Metastasis is the primary cause of death for cancer patients. Cyclooxygenase-2 (COX-2) is over-expressed in many cancers implicating the downstream prostaglandins (PGs) in metastatic progression. While PGE\textsubscript{2} production has been strongly correlated with metastatic disease, potential roles for the remaining PGs are unclear. We aimed to elucidate the actions of individual PGs on the migration potential of breast cancer cells that express COX-2. The *in vitro* migration capacity of MDA-MB-468 breast cancer cells that were engineered to stably express COX-2 was evaluated in 3D-matrigel droplets. In the presence of arachidonic acid (AA, 20 \( \mu \text{M} \)) inhibition of PGE\textsubscript{2} synthase by CAY10526 (20 \( \mu \text{M} \)) and inhibition of the prostacyclin synthase by U-51605 (3 \( \mu \text{M} \)) decreased cell migration relative to AA alone (79\( \pm \)9 versus 110\( \pm \)9 migrated cells, \( P<0.05 \) and 76\( \pm \)10 versus 110\( \pm \)9, \( P<0.05 \) respectively). Consistent with the apparent role for prostacyclin and PGE\textsubscript{2}, the IP receptor antagonist (CAY10441, 10 \( \mu \text{M} \), AA 20 \( \mu \text{M} \)) and EP4 receptor antagonist (L-161,982, 0.05 \( \mu \text{M} \), AA 20 \( \mu \text{M} \)), also decreased cell migration (45\( \pm \)10 versus 123\( \pm \)10, \( P<0.05 \) and 86\( \pm \)6 versus 123\( \pm \)10, \( P<0.05 \) respectively). In contrast antagonists of alternate prostanoid receptors did not influence tumour cell migration out of matrigel droplets. These findings implicate prostacyclin as well as PGE\textsubscript{2} in the metastatic activity of MDA-MB-468 breast cancer cells that over-express COX-2. Current investigations are focusing on potential mediators of increased cell migration caused by activation of IP and EP4 receptors.