Insulin Resistance in children and adolescents after bone marrow transplantation for malignancies. In reply

Haematologica 2006; 91:(10)e143

We thank d'Annunzio and colleagues for their interest in our letter and for bringing to our attention their data concerning the pancreatic function in children treated for malignancies.

In their cross sectional study (period of observation 9 months-10 years), conducted on a small population of children and adolescents treated for acute or chronic leukaemia with chemotherapy (CMT), radiotherapy (RT) and bone marrow transplantation (BMT), the authors show increased levels in insulin resistance (HOMA IR, QUICKI) and insulin secretion (HOMA beta cell function). These results are captivating but are different to ours, as we have shown an impairment of glucose metabolism in children with acute lymphoblastic leukaemia (ALL) treated only with CMT, and reversing of the later observed through a long term follow up after completion of therapy.

The main difference between the two study populations is the BMT which might explain this apparent contrast. In fact, bone marrow transplantation experiments showed that most of the extrapancreatic proinsulin-producing cells originate from the bone marrow.[1]

Furthermore recent studies in normal mice have suggested that transplanted bone marrow cells can transdifferentiate into pancreatic beta cells at relatively high efficiency, although there is no unanimous consensus in the human cells.[2]

These novel findings might explain the increase of HOMA beta cell function after BMT found by d'Annunzio and coll. Furthermore it should be interesting to follow up this patients long-lasting in order to establish the persistence of the improvement of beta cell secretion and/or a possible impairment.

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References

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