“Construction of a complex functional system, such as a living organism, requires not only raw building materials (genes encoding structural and other functional proteins), but also an assembly program, organized into flexible feedback and feed-forward sub-routines that can function within, and readily adapt to a non-stable environment.” This article describes the discovery of a new pan-ontogenic mechanism, Integrative Nuclear FGFR1 Signaling (INFS), which underwrites gene programming during development. Genetic experiments have indicated that the \textit{fgfr1} gene sits on top of the gene hierarchy that governs gastrulation, as well as the subsequent development of the major body axes, nervous system, muscles, and bones, by affecting downstream genes that control the cell cycle, pluripotency and differentiation, as well as microRNAs. The regulatory control exerted by INFS is due to a single protein, the nuclear isoform of FGFR1 (nFGFR1), which integrates signals from development-initiating factors and operates at the interface of genomic and epigenomic information. nFGFR1 cooperates with a panoply of transcription factors, and targets thousands of genes encoding mRNAs, as well as miRNAs in critical ontogenic networks. nFGFR1 binds to promoters of ancient proto-oncogenes and tumor suppressor genes which serve as switches in cell proliferation, binds and regulates the pluripotency core genes as well as metazoan morphogens that delineate body axes, construct the nervous system and the mesodermal and endodermal tissues. The pan-ontogenic gene programming by INFS feed-forward and feedback loops expands our understanding of ontogeny, the roots of cancer, schizophrenia and other developmental diseases, and holds new promise for reconstructive medicine and cancer therapy.