FOLIA MENDELIANA 49/2
Supplementum ad Acta Musei Moraviae *XCVIII, 2013*

CHAPERONES AND EVOLUTION

FILIP TRCKA, BORIVOJ VOJTESEK, PETR MULLER
Regional Centre for Applied Molecular Oncology, Masaryk Memorial Cancer Institute,
Zluty kopec 7, 656 53 Brno

ABSTRACT – Phenotypic robustness is enabled by several mechanisms masking detrimental genetic variations in individuals. These mechanisms involve gene duplication and functional redundancy of cellular pathways. Recently the role of Hsp90 chaperone system was suggested in the process of buffering genetic variation on the protein level. This activity of chaperones has a profound effect on the accumulation of large numbers of phenotypically silent genotypes and thus may play an important role in evolution.

INTRODUCTION

The incorporation of Mendelian genetics and population biology into Darwin's theory of natural selection is referred to as The Modern Synthesis. The major principles of The Modern Synthesis are that populations containing genetic variation (arising by random mutation) evolve by changes in gene frequency caused by genetic drift and especially natural selection and that the most adaptive genetic variants have individually slight phenotypic effects so that phenotypic changes are gradual. The principal of gradual phenotypic change has been (at least in some cases) challenged by another evolutionary model referred to as Punctuated Equilibrium, in which long period of stasis are followed by rapid phenotypic alternations. Nevertheless, the 'substrate' for driving force of evolution - natural selection - is phenotypically manifested genetic variation. Empirically, organisms are remarkably resistant to extrinsic factors and are able to develop unaffected phenotypes even in rapidly changing environments (DE VISSER *et al.*, 2003). This means that 'hidden' genetic variation with no apparent phenotypic manifestation is accumulating in organisms during evolution.

BUFFERING GENETIC VARIATION

There are several mechanisms masking or buffering genetic variation in organisms and thus enabling astonishing phenotypic robustness of individuals under changing environmental conditions. An increasing gene dosage represents the first mechanism. This can be accomplished by increasing ploidy or by duplication of chromosomal regions or individual genes. Gene dosage mechanisms ensure that newly mutated genes are backed up by their unmutated copies. Indeed, only 1% of essential yeast genes have a homolog elsewhere in the genome, compared to 8.5% of non-essential genes (GIAEVER *et al.*, 2002). The second mechanism for buffering genetic variation is the existence of genetic epistasis. Essential cellular processes are accomplished by parallel molecular pathways, where mutation of a single gene affecting one of the pathways is neutralized by functionality of