

VASTox plc
(“VASTox” or “the Company”)

**VASTox Presents Promising Results from Spinal Muscular Atrophy Drug
Discovery Programme at Leading Neuroscience Conference**

Oxford, UK, 17 October 2006 – VASTox (AIM: VOX), a leading UK biotechnology company, has presented exciting progress in its spinal muscular atrophy (SMA) drug discovery programme. VASTox’s Head of Biology, Dr Jon Tinsley, presented the data at the Society for Neuroscience annual meeting, Neuroscience 2006, being held in Atlanta, GA, USA, from the 14-18 October 2006.

The Company has discovered a number of promising ‘hits’ from a proprietary collection of drug-like molecules, which have been shown to improve the symptoms of SMA in an *in vivo* fruitfly (*Drosophila melanogaster*) screen designed to model the disease. The speed with which these hit molecules were identified by screening directly in a genetically-modified fruitfly is an important validation of VASTox’s innovative approach towards drug discovery. This progress will allow the Company to rapidly advance the SMA programme into the lead optimisation phase of pre-clinical development early in 2007, only 18 months after the programme was initiated.

SMA is a severe genetic neurological disease that causes a progressive loss of motor neurons in the spinal cord leading to severe muscle atrophy. SMA patients either do not acquire or eventually lose the ability to move and death occurs primarily as a result of fatal respiratory insufficiency.

SMA is the leading genetic cause of mortality in infants and toddlers in the World. It affects 1 in 6,000 newborns, an incidence comparable to that of other ‘common’ rare diseases, including Cystic Fibrosis, Duchenne Muscular Dystrophy and Sickle Cell Anaemia. There are an estimated 50,000 SMA sufferers in the developed World.

Steven Lee, PhD, CEO of VASTox said: “These results from our spinal muscular atrophy programme illustrate the ‘*in vivo* advantage’ of VASTox’s approach to drug discovery. By using fruitflies and zebrafish at the earliest stages of drug discovery, we are dramatically reducing the time and resources needed and we believe, significantly increasing the chances of producing a drug which is safe in man. SMA is a deadly genetic disease affecting children and our approach towards developing a therapy is both innovative and unparalleled.”

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About VASTox plc

VASTox is a biotechnology company that discovers and develops proprietary novel drugs and provides services to the pharmaceutical industry. The company's most advanced drug development programme is focused on developing a new treatment for Duchenne muscular dystrophy based on the up-regulation of utrophin. A second drug development programme for spinal muscular atrophy is also progressing rapidly. VASTox has four additional programmes focused on osteoarthritis, cancer, tuberculosis and stem cell therapies, which are expected to be out-licensed prior to entering the clinic.

The company's chemical genomics technology platform, which uses zebrafish and fruitflies, has the potential to dramatically decrease the time and cost of drug discovery and development. This is because using whole organisms allows VASTox to carry out high volume, high content screening, which delivers data that are highly predictive of the efficacy and toxicity of potential drug compounds in humans. VASTox is growing revenues based on marketing its unique technology platform and its chemistry expertise. The company listed on the AIM market of the London Stock Exchange in October 2004.

Further information about the company is available at www.vastox.com

Further information on Spinal Muscular Atrophy (SMA)

SMA is an autosomal recessive genetic disease caused by a defect in a single gene called SMN1. SMN protein encoded by this gene is critical to the survival and health of motor neurons. Without this protein, nerve cells atrophy, shrink and eventually die, resulting in the observed muscle weakness.

Approximately 1 in 40 people are carriers of the defective SMN1 gene and in order for a child to be affected by SMA, both parents must be carriers of the abnormal gene and both must pass this gene on to their child.