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Chapter 17
Network Pharmacology: An Emerging Area in Anti-Cancer Drug Discovery

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Following the publication of Chap. 17 by Asfar S Azmi titled 'Network Pharmacology: An Emerging Area in Anti-Cancer Drug Discovery.' In: systems biology in cancer research and drug discovery. Edited by A S Azmi. Pub Springer ISBN 978-94-007-4819-4, it has been brought to our attention that the acknowledgements section was incorrectly printed and there are two references missing from the bibliography that should have also been cited in the text.

The acknowledgements section should have appeared as:

"A substantial proportion of the content of Sect. 4 of this chapter is taken verbatim from the course materials prepared by Dr A V Whitmore, Dr J Wray and Professor M P Young of e-Therapeutics PLC for the Molecular Medicine Tricon Conference 2012, Drug Discovery and Development Channel, De-risking Drug Discovery: SC4 Network Pharmacology, and their contribution is gratefully acknowledged."

The text was also reproduced in another editorial by Dr. Azmi titled 'Network pharmacology for cancer drug discovery: are we there yet?' in the May 2012 issue of Future Medicinal Chemistry (Future Med. Chem. 4 [8], 939–941 [2012]).

The following two references should have been included in the reference list:

Young MP, Zimmer S, Whitmore AV (2012) Drug molecules and biology: network and systems aspects. In: Morphy JR, Harris CJ (eds) Designing multi-target drugs, RSC drug discovery series, vol 21. Royal Soc. Chem, pp 32–49

The online version of the original chapter can be found at
http://dx.doi.org/10.1007/978-94-007-4819-4_17

Azmi AS (2012) Network pharmacology for cancer drug discovery: are we there yet? (in the May 2012 issue of Future Medicinal Chemistry). Future Med Chem 4(8):939–941

The following paragraph ending on line 248, page 399–400 should have cited these two references and appeared as shown below:

Networks are amenable to analysis using several branches of mathematics. The most simple way of representing a biological network is through graph points, more commonly termed nodes or vertices which could be either genes, proteins or even drugs connected by lines representing interactions (called edges). Local and global properties of this map can be evaluated and neighboring substructure or the role of adjacent nodes can be inferred from information in patterns of connections/interactions. This information can be used to identify sets of high value nodes, some of which may serve as targets for drugs, depending on the model of interest. Drugs generally exert their effects through binding to proteins thereby modulating their activity. However, bioactive compounds invariably influence more than one protein, either (a) as a consequence of structural similarities between the intended target and other proteins, (b) through allosteric effects on other proteins, (c) through pleiotropic mechanisms, where an interaction results in multiple downstream effects on other proteins, (d) or through multivalent target binding by different presentations of the active molecule. Many pro-drugs are also converted to active metabolites and these are subject to an exponential range of possible interactions within a target network or distantly un-related network proteins. In whatever way these polyvalent interactions occur, the end result is often unpredictable efficacy and toxicity, or both. It is important to know that the more highly specific and the less promiscuous a drug is for a particular cancer target, the more important that target must be in the cancer network for it to have a significant effect (Young et al. 2012; Azmi 2012).

The authors of this chapter and the editor of this book would like to sincerely apologize for any inconvenience or confusion this may have caused our readers.