In the past several years, knowledge about changes in the dynamic state of the living body by genetic polymorphisms has accomplished rapid progress. Particularly remarkable is the progress in the research on the polymorphism of transporters and metabolic enzymes as shown in the examples of the polymorphisms of transporters and related diseases in Table 1. On the other hand, we have understood from clinical experiences that not all of these polymorphisms always cause pharmacokinetic or pharmacodynamic changes. Therefore, I planned to arrange the latest information on clinical evidence of the pharmacokinetic changes by genetic polymorphism of transporters and metabolic enzymes in this Theme Issue.

This Theme Issue invited four groups of authors, world-leading scientists, to update predictions of the relationship between the genetic polymorphism of transporters and metabolic enzymes, and the clinical events. The first review is “Impact of Genetic Variation in OATP Transporters to Drug Disposition and Response” by Drs. Inna Y. Gong and Richard B. Kim (University Hospital, Ontario, Canada). They summarized the genetic polymorphism of OATP transporters, especially OATP1B1, OATP1B3, OATP2B1 and OATP1A2. They mentioned the relevance of genetic variations in OATPs to drug efficacy and optical therapeutics. The second article is “Polymorphic Transporters and Platinum Pharmacodynamics” by Drs. Jason A. Sprowl, Rachel A. Ness, and Alex Sparreboom (Department of Pharmaceutical Sciences, St. Jude Children’s Research Hospital, Tennessee, USA). They addressed an overview of various transporters that have shown promise in patient populations in facilitating the movement of platinum-based agents across cell membranes, and have also shown how their functions are associated with drug disposition or pharmacological effects. The third is “Impact of Variations in CYP2C Subfamily Genes on the Pharmacokinetics of Clinically Useful Drugs” by Drs. Takeshi Hirota, Shunsuke Eguchi and Ichiro Ieiri (Kyushu University, Japan). They described the genetic polymorphisms of CYP2C, since many studies have shown the clinical importance of these polymorphisms. The final article is “Clinical Evidence of the Pharmacokinetic Changes in Thalidomide Therapy” by Dr. Katsunori Nakamura (Nagoya City University, Japan) and his co-authors. Whereas thalidomide induces multiple birth defects when used in pregnant females, this drug returned to the market in the treatment of several myelomas and erythema nodosum leprosum in Japan. Dr. Nakamura and his co-authors focused on the metabolism of thalidomide and summarized the newest information about the genetic polymorphism of CYP.

I believe that the papers in this Theme Issue are helpful in understanding the current status of clinically relevant effects of genetic polymorphisms in drug transporters and drug-metabolizing enzymes. I sincerely thank all the authors for their contributions. I also cooperated with Dr. Yoshimichi Sai (Department of Pharmacy, Kanazawa University Hospital), Dr. Kousei Ito (Department of Pharmacy, The University of Tokyo Hospital) and Dr. Miki Nakajima (Faculty of Pharmaceutical Sciences, Kanazawa University) in planning and editing this Theme Issue. I would like to give my heartfelt thanks to them.

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