possible that B61 may also be responsible in part for the angiogenic activities of other proinflammatory factors.

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3. The B61-Ig chimera was made with the following primers generated by polymerase chain reaction: 5′ primer with a custom Nhe I site (underlined), CCG CGG CTA GCT GAT CGC CAC ACC GTC TTC TGG AAC AGT, and a 3′ primer with a Bam HI site (underlined), CTC GGG ATC CCT GTG ACC GAT GCT ATG TAG AAC CCG CAC. The control-Ig chimera was made as described (*19*). The amplified fragments were digested and cloned into Nhe I– and Bam HI–cut CD5-IgG1 vector [A. Aruffo, I. Stamenkovic, M. Melnick, C. B. Underhill, B. Seed, *Cell* **61**, 1303 (1990)]. The Ig chimeras were purified from pooled supernatants of transfected 293T cells as described (*19*).
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6. HUVECs were grown in 2% fetal bovine serum (FBS) without any exogenous growth factors for 48 hours prior to all of the following assays. The cells were metabolically labeled for 8 hours with ^{35}S-cysteine and ^{35}S-methionine as described [A. W. Opipari, M. S. Boguski, V. M. Dixit, *J. Biol. Chem.* **267**, 12424 (1992)]. Cells on 100-mm dishes were lysed on ice in lysis buffer containing 1% NP-40, 50 mM tris, and 150 mM NaCl in the presence of protease inhibitors [leupeptin (5 μg/ml), aprotinin (5 μg/ml), soybean trypsin inhibitor (50 μg/ml), and pepstatin (5 μg/ml)] for 30 min. The cells were then scraped, clarified by centrifugation, and the supernatants incubated overnight with the indicated antibody or chimera (10 μg per immunoprecipitation); 50 μl of a 50% slurry of protein A–Sepharose were added and the samples were incubated for 1 hour and then washed three times in lysis buffer. Sample buffer containing 2% β-mercaptoethanol was added, the samples were boiled for 5 min, and the eluted proteins were resolved on 10% SDS-polyacrylamide gels. To deplete Eck, we incubated the samples with 20 μg of anti-Eck followed by addition of protein A/G-Sepharose. In vitro kinase assays were done as described (*8*).
7. For immunoblotting, the cells were lysed in lysis buffer containing 1% NP-40, 50 mM tris, and 150 mM NaCl in the presence of protease inhibitors. Orthovanadate (1 mM) was included for Figs. 1C and 3A. After blocking overnight in 1% bovine serum albumin (BSA) in tris-buffered saline containing 0.1% Tween (TBS-T) at 4°C, the filter was incubated with anti-Eck (*8*) or 4G10 antibody to phosphotyrosine (UBI) at a concentration of 1 μg/ml. Bound primary antibody was visualized with the ECL kit (Amersham). Reprobing to detect Eck protein was done as described [A. Pandey, D. F. Lazar, A. R. Saltiel, V. M. Dixit, *J. Biol. Chem.* **269**, 30154 (1994)].
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10. Angiogenic activity was assayed in the avascular cornea of F344 female rat eyes (Harlan Laboratories, Madison, WI) as described (*20*). Briefly, each sample was combined with an equal volume of sterile Hydron casting solution (Interferon Sciences, New Brunswick, NJ), and 5-μl aliquots were pipetted onto the surface of 1-mm diameter Teflon rods (Dupont Co.) glued to the surface of a glass petri dish. The resulting pellets were air-dried in a laminar hood and refrigerated overnight. Just before implantation, the pellets were rehydrated with a drop of lactated Ringers solution and then placed in a surgically created intracorneal pocket ~1.5 mm from the limbus. Corneas were observed for a period of 7 days; the animals were then perfused with a colloid carbon solution and the corneas removed, flattened, and photographed.
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12. Chemotaxis was assayed as described (*14, 21*). Briefly, we prepared chemotaxis membranes (Nucleopore, 5-μm pore size) by soaking them sequentially in 3% acetic acid overnight and for 2 hours in gelatin (0.1 mg/ml). Membranes were rinsed in sterile water, dried under sterile air, and stored at room temperature for up to 1 month. Bovine adrenal gland capillary endothelial (BCE) cells, maintained in gelatin-coated flasks in Dulbecco's modified Eagle's medium (DMEM) with 10% FBS were used as target cells. Twenty-four hours before use, BCE were starved in DMEM with 0.1% BSA. Twenty-five microliters of cells suspended at a concentration of 1 × 10^6 cells/ml in DMEM with 0.1% BSA were dispensed into each of the bottom wells. A chemotaxis membrane was positioned on top of the bottom wells, and the chambers sealed, inverted, and incubated for 2 hours to allow cells to adhere to the membrane. Chambers were then reinverted, and 50 μl of test medium were dispensed into the top wells and reincubated for an additional 2 hours. Membranes were fixed and stained with Diff-Quick staining kit (Baxter Diagnostics Inc., McGraw Park, IL) to enumerate membrane-bound cells and cells that had migrated through the membrane to the opposite surface.
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17. Quiescent HUVECs were then treated with TNF-α (500 U/ml) or TNF-α plus anti-B61 (20 μg/ml, 30 min before addition of TNF-α and 3 and 5 hours after addition of TNF-α) and grown for 8 hours in 1% BSA (Fig. 3A). Eck was immunoprecipitated with anti-Eck (*8*) and antiphosphotyrosine immunoblotting was done as described (*7*). Polyclonal antibody to B61 was raised against recombinantly expressed human B61 and then affinity purified.
18. Quiescent HUVECs were treated as in (*17*) and then metabolically labeled for 8 hours in the presence of 1% BSA. Cell lysates were incubated with anti-B61 (3E6) (*2*) or anti-Eck (*8*) for 2 hours at 4°C. Immune complexes were precipitated by the addition of protein A/G-Sepharose, washed three times in lysis buffer, dissolved in SDS sample buffer, resolved by SDS–polyacrylamide gel electrophoresis under reducing conditions, and subjected to autoradiography.
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How Baseball Outfielders Determine Where to Run to Catch Fly Balls

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Current theory proposes that baseball outfielders catch fly balls by selecting a running path to achieve optical acceleration cancellation of the ball. Yet people appear to lack the ability to discriminate accelerations accurately. This study supports the idea that outfielders convert the temporal problem to a spatial one by selecting a running path that maintains a linear optical trajectory (LOT) for the ball. The LOT model is a strategy of maintaining "control" over the relative direction of optical ball movement in a manner that is similar to simple predator tracking behavior.

Even recreational baseball outfielders appear to know virtually from the moment of bat contact where to run to catch a fly ball. In this task, the ball's approach pattern renders essentially all major spatial location and depth cues unusable until the final portion of the trajectory. Cues such as stereo disparity, accommodation, image expansion rates, and occlusion help to guide final adjustments in the interception path (*1, 2*). During most of the task, the only usable information appears to be the optical trajectory of the ball (the changing position of the ball image relative to the background scenery). Conceivably, outfielders could derive the destination from an assumed projected parabolic trajectory, but research indicates that observers are very poor at using such a purely computational approach (*3*). In addition, factors such as air resistance, ball spin, and wind can cause trajectories to deviate from the parabolic ideal (*1, 4*).

One proposed model is that outfielders run along a path that simultaneously maintains horizontal alignment with the ball and maintains a constant change in the tangent of the vertical optical angle of the ball, tan α (Fig. 1) (*5–9*). As the ball rises, tan α increases, but at a rate that is a function of the running path selected. If the fielder runs too far in (so that the ball will land behind him), $\partial(\tan \alpha)/\partial t$ will increase. If he runs too far out (so that the ball will land in front of him), $\partial(\tan \alpha)/\partial t$ will decrease. The fielder can arrive at the correct desti-

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