MODULATORY ROLE OF D-CHIRO-INOSITOL (DCI) ON LH AND INSULIN SECRETION IN OBESE PCOS PATIENTS WITH OR WITHOUT FAMILIAR DIABETES
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It is well known that patients with PCOS have the occurrence of a mixture of the many endocrine impairments typical for the syndrome. The metabolic impairment seems to be a relevant aspect that needs to be solved since the compensatory hyperinsulinism that 65% of PCOS patients affects negatively the menstrual as well as the reproductivity ability of these patients. Life-style changes (i.e. exercising, dieting, etc) have a relevant role on body mass index (BMI) though a clear improvement have been demonstrated by the use of insulin sensitizers such as metformin as well as by some specific integrators such as inositol. In fact, growing evidences suggest that a deficiency of d-chiro-inositol (DCI) containing IPG might be at the basis of insulin resistance and of the reactive compensatory hyperinsulinemia, frequent in PCOS patients. It is relevant to know that DCI is synthesized by an epimerase that converts MYO into DCI and that, depending on the specific needs of the two molecules, each tissue has a typical conversion rate. Indeed several studies report the beneficial effects of both MYO and DCI on hyperinsulinemic PCOS patients, though it has not been clarified whether specific clinical evidence might suggest when to use one or the other.

We studied a group of obese PCOS patients (BMI > 26, n=24), with hyperinsulinemia, before and under d-chiro-inositol administration (Chorofol 500, LJPharma, Italy) at the dose of 500 mg every day per 12 weeks. Patients underwent to oral glucose tolerance test before and after treatment. When insulin response was evaluated, the significant reduction of insulin response to glucose load was observed (p<0.01) with a consistent reduction of the maximal insulin response (~38%). When patients were subdivided according to the presence or absence of 1 or more diabetic relatives, the resultas were more interesting since PCOS patients with familiar diabetes had a 50% change of maximal response of insulin to glucose load while PCOS patients with no familiar diabetes showed a 30% (p<0.001).

In conclusion these data support the fact the PCOS patients have a clear impairment at the level of the endocellular (post receptor) signaling where IPG plays a crucial role and such impairment is related to the lack of epimerase-induced conversion of MYO to DCI. PCOS patients with familiar diabetes show a higher impairment, maximally corrected by DCI administration.