REVISTA ESPAÑOLA DE FISIOLOGIA, 26, 147-150. 1970

Plasma Iron in Domestic Fowl *

J. Planas

Laboratory of Animal Physiology. Faculty of Sciences. University of Barcelona
Barcelona (Spain)

(Received on October 11, 1969)

J. PLANAS, *Plasma Iron in Domestic Fowl.* R. esp. Fisiol., 26, 147-150, 1970.

The plasma iron and the total iron binding capacity of the serum have been determined on a series of 273 specimen comprising 5 species. The laying period causes a notable increase in plasma iron without there being any perceptible modification in the total iron binding capacity. It is suggested that in fowl would must contein an auxiliary mechanism to transferrin in serum iron transport. Its existence would explain diverse peculiarities observed in the transportation of serum iron in fowls, in comparison with the situation existing in mammals.

The study of plasma iron in fowls has been the subject of early publications (13, 15, 16, 17, 19), in which it was analysed its concentration and transport mechanism.

Other authors had touched on this subject previously. Thus, MARSHALL and DEUTSCH (9) analysed this problem and pointed out the presence of conalbumin, the egg-white protein, in the serum and for the first time suggested its relationship with the transportation of iron.

RAMSAY and CAMPBELL (22) studied the metabolism of iron in laying hens and demonstrated the increase in plasma levels during this period in view of the fact that the egg production causes a daily loss of 0.5 mg. HALKETT et al. (5) also studied this problem by way of Fe-59 and also showed that iron deposited in the egg and the iron used in the haemoglobin synthesis depend on the same plasma pool.

KLEIN (7) studied the metabolism of iron in ducks with the aid of Fe-59 but paid greater attention to its incorporation in the red blood corpuscles.

The aspect dealing with the transportation of the iron in the plasma is outside the interest of the majority of these authors and the publications that have been found dealing with the serum iron content in fowls are few and far between and very hard to compare (8, 26).

This paper provides new data on plasma iron in fowls and the transport mechanism and they are used as the basis of an overall study.

Materials and Methods

The determination of plasma iron and the total iron binding capacity has been performed according to RAMSAY's methods (20, 21).

In general, samples of heparinised blood taken from the radial vein have been ana-

* This work was supported by the «Fomento de la Investigación en la Universidad», Ministerio de Educación y Ciencia.

lysed. The determinations have been performed individually, using a total volume of 0.5 to 0.7 ml. The photocolorimetric readings have been taken in microcells of a «Spectronic 20».

Results

The plasma iron values and total iron binding capacity for a total of 273 specimens are given. These specimens correspond to 5 species of domestic birds (Table I) and the species *Gallus domesticus* was the most widely analysed (143 specimens).

In the table each species is divided according to sex and differentiated according to approximate age and state of lay.

Discussion

Certain interespecific differences are noted in the plasma iron which are significant in the case of the dove. In the remaining species, it is possible to note an increase with age and, especially, with the state of lay. This last observation was first indicated by RAMSAY and CAMPBELL (22) with reference to the hen and confirmed on the same species (15) and observed in other birds (19).

But the most outstanding fact noted is that during the laying period, the values of plasma iron are higher than the values of the total iron binding capacity of the serum (Table I); thus, the values of the saturation coefficient of transferrin are, in these cases, over 100 % and the direct or

Table I. *Plasma Iron (PI), Total Iron-Binding Capacity (TIBC) and Saturation Coeficient (SC) in Certain Domestic Birds.*
Average values and standard deviations. Age: *a*) 1 ½ month; *b*) 4 month; *c*) prelaying period; *d*) laying period; *e*) adult non-laying; *f*) adult.

SPECIES	MALES				FEMALES			
	Nº	PI µg Fe %	TIBC µg Fe %	SC %	Nº	PI µg Fe %	TIBC µg Fe %	SC %
CHICKEN (Gallus domesticus)	23[a] *	102 ± 22	165 ± 65	67	23[a] *	129 ± 77	262 ± 69	51
	15[b]	156 ± 28	228 ± 30	69	24[b]	143 ± 53	246 ± 70	50
	4[f]	197 ± 33	275 ± 29	71	21[c]	196 ± 65	221 ± 99	89
					16[d] *	500 ± 165	272 ± 85	198
					17[d]	674 ± 136	360 ± 87	187
TURKEY (Meleagris gallopavo)	10[f] *	70 ± 48	302 ± 36	25	11[e] *	105 ± 45	315 ± 46	33
	18[f]	152 ± 41	356 ± 129	42	22[e]	174 ± 50	291 ± 90	59
					2[c]	650 ± 100	483 ± 15	135
					2[d]	990 ± 90	483 ± 15	205
DUCK (Anas platyrhynca)	6[f] *	159 ± 110	634 ± 120	25	6[e] *	132 ± 40	472 ± 108	28
					19[d] *	1065 ± 286	504 ± 131	227
GOOSE (Anser anser)	7[f] *	163 ± 38	547 ± 60	30	6[e] *	160 ± 23	561 ± 80	28
					1[d] *	1260 ±	705 ±	178
PIGEON (Columba livia)	10[f] **	250 ± 53	288 ± 53	88	10[e] **	225 ± 41	288 ± 39	78

*Data from reference 16; ** Data from reference 17

indirect measuring of the latent binding capacity is null (23).

In vitro studies with additions of iron overloads (12) show how the fowl sera may always transport much more iron that is theoretically indicated by the latent capacity of the transferrins.

All of this has suggested to us the possibility of there existing in fowls a second protein fraction, auxiliary to the transferrin or that in this group the transferrin can transport a larger amount of iron joined to the protein molecule in a much more labile way, so that it may be picked up by the $MgCO_3$ used in RAMSAY's method (21).

We have thought that conalbumin could be this protein, because of its identical properties and similar constitution (28, 29) and behaviour to iron (28). Its presence in fowl serum was already pointed out by MARSHALL and DEUTSCH (9) and by KAMINSKI and DURIEUX (6).

Both are glucoproteins differentiated only by their prosthetic group and in the sequency of the amino acids in a polypeptide chain; they are an example of prosthetic allomerism. One same gene controls the synthesis of the transferrin in the liver and of the conalbumin in the oviduct, whereby a polymorphism of both proteins coexists not only hens (4, 11, 24) but also in other species (2, 25).

The conalbumin content in the serum of hens and other birds evaluated immunologically (10, 18) enables one to attend to the increase of plasma iron transport in the laying period and could explain the inversion found in the values of plasma iron and the total iron binding capacity.

In pure solutions it have been checked (3) how the $MgCO_3$ picks up partially the iron attached to the conalbumin-iron complex and to a lesser extent to the transferrin-iron complex.

The effect of the magnesium carbonate in the serum is less noticeable and at the concentration indicated by RAMSAY (21), validity of the results may be relied on. These results have been controlled by us frequently by other methods.

Therefore, the existence of a new protein fraction in fowls appears reasonable. This fraction would cooperate with the transferrin and enable coverage of the increase of plasma transportation which in hens causes a daily excretion of 0.5 mg Fe in the production of eggs (5).

The accuracy of our working hypothesis may be questionable, but is quite evident as shown by *in vitro* work (12, 14), since the fowl sera always bind much more iron than they can theoretically transport in accordance with their unsaturated binding capacity, when in mammals the sera always abide by the theoretical forecasts. The existence of a new protein, assistant in iron transport, could explain these notable differences in transporting capacity of the serum between these two groups of vertebrate animals.

ALI and RAMSAY (1) have apported some proofs to consider the non-transferrin iron in the plasma of laying hen might be bound to phosphoprotein phosvitin which have also iron-binding properties and the magnesium carbonate is able to remove all the iron bound to this phosphoprotein.

References

1. ALI, K. E. and RAMSAY, W. N. M.: *Biochem. J.*, **110**, 36P, 1968.
2. BAKER, C. M. A.: *Comp. Biochem. Physiol.*, **20**, 949, 1967.
3. BALASCH, J. and PLANAS, J.: *R. esp. Fisiol.*, **24**, 169, 1968.
4. CROIZIER, C.: *Ann. Biol. anim. Biochem. Biophys.*, **6**, 379, 1966.
5. HALKETT, J. A. E., PETERS, T. and ROSS, J. E.: *J. Biol. Chem.*, **231**, 187, 1958.
6. KAMINSKI, M. and DURIEUX, J.: *Exp. Cell. Res.*, **10**, 590, 1956.
7. KLEIN, J. R.: *Am. J. Physiol.*, **196**, 656, 1959.
8. KOLB, E. and SCHIMMEL, D.: *Naturwissenschaften*, **49**, 397, 1962.

9. MARSHALL, M. E. and DEUTSCH, H. F.: *J. Biol. Chem.*, **189**, 1, 1951.
10. MARTÍN-MATEO, M.ª C. and PLANAS, J.: *R. esp. Fisiol.*, **21**, 1, 1965.
11. OGDEN, A. L., MORTON, J. R., GILMOUR, D. G. and MCDERMIC, E. M.: *Nature*, Lond., **195**, 1026, 1962.
12. PLANAS, J.: *Nature*, Lond., **215**, 287, 1967.
13. PLANAS, J., and BALASCH, J.: *R. esp. Fisiol.*, **25**, 157, 1969.
14. PLANAS, J., and BALASCH, J.: *R. esp. Fisiol.*, **25**, 195, 1969.
15. PLANAS, J., and CASTRO, S.: *R. esp. Fisiol.*, **16**, 197, 1960.
16. PLANAS, J., CASTRO, S. and RECIO, J. M.: *Nature*, Lond., **189**, 668, 1961.
17. PLANAS, J. and COCHO, D.: *R. esp. Fisiol.*, **18**, 115, 1962.
18. PLANAS, J. and MARTÍN-MATEO, M.ª C.: *R. esp. Fisiol.*, **21**, 9, 1965.
19. PLANAS, J. and RECIO, J. M.: *R. esp. Fisiol.*, **16**, 265, 1960.
20. RAMSAY, W. N. M.: *Clin. Chim. Acta*, **2**, 214, 1957.
21. RAMSAY, W. N. M.: *Clin. Chim. Acta*, **2**, 221, 1957.
22. RAMSAY, W. N. M. and CAMPBELL, E. A.: *Biochem. J.*, **58**, 313, 1954.
23. RODRÍGUEZ, R. and PLANAS, J.: *R. esp. Fisiol.*, **20**, 83, 1964.
24. STRATIL, A.: *X^e Congrés européen sur les groupes sanguins et le polymorphisme biochimique des animaux*, París, julio 1966, pp. 241, 1966.
25. STRATIL, A. and VALENTA, M.: *Folia Biol.*, Praha, **12**, 307, 1966.
26. WARBURG, O. and KREBS, H. A.: *Biochem. Z.*, **190**, 147, 1927. Cited by Buddenbrok «Vergleichende Physiologie», Vol. 111, página 609; Birkhäuser, Basel, 1956.
27. WARNER, R. C. and WEBER, I.: *J. Am. Chem. Soc.*, **75**, 5094, 1953.
28. WILLIAMS, J.: *Biochem. J.*, **83**, 355, 1962.
29. WILLIAMS, J.: *Biochem. J.*, **108**, 57, 1968.