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Treatment with Corticosteroids in Pediatrics: Advances and Problems

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Abstract

This short communication aims to briefly evaluate the current use of corticosteroids (CS) in pediatrics, considering the periodization of early postnatal ontogeny, the phenomena of programming/imprinting and embedding, as well as phylo- and ontopathogenic models. It is concluded that pathophysiological and molecular mechanisms of higher sensitivity to at least some adverse effects of CS, like growth retardation should be studied in much more details, in order to find the ways for their counteraction.

Keywords: Glucocorticoids; Growth; Ontogeny; Corticosteroids; Immunosupressive Agents.

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Introduction

This story has begun in the decade of fourties of the last century, together with discovery, chemical synthesis and first clinical use of corticosteroids (CS). More than 75 years later, we have rather complicated general picture, full of success and some failures at the same time. This short communication has a principal aim of evaluating current use of CS in pediatrics, based on our previous and numerous articles on this topic in open-access journals where interested readers can find citations and references too many original papers of other authors.

Why is this topic so important? At present CS are widely used in various areas of medicine including pediatrics, principally as anti-inflammatory and immunosupressive agents. Their use in pediatric clinics is particularly high in pulmonar and respiratory medicine [1, 2], as well as for the treatment of leukemias [3]. However, CS are also potent hormonal agents and therefore, they are deeply involved in endocrine regulation. Moreover, the role and use of CS may be changing during perinatal and postnatal development; therefore, it is very important to discuss at first the periodization of early postnatal ontogeny.

Stages of Perinatal and Early Postnatal Development

Earlier we have studied postnatal somatic growth in humans and rats; showing that both these species have, at least, two developmental transitions revealed by growth plots in mono- and bilogarithmic coordinates [4]. Juvenile transition occurs in both sexes during the age of 6-8 years in humans and approximately at the age of 3 weeks in rats, whereas pubertal transition takes place during the age of 12-14 years in humans (earlier in females) and approximately at the age of 5 weeks in rats (again slightly earlier in females). Previously we suggested also that juvenile (and not pubertal) transition may be considered as principal transformation from development to aging, because of proximity in both sexes to the minimal value of mortality ini human populations (9-10 years of age, for example in Brazil).

It is interesting that in rats juvenile transition roughly coincides with the period of weaning. Therefore, perhaps it is not surprising that in humans one more transition, infantile one occurs at the age of 1-2 years, also close to weaning in this species. Moreover, recently we proposed that infantile transition in humans can be used as a boundary that separates programming / imprinting from

embedding phenomena [5], to be discussed in details in the next part of this manuscript.

Phenomena of Programming / Imprinting and Embedding

When at the end of last century a group of English epidemiologists headed by David J.P. Barker has demonstrated that low birthweight corresponds to higher risk of several chronic non-infectious disorders 6 decades later (see discussion in [6, 7]) these studies were rapidly followed by a confirmation of epidemiological data in various countries on several continents, as well as by numerous works on experimental models using laboratory animals, thus establishing solid bases for the concept of developmental origins of health and disease (DOHaD).

In particular, biomedical investigations have shown the existence of programming / imprinting phenomena in perinatal development, when adverse factors (undernutrition, psychosocial stress, infections, even exogenous CS in excess) can provoke long-term consequences lasting at least till adult state and perhaps, even until the senescence.

At present, several authors focus the principal attention of DOHaD research area in the first 1000 days of human life, including prenatal development and the first 2 years of life after birth. It is curious that the later boundary of this period roughly coincides with infantile transition revealed in our previous studies, what has given us an opportunity to put the programming / imprinting phenomena before the human age of 1-2 years (see Introduction). In this regard, it is highly pertinent that David Barker and his colleagues have outlined that not only birthweight, but also the weight at the age of 1 year can serve as indicating the predisposition to chronic non-infectious diseases in later life.

What about the phenomena of embedding, their existence was considered by several authors (including the late Bruce S. McEwen) that outlined their important characteristic of occuring in cumulative mode, in accord with the paradigm of allostatic load and overload [8]. Let us discuss now the evidence concerning CS.

The Role of Corticosteroids Ini the Phenomena of Programming / Imprinting and Embedding

At first the research groups of Simon Langley-Evans and Jonathan Seckl have shown the important role of CS in programming / imprinting phenomena in experiments on rats (see discussion in [9, 10]). Thereafter many other authors have confirmed and extended these data. Here we should outline that principal attention of researchers was attracted at first to prenatal CS action, in order to correspond to epidemiological data of David Barker on lower birthweight as a consequence of intrauterine growth restriction.

However, in our own experiments on rats we used mainly neonatal CS administration, showing much more expressive somatic growth retardation, as compared to prepubertal treatment [11, 12]. It is interesting that these data in vivo corroborated higher sensitivity of cultured pituitary cells from neonatal rats to inhibitory CS action on DNA and total protein synthesis, as compared to cells from prepubertal and adult animals [13].

However, it is essential to remember here that because of difference in the velocity of maturation between rats and humans, neonatal rats correspond to human fetus in the 3rd trimester of pregnancy and even better to premature human newborn [6]. Therefore, our data obtained in experiments on neonatal rats were discussed by us mainly in relation to the use of CS in premature human infants, in order to enhance their lung maturity [14].

It is interesting that according to our unpublished data, administration of CS to lactating mother rats have not provoked any change in somatic growth of their offspring. This corrresponds perfectly to well established safety of CS use by lactating women, as referred to their offspring, because of rather low infant exposure to CS in maternal milk [15,16].

However, direct systemic administration of CS to infants, children and adolescentes has well-known inhibitory action on somatic growth [17], although in our experimental models younger rats were more sensitive to inhibitory CS action on the growth of target organs of immune and endocrine systems than older animals [18].

In this regard, earlier we have offered to use new terms of pharmacotoxicologic programming / imprinting and embedding, in order to describe adverse CS effects, especially in pediatrics [8, 19]. Obviously, in no way we try to promote steroid phobia, considering also well-established safety of topical CS use, including inhalatory and intranasal administration. Nevertheless, we would like to put forward firmly that on our opinion, it is not prudent to ignore the notable adverse actions of systemically administered CS. Moreover, earlier we have gathered bibliographic evidence of several antistress agents that can be employed for diminishing unfavourable CS effects [7, 20-22].

Conclusion

In conclusion, general rules of CS use in pediatrics appear to be well established, but many investigative efforts are still necessary for better understanding the age-related diferences in CS actions, especially as referred to the phenomena of programming / imprinting and embedding. In this regards, we would like to attract particular attention to phylo- and ontopathogenic models that evaluate respectively inter-, multi- or transgenerational transfer of disease risk, i.e. across generations and the establishment of etiopathogenic mechanisms along the whole ontogeny that includes both pre- and postnatal development until adult state, continuing through middle-age categories to the senescence [19, 23].

In any case, as for David Barker and many other researchers of DOHaD area, our main goal is invariably the same: to promote better health and well-being for the current, as well as future generations.

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