Undergraduate Research Seminar
Wednesday, April 2nd 5:30 p.m.
Leigh 309

Matthew Boles

“Can corporations prevent transnational communication crises?”

With the increase in technology, news and information are disseminated to audiences in multiple countries by media outlets, netizens and nongovernmental organizations (NGO). Transnational organizations can no longer believe a communications crisis will stay in one country.

In October, 2012, an award-winning investigative news show, *Kalla Fakta*, broadcasted a 22-minute segment about the unethical practices of H&M in Cambodia. H&M, which is headquartered in Sweden, is the second-largest apparel chain in the world and has operations in 53 markets. News of the segment spread to other countries, both in and out of Europe, as governments requested to meet with H&M, petitions were created, and demonstrators organized “fashion mobs” in different cities.

H&M’s situation in Cambodia is an example of a Cross-National Conflict Shift (CNCS), which was first defined in the field of public relations by Molleda and Connolly-Ahern (2002). This presentation comes from a case study that analyzed how the news traveled, as well as the reactive strategies used by H&M to rectify the situation. After analyzing two of the six types of archival records described by Yin (1994) and communicating with an H&M public relations manager overseas, a thorough timeline was created so that an analysis could be performed.

Although a limitation to the case study is that it is not generalizable, other transnational organizations can still learn from the mistakes of H&M and be more proactive and communicate with its publics in multiple countries.

Lindsey Backman

“Oxidative stress shapes breast cancer phenotype through chronic activation of ATM-dependent signaling.”

Oncogenic transformation of normal mammary epithelium to invasive adenocarcinoma is understood to arise from a complex set of cellular and molecular alterations. Less understood is the nature of the stresses and forces that propel such alterations. Oxidative stress is known to be commonly dysregulated in cancer and is thought to be amongst the initiating insults leading to the onset of carcinogenesis. However, it is unclear how oxidative stress, and response to this form of cellular insult, may shape cancer phenotype.

Our research shows that the ATM-dependent NF-kB activation occurs through canonical DNA damage-responsive signaling, as knockdown of two proteins involved in this signaling pathway, ERC1 and TAB1, results in loss of both DNA damage-activated and basal activity of NF-kB. We further show that long-term culture of MDA-MB-231 cells on the antioxidant N-acetyl cysteine (NAC) and lowered amounts of reactive oxygen species (ROS) results in decreased NF-kB signaling and a dramatic effect on cellular phenotype, including acquisition of an epithelial morphology, upregulation of epithelial marker genes, and downreglation of mesenchymal marker genes. Moreover, these effects are attributable to downregulation of ATM-dependent signaling as RNAi-mediated ATM knockdown in MDA-MB-231 cells produced the same alterations in phenotype.

From these studies, we conclude that chronic activation of ATM-dependent signaling in response to persistent ROS insult promotes more aggressive cellular phenotype by continual activation of the oncogenic NF-kB transcriptional complex.