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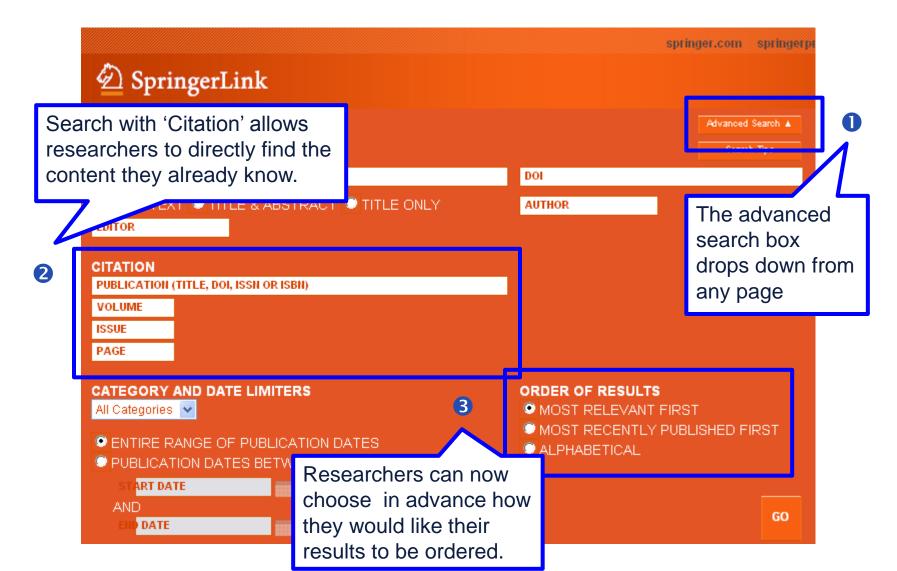
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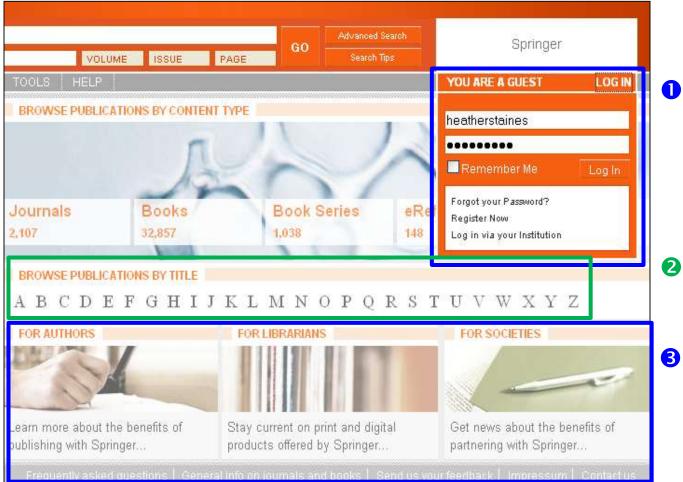
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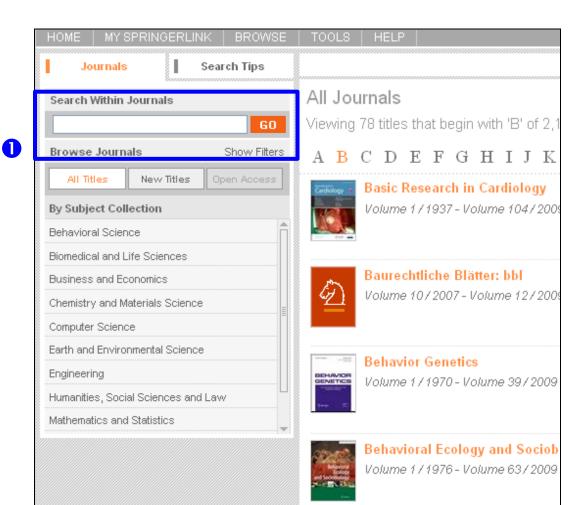


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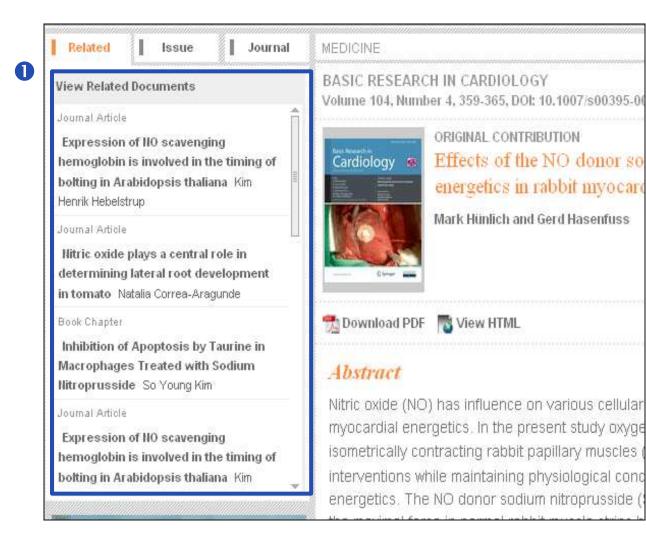
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<b>Abstract</b> Our previous studies have shown that morphine withdrawal induced hyperactivity of cardiac noradrenergic p purpose of the present study was to evaluate the effects of morphine withdrawal on site-specific tyrosine hyp phosphorylation in the rat left ventricle. Dependence on morphine was induced by a 7-day s.c. implantation pellets. Morphine withdrawal was precipitated on day 8 by an injection of naloxone (2 mg/kg, s.c.). TH phosp determined by quantitative blot immunolabelling using phosphorylation state-specific antibodies. Ninety mit administration to morphine-dependent rats there was an increase in phospho-Ser40-TH (139.0 ± 13%, $P <$ Ser31-TH (135.5 ± 11%, $P < 0.05$ ) in the left ventricle which is associated with both an increase in total TH le (114.4 ± 4.6%, $P < 0.05$ , $P < 0.01$ ) and an enhancement of TH activity (51.0 ± 11 dm/µg protein, $P < 0.001$ ). V (40 nmol/day), inhibitor of cyclic AMP dependent protein kinase (PKA) was infused, concomitantly with morp diminished the increase in noradrenaline (NA) turnover, total TH expression (95.76 ± 4.1 %, $P < 0.01$ ) and T phosphorylation at Ser40 (85.5 + 11%, $P < 0.01$ ) in morphine-withdrawn rats. In addition, we showed that the	droxylase (TH) of morphine ohorylation was n after naloxone 0.05) and evels Vhen HA-1004 hine, it 'H

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Plants contain three classes of hemoglobin genes of which two, class 1 and class 2, have a structure similar to classical vertebrate globins. We investigated the effect of silencing the class 1 non-symbiotic hemoglobin

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