

the lipogenic response to insulin coincide in fat animals and not in lean animals.

The phase relations of both prolactin and insulin rhythms as well as the rhythms of tissue responses to the hormones are important elements in the regulation of lipogenesis. Phase malfunctions in these and perhaps other rhythms may also account for insulin resistance.

It is apparent that various modification and changes can be made without departing the spirit and scope of this invention.

Having described the invention, what is claimed is:

1. A method for modifying and regulating at least one of the lipid and glucose metabolism in a vertebrate animal or human subject in need of such treatment comprising:

administering a dopamine agonist to said subject on a times daily basis at a first predetermined time of day in a first dosage amount sufficient to reduce the hormonal prolactin level in the blood of said subject; and additionally

administering a prolactin stimulator to said subject on a time daily basis at a second predetermined time of day in a second dosage amount sufficient to increase hormonal prolactin level in the blood of said subject;

continuing said administrations for periods of time sufficient to modify and reset on a long-term basis the subject's daily prolactin cycle to cause it to mimic the low level and peak of the daily prolactin cycle of a lean healthy member of the same species, thereby achieving in said subject at least one of the following modifications in lipid and glucose metabolism: decrease in insulin resistance, reduction in fat stores, suppression of hyperinsulinemia and reduction of hyperglycemia.

2. The method of claim 1 wherein said subject is a human and wherein said administration are continued for periods of time sufficient to modify and reset on a long-term basis the subject's daily prolactin cycle to cause it to mimic the low day level and the nightly time peak of the daily prolactin cycle of a lean healthy human.

3. The method of claim 1 further comprising discontinuing said administrations after lapse of said periods of time, said modifications persisting over the long-term after cessation of said administrations.

4. The method of claim 1 wherein the dopamine agonist and prolactin stimulator are administered respectively on timed daily bases to said subject in amounts and for periods of time sufficient to modify and reset on a long-term basis the daily cycles of both prolactin and glucocorticosteroid in said subject.

5. The method of claim 1 wherein the timed daily dosages of the dopamine agonist and prolactin stimulator are given daily, once a day, over a period ranging from about 10 days to about 150 days, the dopamine agonist being administered in an amount within the range from about 3 micrograms to about 100 micrograms, per pound of body weight, and the prolactin stimulator being administered in an amount within the range from about 10 micrograms to about 100 micrograms per pound of body weight.

6. The method of claim 1 wherein said subject is a human and the timed dosages of the dopamine agonist are given daily, once a day, in an amount within the range from about 3 micrograms to about 20 micrograms, per pound of body weight, and the timed dosages of the prolactin stimulator are given daily, once a

day, in an amount ranging from about 10 micrograms to about 100 micrograms, per pound of body weight.

7. The method of claim 6 wherein the subject is an obese human and the dopamine agonist is given daily at a time ranging from about 2 hours to about 8 hours after the time at which the prolactin concentration peaks in a lean human to modify and reset the lipid metabolism of the obese human to that of a lean human.

8. The method of claim 1 wherein the dopamine agonist is selected from the group consisting of 6-methyl-8-beta-carbobenzyloxy-aminoethyl-10 alpha-ergoline; 1,6-dimethyl-8-beta-carbobenzyloxy-aminoethyl-10 alpha-ergoline; 8-acylaminoergolines; ergocornine; 9,10-dihydroergocornine; bromocriptine, and D-2-halo-6-alkyl-8-substituted ergolines.

9. The method of claim 1 wherein the prolactin stimulator is selected from the group consisting of metoclopramide, haloperidol, pimozide, phenothiazine, sulpiride, chlorpromazine and serotonin agonists.

10. The method of claim 1 comprising further administering to said subject thyroid hormone, daily, in an amount within the range from about 0.1 milligrams to about 0.4 milligrams.

11. A method for modifying and resetting the neutral phase oscillations of the brain which control prolactin levels in the bloodstream of a vertebrate animal or human subject, the method comprising administering a dopamine agonist and a prolactin stimulator to said subject on a daily basis at different times predetermined to produce a change of the plasma prolactin rhythm of said subject to mimic that of a lean insulin-sensitive subject, said administrations continuing for periods of time sufficient to increase the cellular sensitivity of the subject to insulin and reduce hyperglycemia, said increase and reduction persisting after cessation of said administrations.

12. The method of claim 11 wherein the dopamine agonist and prolactin stimulator are administered to the subject to also reduce hyperinsulinemia.

13. A method for modifying and resetting the neutral phase oscillations of both the prolactin and glucocorticosteroid rhythms in an obese insulin resistant vertebrate animal or human subject, which comprises

administering a dopamine agonist to said obese insulin-resistant subject on a daily basis at a time of day just after the time that the prolactin level will reach its daily peak in the blood of a lean insulin-sensitive subject, in a dosage amount ranging from about 3 micrograms to about 20 micrograms, per pound of body weight, and additionally

administering a prolactin stimulator to said obese insulin-resistant subject on a daily basis at a time of day just before the prolactin level will peak in the blood of a lean insulin-sensitive member of the same species, in a dosage amount ranging from about 10 micrograms to about 100 micrograms, per pound of body weight, and continuing the administrations over periods of time ranging from 10 days to 150 days sufficient to at least begin reducing the fat deposits in the body of the obese insulin-resistant subject to that of the lean insulin-sensitive member, such that on cessation of said administrations the fat deposits in the body of the treated subject, will continue to be reduced until they correspond substantially with that of the lean insulin-sensitive member, said substantial correspondence being thereafter maintained on a long-term basis.