POINT OF VIEW

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Induction and deinduction in insect photoperiodism: Experimental evidence and a model

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Abstract. Experiments with the aphid *Megoura viciae* Buckton were directed toward quantitative analysis of the cumulative effects of different long- and short-day photoperiods, as dependent on the numbers of light-dark cycles. It has been shown that the accumulation of these effects is a nonlinear process since, as the number of the cycles experienced increases, this process gradually slows down and finally stops. From this observation a suggestion was derived that the kinetics of the cumulative effects depends on interaction of two opposite processes, viz. the increase of the effect due to summation of consecutive daily impulses (induction), and its spontaneous decrease due to degradation (deinduction). The dependence of the kinetics of the cumulative effect on the rates of induction and deinduction was analyzed in the framework of a model proposed earlier. Incorporation of these parameters into the model produced feasible explanations for (a) the compatibility of the quantitative perception of photoperiod with the evident "all-or-nothing" overt responses; (b) the dynamics of switches caused by photoperiod reversals, and (c) other aspects of insect photoperiodism. Since temperature coefficients of both parameters may be unequal, the outcome of interaction between induction and deinduction may be different at different temperatures. This provided novel explanations (1) of the well known variations in interaction between the photoperiodic and temperature reactions and (2) of the promotion of development in diapausing insects by chilling.

INTRODUCTION

It is widely recognized that for a particular response (e.g. diapause) to be photoperiodically induced, two specific thresholds need to be surpassed. These are the critical day-length (or, more precisely, night-length), and the critical number of inductive events ("required day number") (Danilevskii, 1961; Saunders, 1976; Tyshchenko, 1977; Beck, 1980; Page, 1985). It is a common belief (Saunders, 1981; Vaz Nunes & Veerman, 1982; Veerman & Vaz Nunes, 1987; Vaz Nunes, 1990) that these thresholds are determined at two consecutive levels of the photoperiodic machinery. The first is the "photoperiodic clock" which qualifies photoperiods as "long-day" or "short-day", and the second is the "photoperiodic counter" which summates long-day and/or short-day inductive events.

A largely different concept has, however, been developed (Zaslavski, 1972, 1988) according to which it is the second level of the mechanism that (1) distinguishes between short and long days, (2) determines both thresholds mentioned, and (3) is responsible for typology of the photoperiodic reactions. The concept was based upon increasing evidence that the thresholds, despite their overt function of demarcation are, in fact, non-fixed points on a quantitative continuum (Zaslavski, 1975a,b, 1976, 1978, 1979, 1988, 1992; Zaslavski & Fomenko, 1980, 1983, 1986, 1990; Spieth & Sauer 1991). Firstly, different photoperiods that cause identical qualitative response require different times to evoke them, thus exhibiting different inductive strength. In consequence, with varying numbers of inductive events the critical day-length itself becomes variable. Secondly, the gradual series in inductive strength that have been traced at opposite sides of the photoperiodic threshold, join with and merge into each other (Zaslavski, 1988). Thirdly, after the required day number has been passed, the ongoing exposure to the same photoperiod results in the ongoing induction which, in turn, entails quantitative strengthening of a response (Gibbs, 1975; Hodek, 1974, 1975; Zaslavski, 1988; present comm.). Fourthly, the quantitative effects of long- and short-day photoperiods are summed to a common cumulative effect (Zaslavski, 1988). Several conclusions were drawn from these findings. (1) It is unlikely that the photoperiodic clock does, in fact, distinguish qualitatively between short and long days. Its more probable function is the uniform quantitative evaluation of all the variety of photoperiods. (2) Neither the strength of the photoperiodic signals, nor their number, but the value of their "induction sum" (cumulative effect) contributes to the qualitative and quantitative characteristics of the photoperiodic reaction that we examine in our experiments. (3) Not only summation of the day-length induced impulses generated by the photoperiodic clock, but also formation of the characteristics mentioned are performed by the mechanism that follows the clock. This "managing" or "commanding" mechanism is, therefore, quite different from a mere "counter" (Zaslavski, 1988).

The present work concerns the kinetics of formation of the cumulative effect. Gibbs (1975) was the first to approach this process quantitatively. In his model the process is considered a gradual accumulation of a "diapause titer", its actual level interacting with the inborn individual threshold. Shimada (1985) added to this model the suggestion that not only consecutive increments but also losses due to degradation participate in formation of the "titer of diapause". These ideas were incorporated to a greater or lesser extent in several works (Zaslavski, 1984, 1988, 1990; Lewis & Saunders, 1987; Vaz Nunes, 1990). The present paper offers experimental evidence of the involvement of both increments and losses, and a model interpretation of their importance in formation of the photoperiodic response.

MATERIAL AND METHODS

A clone of *M. viciae* from St Petersburg (Leningrad, 60°N.L.) established in 1979 and maintained under a constant regime (20L : 4D, 20°C) was employed in this study. Aphids were fed on seedlings of *Vicia faba* with roots inserted into glass funnels put into flasks with water. Fourth instar nymphs of apterous virginoparae (grandmother generation, P) were transferred from these conditions to the tested initial regimes. Some 5–6 days later, the newborn individuals (mother generation, G1) were isolated. Then, according to the experimental protocol, they were submitted to the following regimes. Photoperiodic reaction in *M. viciae* is an attribute of the viviparous parthenogenetic females (virginoparae) and manifests itself as maternal determination of the morph in the progeny. In the present work the photoperiodic response was expressed not as the proportion of virginopara-producers in the maternal generation itself (cf. Lees, 1966), but as the rate of virginoparae in pooled progenies (daughter generation, G2), obtained from 10–20 G1 females. To check morphs in the progenies they were raised to adult. The response to photoperiod reversals was expressed as the percentage of virginoparae in consecutive portions of the progenies taken simultaneously in all replicates of a given experiment at two-day intervals. Such a method is suitable for examination of the dynamics of switches and appears adequate for the study of quantitative effects. As in previous work, the differences in the dynamics of switches, caused by photoperiodic reversals, served as indices of values of the cumulative effects. The higher the cumulative effect of the preceding photoperiod, the slower will be the switch in the progeny following transfer to the next photoperiod. It is clear that the difference in cumulative effect may serve as estimate of the relative power of different long-day or different short-day photoperiods provided the exposure time was the same, while at different exposures to the same photoperiod changing levels of the cumulative effect reflect its dynamics.

The number of photoperiodic cycles that contributed to the morph determination, in each consecutive batch of progeny, was counted as a number of nights spent by the maternal generation from the onset of regime under study until day 3 before the birth of a given portion of the progeny (Zaslavski & Fomenko, 1990).

The experiments were performed in special automatic chambers installed in thermostatic room (20°C) which provided alternation of light and darkness by opening and closing the light-tight shutters.

RESULTS

Aphids as suitable animals from which to observe the dynamics of photoperiodic induction

The process of accumulation of the photoperiodic signals by an individual is covert in those insects in which the photoperiodic response is a single event in its life-history. The reverse is peculiar to aphids, whose response to the daylength manifests itself in the maternal determination of polyphenism in the progeny. The parthenogenetic female of *M. viciae* perceives the daylength prenatally, when it is still an embryo some 3–4 days before birth, and photoperiodic sensitivity lasts throughout the entire nymphal and at least the first half of the imaginal life (Lees, 1966). The run of such a prolonged induction may well be observable in a long sequence of progeny throughout the entire period of parturition. For example, an individual female, as Fig. 1 demonstrates, could respond during its life to alternating long- and short-day regimes up to 4 times. Thus, due to a combination of the prolonged photoperiodic induction and its prolonged overt outcome typical of aphids, it becomes evident that the sum effect of the photoperiodic signals is a continuously changing entity.

Does counting daily impulses or summing their values contribute to the photoperiodic induction sum?

A particular long-day photoperiod introduced at a particular point in the aphid life, may induce the corresponding switch after different exposure times depending on the strength of preceding short-day photoperiods, their numbers being equal (Fig. 2), or on their number, the strength being equal (Fig. 3). However, for the equal short-day induction sum to be overridden, different numbers of long days are needed if their strengths are different (Fig. 2). Therefore, the data given in Figs 2 and 3 show again (cf. Zaslavski & Fomenko, 1983; Zaslavski, 1988), that the process of accumulation of the photoperiodic information proceeds as a summation of the values of impulses caused by the photoperiodic signals.

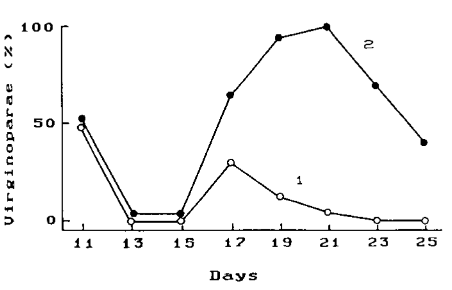

Fig. 1. An example of reversibility of the photoperiodic response within a single maternal generation of *Megoura viciae*. The maternal generation was exposed to a sequence of photoperiodic regimes (temperature 20°C): the weak long day 18L : 6D for all prenatal development and 3 post-natal nights – short day 12L : 12D for 4 nights – long day 18L : 6D for 4 (curve 1) or 7 nights (curve 2) – short day 12L : 12D until the end of parturition. Ordinate – virginoparae in the progeny. Abscissa – the post-natal age of the parents on the day of the progeny sampling.

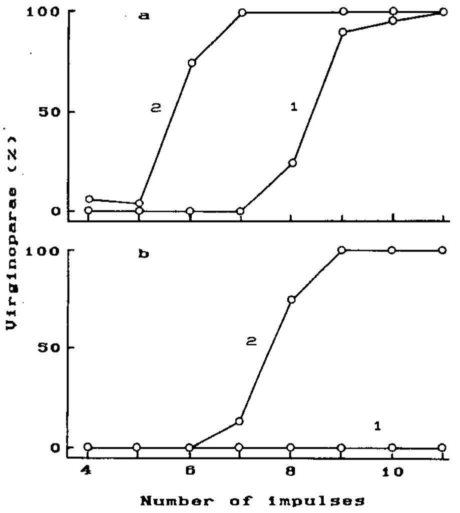

Fig. 2. The dynamics of response of *Megoura viciae* to the short-day/long-day photoperiod reversal as dependent on the photoperiods' inductive strength. The maternal generation was kept for 6 prenatal and 3 post-natal nights in initial short-day photoperiods, then transferred to the long-day photoperiods. a – transfer from the short days 12L : 12D (1) and 17L : 7D (2) to the long day 20L : 4D; b – transfer from 12L : 12D (1) and 17L : 7D (2) to the long day 23L : 1D. Ordinate – virginoparae in the progeny. Abscissa – numbers of the long-day photoperiodic impulses in the parental generation.

Covert changes of the saturated induction sum

It may be inferred from the above data that after a minimal number of inductive events required to evoke saturated response (100% oviparae at short day, 100% virginoparae at long day) is over, the accumulation of the photoperiodic impulses does not stop, since the saturated inductive sums appear to be quite different. This results from noting that, after different saturating exposures, the resulting induction sums can be compensated through alternative regime exposures of different lengths.

Some further details of changes of the saturated induction sum can be seen in the data presented in Figs 4 and 5. In one of the experiments (Fig. 4) the maternal generation was first raised at the short day 12L : 12D, which caused 100% oviparae in corresponding batches of the progeny. Then it was exposed for 2–6 days to the long-day photoperiod 20L : 4D. To induce a complete switch (100% virginoparae), this long-day regime needed 4-days exposure. As was supposed, the induction continued after the saturation was reached and, to observe this, the alternative short-day photoperiod was reintroduced. The results were somewhat surprising. A rise of the value of the induction sum lasted until the 5th long day (8th post-natal day), and ceased thereafter. One might attribute this to decline in photoperiodic sensitivity near post-natal day 8. However, this view contradicts an other experiment (Fig. 5,A) in which the long-day induction was started prenatally and

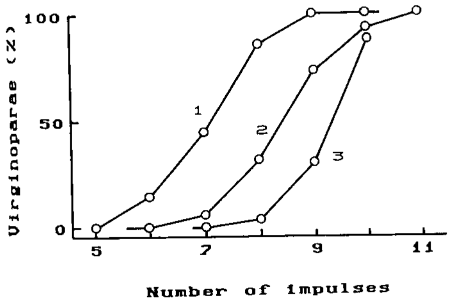

Fig. 3. The dynamics of response of *Megoura viciae* to the short-day/long-day photoperiod reversals as dependent on the length of the initial short-day exposure. The parental generation was kept for 6 prenatal and 1 (1), 2 (2) and 4 (3) post-natal nights in the short day 12L : 12D, then transferred in the long day 19L : 5D. Other designations – as in Fig. 2.

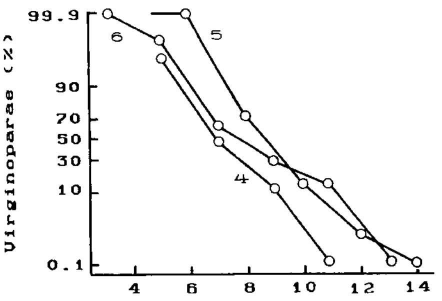

Fig. 4. The dynamics of response of *Megoura viciae* to the long-day/short-day photoperiod reversal as dependent on the length of the long-day exposure. The parental generation was kept for 6 prenatal and 3 pos-tnatal nights in the short day 17L : 7D, then transferred for different times in the long day 20L : 4D, and then returned to 17L : 7D. Numbers at the curves designate numbers of the long-day impulses. Ordinate – virginoparae in the progeny, probit scale. Abscissa – numbers of the final short-day impulses in the parental generation.

terminated earlier, at post-natal days 2 and 5. Here again, the effect of 8 long days was similar to that of 5 long days.

In the experiment documented in Fig. 3, the maternal generation experienced 6 prenatal and from 1 to 4 post-natal short days. The data showed that post-natal prolongation of the short-day exposure for either 1 (curves 1 and 2) or 2 days (curves 2 and 3) was compensated for evenly by 1 additive long day. A further example of this is given in Fig. 5,B. In this experiment 3 prenatal short days were combined with 2, 5 or 8 postnatal short days. Again, it was observed that an initial enhancement of the induction sum is followed by its stabilization or even its decline. To be compensated, 5 short days should be followed by 8–9, 8 – by 11–13, and 11 – by 10–12 long days. Thus, the evidence concerning both the long- and short-day photoperiodic induction shows that (1) the value of the induction sum continues to increase after the saturated qualitative response has been reached, but (2) its rise gradually slows down and then apparently stops. A feasible interpretation of these observations is that, not only daily increments, but also, simultaneous losses do contribute to the kinetics of the photoperiodic induction sum.

DISCUSSION

Induction and deinduction

The behavior of the induction sum shown in *M. viciae* is unlikely to be a unique property of this species. The results reported above evidence clearly in favour of fundamental role of quantitative principles in insect photoperiodism. Hence, it would be reasonable to interpret these findings in the framework of a model elaborated with emphasis on the

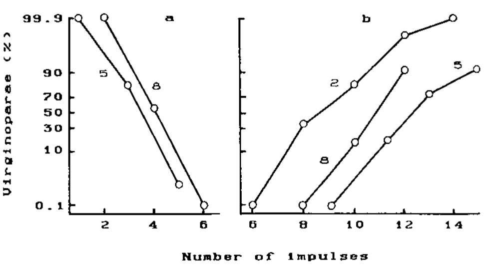

Fig. 5. The dynamics of response of *Megoura viciae* to the final long-day (18L : 6D) or short-day (12L : 12D) photoperiods as dependent on the length of initial exposure to the alternative photoperiod. a – the parental generation was kept for 3 prenatal and 5 or 8 post-natal nights in 18L : 6D, then transferred to 12L : 12D; b – the parental generation was kept for 3 prenatal and 2, 5 or 8 post-natal nights in 12L : 12D, then transferred to 18L : 6D. Numbers at the curves designate the length of exposition to the initial photoperiods. Other designations – as in Fig. 4.

primary role of the quantitative photoperiod perception (Zaslavski, 1972, 1978, 1984, 1988, 1992). According to the model, the perceiving mechanism (the photoperiodic clock) generates quantitative impulses of different strength, specific for each photoperiod. These impulses affect the rates of synthesis of neurohormones in two antagonistic neurosecretory centers, activatory and inhibitory. In other words, photoperiodic signals of different strength are thought to be converted into the hormonal impulses of different intensity.

Thus, similar features of hormonal and photoperiodic induction appear unsurprising. It is known that positive hormonal induction proceeds with the opposite process of deinduction. The latter may, for instance, be degradation of an enzyme the synthesis of which the hormone initiated (Schimke & Doyle, 1970; Mertvetsov, 1986).

Similarly, along with the process of photoperiodic induction a parallel process of deinduction appears discernible (see above). Thus, it would be reasonable to discuss the possible significance of these similarities in the understanding of some aspects of the control of insect seasonality, and to assess their relevance to the model.

Cumulative effect: induction and deinduction rates

It will be assumed below that the kinetics of the photoperiodic (and temperature) induction depends on an interaction between the rate of induction and the rate of deinduction. In the framework of the model this relationship is expressed as the kinetics of the cumulative effects of the presumed activatory and inhibitory neurohormones on their target.

In the analysis of several typical situations some simplifications could be adopted.

1. Only cumulative effects of the activator, but not the inhibitor, on its target will be considered.

2. The increase of the cumulative effect (induction) is assumed to be comprised of two levels at least: accumulation of the hormone itself, and accumulation of its proximate effect on its target. Accordingly, the process of deinduction may comprise degradation of the hormone itself and of its effect.

3. It is clear that even if the degradation rate remains constant, the progressive absolute rise of the cumulative effect will be accompanied by a corresponding absolute rise of loss. Due to this, both the hormone titer and its effect will tend to their respective equilibrium levels. As mentioned earlier, the value of the latter alone is thought to contribute genuinely to the photoperiodic response. This allows the simplification of the forthcoming reasoning. It is assumed that the activating hormone is released once a day, and its daily amount determines the induction rate. Since the half-life of insect hormones may be extremely short (Hammock, 1985; Koolman & Karlson, 1985), the same could occur with neurohormones. Thus, it is assumed here that there is no accumulation of the activator itself, but that each daily hormonal impulse exerts its action on the target before the next impulse, and, therefore, that only their effects are accumulated.

4. The value ("strength") of the hormonal impulse may then be measured as the value of its effect. Then, both the value of impulse (I) and the value of effect (E) could be expressed in the same arbitrary units, as illustrated in Figs 6–9. The deinduction rate (K_d) may be expressed as a proportion of the daily diminution of the cumulative effect. If photoperiodic and temperature conditions remain constant, then the cumulative effect would tend asymptotically to the limit $E_{lim} = I : K_d$. Each parameter, I and K_d has to play its respective role in inductive and spontaneous processes that control seasonal phenomena in insects.

An impact of the impulse value (I)

According to the model (Zaslavski, 1988), two constituents contribute to the value of daily impulse. The first is the basal level of synthetic activity of the "commanding" (here activatory) centre. The second constituent is an action of the photoperiodic clock that enhances the activity level above the basal one. In an insect endowed with photoperiodic reaction the basal level is taken to be the lowest one within the entire range of photoperiods at the given temperature. A simple suggestion was adopted that intensity of synthesis in both activatory and inhibitory centres depends, primarily, on the ambient temperature in the common positive fashion.

The curves presented in Fig. 6 were computed with the constraint that initial levels of E were equal, and K_d remained constant. It may be seen that I itself determines, first, the level of plateau (E_{lim}) and, second, the absolute rate of increase of E.

The impact of I may also be examined when initial levels of E are different (Fig. 8). For example, it is assumed that at particular temperature $K_d = 0.2$, the basal equilibrium level (E_{lim}) corresponding to the weakest photoperiod equals 5.0, the maximal E_{lim} corresponding to the strongest photoperiod equals 20.0, and the threshold value of E is 12.5. These cumulative effects correspond, respectively, to the short-day photoperiod causing $I = 1.0$, the long-day photoperiod with $I = 4.0$, and the threshold photoperiod with $I = 2.5$. The near-threshold $E_{lim} = 11.0$ and $E_{lim} = 14.0$, corresponding to photoperiods short of the critical daylength with $I = 2.2$ and $I = 2.8$, are also exemplified.

The computed kinetics of E illustrates the effects of changes of the photoperiodic regime from short to long day (Fig. 8, 1–5) and from long to short day (Fig. 8, 6,7). An inspection of these curves shows that, with a constant degradation rate, E_{lim} depends solely on I. The initial level determines the direction and the slope of change only.

The data given in Figs 6 and 8 provide some bases for the discussion of three topics of importance.

1. According to the model used here, both short- and long-day photoperiods evoke innate physiological impulses which are common in their nature and differ in their value (strength) only. Therefore, one could suppose that if short days with their weak impulses are permitted to act for very prolonged exposure time, they would be able to induce eventually the same response (e.g. non-diapause) as the long day. However this does not occur.

The curves in Fig. 8 explain the existence of the qualitative border between short and long days as follows. At short-day photoperiods the diel impulsion is insufficient. As a consequence, the degradation of the cumulative effect halts its increase at a level which is lower than the threshold (curve 1). On the contrary, the diel impulsion of long-day

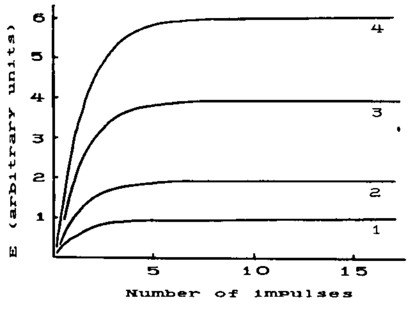

Fig. 6. Kinetics of the cumulative effect (E) of a hormonal centre on its target as dependent on the value (I) of consecutive daily impulses that the centre generates. The degradation rate remains constant ($K_d = 0.5$), initial level of E = 0. 1 – I = 0.5, 2 – 1.0, 3 – 2.0, 4 – 3.0. Ordinate – E (arbitrary units). Abscissa – number of impulses. For other explanations see the text. From Zaslavski, 1990.

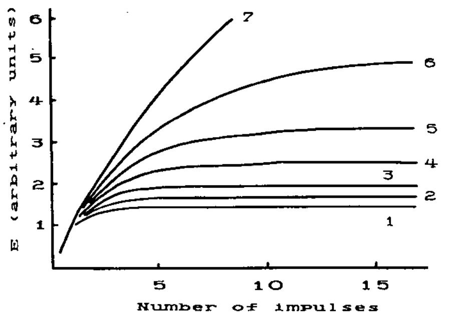

Fig. 7. Kinetics of the cumulative effect (E) of a hormonal centre on its target as dependent on the degradation rate (K_d). The value of daily impulses remains constant (I = 1.0), initial level of E = 0. 1 – K_d = 0.7, 2 – 0.6, 3 – 0.5, 4 – 0.4, 5 – 0.3, 6 – 0.2, 7 – 0.1. Other designations – as in Fig. 6. From Zaslavski, 1990.

photoperiods brings the cumulative effect to a level higher than the critical, and maintains that level despite losses due to degradation (curves 2–5).

With the data of Fig. 8 the concept of unfixed photoperiodic threshold (see Introduction) necessitates further consideration. Variation of threshold is clearly observable in experiments with photoperiodic reversals, in which only a part of the sensitive period of development is exposed to the photoperiods under study, whereas the remainder is exposed to some alternative regime. In experiments of such a type with long-day photoperiods, the shorter the exposure of an insect to them, the more the short-day zone replaces that of long-day, and the reverse replacement is observable in experiments with short-day photoperiods (see references in Introduction). However, as Fig. 8 shows, when exposure time is sufficient ("infinite"), the cumulative effect of any photoperiod becomes fixed, the particular day-length that induces the both alternative responses with equal frequencies (the critical day-length) being no exclusion. Hence, the position of the photoperiodic threshold *is* ultimately fixed. However, this derives from the interactions of I and K_d and by no means favours the concept of qualitative difference between long and short days. Therefore, the quantitative basis of photoperiodic reaction (Zaslavski, 1988) and the ordinary "all-or-nothing" outcome of this reaction may be reasonably reconciled.

2. The curves 6 and 7 (Fig. 8) offer a model description of switches that follow changes from long to short day. They explain quantitative differences between strong and weak short-day photoperiods as a consequence of difference in times needed for degradation of the cumulative effects from the level attained under long days to the level below the threshold. Correspondingly, due to different strengths of long-day photoperiods their

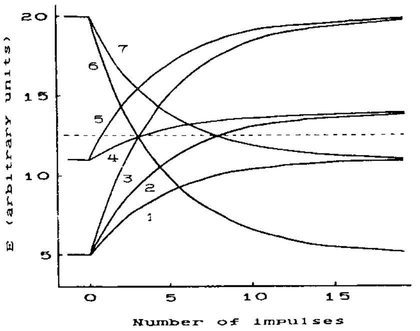

Fig. 8. Kinetics of changes of the cumulative effect (E) induced by photoperiodic switches. The degradation rate remains constant (K_d = 0.2). 1 – transfer from photoperiod generating the impulse with I = 1.0 to photoperiod with I = 2.2; 2 – from 1.0 to 2.8; 3 – from 1.0 to 4.0; 4 – from 2.2 to 2.8; 5 – from 2.2 to 4.0; 6 – from 4.0 to 1.0; 7 – from 4.0 to 2.2. Dashed line – the threshold value of E = 12.5. Other designations – as in Fig. 6. For other explanations see the text. From Zaslavski, 1990.

cumulative effects starting from the level created under short days take different times to exceed the threshold value (curves 1–5).

3. Not infrequently, the long-day induction requires fewer inductive cycles to be accomplished than the short-day induction does. Sometimes this has been interpreted (Lees, 1966; Zaslavski, 1988) as a reflection of positive hormonal induction under long-day photoperiods. However, comparison of curve 2 with 7, and 3 with 6 (Fig. 8) predicts that, if both the strengths of short- and long-day photoperiods and the initial cumulative effects are symmetrical to the threshold values, then the dynamics of switch will also be symmetrical. Therefore, it appears probable that asymmetry in response could be caused by different relative strengths of the compared long- and short-day photoperiods rather than by the positive or negative mode of the hormonal induction per se.

This hypothesis is in close agreement with the experimental evidence. Steel & Lees (1974) have shown, convincingly, by means of microcautery in the pars intercerebralis region of the brain of *M. viciae*, that the long-day effect occurs via positive hormonal induction, while the negative induction is associated with short-day response. Nevertheless, if the weak short day (17L : 7D) and the strong long day (20L : 4D) are matched, then the more rapid switch occur at the short-day/long-day transition, but when the strong short day (12L : 12D) and the weak long day (18L : 6D) are compared, the faster is the switch from long- to short-day response (Zaslavski & Fomenko, 1983; Zaslavski, 1988).

An impact of the rate of degradation (K_d)

Two characteristics of the accumulation process depend on K_d. First, as seen from the expression $E_{lim} = I/K_d$, K_d defines the value of the cumulative effect, because the equilibrium level of the latter is inversely dependent on the rate of its degradation (Fig. 7). Due to such a dependence even very weak impulses, coupled with low rate of degradation, could result in higher cumulative effect than that of the strong impulses combined with intense degradation.

Second, the rate of deinduction is, by definition, a time-based parameter, and due to this it affects the dynamic aspects of the process of accumulation. It is possible to visualize this by counting the number of impulses that are required to pass a certain part in the transition from the initial level attained before onset of the regime tested, to the new equilibrium level. For instance, if it is assumed that this point equals 0.9, then at $K_d = 0.6$ only 2–3 impulses are sufficient for 90% of the entire path, while 3–4 impulses at $K_d = 0.5$ and 10–11 impulses at $K_d = 0.2$ are needed. It should be noted that neither the impulse value nor the amplitude of the cumulative rise (or decline) exert any effect on these numbers.

Thus, the rate of deinduction may define the rate of induction. From this, a feasible interpretation of the "elementary reaction rapidity" can be derived. The latter is a characteristic employed earlier to explain the so-called "staged" or "stepwise" reactions, i.e., the specific responses to directed changes of photoperiod or temperature [(Zaslavski 1972, 1975a, 1988 (Figs 93, 94)]. Different rapidity of the two elementary reactions may be a consequence of different degradation rates of the cumulative effects of activator and inhibitor (see for detail: Zaslavski, 1988).

Interactions between I and K_d

As mentioned above, the model (Zaslavski, 1988) states that the intensities of synthesis in both activatory and inhibitory centres depend positively on the ambient temperature. By logical extension, the same would be true for degradation of activator and inhibitor and of their cumulative effects. Hence, higher temperatures will enhance the intensity of both synthesis and degradation, whereas lower temperatures will result in a drop in both.

It is easily understandable that if the Q_{10} of both processes are equal, the equilibrium level (E_{lim}) would be stable irrespective of temperature, though the time lapsed to attain this level would vary. However, there is little doubt that inequality of Q_{10} of the two processes is more probable. This, in turn, could have some important consequences which are envisaged below. They concern two temperature-dependent phenomena, viz. the variability of the photoperiodic reaction within the temperature range optimal for manifestation, and diapause development at low temperatures.

1. Temperature-induced variation in the interaction between induction and deinduction should be influential in interaction between photoperiodic and temperature reactions.

The first aspect of this interaction is the temperature-dependent variability in the configuration of the photoperiodic response curve, variation of the critical day-length, etc. An interpretation of this variability in the framework of the model has been given elsewhere (Zaslavski, 1988). However, the amplitude of such variability differing so greatly from one species to the next requires discussion. The model postulates that the influence of temperature on photoperiodic response is realized via its influence on the basal level of activity of the two "commanding" centres. The "level of activity" is not intended to represent a level of synthesis, but an attainable level of the cumulative effect (Zaslavski, 1988). Relying on such an interpretation, it may be surmised that, if the Q_{10} of induction and deinduction are not linked, the amplitude of the temperature-induced variation of the "activity level" would vary greatly and even achieve stability due to complete compensation. This may provide the physiological background for the well known diversity in the extent of influence which temperature exerts on photoperiodic responses in different insect species.

The second aspect of the interrelation between photoperiod and temperature comes from many observations made that the number of required inductive events appears to be relatively stable at different temperatures (Saunders, 1966, 1976; Goryshin & Tyshchenko, 1970; Tyshchenko, 1977), although exceptions are known (Hodková & Hodek, 1987; Kimura & Masaki, 1993). It is the rise of K_d accompanying the rise of I that may cause compensation in this case.

However, shifts of the I/K_d ratio may be adaptive when adjustment of the required day number to the temperature-dependent length of development is needed. For example, at high temperatures a shortened day-length-sensitive period may require more rapid growth of the cumulative effect.

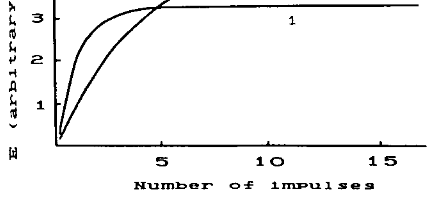

Fig. 9. An example of alteration of kinetics of the cumulative effect when dependencies of impulse value (I) and degradation rate (K_d) are different. 1 – high temperature, I = 2.0, K_d = 0.6; 2 – low temperature, I = 1.0, K_d = 0.2. Other designations as in Fig. 1. From Zaslavski, 1990.

2. It is widely known that diapause development in insects occurs rapidly at low temperatures. It is commonly believed that the cold acts as an activating factor per se. According to another view (Hodek, 1978, 1983; Zaslavski, 1978, 1988), exposure to cold is insufficient since it acts in combination with the subsequent rise in temperature. Hence, the phenomenon of "cold reactivation" was interpreted as a two-stage reaction (Zaslavski, 1978, 1988) consisting of the sensibilization by cold (1st stage) and activation by heat (2nd stage). The role of the cold was believed, first, to maintain very long, though slow, progress of sensibilization (horothelic process: Hodek, 1983), and second, to provide a contrast for the subsequent temperature increase (Hodek, 1978; Hodek & Hodková, 1988, Zaslavski, 1978).

Here a more comprehensive explanation may be proposed for the causes of effectiveness of sensibilization by cold. The following is an extension of the idea of Lees (1955) that the phenomena exhibited by diapausing insects in cold were unlikely to be considered as evidence of unusual inverse dependence of a physiological process on temperature, but rather a consequence of the interaction of two usual processes, both with positive but different temperature coefficients.

The Q_{10} of K_d greater than the Q_{10} of I is a particular condition which would increase the equilibrium level in response to a decline in temperature. This may be seen in Fig. 9. By conditions adopted there, certain low temperature causes a two-fold drop in I vs. a three-fold drop in K_d (as compared with high temperature). As a consequence, despite cold conditions, the cumulative effect attains a high level that is impossible to achieve under warm conditions. The usual positive dependence on temperature of two opposite, but interacting, processes may thus result in phenomena that seem to be an exception from the general rule.

Relevance of the presented data to the model

The principle of regulatory interactions of antagonistic processes is one of the cornerstones of the present model (Zaslavski, 1972, 1988). As a consequence, the model is dynamic and covers and interprets a broad variety of phenomena including those which proceed in time (e.g., stepwise reactions, long-running spontaneous processes). Originally only interactions between activatory and inhibitory centres were considered by the model. Now, antagonistic interactions within each of the centres are also portrayed, and this provides a diversity of pathways to the same physiological and/or adaptive goal. Some phenomena (e.g., stepwise photoperiodic reactions, complicated photoperiodic curves with 4–5 slopes, some thermoperiodic reactions) appear to remain explicable, exclusively, on the basis of interactions between the two centres, whilst two and more explanations may fit the others.

For example, the novel interpretation of the temperature stepwise reaction proposed here appears to be sufficient and more universal than one proposed previously (Zaslavski, 1988). Another example of the multiple solutions within a single model concerns the often observable thermostability of the photoperiodic threshold. This was suggested to be caused either by the steepness of the slope of the "primary photoperiodic curve", or by compensation of changes of the activator level by parallel changes of the inhibitor (Zaslavski, 1988). The third explanation has been proposed above, and there is little doubt that each of these alternative possibilities could be selected in different species.

Thus, incorporation into the model of the new category, deinduction and its rate, has expanded the explanatory capacities of the model without alteration of its earlier postulates and statements.

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