

S5-3 "Public - Private sector partnership for human development -A case study of Iodine Deficiency Disorders elimination program

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Background

Moving from pilot projects to national programs and effective and sustainable implementation of national programs in micronutrient malnutrition involves decisions with implications for health status, service delivery and resource allocation. These decisions in turn require detailed consideration of a number of demographic factors. In reviewing these factors, this presentation will use the case study of Iodine Deficiency Disorders Elimination Programs (IDDEP).

Objective

To understand the process require to move from pilot projects to effective and sustainable implementation of national programmes related to Iodine Deficiency Disorders Elimination.

Methodology

Experiences related to Iodine Deficiency Disorders Programme from countries of the South Asian Region over a period of last 40 years have been synthesized based on various national, regional, country reports, conferences, meetings, published and unpublished reports, conferences, meetings, published and unpublished reports, site visits & in depth the discussions with national regional programme managers alongwith representatives of relevant sectors involved in formulation of programming and its implementation.

Results

Some of the salient factors that would determine the sustainability of IDDEP are political commitment; financing and resource allocation; identification of a focal point (Program Manager), partnership with private sector; scientific and technical consensus on assessment, intervention strategies, quality assurance, monitoring and evaluation; legislation and enforcement; institutional and individual capacity enhancement with focus on continuing training and supervision; advocacy, social marketing and communication; collaboration / integration with other national program activities, in addition to health sector; community participation, involvement of NGO's etc. Supported by periodic inter-country and regional meetings and on-going interaction with bilateral and international agencies and NGO's at national, regional and global level, the sustainability of IDDEP would be ensured.

Conclusion

The lessons learnt from these would be applicable to other micronutrient programs and possibly to other nutrition and health programs.

S6-2 **Leydig cell insulin like-hormone (InsL3): Genetic structure and function in male reproduction**

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Using techniques for isolation of differentially expressed genes, we isolated a testis-specific gene. Sequencing analysis showed that the cDNA of this novel gene has homology with insulin-like hormones and is specifically expressed in Leydig cells of fetal and adult testis and in the theca cells of postnatal ovary and therefore designated as Leydig cell insulin-like hormone (InsL3). The InsL3 gene is expressed at high levels in the adult testis and at much lower levels in the adult ovary. Analysis of InsL3 transcripts in testis and ovary throughout the pre-and postnatal life of the mouse revealed a sexual dimorphic pattern of InsL3 expression during development. No InsL3 transcripts were detected in female embryos of any stage, whereas in male embryos transcripts were first detected at 13.5 dpc. After birth, the level of InsL3 transcription in testis remains constant during the first 3 weeks, increases at the time at which the first wave of round spermatids undergoes spermatogenesis, and reaches the highest level in adult testis. These results led us to suggest that the InsL3 factor plays an essential role in differentiation and maintenance of male phenotype and spermatogenesis. To determine the role of InsL3 in sexual differentiation and spermatogenesis, we have generated mice containing a targeted disruption of InsL3 gene using homologous recombination. Morphological abnormalities were only observed in male mutant mice, which exhibited cryptorchid testis located high in abdomen. These malformations are due to failure of gubernaculum development during embryogenesis. No sperm was found in the testis of InsL3 mutant mice due to an arrest in spermatogenesis.