MCR remained low, at only 63% of baseline levels with a corresponding mean BBF which was roughly 1.7X baseline, demonstrating a reperfusion phenomenon. Calculated bladder compliance over the entire filling curve correlated directly with BBF ($p = .025$); i.e. low compliance was associated with low bladder blood flow.

Conclusions: Human bladder blood flow and microcirculatory resistance vary with the degree of bladder filling and the phase of the filling/emptying cycle. In the empty state, despite low intravesical pressure, bladder microcirculatory resistance and blood flow are at their maximum and minimum values respectively. Once filling commences, microcirculatory resistance falls and correspondingly, blood flow increases. When the bladder is filled beyond 75% of its maximum capacity, detrusor pressure rises and microcirculatory resistance is subsequently increased, lowering blood flow. These observations suggest that elevations in bladder wall tension only become significant in reducing BBF when the bladder is near maximal capacity. Acutely after bladder drainage, microcirculatory resistance falls, allowing reperfusion in the collapsed state. Our studies also demonstrated a close correlation between decreased bladder wall compliance and decreased bladder blood flow.

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Title (type in CAPITAL LETTERS, leave one blank line before the text):
CYSTOMETRIC EVALUATION OF BLADDER FUNCTION IN NON-ANESTHETIZED MICE WITH AND WITHOUT BLADDER OUTLET OBSTRUCTION

Aims of Study
Transgenic knockout mice may be a useful tool for examination of the molecular mechanisms underlying various forms of bladder dysfunction.
There are several published reports on cystrometry in mice both without and with bladder outlet obstruction (1-4). However, these investigations have been performed in anesthetized animals, which precludes information on active micturition. Development of a reliable and reproducible cystometric model in the non-anesthetized mouse is desirable.
The aim of the present study was to develop a model for cystrometric study of bladder function in the awake mouse, and to characterize urodynamically and immunohistochemically the normal and infravesically obstructed mouse bladder.
Methods
Normal Balb/CJ mice, and mice with bladder outlet obstruction after surgical, partial ligation of the urethra underwent continuous cystometry as previously described for rats (5). Bladders were also investigated by immunohistochemistry.
Results
During the period of cystometry, reproducible micturition patterns were obtained. Bladder overactivity could be evoked by intravesical administration of capsaicin and prostaglandin E2, and by subcutaneous apomorphine. Marked differences in the urodynamic parameters between normal and obstructed mice were revealed. In mice subjected to urethral obstruction, micturition pressure ($p<0.05$), threshold pressure ($p<0.05$), bladder capacity ($p<0.001$), micturition volume ($p<0.001$), and residual volume ($p<0.05$) increased significantly. There was no difference in basal pressure or compliance between normal and obstructed mice. Non-voiding bladder activity was consistently recorded in obstructed mice; both frequency and amplitude increased significantly ($p<0.01$). Compared to normal bladders, obstructed bladders showed hypertrophy of the bladder wall and various degrees of ìpatchy denervationî of the detrusor.
Conclusions
Continuous cystometry can be reproducibly performed in awake, freely moving normal mice and mice with bladder outflow obstruction. The changes induced by

infravesical obstruction in mice were similar to those previously found in rats. This model may be useful for investigations of genetically modified mice.

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Title (type in CAPITAL LETTERS, leave one blank line before the text):

THE DISTRIBUTION OF $P2X_1$ AND $P2X_3$ RECEPTORS IN THE RAT AND HUMAN URINARY BLADDER

Aims of the Study Adenosine 5'-triphosphate (ATP) is well recognized as a neurotransmitter in smooth muscle preparations [1,2,3]. There is evidence to show that ATP both causes bladder contractions [1,2,4] and may have a sensory role in processing physiological information in the urinary bladder [5]. These effects are likely to be mediated by P2X receptors [2,4], namely $P2X_1$ and $P2X_3$, respectively. This study set out to investigate their distribution using subtype-specific antibodies to localise these receptors in the rat and human urinary bladder.

Methods Sections of rat and human urinary bladder, the latter obtained from male donor subjects, were incubated with antibodies to $P2X_1$ and $P2X_3$ receptors. Antibodies to the sensory neuropeptide, calcitonin gene-related peptide (CGRP) were used to identify sensory neurones in the rat [6] and human urinary bladder. Colocalisation studies with the CGRP and $P2X_3$ receptor antibodies were also performed.

Results $P2X_1$ receptor immunoreactivity was found on detrusor muscle fibres of both species. $P2X_3$ receptor immunoreactivity was mainly found in the urothelium and labelling was also seen in the suburothelial layers of the rat and human urinary bladder. The sensory innervation of the urinary bladder of both species was shown using the antibodies to CGRP. No clear evidence for colocalisation of CGRP and $P2X_3$ immunoreactivity was seen in the urinary bladder of either species.

Conclusion This study has confirmed the presence of $P2X_1$ receptors on the detrusor muscle of the rat [7] and human urinary bladder. Interestingly, $P2X_3$ receptors were found on urothelial cells, the first demonstration of a non-neuronal localisation for $P2X_3$ receptors. No clear evidence was found for the presence of $P2X_3$ receptors on CGRP-containing nerves and therefore $P2X_3$ receptors may not mediate the sensory response to ATP in the urinary bladder.

References:

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