Metformin is a cationic antihyperglycaemic agent, which is mostly excreted into urine as an unchanged form. Although metformin undergoes tubular secretion, the molecular mechanisms responsible for the tubular transport of metformin have not been clarified. Our recent study demonstrated that hOCT2 is the most abundant organic cation transporter expressed in the basolateral membrane of human kidney proximal tubules (Motohashi et al., J. Am. Soc. Nephrol. 13(4): 866-874, 2002). In the present study, we characterized the transport of metformin in hOCT2-expressing HEK 293 cells by the tracer uptake method. The uptake of [14C]metformin was markedly stimulated by hOCT2-expressing cells. The accumulation of [14C]metformin was concentrative and was dependent on the membrane potential, showing consistency with the functional characteristics of hOCT2. The apparent Km and Vmax values of [14C]metformin transport by hOCT2-expressing HEK293 cells were 1.38 mM and 11.9 nmol/mg protein/min, respectively. The transport of [14C]metformin was inhibited slightly or moderately by some cationic drugs at respective therapeutic concentrations. These results suggest that hOCT2 should be a key factor regulating tubular secretion of metformin in the human kidney.