<table>
<thead>
<tr>
<th>Title</th>
<th>Understanding the impact of angiopoietin-like protein 4 (ANGPTL4) on keratinocytes migration and proliferation using time lapse microscopy and live cell imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author(s)</td>
<td>Teo, Ziqiang</td>
</tr>
<tr>
<td>Citation</td>
<td>Teo, Z. (2008, March). Understanding the impact of angiopoietin-like protein 4 (ANGPTL4) on keratinocytes migration and proliferation using time lapse microscopy and live cell imaging. Presented at Discover URECA @ NTU poster exhibition and competition, Nanyang Technological University, Singapore.</td>
</tr>
<tr>
<td>Date</td>
<td>2008</td>
</tr>
<tr>
<td>URL</td>
<td><a href="http://hdl.handle.net/10220/8992">http://hdl.handle.net/10220/8992</a></td>
</tr>
<tr>
<td>Rights</td>
<td>© 2008 The Author(s).</td>
</tr>
</tbody>
</table>
Introduction
Skin wound healing involves both epithelia cell proliferation and migration, which are modulated by numerous secretory factors. Fasting-induced Adipose Factor (FIAF) was shown to be upregulated during wound healing, however its effects on migration and proliferation of keratinocytes remains unclear. By using immortalized form of keratinocytes (HaCaT) and FIAF-knockdown keratinocytes (HaCaT<sub>KD</sub>) with and without proliferation inhibitor (Mitomycin C), we aim to determine the effect of FIAF on keratinocyte migration and proliferation during wound healing.

Aims
To determine if FIAF is involved in the migration and proliferation during in vitro scratch wound

Results & Discussions
Subculturings/
Seedings of
cells
Incubate for 1
day to
confluency
Scratching
d of cells
Live cell imaging
using time-lapse
microscopy

Our results reveal that the deficiency in FIAF expression impairs cell migration. HaCaT cells migrate faster to close the in vitro wound when compared to HaCaTKD cells. In a separate experiment, Mitomycin C was added to determine the rate of migration of the cells independent of cell proliferation. Our findings showed that the migration rate of HaCaT cell was significantly faster HaCaTKD cells in the absence of cell proliferation, but still slower than cells under normal condition. These observations underscore the role of FIAF in both cell proliferation and migration during wound healing. Further studies will establish the molecular mechanism of its action.

Conclusion
Our results reveal that the deficiency in FIAF expression impairs cell migration. HaCaT cells migrate faster to close the in vitro wound when compared to HaCaTKD cells. In a separate experiment, Mitomycin C was added to determine the rate of migration of the cells independent of cell proliferation. Our findings showed that the migration rate of HaCaT cell was significantly faster HaCaTKD cells in the absence of cell proliferation, but still slower than cells under normal condition. These observations underscore the role of FIAF in both cell proliferation and migration during wound healing. Further studies will establish the molecular mechanism of its action.