mass compared to surface area available for heat loss results in a precarious juggling between the production of heat and its dissipation to the environment. This is manifested in the wide diurnal variations in body temperature seen in the small birds. In the small mammals, with a less efficient insulation, a body temperature as high as that of the small birds apparently cannot be maintained. This is evidenced in the hypothermic response of small rodents to infection or to the injection of foreign proteins (*6*), which in larger animals leads to a febrile response. This poorly developed homeothermism is also seen in the facility with which the body temperature of small mammals falls and a state of hibernation ensues on exposure to cold. By contrast, animals such as the bear with a large ratio of mass to surface area have only a slight fall in body temperature on entering the hibernating state (*1*).

A number of physiological measurements such as heart rate, cardiac output, and oxygen consumption have been correlated with weight (*4*). However, body temperature also play a significant role in the determination of these functions. This is illustrated by the fact that the mouse has a heart rate of about 670, while the canary, also weighing about 20 g, has a heart rate of 1000 per min. The mouse has a normal body temperature of about 37° C, while the body temperature of the canary is about 44° C. We have recently shown that in some species, the blood pressure and the level of the blood sugar are related to the body temperature (*7–9*). The blood pressures and blood sugars of birds are generally higher than those for mammals, in accord with their higher body temperatures. That these effects are related to body temperature rather than to weight is shown by the fact that small mammals (*4*) have much lower blood pressures than do birds of the same weight (*13*).

Studies on the metabolic rates of animals have been based on a calculated surface area based upon approximately the 0.7 power of the weight (*5*). However, since weight of the animal appears to affect the body temperature, which in itself plays a significant role in the metabolic rate of the animal, simple conversion of weight to surface area may lead to considerable error. This is apparent upon consideration of the fact that a variation of only 1° C in body temperature may increase the resting metabolism by 10%. It would therefore appear that comparisons of the metabolic activity of various species on the basis of surface area alone, without regard to weight and body temperature, are likely to be misleading.

Individuals of a given species may all have approximately the same body temperature, despite fairly large variations in body size. Adequate data on this point are not available. The weight-body temperature relationship may, however, appear in the fact that young animals have slightly higher body temperature than adults of the same species.

It is noteworthy that closely related species may have fairly deviant body temperatures in accordance with their body weights. Since the setting of the normal body temperature determines the lethal level in fever, a bird of large size with a normal temperature of 40° C will have lethal body temperature of about 44° C, while a very small but closely related species with a normal temperature of 44° C may have a lethal temperature of 47° C. This difference in lethal thermal levels suggests that the mechanisms leading to death in pyrexia devolve upon disturbances in specific physiological adjustments induced by change in body temperatures, rather than upon the absolute temperature level itself.

The fact that various unrelated species of large size have body temperatures in the same range indicates the independent achievement of these thermal levels. This may be dependent upon survival factors related to the size of animal. It suggests that for large homeotherms of a given weight, a particular level of body temperature is optimal for survival. In small homeotherms, other factors predominate, resulting in a marked disparity between the body temperatures of birds and mammals of the same size.

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On the Amendment of the Nomenclature of the Rh-CDE System

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The recent article by Castle, Wintrobe, and Snyder on the nomenclature of the anti-Rh-CDE typing serums (*1*) has served to clarify the situation but poorly, and has brought out into the open the confusion that exists in the terminology of the Rh-CDE factor. The Wiener classification (*6, 7, 10*) using the Rh and Hr terms is unwieldy. Anyone who has tried to learn (and teach) the terminology of Wiener, with the hat, arm, and glove symbols (*5*), has soon become lost in flights of fantastic conjectures. The steps taken by Race (*2*), and later elaborated and expanded (*3*), have pointed a path through the forest of the Wiener complexity.

But the recommendation of the Review Board (*1*) that both systems be used concurrently has done the field of immunology an unintentional disservice. The Rh-CDE

terminologies are too confusing to exist side by side. Eventually one or the other must achieve universal acceptance. The Fisher-Race terminology (*2, 3*) is familiar to everyone. There are many reasons why it is easier to teach and learn.

For example, Wiener (*9*) must use a different set of terms for the genotypes and the phenotypes (Rh_1, Rh_0, and R_1, R_0, etc.). So the worker in the field must learn another terminology. This is obviated in the British system. $\frac{Cde}{Cde}$ clearly indicates all the information necessary.

Another point is that the symbol Rh_1 does not indicate whether the individual referred to is homozygous or heterozygous (*4, 8, 9*). The corresponding symbol of Race (*3*) does, $\frac{CDe}{CDe}$ or $\frac{CDe}{cde}$. The importance of this can be seen in the following example. In the mating of two individuals—one, Rh_1, the other, rh—the possible progeny are Rh′, Rh_1, Rh_0, and rh. (It reminds one of multiplying with Roman numerals, i.e., XIV × VIII = CXII!) One must memorize the entire table (*9*); one cannot easily work out the genotypes involved. There are also multiple matings possible, but the Wiener terminology does not take that in consideration. When he feels additional explanation of a phenotype is needed, he mentions it in a footnote (*11*).

TABLE 1

Wiener		Race		Proposed	
Antigen	Agglutinin	Antigen	Agglutinin	Antigen	Agglutinin
Rh_0	Anti Rh_0	D	Anti D	D	Anti d
rh′	Anti rh′	C	Anti C	C	Anti c
rh″	Anti rh″	E	Anti E	E	Anti e
Hr_0	Anti Rh_0	d	Anti d	D′	Anti d′
rh′	Anti rh′	c	Anti c	C′	Anti c′
rh″	Anti rh″	e	Anti e	E′	Anti e′

How much simpler is the Fisher-Race nomenclature: The Rh_1 individual is either homozygous $\frac{CDe}{CDe}$ or heterozygous $\frac{CDe}{cde}$, and in matings with an rh $\frac{cde}{cde}$ individual, the results would be (in the first mating) $\frac{CDe}{cde}$; in the second mating: $\frac{CDe}{cde}$ or $\frac{cde}{cde}$. There is nothing esoteric or far-fetched about it.

But the Fisher-Race nomenclature has still some confusing terms. The use of the lower case letters c, d, and e, to denote the Hr antigens leads to ambiguity when it is remembered that in the major groups, a and b indicate agglutinins. It is therefore proposed that the lower case letters be reserved for agglutinins, leaving the capital letters to indicate antigens.

Table 1 will clarify the proposed change. The advantages are quite obvious. No confusion can exist in either the reading or the speaking of these terms. Allowance is made for the discovery and naming of new antigens and agglutinins. Also their reciprocal relation is retained: D and D′; c and c′.

Conclusion. The great contributions made by Dr. Wiener in the field of immunology cannot be denied. However, although a subject must necessarily be scientifically correct, it must also be as clear and intelligible as possible. The adoption of the Fisher-Race terminology is a step forward, but there is still some confusion. It is hoped that the proposed changes will also lead along the same path to a clearer understanding.

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Rate of Elimination of C^{14} Administered as $BaC^{14}O_3$[1]

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A problem of particular importance is the evaluation of the hazard of the long-lived radiocarbon isotope C^{14}. Armstrong and his associates (*1*) studied the rate of elimination of C^{14} by rats after intraperitoneal injections of sodium carbonate containing C^{14}. The incorporation of C^{14} could be detected in the muscle and liver by implantation of $CaC^{14}O_3$ in the peritoneal cavity and maintaining the isotopic inorganic C^{14} content of the body fluid at a high level over a long period of time. A comparison was also made between the elimination of C^{14} from the tissues by mature and growing rats (*3*). In general, the rate of excretion of the isotope was very fast. The specific activities of the tissues of growing rats greatly exceeded those of mature animals. The over-all retention of C^{14}, however, was greater in mature rats. In the latter, no significant change in the C^{14} retention was observed from the 15th day after injection, whereas an appreciable decrease in the C^{14} retention was still observed in growing animals. A rapid excretion of C^{14} has been observed by Gould *et al.* (*2*) after intraperitoneal injection of labeled sodium bicarbonate, acetate, or succinate. After 4 hr the

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[2] While on leave from the Laboratory of Radioactivity and Nuclear Physics, University of Liége, Liége, Belgium.

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