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# ESTIMATION OF SENSIBLE HEAT LOSS IN CAPSAICIN-DESENSITIZED CHICKEN AFTER EXPOSURE TO DISRUPTION OF THERMAL HOMEOSTASIS

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## ABSTRACT:

Background: Thermal conditioning in newly hatched chicken is of great importance. Induction of thermotolerance to cold, heat and disrupted thermal balance is one of the most important managemental tools used to improve survival in chicken. It is well known that chicken is in sensitive to capsaicin (main ingredient of hot chilli peppers) due to insensitivity of Transient membrane potential of vanilloid subtype-1 (TRPV1) receptors to capsaicin. These receptors are responsible for perception of pain burning sensation and thermoregulation in mammals. Owing to the capsaicin's availability as an rodent repellent function in poultry ration and its preference by many birds, we investigated the mechanism of thermoregulation of capsaicin in chicken The purpose: To interpret the phenomenon of thermotolerance in capsaicin (CAP) desensitized chicken and to study its effect on sensible heat loss mechanisms in newly hatched chicks. Methods: In this study, chicken were treated intravenously (IV) in wing vein once with CAP (10 mg/kg, body weight) at 1st, 2nd & 3rd days of age. Then after one week from CAP- pretreated chicken were exposed to cold (8° C), heat (38° C) or injected with lipopolysacccharide (LPS; outer memebrane of Gram negative bacteria). LPS at a low dose (1 mg/kg, body weight, IV) induce fever or at a high dose (10 mg/kg, IV) to induce hypothermia. Surface (skin of back) and colonic temperatures were measured to calculate heat loss index (HLI) as an indicator to the sensible heat loss. Main results: The HLI was 0.95 in control non-treated chicken at ambient temperature (Ta 25° C). In CAP-desensitized chicken HLI was increased to a maximum of 0.97 at Ta 38° C and up to 0.99 at climax of fever induced by LPS. The controversial finding was observed in chicken exposed to cold; HLI in CAP-desensitized chicken was not increased but reduced to 0.94, however no such effect of capsaicin at nadir of hypothermia induced by high dose of LPS.
Conclusion: CAP-sensitive receptor (Transient membrane potential of vanilloid subtype-1;TRPV1)-independent pathway may exert a thermoregulatory role during heat and cold exposure, and in LPS-induced fever in part through affecting sensible heat loss in chicken.

# INTRODUCTION:

**Chicken as homeothermic species is able to keep their body core temperature balanced within a very narrow range (within 1°C) even when exposed to a wide range of ambient temperatures (Romanovsky, 2007). At high temperature, heat production decreases while heat dissipation increases. The main pathway of heat dissipation for birds under hot environment is via panting (Richard, 1971 and Shinder *et al*., 2007). Nonevaporative heat loss takes place at the surface of bare skin and plumage. The resultant level of skin temperature depends on the rate of heat loss and the rate at which warm blood flows from core to the skin. Therefore, the skin temperature could give a certain reflection of the response of thermoregulation (Nääs *et al*., 2010).**

**Disruption of thermal balance can occur by thermal stimuli such as heat and cold, and non-thermal stimuli such as Lipopolysachharide (LPS) injection. LPS causes fever or hypothermia depending on the dose of injection. The fact that CAP pretreatment attenuates both LPS-induced fever (Mahmoud *et al*., 2007) and LPS-induced hypothermia (Nikami *et al*., 2008) as they suggested that the CAP-sensitive, TRPV1-independent pathway was not only specifically involved in hyperthermic reactions but also was commonly related to disruption of thermoregulation. Both LPS induced fever and hypothermia that arise from processing of LPS by macrophages and endothelial cells in major organs (liver, lung and brain), where cytokines and free radicals like NO are released in animals (Kluger, 1991; Romanovsky *et al*., 1996, 2005; Saia & Carnio, 2006 and Steiner *et al*., 2006) and in chicken (Nikami *et al*., 2008). In mammals, LPS induces hypothermia by reducing metabolic heat production and by an induction of cold-seeking behavior, even though body temperature is decreasing (Kluger, 1991; Steiner and Branco, 2002; Romanovsky *et al*., 2005; Rudaya *et al*., 2005 and Almeida *et al*., 2006a, b). However, the thermoeffector mechanisms initiating cold- and high dose of LPS- induced hypothermia which seemed to oppose each other, and despite of the similar responses of CAP-pretreated chicks to both inflammatory (LPS) and thermal (cold) stimuli, as one major difference. Exposure to cold may activate autonomic and behavioral mechanisms to increase heat production and reduce heat loss at maxima, and body temperature drops when heat loss exceeds heat production. In contrast, LPS-induced hypothermia is accompanied by decrease of heat production and cold-seeking behavior in animals (Romanovsky *et al*., 1996, 2005; Mailman *et al*., 1999 and Almeida *et al*., 2006a, b) and chicken (Mahmoud *et al*., 2007).**

**Therefore, the mechanisms responsible for prevention of cold- induced hypothermia by CAP-pretreatment would be independent of those operating in the prevention of LPS-induced hypothermia. CAP pretreatment also rescued cold-induced hypothermia (Nikami *et al*., 2008). Although, CAP has wide rage of anti-inflammatory actions (Kim *et al*., 2003 and Chen *et al*., 2003) this study was focused on the**

**effect of CAP of sensible heat loss that indicated by heat loss index.**

**The present study was achieved to illustrate the role of desensitization of CAP-sensitive, TRPV1-independent pathway in ameliorating the effect of thermal (cold/heat) and inflammatory stimuli (LPS-induced fever/ hypothermia) through estimating heat loss index in chicken.**

## MATERIALS & METHODS:

**1-Experimental birds: Male, newly hatched white leghorn chicks, specific pathogen free, were brought from Goto Chick Company (Gifu, Japan) with a body weight range of 40±7 g. Chicks were kept on a 12:12 h light–dark cycle in thermostatically controlled cages. To match the chicks' requirements, the temperature of the cage for the newly hatched chicks was set at 38°C and then decreased 0.5°C every day until day 4. All procedures were approved by the Local Committee for Ethics of Animal Experimentation, Care and Use of Gifu University.**

**2-Desensitization of capsaicin-sensitive pathways: As described previously (Mahmoud *et al.*, 2007), CAP-sensitive pathways were desensitized by repetitive injections of CAP into newly hatched chicks. In brief, CAP (10 mg/kg; Sigma, St. Louis, MO, U.S.A.) was dissolved in 0.1 ml of vehicle (10% ethanol, 10% Tween 80 and 80% saline) and injected intravenously at 1, 2 and 3 days of age. Control groups were injected with vehicle only.**

**3-Experimental model of disrupted thermal homeostasis: Chicks at 10 days of age were injected in wing vein with hyperthermic (1 mg/kg, body weight) and hypothermic (10 mg/kg, body weight) doses of LPS from Escherichia coli (0111:B4; Sigma). LPS injected chicks showed signs of sickness behavior: lethargy, ruffled feathers and white diarrhea. For comparison, hypothermia and hyperthermia were induced by exposing chicks to an ambient temperature of 8°C and 38°C for 5 hours. The latter was considered a mild form of heat stress where panting behavior was not observed in 10 days old chicken (Gerken *et al.*, 2006).**

**4-Measurement of temperature: Colonic temperature (Tc) was measured by using a lubricated thermistor probe as described previously (Mahmoud *et al.*, 2007). Skin temperature (Tsk) was measured by using special kin probe (model XN-64, Technol Seven, Yokohama, Japan). To prevent stress fever, chicks were allowed to adapt to the handling and experimental cage (Jones *et al.*, 1983), where the ambient temperature was kept at 30°C. The thermistor probe was inserted gently 5 cm beyond the vent, and the colonic temperature was monitored using a peripheral processor connected to a computerized medical system (Chuo Electronic Co., Hong Kong). The baseline temperature recordings were determined for 1 h, and the chicks exhibiting no stress fever were used for experimentation. Each chick was used only once. To avoid**

**circadian variations in colonic temperature recordings, measurements were started at 8 a.m.**

**5-Heat-loss index (HLI): The differences between colonic and surface temperature (ΔT) and heat loss index (HLI) were calculated from the following equation; ΔT=body temperature (Tb)- Skin temperature (Tsk), and HLI = Tsk-(Ta)/(Tb)-(Ta). HLI value ranges theoretically from zero (state of complete vasoconstriction) to one (state of complete vasodilatation) (Steiner & Branco, 2002).**

**6-Statistical analysis: All statistical analysis of data was performed using SPSS (2007) Software. All values were presented as means±standard error (SEM). Descriptive statistics of data were analyzed by one way analysis of variance (ANOVA) at time points of climax and nadir of temperature curve. Tukey's HSD was used for comparisons among mean values.**

## RESULTS & DISCUSSION:

### 1-Effect of CAP-desensitization on sensible heat loss after exposure to hyperthermic stimuli in chicken at 10 days of age:

**CAP (10 mg/kg, body weight, IV) was injected at 1, 2 and 3 days of age. Chicks at 10 days of age were injected intravenously with hyperthermic (1 mg/kg, body weight, IV) doses of LPS. For comparison, hyperthermia was induced by exposing chicks to an ambient temperature of 38°C for 5 hours. Data are presented as means and standard error bars (SEM.) and values with asterisk means significantly different ($P<0.05$).**

**Data in Table 1 showed the differences between colonic and surface temperatures (ΔT; Tc-Tsk) and Figures 1&2 illustrated ΔT and calculated sensible heat loss in terms of HLI at climax of body temperature. Chicks were exposed to mild heat (Ta 38°C) for 5 hours (Fig. 1) or injected intravenously with hyperthermic doses of LPS (LPS 1 mg/kg, body weight Fig. 2).**

**In Fig. 1A chicks of control non treated groups, the ΔT ranged from 0.8 C to 1.0°C and the HLI at ambient temperature (Ta 25°C) was 0.95±0.02. Exposure to high Ta increased surface temperature and lowered the ΔT during the first 2 to 3 hr (Table 1: $P<0.05$), and calculated HLI was 0.93 ±0.1°C at a 2.5 hr time point (Fig. 1B). While heat exposure in CAP-desensitized group elevated the surface temperature, decreased the ΔT to a minimum of 0.2 ±0.1°C (Table 1*: P*<0.01), and increased HLI to 0.97±0.02 at a 2.5 hr time point (Fig. 1B). These results could be explained in part from the following facts; It is long known that TRPV1 agonist (like CAP) affect vasomotor tone and causes hypothermia by skin vasodilatation (increased heat loss through the skin) and reduction in metabolism (a decreased oxygen consumption; VO2 = decreased heat production, Ayoub *et al*., 2009). However, this thermoteloerance to mild heat in CAP is difficult to interpret at present study and needs further investigations.**

**Although injection of a low dose of LPS (1 mg/kg, body weight, IV) which is known to**

**cause fever (Mahmoud *et al*., 2007), ΔT was not affected and HLI (0.94±0.02) was not different from the control group (Table 1, Fig. 2 A &B). Despite injection of LPS in CAP-desensitized chicks (in absence of fever) did not change ΔT, HLI was increased to the maximum 0.99±0.01 at 2.5 hr time point or climax of fever in LPS-injected group (Fig. 2B). Increasing heat loss in this experiment could explain in part our previous finding (Mahmoud *et al*., 2007) in which CAP–pretreatment abolished LPS-induced fever in chicken through TRPV-1-independent pathway.**

**Although injection of a low dose of LPS (1 mg/kg, IV) led to fever, ΔT was not affected and HLI (0.94±0.02) was not different from the control group (Table 1, Fig. 2). However, injection of LPS in CAP-desensitized chicks (absence of fever), did not change ΔT but, HLI was increased to the maximum 0.99±0.01 at 2.5 hr time point (Fig. 2). Increasing heat loss in that experiment could explain in part our previous study (Mahmoud *et al*., 2007) in which CAP–pretreatment abolished LPS-induced fever in poultry chicken.**

**Table 1: Differences between colonic temperature and skin temperature (ΔT) after exposure to hyperthermic stimuli in CAP-desensitized chicks at 10 days of age**

| Time after Exposure (hour) | Control (Ta; 25°C) | Hyperthermic Stimuli [Heat (Ta 38°C) or LPS (1 mg/kg, IV)] | | | |
|---|---|---|---|---|---|
| | | Heat | Heat /CAP | LPS | LPS/CAP |
| -1.0 | 0.9±0.1 | 0.9±0.1 | 0.8±0.1 | 0.9±0.0 | 0.9±0.0 |
| -0.5 | 1.0±0.0 | 0.8±0.1 | 0.9±0.1 | 0.9±0.1 | 0.9±0.1 |
| 0.0 | 0.9±0.1 | 0.9±0.0 | 0.9±0.0 | 0.8±0.0 | 0.8±0.0 |
| 0.5 | 0.8±0.0 | 1.0±0.1 | 1.0±0.1 | 0.9±0.1 | 0.9±0.1 |
| 1.0 | 0.9±0.1 | 1.9±0.0 | 0.8±0.1 | 1.0±0.1 | 1.0±0.0 |
| 1.5 | 0.9±0.0 | 0.8±0.0 | 0.6±0.1* | 0.9±0.0 | 0.9±0.1 |
| 2.0 | 0.9±0.1 | 0.6±0.1* | 0.4±0.0* | 1.0±0.1 | 0.9±0.0 |
| 2.5 | 0.8±0.0 | 0.5±0.0* | 0.2±0.1** | 1.1±0.2 | 0.9±0.1 |
| 3.0 | 0.9±0.0 | 0.6±0.0* | 0.4±0.0* | 1.1±0.1 | 0.9±0.1 |
| 3.5 | 0.9±0.0 | 0.7±0.1 | 0.5±0.1* | 0.9±0.0 | 1.0±0.0 |
| 4.0 | 0.8±0.1 | 0.8±0.0 | 0.6±0.1* | 0.9±0.1 | 0.9±0.1 |
| 4.5 | 0.9±0.0 | 0.7±0.1 | 0.7±0.1 | 1.0±0.0 | 0.9±0.0 |
| 5.0 | 0.8±0.0 | 0.7±0.0 | 0.8±0.0 | 0.9±0.0 | 0.9±0.1 |

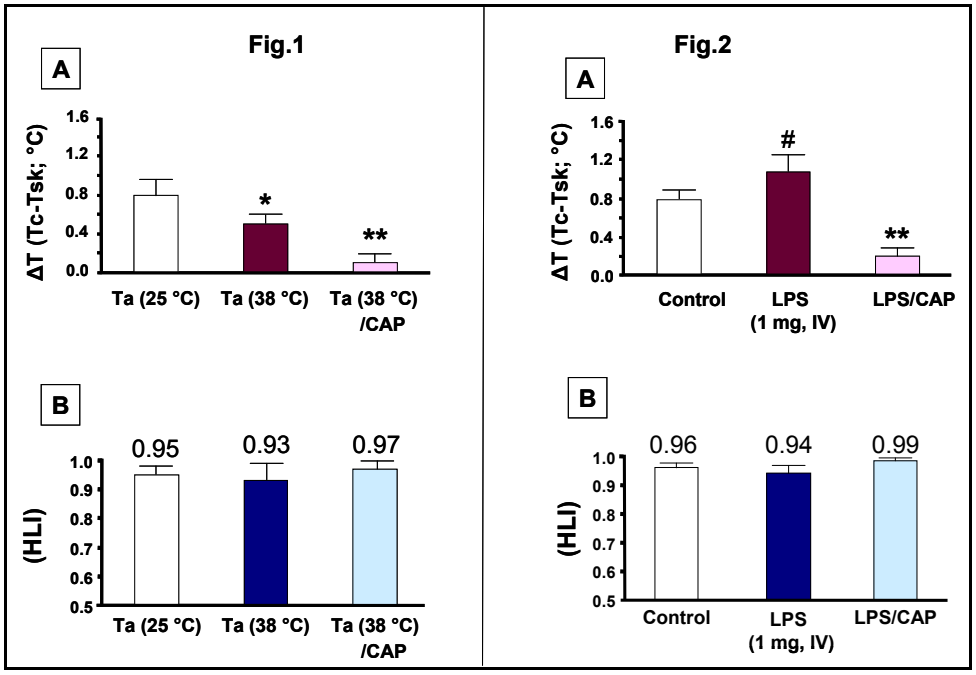


**Fig. 1 & 2: Temperature differences (ΔT: Tc-Tsk) and heat loss index (HLI) 2.5 hr after exposure to heat (Fig. 1: Ta 38°C) or at a body temperature climax of LPS (Fig.2: LPS 1 mg/kg, body weight, IV)-induced fever in 10 days old chicken. Values with asterisk mean significant (*P*<0.05) differences versus # in Tukey's HSD tests**

## 2-Effect of CAP-desensitization on sensible heat loss after exposure to hypothermic stimuli in chicken:

**CAP (10 mg/kg, IV) was injected at 1st, 2nd and 3rd days of age. Chicks at 10 days of age were injected intravenously with hypothermic (10 mg/kg, body weight) doses of lipopolysaccharide (LPS). For comparison, hypothermia were induced by exposing chicks to an ambient temperature of 8°C for 5 hours. Data are presented as means and standard error bars (SEM.) and values with asterisk means significantly different (*P*<0.05) versus #.**

**Chicks at 10 days of age were exposed to cold (Ta 8°C) for 5 hours or injected with hyporthermic doses of LPS (LPS 10 mg/kg, body weight, IV). The differences between colonic and surface temperatures (ΔT; Tc-Tsk) were shown in Table 2, and ΔT and HLI at nadir of body temperature were shown in Figures 3&4.**

**On other side, Data in Table 2 indicated that exposure to cold lowered skin temperature and decreased the ΔT from time point of 1.5 hr on (*P*<0.05), and HLI reached the maximum (0.99±0.01) as shown in Fig. 3. While cold exposure in CAP-desensitized group did not affect the skin temperature and the ΔT started to decrease only after 4.5hr from cold exposure (Table 2). The controversial finding was that HLI in CAP-desensitized chicken was not increased but was similar to control non treated group (0.94±0.02) at time point 2.5 hr (Fig. 3). The present results indicated that the non treated group responded immediately to cold exposure by significantly increasing its HLI to**

**the maximum level, however, CAP-desensitization clearly enhanced the chicks' ability to maintain on-chick body surface temperatures during exposure to 8°C and can recover much faster from cold exposure than non-treated group (Nikami *et al.*, 2008). Injection of high dose of LPS (10 mg/kg, IV) which is known to cause hypothermia in chicken decreased ΔT along 1.0 to 3 hr time points post-injection ($P$<0.05, Table 2). As a result HLI was increased to 0.97±0.01. But no such effect was observed in CAP-desensitized group (Fig.4). Preservation of heat loss by CAP-desensitization after cold exposure could be explain in part the previous study (Nikami *et al.*, 2008) in which CAP–pretreatment attenuated cold-induced hypothermia in newly hatched chicken. However ion case of LPS-induced hypothermia that could lead to shock, it is plaucible to relate this action to the anti-oxidant effect of CAP as reported by Nikami *et al.* (2008) rather than its effect on vasomotor tone of skin indicated by changes in heat loss index.**

**Values with asterisk mean significant ($P$<0.05) differences versus# in Tukey's HSD tests.**

**Chicks at 10 days of age were exposed to cold (Ta 8°C) for 5 hours or injected with hyporthermic doses of LPS (LPS 10 mg/kg, body weight, IV). The differences between colonic and surface temperatures (ΔT; Tc-Tsk) were shown in Table 2, and ΔT and HLI at nadir of body temperature were shown in Figures 3&4.**

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**Table 2: Differences between colonic temperature and skin temperature (ΔT) after exposure to hypothermic stimuli in CAP-desensitized chicks at 10 days of age**

| Time after Exposure (hour) | Control (Ta; 25°C) | Hyporthermic Stimuli [Cold (Ta 8°C) or LPS (10 mg/kg, IV)] | | | |
|---|---|---|---|---|---|
| | | Cold | Cold/CAP | LPS | LPS/CAP |
| -1.0 | 0.9±0.1 | 0.9±0.0 | 0.9±0.0 | 0.9±0.0 | 0.9±0.0 |
| -0.5 | 1.0±0.0 | 0.9±0.1 | 0.9±0.1 | 0.9±0.0 | 0.9±0.0 |
| 0.0 | 0.9±0.1 | 0.9±0.0 | 0.9±0.0 | 0.9±0.0 | 0.9±0.0 |
| 0.5 | 0.8±0.0 | 0.6±0.0* | 0.8±0.1 | 0.8±0.1 | 0.8±0.1 |
| 1.0 | 0.9±0.1 | 0.7±0.1 | 0.9±0.0 | 0.6±0.1* | 0.6±0.0* |
| 1.5 | 0.9±0.0 | 0.6±0.1* | 0.9±0.1 | 0.5±0.0* | 0.7±0.1 |
| 2.0 | 0.9±0.1 | 0.5±0.1* | 1.0±0.1 | 0.4±0.1* | 0.8±0.0 |
| 2.5 | 0.8±0.0 | 0.4±0.1* | 1.2±0.0# | 0.4±0.1* | 0.9±0.0 |
| 3.0 | 0.9±0.0 | 0.5±0.1* | 1.0±0.1 | 0.6±0.1* | 0.9±0.1 |
| 3.5 | 0.9±0.0 | 0.6±0.1* | 0.8±0.0 | 0.8±0.1 | 0.8±0.1 |
| 4.0 | 0.8±0.1 | 0.5±0.1* | 0.7±0.1 | 0.9±0.0 | 0.9±0.1 |
| 4.5 | 0.9±0.0 | 0.6±0.1* | 0.6±0.0* | 0.8±0.1 | 0.8±0.0 |
| 5.0 | 0.8±0.0 | 0.5± 0.0* | 0.6±0.1* | 0.9±0.0 | 0.9±0.0 |

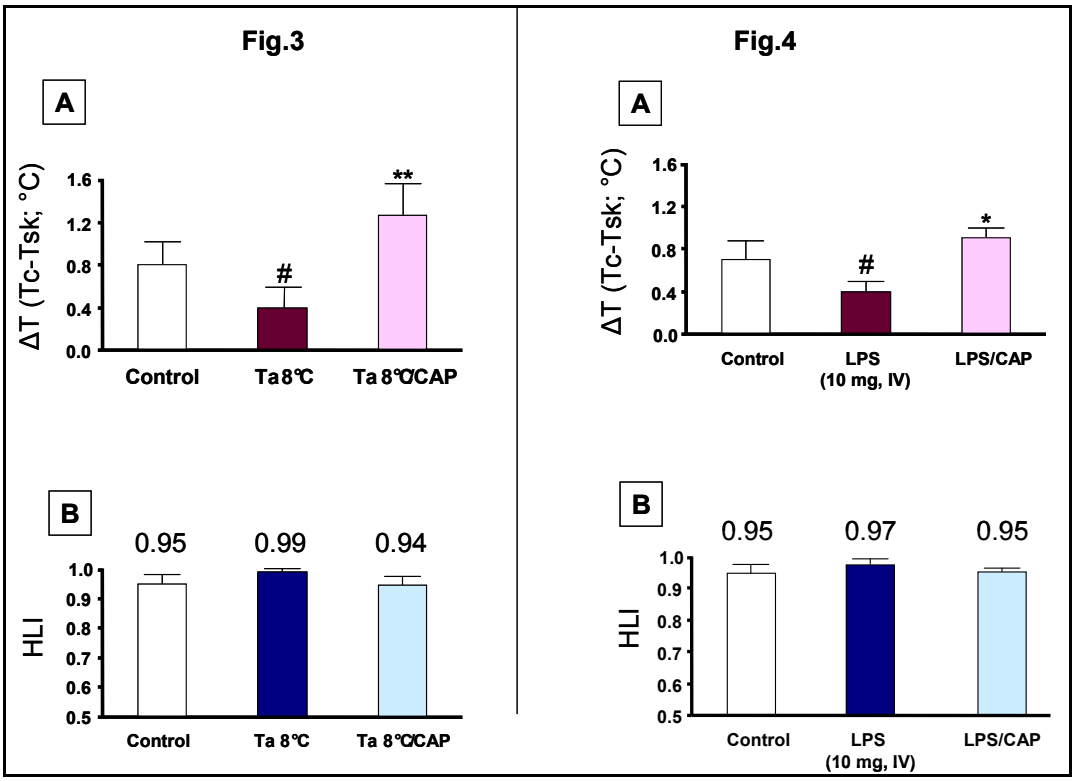


**Fig. 3 & 4. Temperature differences (ΔT: Tc-Tsk) and heat loss index (HLI) 2.5 hr after exposure to cold (Fig. 3: Ta 8°C) or at a body temperature nadir induced by LPS (Fig. 4: LPS 10 mg/kg, body weight, IV) in 10 days old chicken**

**To sum, a CAP-sensitive, a TRPV1-independent pathway is involved in thermal (heat and cold) and non-thermal stimuli (LPS-induced fever and hypothermia), and CAP pretreatment improved thermotolerance in chicken. Since preservation of body temperature balance is crucial for survival of newly hatching chicks, which are inevitably exposed to a wide range of ambient temperature every day, It could be considered that the present study has implications for the poultry industry. Chickens are indifferent to the burning pain sensation induced by CAP (Mason & Maruniak, 1983; Tewksbury and Nabhan, 2001), and we can simply add CAP to their feed. In addition to the well known effect of hot chilli pepper (or CAP) on derratization of rodent (Tewksbury and Nabhan, 2001).**

**Finally it could be concluded that, capsaicin desensitization enhanced thermoregulatory tolerance to both thermal stimuli in terms of cold and heat and to inflammatory agent in part by affecting heat loss mechanisms in newly hatched chicks.**

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## ANNEX OF TERMS

**CAP: Capsaicin: main ingredient of hot chilli pepper.**

**LPS: Lipopolysachharide (outer membrane of Gram negative bacteria).**

**HLI: Heat loss index.**

**Tc: Colonic temperatures.**

**Tb: Body temperature.**

**Tsk: Skin temperatures.**

**Ta: Ambient temperature.**

**$\Delta$ T: Differences between 2 temperatures or heat increment.**

**TRPV1: Capsaicin receptors: transient membrane potential of vanilloid subtype-1**

**HLI: Heat loss index.**

**Hypothermia: Lowered body temperature.**

**Nadir: Lowest point at temperature curve in case of hypothermia.**

**Hyperthermia: Elevated body temperature.**

**Fever: Elevated body temperature in case of infection.**

**Climax: Highest point at temperature curve in case of fever.**

**ANOVA: One way analysis of variance.**

# قياس الفقد الحراري المحسوس في كتاكيت الدجاج فاقد الحساسية للكابسايسين عند التعرض للاختلال في الاتزان الحراري

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**الخلفية البحثية: الحفاظ على الاتزان الحراري في كتاكيت الدجاج حديثة الفقس له أهمية قصوى، ومن أهم وسائل الرعاية للحفاظ على كتاكيت الدجاج حديثة الفقس من النفوق هو إحداث التحمل الحراري عند التعرض للجو البارد أو الحار أو أثناء الحمى أو الهبوط في درجة الحرارة المصاحب للمرض.**

**الغرض من الدراسة: هو قياس معامل الفقد الحراري في كتاكيت الدجاج فاقد الحساسية للكابسايسين عند التعرض للمؤثرات الحرارية مثل: البرد والحرارة والعوامل المسببة للحمى أو الهبوط في درجة الحرارة.**

**طرق البحث: تم معالجة كتاكيت اللجهورن الأبيض فى اليوم الأول، الثاني، الثالث بعد الفقس بمادة الكابسايسين (١٠ مج/كجم من الوزن الحي) في الوريد يومياً، ولمدة ٣ أيام وذلك لإحداث ظاهرة فقد الحساسية للكابسايسين، والتي تم الكشف عنها عن طريق فقدان انخفاض درجة الحرارة الجسم المصاحب لحقن الكابسايسين، وبعد مرور أسبوع من المعالجة تم تعرض مجاميع من الكتاكيت للمؤثرات التالية: البرد (٨° مئوية في غرفة التبريد)، الحرارة (٣٨° مئوية في الأقفاص الحرارية)، حقن مركب الليبوبوليسكاريد بجرعة (١ مج/كجم من الوزن الحي) في الوريد لإحداث الحمى، كما تم حقن جرعة أخرى (١٠ مج/كجم من الوزن الحي) في الوريد لإحداث الهبوط في درجة الحرارة، وفي جميع هذه المجموعات تم قياس درجة الحرارة الجلد والقولون كل نصف ساعة، وبدأ القياس قبل الحقن بساعة، وأيضاً لمدة ٥ ساعات بعد الحقن، وذلك لحساب معدل الفقد الحراري المحسوس عن طريق الجلد.**

**النتائج: معدل الفقد الحراري كان حوالي ٠.٩٥ في كتاكيت المجموعة الضابطة الغير المعاملة، والتي لم تتعرض للأجهاد وفي درجة حرارة الغرفة، أما في الكتاكيت التي تم معالجتها بالكابسايسين تم زيادة الفقد الحراري عند تعرضها لكل من الحرارة العالية (٣٨° مئوية في الأقفاص الحرارية) أو بعد حقن جرعة الليبوبوليسكاريد المسببة للحمى، على النقيض فإن تأثير الكابسايسين لم يقتصر فقط على زيادة الفقد الحراري، ولكن أيضاً أدى إلى التقليل من الفقد الحراري عند التعرض للبرد (٨° مئوية في غرفة التبريد).**

**الاستنتاج: يعتبر التأثير المعالج للكابسيسين فى التنظيم الحراري للكتاكيت تأثيراً مزدوجاً أثناء التعرض للجو الحار، لحمى الليبوبوليسكاريد أو أثناء تعرض الكتاكيت للبرد. وقد يعذى ذلك لمدى كفاءة الكابسيسين فى الزيادة أو التقليل من الفقد الحراري سوياً. ولهذه الدراسة أهمية تطبيقية خاصة فى حماية الكتاكيت الصغيرة عند تعرضها لمؤثرات التغير فى درجات الحرارة المحيطة للكتاكيت، والتي تؤدي الى اختلال في التنظيم الحراري لصغار الكتاكيت ومن ثم النفوق.**