± SEM.



Fig. S3. IL-6 secretion from human monocytes in response to MIF. Data shown are representative of two donors. Data are the mean of triplicates per one assay \pm SEM.

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