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Tularik Granted U.S. Patents For Novel Human Kinases

IKKα and hRIP Play Roles In An Important Cell Signaling Pathway For Inflammation

South San Francisco, Calif., June 12, 2001 – Tularik Inc. (Nasdaq: TLRK) today announced that it has been granted three United States patents relating to IKKα, a novel human kinase involved in important intracellular signaling pathways for inflammation. U.S. Patent Nos. 6,235,492, 6,235,512 and 6,235,513 cover polypeptides and nucleic acids relating to IKKα, as well as methods of high throughput screening.

IKKα, a component of human IκB kinase complex, is part of a biochemical relay system that triggers the body's response to injury and infection. Under normal circumstances, inflammation is a desirable defensive response to such threats. In its initial stages, the inflammatory response involves the recruitment of leukocytes, or white blood cells, from the circulatory system to the site of damaged, infected, or otherwise sensitized tissue. Excessive or prolonged accumulation of leukocytes is often unhealthy, however, and can result in a variety of unwanted inflammatory conditions such as asthma, inflammatory bowel disease, psoriasis and rheumatoid arthritis.

The discovery of IKKα is representative of the strength of Tularik's research in the inflammation field. Tularik scientists have taken a leading role in the discovery of regulatory proteins in pathways leading from cell-surface receptors for molecules, such as TNF (tumor necrosis factor), that carry inflammation "messages" to specific genes governing the inflammatory response. The issuance of the three patents relating to IKKα complements Tularik's intellectual property portfolio in the inflammation field, which includes several previously issued patents relating to IKKβ, another component of the IκB kinase complex.

Tularik's intellectual property portfolio in the inflammation area has also been enhanced in recent weeks by the receipt of U.S. Patent No. 6,211,337, which contains claims relating to a human Receptor-Interacting Protein (hRIP). The hRIP molecule is another protein kinase involved in signal transduction within the cell. Indeed, hRIP, like IKKα and IKKβ, is a component of the inflammation pathway that is activated by the binding

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of TNF to the surface of certain cells. In knockout gene studies in mice, it has been shown that when the gene for RIP is deleted, activation of the inflammatory response by TNF signaling is blocked.

Tularik holds over 65 issued U.S. patents and has over 80 other pending U.S. patent applications. Tularik believes many of the regulatory proteins in its intellectual property portfolio —now enhanced with the receipt of the recent patents relating to IKKα and hRIP— are important drug targets. The company believes that its discoveries and expertise in the inflammation area place it in a leading position to identify the next generation of anti-inflammatory drugs.

Tularik is engaged in the discovery and development of a broad range of novel and superior orally available drugs that act through the regulation of gene expression. Tularik programs address cancer, viral diseases, inflammation, immune disorders, lipid disorders, diabetes and obesity. Tularik has established strategic partnerships with Japan Tobacco Inc., Roche Bioscience and Knoll AG. For more information, visit Tularik's Internet website at www.tularik.com.

This press release contains "forward-looking" statements, among these statements about U.S. Patent Nos. 6,211,337, 6,235,492, 6,235,512 and 6,235,513. For this purpose, any statements contained in this press release that are not statements of historical fact may be deemed to be forward-looking statements. Words such as "believes," "anticipates," "plans," "expects," "will," "intent" and similar expressions are intended to identify forward-looking statements. There are a number of important factors that could cause the results of Tularik to differ materially from those indicated by these forward-looking statements, including, among others, risks detailed from time to time in Tularik's SEC reports, including its Company's Form 10-Q for the quarter ended March 31, 2001.

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