

DAY 1

Keynote Forum



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Berlin, GermanyHarpal S Buttar, J Heart Cardiovasc Res 2019, Volume 3
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Recent trends and advancements in the prevention of mortality and morbidity associated with cardiovascular diseases

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Health care burdens associated with premature mortality and morbidity due to non-communicable diseases such as cardiovascular diseases (CVDs), respiratory disorders, obesity, diabetes mellitus, cancers, anemia, musculoskeletal abnormalities, and neurodegenerative problems are escalating worldwide. Though these diseases generally manifest in middle age and beyond, it is now recognised that roots of these diseases lie in childhood and adolescence. The conventional risk factors of CVDs consist of hypertension, hyperlipidemia, atherosclerosis, and hyperglycemia. Lifestyle factors including tobacco use, lack of exercise, unhealthy dietary habits, and low socioeconomic status contribute heavily to the development of obesity, diabetes and CVDs in children and adults. Sugar-loaded beverages and excessively salted foods are also potential risk factors. Diets rich in whole grains, fruits and vegetables, olive oil, fish, low-fat dairy products, probiotics, and moderate wine consumption are linked with lower incidence of CVDs. Lifestyle modifications such as regular physical activity (about 30 min/day), restriction of caloric and sodium intake, smoking cessation and moderate alcohol consumption are recommended for improving cardiovascular health and quality of life. Ingestion of phytosterol-enriched foods, micronutrients (vitamins, minerals), and amino acids assist to improve overall health beyond basic nutritional functions. Emerging

evidence suggests that dietary supplements containing flavonoids and antioxidants modulate gene and protein expression and thereby modify endogenous metabolic pathways and homeostasis, and consequently reduce the risk of CVDs and chronic diseases multifactorial in origin. Given the scope and prevalence of CVDs, a cost effective population health strategy - 'prevention is better than cure' - would be the most appropriate model to adopt to curb CVD-related mortality/morbidity and to reduce health care cost.

Biography

Dr. Buttar received his degree in Veterinary Medicine in 1961 from the Punjab University, Chandigarh, India. Before coming to Canada, he was Lecturer for about 2 years in the Department of Pharmacology (1961-1963) in his first alma mater, College of Veterinary Medicine, Hissar. In January 1964, he was awarded an overseas scholarship by the University of Alberta, Edmonton, Alberta, Canada, where he completed his MSc and PhD degrees in Pharmacology in 1966 and 1970, respectively. After a post-doctoral stint at the Wayne State University, Detroit, Michigan, USA, he joined as a Research Scientist level-1 (August 1971) at the Health Protection Branch, Ottawa, and was promoted to the rank of Research Scientist level-5 in April 1997 (highest scientific rank in the Federal Govt.). Since May 1994-Present, he has held cross-appointment of Adjunct Professorship in the Department of Pathology & Laboratory Medicine, Medical College, University of Ottawa, Canada. Previously, he also held the positions of Adjunct Professor in the Faculty of Pharmaceutical Sciences, University of British Columbia, Vancouver, and at the Memorial University of Newfoundland, St.

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John's, Canada. He is also a scientific consultant to the Institute of Cardiovascular Sciences, Faculty of Medicine, University of Manitoba, Winnipeg, Canada. In June 2013, Dr. Buttar was appointed Visiting Professor in the Department of Pharmacology, School of Medicine, Democritus University of Thrace, Alexandroupolis, Greece. He is currently serving as Visiting Professor in the Faculty of Pharmaceutical Sciences, Guru Nanak Dev University (GNDU), Amritsar, Punjab, India.

At GNDU, Professor Buttar teaches a special course in Basic & Clinical Pharmacology, and helps the MPharm students in doing in vitro and in vivo studies and designing experimental protocols to evaluate the therapeutic potential of drugs and plant-derived products in animal models

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Which cells are involved in cardiomyogenesis in mammalian and zebrafish heart?

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There is no clear evidence on which cells are able to renew the adult mammalian myocardium. Studying cardiomyogenesis in the heart of newborn mammals and adult zebrafish, many researchers have concluded that new cardiomyocytes (CMs) are formed by dedifferentiation and division of pre-existing mature CMs. In addition, it is supposed that mature CMs of zebrafish are divided throughout life, not only renewing the myocardium, but also regenerating it after injury. In turn, it has been shown that mature CMs of mammals are divided only in the first 5-7 days after birth, and then permanently lose this ability. Investigating the phenomenon of intracellular development of resident cardiac stem cells (CSCs) with the formation of "cell-in-cell structures" (CICSSs), we've found that transitory amplifying cells (TACs), being released after CICSSs opening, are 2 times larger than the original CSCs (12-13 μm vs. 5-6 μm) and are able to divide and differentiate. We observed the presence of CICSSs not only in the myocardium of adult mammals, but also in 18-day-old embryos and the neonatal rats. We found that CICSSs, formed in the embryonic phase, not only provide TACs for embryonic cardiomyogenesis, but, opening immediately after birth, release large numbers of proliferating TACs to support neonatal cardiomyogenesis. Counting the number of mitotic cells and measuring their size showed that only small cells

with $D < 13 \mu\text{m}$ are able to divide in the neonatal period. After that, their proliferation stops, and they transit from hyperplasia to hypertrophy. We demonstrated that adult myocardium of *Danio rerio* also contains CICSSs. Upon opening, they release a large number of TACs, the dimensions of which are comparable to the dimensions of cells that divide inside the myocardium of newborn mammals. We assume that specifically TACs, but not mature CMs, form new CMs in mammals and zebrafish throughout life.

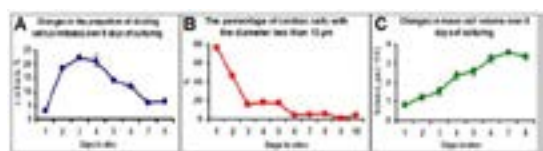


Figure 1: Growth of myocardial cells obtained from newborn rat heart in vitro. Proportion of dividing cells (A), proportion of cells with $D < 13 \mu\text{m}$ (B) and mean cell volume (C).

Recent Publications

1. Belostotskaya G B and Golovanova T A (2014) Characterization of contracting cardiomyocyte colonies in the primary culture of neonatal rat myocardial cells: A model of in vitro cardiomyogenesis. *Cell Cycle* 13(6):910-

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2. Belostotskaya G, Nevorotin A and Galagudza M (2015) Identification of cardiac stem cells within mature cardiac myocytes. *Cell Cycle* 14(19):3155-3162.
 3. Filippov S K, Sergeeva O Yu, Vlasov P S, Zavyalova M S, Belostotskaya G B, Garamus V M, Khrustaleva R S, Stepanek P and Domnina N S (2015) Modified hydroxyethyl starch protects cells from oxidative damage. *Carbohydrate Polymers* 134:314–323.
 4. Belostotskaya G B, Golovanova T A, Nerubatskaya I V and Galagudza M M (2018) Discovery of the phenomenon of intracellular development of cardiac stem cell – a new step in understanding of biology and behavior of tissue-specific stem cells. In the book “Evolutionary Physiology and Biochemistry: Advances and Perspectives”, Chapter 5:45-60.
 5. Belostotskaya G B, I V Nerubatskaya and M M Galagudza (2018) Two mechanisms of cardiac stem cell-mediated cardiomyogenesis in the adult mammalian heart include formation of colonies and cell-in-cell structures. *Oncotarget* 9:34159-34175.

Biography

Galina B Belostotskaya graduated from Leningrad State University and defended her thesis on a specialty “Radiobiology”. During late 15 years she has been studying the physiology of skeletal muscle cells and the behavior and cardiomyogenic potential of resident cardiac stem cells. Being the head of investigations she released 7 specialists and 2 graduate students. The works have been supported by the 11 Russian grants.

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